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FINAL REPORT

REMEDIAL INVESTIGATION REPORT

ASHLAND/NORTHERN STATES POWER LAKEFRONT SUPERFUND SITE

VOLUME 10 OF 12
HUMAN HEALTH RISK ASSESSMENT (PART 1 OF 2)

Prepared for

Northern States Power Company - WI
1414 West Hamilton Avenue
Eau Claire, WI 54701

August 31, 2007

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APPENDIX H

NEWFIELDS

September 26, 2007

Mr. Scott Hansen
USEPA
77 West Jackson Blvd.
Superfund Division (SR-6J)
Chicago, IL 60604

RE: Ashland/NSP Lakefront Site
Errata for Remedial Investigation Report
Final Human Health Risk Assessment
CERCLA Docket No. V-W-04-C-764
USEPA ID# WISFN057952
WDNR BRRTS #02-02-00013

Dear Mr. Hansen:

In accordance with USEPA's September 19, 2007 letter, modifications to the Final Remedial Investigation (RI) report (August 31, 2007) and the most recent version of the Human Health Risk Assessment (HHRA) report (September 6, 2007) are attached. An errata sheet has been prepared to show the modifications to the RI Report. Please note that the RI Report errata sheet includes changes to Section 7 of the RI Report consistent this revision of the HHRA.

This HHRA document has been prepared in accordance with the approved RI/FS work plan, which in turn is based upon the Scope of Work (Task 7 (A) 2) appended to the November 2003 Administrative Order on Consent between USEPA and NSPW. The attached pdf files includes the HHRA report text, tables, and Appendices B, D, and F. A separate file of the report showing edits to the HHRA text is also attached. The remaining segments of the HHRA are not included because they have not changed since they were submitted with the previous version.

Sincerely,

NEWFIELDS

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Hansen_RI_HHRA_09_26_07_Cov_Ltr

USEPA Comment:

1. *As part of the RI report, do you think that the site contamination boundaries might need to be re-drawn? For example, the residential area west of the Church property has not been impacted. It is recommended that a re-draw from Highway 2, north down 2nd Ave., west to the RR tracks and west to Ellis Ave. This would remove the un-impacted properties from the site contamination boundaries.*

Response:

The Site boundary shown on report figures were not revised. The boundary was established by USEPA as part of the AOC and incorporated into the Remedial Investigation/ Feasibility Study Work Plan.

USEPA Comment:

2. *Figure 1-2 – The "underground clay tile remnants" needs to be stated "underground clay tile pipe remnants."*

Response:

The note on Figure 1-2 has been modified "underground clay tile remnants" to "underground clay tile pipe remnants."

USEPA Comment:

3. *Figure 1-3 – This figure should include the clay tile pipe trace.*

Response:

The clay tile pipe trace has been added to Figure 1-3

USEPA Comment:

4. *Figure 5-2 – The 100,000 ppb. There has been no "clean" area documented at St. Clarie Street or the RR tracks.*

Response:

Figure 5-2 has been modified by enclosing the 100,000 ppb contour to include the filled ravine at the upper bluff area and the coal tar dump area at Kreher Park.

USEPA Comment:

5. *Figure 5-4 – The 10,000 ppb isoconcentration lines should be connected from the "court yard" through to the seep area. MW-2R had a total PAH concentration of 10,860 (3/17/05).*

Response:

Figure 5-4 was not modified because the total PAH concentrations at MW-2R for 3/17/05 is 4,530 ppb.

USEPA Comment:

6. *Figure 5-5 – The 1,000,0000 ppb isoconcentration lines should be connected from the "court yard" through to the seep/coal tar dump area.*

Response:

Figure 5-5 has been modified by enclosing the filled ravine in the upper bluff area and the coal tar dump area in Kreher Park within the 1,000,000 ppb contour. The total PAH concentration at GP-122 at the upper bluff area has also been labeled. The footnote explaining the highest total PAH concentrations at Kreher Park has also been modified to reference the GP-122 data.

USEPA Comment:

7. *Figure 5-7 – The "clay tile pipe" from the MGP location to the seep area needs to be included in this drawing.*

Response:

The clay tile pipe located in the buried ravine (identified during the 2001 investigation) has been added to Figure 5-7

The following changes (shown in yellow) to the Section 7 of the RI Report have been made in accordance with changes to the final HHRA report.

Executive Summary – Human Health Risk Assessment Results Subsection.

Insert the following as the first paragraph of this subsection.

The results of the human health risk assessment (HHRA) indicate that only three exposure pathways result in estimated risk levels exceed USEPA's target risk levels: residential exposure pathways (for soil depths between 0 and 3 feet or all soil depths to 10 feet bgs), construction worker exposure pathway (for soil depths between 0 and 10 feet) and worker exposures to indoor air. These include estimates for the reasonable maximum exposure conditions for potential cancer risks (greater than 10^{-4}), and non-cancer risks (greater than a hazard index of 1). These conclusions are based on exposures to soil in the filled ravine area (for residential receptors) and the Kreher Park area (for construction worker receptors), and to indoor air samples collected at NSPW Service Center. Carcinogenic risks based on average exposure conditions indicate that only the residential receptor exposure to soil (all soil depths to 10 feet bgs) are estimated to be at 1×10^{-4}, the upper-end of the target risk range. Noncarcinogenic risks for the residential receptor (for all soil depths to 10 feet bgs) and risks associated with the construction scenario are within acceptable levels. However, residential receptor exposure to subsurface soil is not expected, given the current and potential future land use of the Site. For this Site, residential risks associated with exposures to surface soil (0 to 1 foot bgs) are within the target risk ranges.

The last paragraph of this subsection has been modified as follows:

Risks to recreational users (surface soil), subsistence fishers (finfish), waders and swimmers (sediments), industrial workers (surface soil), and maintenance workers (surface soil) are all within USEPA's target risk range of 10^{-4} to 10^{-6} for lifetime cancer risk and a target hazard index of less than or equal to 1 for non-cancer risk. However, the cancer risk for waders and swimmers exposed to sediment is greater than the WDNR target risk of 1×10^{-5}.

Section 7.2.2.2 Groundwater Use – First sentence of first paragraph of this subsection has been changed as follows:

Groundwater contamination is present in both a shallow aquifer and a confined deep aquifer. Currently the shallow groundwater is not used as a potable water source.

Section 7.2.3.4 Analytical Data Used to Evaluate Risk – Footnotes to imbedded table have been changed as follows:

ANALYTICAL DATA USED FOR THE HHRA					
Exposure Scenario	**Soil**	**Sediment**	**Surface Water**	**Air**	**Biota**
Residential	Surface and Subsurface Soil (0-1 foot bgs) (0-3 feet bgs) (0-10 feet bgs)	Not Evaluated	Not Evaluated	Soil Vapor	Not Evaluated
Industrial/Commercial	Surface Soil (0-1 foot bgs)	Not Evaluated	Not Evaluated	Indoor Air	Not Evaluated
Maintenance Worker	Surface Soil (0-1 foot bgs)	Not Evaluated	Not Evaluated	Not Evaluated	Not Evaluated
Construction Worker	Surface and Subsurface Soil (0-10 feet bgs)	Not Evaluated	Not Evaluated	Not Evaluated	Not Evaluated
Recreational	Surface Soil (0-1 foot bgs)	First 0 – 2 feet[a]	All available data[b]	Not Evaluated	Not Evaluated
Subsistence Fishing	Not Evaluated	Not Evaluated	Not Evaluated	Not Evaluated	Fin Fish[c]

[a] Only those 0 – 2 feet sediment locations with four feet or less of surface water cover were used in the HHRA. The data set used to evaluate sediment exposures has been modified from previous versions of the report. This new data reflects two foot drop in the Lake Superior water levels observed in 2007.

[b] Includes both the 2005 RI data and the 1998 SEH data.

[c] Only the edible portions of the fish species evaluated were included in the HHRA.

Section 7.5.1 Risk Characterization Results – Values for adult swimmer, adolescent swimmer, adult wader, and adolescent wader for sediment in the Summary of RME Carcinogenic and Noncarcinogenic Risks table have been changed, and footnote d has been changed as follows:

Summary of RME Carcinogenic and Noncarcinogenic Risks[a]

Receptor	Table	Soil		Oily Materials in Surface Water[d]		Sediment		Oily Materials in Groundwater[d]		Biota		Indoor Air[b, e]	
		CR	HI	CR	HI	CR	HI	CR	HI	CR	HI	CR	HI
Resident	20	5×10^{-4}	15	–	–	–	–	–	–	–	–		
Recreational Adult	21	3×10^{-6}	0.002	–	–	–	–	–	–	–	–	–	–
Recreational Adolescent	22	2×10^{-6}	0.003	–	–	–	–	–	–	–	–	–	–
Recreational Child	23	1×10^{-5}	0.04	–	–	–	–	–	–	–	–	–	–
Adult Swimmer	I1–6b	–	–	9×10^{-2}	6	5×10^{-9}	2×10^{-5}	–	–	–	–	–	–
Adolescent Swimmer	25, I1-12b	–	–	4×10^{-2}	6	3×10^{-9}	2×10^{-5}	–	–	–	–	–	–
Adult Wader	26,I1-18b	–	–	5×10^{-2}	4	1×10^{-5}	0.002	–	–	–	–	–	–
Adolescent Wader	27,I1-24b	–	–	2×10^{-2}	4	5×10^{-6}	0.002	–	–	–	–	–	–
Industrial Worker	28 29	6×10^{-6}	0.007	–	–	–	–	–	–	–	–	8×10^{-5}	3
Maintenance Worker	30	1×10^{-6}	0.001	–	–	–	–	–	–	–	–	–	–
Construction Worker[c]	31,I1-28, J-2a 2b & 2c	1×10^{-4}	35	–	–	–	–	7×10^{-3}	59.5	–	–	8.34E-03 (KP) 2.14E-05 (UB) 3.29E-02 (FR)	17152 (KP) 228 (UB) 646601 (FR)
Subsistence Fisher	32	–	–	–	–	–	–	–	–	1×10^{-4}	0.01	–	–

[a] No COPCs were identified for soil gas and surface water. Risks based on exposure to these media were not quantified.

[b] For the industrial worker, the air risks were estimated using indoor air data from sample locations NS-GSINDOOR-0405 and NS-GSINDOOR-0705.

[c] For the construction worker, the groundwater risks were calculated using a derived concentration of "oily materials" in groundwater estimated using the laboratory analytical data of the DNAPL samples collected from the product stream recovered from the active free product recovery system for the Copper Falls aquifer.

[d] Represents the linear low dose risks calculated for both the recreational and construction receptors. The non-linear low dose risks are presented in Attachments I1 and I2.

Risks in bold are greater than the USEPA range for acceptable risk (1×10^{-4} to 1×10^{-6}). Cancer risks that are underlined are greater than the Wisconsin Department of Public Health threshold of 1×10^{-5}.

KP – Calculated using the Virginia Department of Environmental Quality Equation 3-8: Exposure of Workers to Volatiles in a Construction/Utility Trench (Groundwater less than 15 feet deep). Maximum detected concentrations in groundwater from Kreher Park were used as the exposure point concentration. Detailed calculations for this exposure pathway are presented in Attachment J.

UB – Calculated using the Virginia Department of Environmental Quality Equation 3-8: Exposure of Workers to Volatiles in a Construction/Utility Trench (Groundwater less than 15 feet deep). Maximum detected concentrations in groundwater from the Upper Bluff were used as the exposure point concentration. Detailed calculations for this exposure pathway are presented in Attachment J.

FR – Calculated using the Virginia Department of Environmental Quality Equation 3-8: Exposure of Workers to Volatiles in a Construction/Utility Trench (Groundwater less than 15 feet deep). Maximum detected concentrations in groundwater from the Filled Ravine were used as the exposure point concentration. Detailed calculations for this exposure pathway are presented in Attachment J.

Section 7.5.1 Risk Summary for the Recreational Scenario – in the Risk Summary for Recreational Swimmers Exposure to Sediment and Surface Water subsection, the last sentence of the first paragraph of the *Adult Swimmers Exposed to Oil Slicks in Surface Water* subsection has been changed as follows:

> Risks associated with exposures to oil slicks in surface water were evaluated. This pathway was evaluated because a tar slick was reported and photographed by a citizen. Although no slicks were observed by sample collectors and the subsequent RI data do not indicate notable surface water impacts, the 1998 SEH report calculated unacceptable levels of current and future health risks for workers, trespassers, and people engaged in recreational activities on the site. Since this exposure pathway poses one of the greatest potential health risks at the site, the revised HHRA report includes an evaluation of exposures to "oil slicks" in surface water in addition to the evaluation of the 1998 SEH data (Attachment K).

Section 7.5.1 Risk Summary for the Recreational Scenario – in the Risk Summary for Recreational Swimmers Exposure to Sediment and Surface Water subsection, the first paragraph of the *Adult Swimmers Exposed to Sediment* subsection has been changed as follows:

> Risks associated with exposure to sediment for adult swimmers are a CR of 4×10-5 and an HI of 0.05 for samples collected within Chequamegon Bay. Both the cancer and noncancer risk are below the USEPA target risk range of 10-4 to 10-6 for cancer and an HI of 1 for noncancer endpoints, respectively. However, the cancer risk is greater than the WDNR target risk goal of 1×10-5.

Section 7.5.1 Risk Summary for the Recreational Scenario – in the Risk Summary for Recreational Swimmers Exposure to Sediment and Surface Water subsection, the last sentence of the first paragraph of the *Adolescent Swimmers Exposed to Oil Slicks in Surface Water* subsection has been changed as follows:

> Risks associated with exposures to oil slicks in surface water were evaluated. This pathway was evaluated because a tar slick was reported and photographed by a citizen. Although, no slicks were observed by sample collectors and the subsequent data does not indicate notable surface water impacts, the 1998 SEH report calculated unacceptable levels of current and future health risks for workers, trespassers, and people engaged in recreational activities on the site. Since this exposure pathway poses one of the greatest potential health risks at the site, the revised HHRA report includes an evaluation of exposures to "oil slicks" in surface water in addition to the evaluation of the 1998 SEH data (Attachment K).

Section 7.5.1 Risk Summary for the Recreational Scenario – in the Risk Summary for Recreational Swimmers Exposure to Sediment and Surface Water subsection, the last sentence of the first paragraph of the *Adolescent Swimmers Exposed to Sediment* subsection has been changed as follows:

Risks associated with exposure to sediment for adolescent swimmers are a CR of 2×10-5 and an HI of 0.05 for samples collected within Chequamegon Bay. Both the cancer and noncancer risk are below the USEPA target risk range of 10-4 to 10-6 for cancer and an HI of1 for noncancer endpoints, respectively. However, the cancer risk is greater than the WDNR target risk goal of 1×10-5.

Section 7.5.1 Risk Summary for the Recreational Scenario – in the Risk Summary for Recreational Waders Exposure to Sediment and Surface Water subsection, the first paragraph of the *Adult Waders Exposed to Sediment* subsection has been changed as follows:

Risks associated with exposure to sediment for adult waders are a CR of 4×10^{-5} and an HI of 0.05 for samples collected within Chequamegon Bay. The cancer risk is within the USEPA target risk range of 10^{-4} to 10^{-6} for cancer and noncancer risk is less than the target HI of 1 for noncancer endpoints. However, the cancer risk is greater than the WDNR target risk goal of 1×10^{-5}.

Section 7.5.1 Risk Summary for the Recreational Scenario – in the Risk Summary for Recreational Waders Exposure to Sediment and Surface Water subsection, the first paragraph of the *Adolescent Waders Exposed to Sediment* subsection has been changed as follows:

Risks associated with exposure to sediment for adolescent waders are a CR of 2×10^{-5} and an HI of 0.05 for samples collected within Chequamegon Bay. The cancer risk is within the USEPA target risk range of 10^{-4} to 10^{-6} for cancer and an HI of 1 for noncancer endpoints. However, the cancer risk is greater than the WDNR target risk goal of 1×10^{-5}.

Section 7.5.3.1 Residential Scenario Evaluation – The values in the embedded table following the first two paragraphs of this subsection have been changed as follows:

Receptor	RME Table	RME		CTE Table	CTE	
		CR	HI		CR	HI
Resident (0 – 10 feet soil depth)	20	5×10^{-4}	15	35	1×10^{-4}	5
Resident (0-1 foot soil depth)	33	1×10^{-5}	0.2	36	5×10^{-6}	0.1
Resident (0 – 3 feet soil depth)	34	3×10^{-4}	0.9	37	5×10^{-5}	0.3

Section 7.5.3.3 Surface Water Evaluation – The last sentence of the first paragraph has been changed as follows:

Prior to selecting the COPCs in surface water, all surface water data were evaluated to determine if the data were considered usable for the purposes of estimating risks to recreational receptors. The surface water data reviewed included the 1998 SEH data along with the 2005 high-energy and low-energy data. However, the surface water data from the 1998 SEH report were used to in the quantitative risk assessment because no COPCs were identified in surface water samples collected in 2005.

FINAL REPORT

HUMAN HEALTH RISK ASSESSMENT - ASHLAND/NORTHERN STATES POWER LAKEFRONT SUPERFUND SITE

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September 26, 2007

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Acronyms

ACGIH	American Conference of Governmental Industrial Hygienists
ALM	Adult Lead Model
ATSDR	Agency for Toxic Substances and Disease Registry
AWQC	Ambient Water Quality Criteria
bgs	below ground surface
CDC	Centers for Disease Control
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CFR	Code of Federal Regulations
cm/sec	centimeters per second
COPC	Chemical of Potential Concern
CR	Cancer Risk
Csat	Chemical-specific Saturation Limit
CSM	Conceptual Site Model
CTE	Central Tendency Evaluation
DQO	Data Quality Objectives
EPC	Exposure Point Concentration
ft/ft	foot per foot
HEAST	Health Effects Assessment Summary Tables
HHRA	Human Health Risk Assessment
HI	Hazard Index
HQ	Hazard Quotient
IR	Intake Rate
IRIS	Integrated Risk Information System
IEUBK	Integrated Exposure Uptake Biokinetic Model
m^3/day	cubic meters per day
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
mg/m^3	milligrams per cubic meter
MGP	Manufactured Gas Plant
MSL	mean sea level
MVUE	Minimum Variance Unbiased Estimate
NAPL	Non-aqueous Phase Liquid
NCP	National Oil and Hazardous Substances Pollution Contingency Plan
NSPW	Northern States Power Wisconsin

ORNL	Oak Ridge National Laboratory
OSHA	Occupational Safety and Health Administration
OSWER	Office of Solid Waste and Emergency Response
PAH	Polycyclic Aromatic Hydrocarbon
PEF	Particulate Emission Factor
ppm	parts per million
PPRTV	Provisional Peer-reviewed Toxicity Value
PRG	Preliminary Remediation Goal
QAPP	Quality Assurance Project Plan
RAGS	Risk Assessment Guidance for Superfund
RAIS	Risk Assessment Information System
RBC	Risk-based Concentration
RBSC	Risk-based Screening Concentration
RfC	Reference Concentration
RfD	Reference Dose
RI/FS	Remedial Investigation and Feasibility Study
RME	Reasonable Maximum Exposure
RV	Recreational Vehicle
SF	Slope Factor
SQL	Sample Quantitation Limit
TIC	Tentatively Identified Compound
UCL	Upper Confidence Limit
U.S.C.	United States Code
USEPA	United States Environmental Protection Agency
VF	Volatilization Factor
VI	Vapor Intrusion
VOC	Volatile Organic Compound
WDHFS	Wisconsin Department of Health and Family Services
WDNR	Wisconsin Department of Natural Resources
WQS	Water Quality Standard
WWTP	Wastewater Treatment Plant
µg/L	micrograms per liter
µg/dL	micrograms per deciliter

The results of the human health risk assessment (HHRA) for Ashland/NSP Lakefront Superfund Site (Site) in Ashland, Wisconsin (Site) indicate that seven exposure pathways result in estimated risks that exceed U.S. Environmental Protection Agency's (USEPA's) target risk levels and eight exposure pathways result in estimated risks that are either equivalent to or exceed the Wisconsin Department of Natural Resources (WDNR) threshold of an incremental cancer risk (CR) of one in one hundred thousands (1×10^{-5}). These exceedances are indicated below.

Exceeds USEPA Threshold ($CR \geq 1\times10^{-4}$ or HI >1)	**Exceeds WDNR Threshold ($CR \geq 1\times10^{-5}$ or HI >1)**
Residents (Soil[0-3 feet and all soil depths] - Cancer)	Residents (Soil[0-3 feet and all soil depths] - Cancer)
–	Residential Child (Soil – Noncancer)
Construction Worker (Soil [0-10 feet bgs]/Groundwater)	Construction Worker (Soil [0-10 feet bgs]/Groundwater)
Construction Worker (Trench Air)	Construction Worker (Trench Air)
Adult Swimmer (Surface Water)	Adult Swimmer (Surface Water)
Adult Wader (Surface Water/Oil slicks)	Adult Wader (Surface Water/oil Slicks/Sediment)
Industrial Worker (Indoor Air)	Industrial Worker (Indoor Air)
Subsistence Fisher (Biota)	Subsistence Fisher (Biota)

HI: Hazard index for noncarcinogenic effects

These include estimates for the reasonable maximum exposure (RME) scenarios for potential cancer risks and non-cancer risks. These conclusions are based on assumed exposures to soil in the filled ravine area (for residential receptors) and the filled ravine, upper bluff and Kreher Park area (for construction worker receptors), and to indoor air samples collected at the Site.

Carcinogenic risks based on central tendency evaluation (CTE) scenarios indicate that only the residential receptor exposure to soil (all soil depths to 10 feet bgs) are estimated to be at 1×10^{-4}, the upper-end of the USEPA target risk range or greater than the WDNR threshold. Carcinogenic risks based on the RME scenarios for residential receptor exposure to soils for all depths exceed the 1×10^{-4} the upper-end of the USEPA target risk range. Noncarcinogenic risks for the residential receptor (for soil depths 0-1 foot and 0-3 feet bgs) and risks associated with the construction scenario are within acceptable levels. However, residential receptor exposure to subsurface soil is not expected, given the current and potential future land use of the Site. For this Site, residential risks associated with CTE exposures to surface soil (0 to 1 foot bgs) are within the target risk ranges, but the RME exposures exceed the target risk range.

Although the results of the HHRA indicate risks for the construction workers under the RME conditions exceed USEPA's target risk levels, the assumptions used to estimate risks to this

receptor were conservative and assumed the worst case. Given both the current and future land use of the Site, it is unlikely that construction workers would be exposed to soil in the filled ravine and Upper Bluff. The most likely scenario for the future construction worker is exposure to soil within 0 to 4 feet bgs in Kreher Park (a typical depth for the installation of underground utility corridors), as most activities associated with the implementation of the future land use would be associated with regrading, landscaping, and road or parking lot construction. However, the depth to groundwater in Kreher Park is relatively shallow due to the lake-filled material comprising most of the park. Consequently, it is possible that construction workers excavating and installing utilities in such underground corridors in certain portions of Kreher Park may encounter chemicals of potential concern (COPC) impacted sub-surface soils and non-aqueous phase liquids (NAPLs) in groundwater.

An HI of 3 was calculated for the general industrial worker exposure to indoor air pathway under the RME conditions. This risk level is likely to be an overestimate because:

- It was estimated using the maximum detected concentrations as the concentrations at points of exposure.
- It was calculated based on USEPA default exposure parameters for the industrial /commercial workers (i.e., an individual works at the Site for 8 hours per day, 5 days per week, 50 weeks per year for a total of 25 years). The NSPW Service Center is used as a warehouse; there is an office space inside the building, but used only on a part-time basis.

Cancer risks to subsistence fisher (finfish) are equivalent to 1×10^{-4}, the upper-end of the USEPA target risk range, and greater than the WDNR threshold of 1×10^{-5}. Noncarcinogenic risk is within acceptable limits for both USEPA and WDNR.

Risks to recreational children (surface soil) are equivalent to1×10^{-5}, which is the WDNR cancer risk threshold. However, risks to adolescent and adult receptors exposed to surface soil are below the USEPA acceptable risk range and below the WDNR risk threshold.

Risks to waders and swimmers (sediments), industrial workers (surface soil), and maintenance workers (surface soil) are all within USEPA's target risk range of 10^{-4} to 10^{-6} for lifetime cancer risk and a target HI of less than or equal to 1 for non-cancer risk and are greater than the WDNR threshold of 1×10^{-5} for lifetime cancer risk and a target HI of less than or equal to 1 for non-cancer risk.

At the request of the Wisconsin Department of Health and family Services (WDHFS), risks were also estimated for construction workers exposed to "oily materials" in groundwater via dermal contact and swimmers and waders who may be exposed to oil slicks in surface water via ingestion and dermal contact. Because no media-specific concentrations are available for either scenario, risks were estimated using analytical data collected from the product stream from the active free product recovery system for the Copper Falls aquifer or chemical-specific solubility values detected in the dense non-aqueous phase liquid (DNAPL) sample. Risks to construction workers exposed to "oily material" in groundwater and adult swimmers and waders exposed to "oil slicks" in surface water is greater than both the USEPA upper risk range (CR 1×10^{-4} and HI of 1) and than WDNR threshold (CR 1×10^{-5} and HI of 1). However, it is important to note that there is much uncertainty associated with estimating risks to oily material in groundwater or oil slicks in surface water. The primary uncertainties are associated with the lack of:

- Established methodology for estimating this exposure pathway
- Relevant oily material data resulting in the use of DNAPL data that are expected to result in an overestimate of risk.

Northern States Power Company, a Wisconsin corporation, d/b/a Xcel Energy (hereafter "NSPW"), submits this baseline Human Health Risk Assessment (HHRA) in accordance with the United States Environmental Protection Agency (USEPA) approved Remedial Investigation and Feasibility Study (RI/FS) Work Plan (URS, 2005), as amended (RI/FS Work Plan). This HHRA has been prepared to support the Ashland/NSP Lakefront Superfund Site (Site) RI/FS being conducted under the regulatory framework of the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), 42 U.S.C. 9601, et seq. and the National Oil and Hazardous Substances Pollution Contingency Plan (NCP), 40 CFR Part 300.

1.1 PURPOSE

The purpose of the baseline HHRA is to provide a risk-based interpretation of the data collected during the RI and to provide conservative estimates of potential human health risks posed by chemicals that are present at or migrating from the Site. The results of the HHRA may also be used as the basis for risk management decisions. In summary, the objectives of the baseline HHRA are to:

- Quantify exposures and characterize baseline risks to potentially exposed individuals (both current and future) at or near the Site;
- Identify those chemicals that may pose risks to human health; and
- Provide the basis for risk management decisions.

1.2 APPROACH

This HHRA was completed using the data collected as part of RI/FS along with historical data from work previously completed by the Wisconsin Department of Natural Resources (WDNR) and the Wisconsin Department of Health and Family Services (WDHFS). The methodology for completing the HHRA follows guidance presented in *Risk Assessment Guidance for Superfund (RAGS): Volume I. Part A – Human Health Evaluation Manual* (USEPA, 1989) and several more recent regulatory guidance documents and resources as appropriate such as:

- *Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites* (OSWER 9355.4-24, March 2002)(USEPA, 2002a);
- *Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual, Part E, Supplemental Guidance for Dermal Risk Assessment* (EPA/540/R/99/005, OSWER 9285.7-02EP, PB99-963312, July 2004)(USEPA, 2004a);

- *Calculating Upper Confidence Limits for Exposure Point Concentrations at Hazardous Waste Sites* (OSWER 9285.6-10 December 2002)(USEPA, 2002b);
- *Exposure Factors Handbook* (EPA/600/P-95/002) August, 1997 (USEPA, 1997a); and
- A summary of up-to-date guidance and screening criteria presented in http://risk.lsd.ornl.gov/homepage/rap_docs.shtml, (Oak Ridge National Laboratory [ORNL], On-line).

A draft HHRA was submitted for review on April 7, 2006 as a stand alone report and on June 5, 2006 as part of the draft RI Report. The draft HHRA has been revised based on agency review comments provided on August 25, 2006 and October 27, 2006 and decisions agreed upon during the November 12, 2006 meeting between USEPA, WDNR, WDHFS and NSPW. By letter dated December 22, 2006, USEPA sent NSPW a notice of deficiency regarding the HHRA. USEPA provided a second notice of deficiency on July 10, 2007, giving NSPW 21 days to cure the deficiency by incorporating USEPA's modifications. NSPW submitted the revised HHRA on July 31st. USEPA, in consultation with WDNR and the WDHFS, reviewed NSPW's revised HHRA. In a letter dated August 23, 2007, USEPA agreed to incorporate most of NSPW's language changes, but contained other modifications. This document contains NSPW's language changes and addresses remaining issues outlined in USEPA's letter and attached document dated August 23, 2007. This HHRA incorporates the following components:

- Section 2 Data Evaluation
- Section 3 Exposure Assessment
- Section 4 Toxicity Assessment
- Section 5 Risk Characterization
- Section 6 Uncertainty Analysis

1.3 SITE DESCRIPTION

The Site is located in S 33, T 48 N, R 4W in Ashland County, Wisconsin, shown on **Figure 1**. The Site consists of property owned by NSPW, a portion of Kreher Park, and sediments in an offshore area adjacent to Kreher Park. Existing site features showing the boundary of the Site are shown on **Figure 2**. The Site includes the following:

- NSPW's property (a former manufactured gas plant [MGP]), and potentially the areas beneath residences located on the upper bluff,

- Private, non-industrial areas including 17 single-family homes, hotel, a school, a playground, and a church (also located on the upper bluff);
- Soils along the flat terrace adjacent to the Chequamegon Bay shoreline including Kreher Park (filled lakebed areas north of the bluff face);
- Other areas of the filled former lakebed not within the Kreher Park boundary including a former City Wastewater Treatment Plant (WWTP) and buildings, grassed areas, and boat storage; and
- Impacted sediment in the lake adjacent to the filled lakebed area north of Kreher Park.

On the upper bluff, the NSPW property includes a small office building and parking lot fronting on Lake Shore Drive, and a larger vehicle maintenance building and parking lot area located south of St. Claire Street between Prentice Avenue and 3rd Avenue East. The office building and vehicle maintenance building are separated by an alley. A gravel-covered parking and storage yard area, with a large microwave tower, is located north of St. Claire Street between 3rd Avenue East and Prentice Avenue. A filled ravine formerly opening to the north underlies this storage yard. The area occupied by the buildings and parking lots is relatively flat, at an elevation of approximately 640 feet above mean sea level (MSL). Surface water drainage from the NSPW property is to the north. Seven residences bound the Site north the NSPWs building. Our Lady of the Lake Church and School is located immediately west of NSPW's buildings, with nine private homes further west of the school. Private homes are located immediately east of Prentice Avenue. To the northwest of the upper bluff, the Site slopes abruptly to the Canadian National (formerly Wisconsin Central Limited) Railroad property that marks the former Lake Superior shoreline and then to the City of Ashland's Kreher Park, beyond which is Chequamegon Bay.

Based on current data, the impacted area of Kreher Park consists of a flat terrace overlaying fill material adjacent to the Chequamegon Bay shoreline. The surface elevation of the park varies approximately 10 feet, from 601 feet MSL, to about 610 feet MSL at the base of the bluff overlooking the park. The bluff rises to an elevation of about 640 feet MSL, which corresponds to the approximate elevation of the NSPW property. The lake elevation generally fluctuates about two feet, from 601 to 603 feet MSL, however, in 2007 lake levels were notably lower. At the present time, the park area is predominantly grass covered. A gravel overflow parking area for the marina occupies the west end of the Kreher Park property, while a miniature golf facility formerly occupied the east end of the Site. The former City of Ashland WWTP and associated structures front the bay inlet on the north side of the Kreher Park property. The impacted area of Kreher Park (excluding the affected sediments area) occupies approximately 13 acres and is

bounded by Prentice Avenue and a jetty extension of Prentice Avenue to the east, the Canadian National railroad to the south, the Ellis Avenue and the marina extension of Ellis Avenue to the west and Chequamegon Bay to the north.

The offshore area with approximately 10 acres of impacted sediments is located in an inlet created by the Prentice Avenue jetty and marina extensions previously described. For the most part, impacted sediments are confined in the inlet bounded by the northern edge of the line between the Prentice Avenue jetty and the marina extension. Data collected to date indicate that impacted sediment levels sharply decline beyond this boundary. The affected sediments consist of lake bottom sand and silts, and are overlain by a layer of wood chips and larger wood waste fragments (slab wood, logs), likely originating from former lumbering operations. The wood waste layer varies in thickness from zero to seven feet, with an average thickness of nine inches. Based on current data, the entire area of impacted sediments encompasses approximately ten acres.

1.3.1 Population and Land Use

The Site is located in Ashland County, Wisconsin. Ashland County has a population of 16,866 and covers a land area of 1,047 square miles. The City of Ashland (population 8,620 based on the 2000 Census) is the largest city in Ashland County, as well as the county seat. The Bad River Indian Reservation, an area of 200 square miles, is located entirely within Ashland County and has a population of 1,538.

According to census estimates, the population of Ashland County and the City of Ashland has changed little since 1990. Ashland County grew by 3.3% between 1990 and 1999 (16,307 to 16,866). The City of Ashland dropped in population by 0.8% (8,695 to 8,620). This is consistent with the limited population growth in the region over the last 20 years.

Residents are served by the city's municipal water supply, which is provided from Chequamegon Bay surface water. The surface water intake is located at Longitude 90° 50' 29" E and Latitude 46° 36' 25"N. The intake is located in approximately 23 feet of water and is approximately one mile northeast of the Site, and not affected by site-related contamination. The area is located in the Lake Superior Lowland Physiographic Province characterized by flat to undulating topography underlain by red glacial clay (Miller Creek Formation). Uplands lie to the south of Ashland and are characterized by rolling hilly topography and underlain by sand and gravel soils (Copper Falls Formation). Elevations in the Ashland area range from 601 feet MSL datum (Lake

Superior surface elevation) to approximately 700 feet MSL. Regional slope is generally to the north.

1.3.2 Geological and Hydrogeological Setting

The filled ravine at the upper bluff is a former drainage feature that begins near the NSPW administration building fronting on Lakeshore Drive, and deepens and widens to the north **(Figure 3)**. The mouth of the ravine opens to Kreher Park through the bluff face at the north end of the gravel storage yard. The maximum depth of fill in the ravine at the mouth is approximately 33 feet.

The Copper Falls Aquifer is a confined, variably coarse to fine-grained sand (reworked glacial till) that underlies the entire Lakefront site **(Figure 4)**. The formation is overlain by the surficial Miller Creek Formation, which is a lacustrine clay to silt till unit. At the NSPW property, the Miller Creek Formation has a maximum thickness of about 35 feet; the thinnest portion of the unit is at the mouth of the former ravine, at approximately four feet.

Surficial soils at the Site are underlain by a variety of fill materials, including wood waste (slabs and sawdust), solid waste (including concrete, bricks, bottles, steel, wire, and cinders), and earthen fill (including a buried clay berm along the shoreline on the northeast side of the Site near the former WWTP). The fill materials at Kreher Park are underlain by a variably 0 to 5.5 foot thick layer of beach sand separating the fill from the underlying Miller Creek Formation. The Miller Creek soils encountered at the Site consist of clays and silts and range in thickness from 7 to 40 feet (the Miller Creek Formation thickens from the bluff face toward the shoreline and beyond to the north). Silty sand and gravel soils of the Copper Falls Formation are present beneath the Miller Creek soils. Thickness of the Copper Falls Formation at the site has not been determined, though monitoring wells installed in December 2003 suggest that the bedrock is at least 190 feet below ground level in at least some locations. The Copper Falls Formation consists of granular, cohesionless material deposited by glacial melt waters. Bedrock was encountered at 192 feet during the latest exploration drilling program at the NSPW property during December 2003 (monitoring well MW-2C). Bedrock in the Ashland area consists of Precambrian sandstones. To the south, beneath the NSPW facility, the Copper Falls consists of silty sands with discontinuous lenses of silty clay and silt. To the north, beneath Kreher Park, the Copper Falls formation consists of outwash sediments (i.e., clean sands with occasional gravel intervals).

Geology of the upper bluff area in the vicinity of the former ravine consists of earthen fill materials, with clay soils of the Miller Creek Formation on the flanks of the former ravine. The ravine fill unit consists of silty clay fill material mixed with ash, cinders, slag, and fragments of bricks, concrete, glass, wood, and other solid waste. The thickness of the fill diminishes to less than three feet beyond the flanks of the ravine to the east and west. Miller Creek clay soils are present at the base of the former ravine; however, the thickness of these soils has been measured at as little as four feet at one soil boring location (at the mouth of the ravine where it opened to the former lake shoreline). Sand and gravel layers interbedded with silty clay lenses have been encountered near the contact of the Miller Creek Formation and the underlying Copper Falls aquifer.

Offshore geology consists of a discontinuous layer of submerged wood chips on the lake bottom underlain by variably fine to medium grained sediments. The sediments are underlain by silts and clays of the Miller Creek Formation. The Copper Falls Formation was not encountered during earlier investigations of the offshore sediments. Consequently, the thickness of the Miller Creek Formation below the bay is unknown.

The water table is found within the fills overlying the Miller Creek Formation at the Site. (Where the Miller Creek is the surficial soil unit, the water table is also present within the Miller Creek Formation.) The hydraulic conductivity of the shallow soils and fill materials ranges from approximately 0.1 to 5×10^{-5} centimeters per second [cm/sec] (URS, 2005). The higher hydraulic conductivity values are typically found in locations with saturated wood waste fill. The horizontal hydraulic gradient is very flat (< 0.0004 foot per foot [ft/ft] to the north measured during June 2004) due to the high hydraulic conductivities on the Site.

Hydrogeology of the upper bluff area (the former MGP plant location of the Site) includes low permeability conditions (3×10^{-6} to 4×10^{-8} cm/sec) in the Miller Creek Formation comprising most of the shallow saturated soil in the area. Fill soils located in the former ravine area exhibit hydraulic conductivities approximately 1,000 times higher than the surrounding Miller Creek soils. The horizontal hydraulic gradient in the fill soils of the former ravine is approximately 0.09 ft/ft. Direction of the groundwater flow in the ravine fill is to the north (toward the mouth of the former ravine). An intermittent groundwater discharge to the surface used to be present at the base of the bluff in the proximity of the mouth of the former ravine in the form of a seep. This seep was found to be caused by a buried 12-inch clay tile pipe that traversed the length of the ravine at its base. The elevation of the seep was over five feet above the water table levels

measured in MW-7, formerly located immediately adjacent to the seep. The buried pipe was located and the seep area capped as part of the 2002 interim action response (URS, 2002).
Artesian conditions are present at the Kreher Park areas of the Site in the Copper Falls aquifer. Hydraulic head levels of approximately 17 feet above ground surface have historically been measured in an artesian well located at Kreher Park. However, artesian conditions have not been identified in the Copper Falls aquifer in the vicinity of the former ravine area or the upper bluff area. An upward hydraulic gradient is present in the Copper Falls aquifer in the northern portion of the upper bluff area, and diminishes and eventually changes to a downward gradient south of the alley separating the NSPW Service Center Building from the Administration Building parking area. The general direction of flow in the Copper Falls aquifer is to the north (toward Chequamegon Bay). Hydraulic conductivity values for the Copper Falls aquifer ranging from 5.9×10^{-4} cm/sec to 9.6×10^{-4} cm/sec were derived from a 48-hour aquifer performance test at the NSPW property in 1997. These data were used to later design an interim coal tar removal system installed by NSPW during 2000 (URS, 2005).

1.3.3 Surface Water Features

The Site is located on the shore of Chequamegon Bay. Regional surface water drainage flows to the north through Fish Creek and several small unnamed creeks and swales into Chequamegon Bay. Surface water at the Site flows either to the City of Ashland storm sewer system, or discharges directly to Chequamegon Bay. An open sewer is depicted on historic Sanborn Fire Insurance maps dating from 1901 to 1951 on the western portion of the Kreher Park area. The head of the sewer is shown at a location about two-thirds of the distance from the shoreline to the bluff face with no identified upstream inlet. It is not clear whether the open sewer was used for discharging storm water, sanitary wastewater or both to Chequamegon Bay.

Surface water sampling was conducted by Short Elliot Hendrickson Inc. (SEH) in 1998. No chemicals were detected above ambient water quality criteria (AWQC) in twelve unfiltered surface water samples collected on January 14 and 15, 1998. However, in one unfiltered water column sample collected during a period on May 14, 1998, when wave heights were estimated to be between 60 and 90 cm[1], benzo(a)anthracene and benzo(a)pyrene exceeded secondary chronic and acute water quality criteria values, respectively. No VOCs exceeded AWQC in that sample. It is unknown whether the contaminants in this sample were adsorbed onto suspended particulates or in a dissolved state.

The WDNR received a report from a citizen on November 15, 2005 that high winds at the time likely caused several oil slicks to form in the affected area of the bay inlet. USEPA subsequently forwarded several photos taken of these slicks to NSPW. This event corresponded to the high energy surface water sampling. In follow-up, NSPW's sampling crew inspected the area and did not observe any slicks. Additionally, Coleman Engineering personnel inspected the area from the shoreline and also reported no slick observations. The occasional formation of slicks or intermittent releases may occur during high energy events stronger than conditions observed during the November sampling event.

The high-energy samples were collected on November 14 and 15, 2005 during a period where wave conditions exceeded 30 centimeters during the 24-hour period prior to sampling. This investigation was conducted in accordance with the approved RI Work Plan. Details are provided in Appendix D to the BERA. For the majority of samples (both low energy and high energy), no contaminants were detected, including those collected during the high energy sampling event. VOCs including benzene, ethylbenzene, toluene and xylenes, along with a few PAHs were detected at very low levels in a few samples. Most of these detections were reported as estimated values because the chemicals were detected between the method detection limit (MDL) and limit of quantitation. Only one ecological sample and one human health sample yielded quantifiable values of benzene (ERA 07 1105-NB-FIL at 0.53 μg/L, and HHRA3-1105-UNF at 0.74 μg/L).

All reported detections for PAHs were estimated concentrations; the highest estimated concentration for naphthalene was 2 μg/L. No reported concentrations exceeded U.S. EPA Region V ecological screening levels (ESLs) or comparable screening criteria Sample results are included in the Surface Water Investigation report included in Appendix D of the BERA.

The polycyclic aromatic hydrocarbon (PAH) and volatile organic compound (VOC) impacted sediment is concentrated at the wood debris/sediment-water interface and concentrations generally decrease with depth, although exceptions are found in a few locations. The presence of impacted sediment and non-aqueous phase liquids (NAPLs) across the surface of the lakebed is consistent with the physical-chemical characteristics of the Site-related chemicals. The mode of chemical transport to sediments was likely through backfilling (i.e., construction activities associated with the former WWTP), historic surface water runoff, or possible discharge from one or more source areas (e.g., MGP plant, coal tar dump at Kreher Park, etc.).

[1] It is likely this estimate was based upon crest to trough height rather than wave height compared to lake surface.

Information provided by the City of Ashland's Department of Public Works indicates that the City had a combined storm and sanitary sewerage system until the early to mid-1980s. The storm sewer system was separated from the sanitary system at that time to reduce flow to the former WWTP. In the past, storm water discharged directly to Chequamegon Bay through three known outfalls within the Site. Those outfalls have been closed and storm water is now re-routed to a discharge point east of the Site.

1.3.4 Groundwater Use

Groundwater is present in both a shallow aquifer and a confined deep aquifer. Currently the shallow groundwater is not used as a potable water source. There are two artesian wells in the Site vicinity—one located near Prentice Avenue on the eastern boundary of the Site and the other located near the marina on the western boundary. Both wells draw water from the Copper Falls aquifer, which is a deep aquifer separated from the shallow groundwater by the Miller Creek Formation (URS, 2005; ATSDR, 2003). The City of Ashland temporarily closed these wells for public use in August 2004. The City of Ashland will determine when the wells will be reopened pending the outcome of the RI/FS and subsequent cleanup actions. To date water from these wells have met all federal and state safe drinking water standards. Water from these artesian wells is considered safe to drink as Site-related chemicals have not been detected in these wells at levels of concern (ATSDR, 2003).

Except for the two artesian wells at Kreher Park, the Copper Falls aquifer is not used for drinking water and is not considered a source of human exposure. Shallow groundwater at the Site is not a drinking water source for the City of Ashland. Drinking water at the Site is provided by the City of Ashland that draws its water from intakes in Lake Superior, located approximately one mile northeast of the Site, which is outside the known extent of Site-related sediments and surface water impacts. Therefore, there are no known current receptors to shallow groundwater beneath the Site. However, workers at the former WWTP had reported previous direct contact with tar-like product floating on shallow groundwater when they were in trenches where pipes transferred sewage to the plant (ATSDR 2003). Such activities are currently not occurring, but could occur to the future workers when performing construction activities below the water table at Kreher Park.

1.3.5 Current and Potential Future Land Use Patterns

The upland area (upper bluff/ravine area) is primarily used for industrial or commercial purposes.[2] Portions of the Site (e.g., the abandoned WWTP) are subject to trespassing activities. These areas, some of which are public streets, are readily accessible to the public although they are generally covered by clean fill or roadways.

The area near the lakefront is zoned conservancy district; i.e., acceptable for use as parkland. The filled lakebed portions of the Site are comprised of City parkland (Kreher Park). The area is readily accessible by the public and a majority of the Kreher Park area of the Site is mowed and maintained for public use. No physical barrier exists at the shoreline to prevent swimming or wading in the bay where the impacted sediments have been found, although warning signs are posted along the shore of the affected area. Kreher Park and the impacted sediments are surrounded by facilities that draw the public to the lakefront—a city marina, public swimming beach, a boat ramp and a recreational vehicle (RV) park and campground. Warning buoys also prohibit boats into the affected area.

According to the *Ashland Wisconsin Waterfront Development Plan*, prepared by SmithGroup JJR for the City of Ashland, Wisconsin in March 2002, the swimming beach will be retained but the existing RV park, located immediately adjacent to the Ashland property to the east, will be relocated to the Clarkson Dock farther to the east. The plan proposes that the existing RV park land will be redeveloped into a parking lot and an interpretive center for the ore freighter and/or the Great Lakes Shipping and Mining Museum. The future reuse plan for the Site has not been determined pending remedies to be implemented at the lakefront.

[2] Although neighboring residences and the Our Lady of the Lake school and parish grounds are designated within the Site boundary, these areas have been characterized as affected by contaminated groundwater only.

One of the first steps of the baseline HHRA process was to review data collected during site investigations to develop a data set to support the site-specific HHRA. The analytical data from the Site were reviewed to:

- Validate and organize sampling data that were of acceptable quality for their use in the detailed HHRA; and
- Identify a set of chemicals that are Site-related.

Data evaluation was conducted as follows.

2.1 DATA REVIEW PROTOCOL

RI analytical and field data were first compiled. Validated data were entered into the USEPA-specified database and tabulated for use. The data from previous sampling efforts and this RI were reviewed to:

- Identify the nature and extent of Site-related chemical; and
- Evaluate the usability, including any uncertainties associated with the data.

The data were checked against the data quality objectives (DQOs) identified in the approved Quality Assurance Project Plan (QAPP) (URS, 2005). Details of the procedures for assessing the precision, accuracy, representativeness, completeness and comparability of field data and analytical laboratory data are described in the QAPP. Qualifications to the data usability are discussed in the quality assurance section of any reports presenting the data. Data generated under this program were considered technically sound and of sufficient quality and quantity to support the needs of the data users.

Methods used to develop a data set to support the development of the HHRA are described in the following sections.

2.1.1 Tentatively Identified Compounds

Both the identity and reported concentrations of tentatively identified compounds (TICs) are highly uncertain. As outlined in the approved RI/FS Work Plan (URS, 2005), TICs were excluded from further evaluation in the baseline HHRA.

2.1.2 Qualified Data

Qualifiers pertaining to uncertainty in the identity or the reported concentration of an analyte were assigned to certain analytical data by the laboratories or by persons performing data validation. The following qualifiers were used for HHRA data.

QUALIFIER	DEFINITION	USE OF QUALIFIED DATA IN HHRA
U	The analyte was analyzed for, but was not detected above the reported sample quantitation limit (SQL).	If the analyte is selected as a chemical of potential concern (COPC), then it is assumed to be present at one-half the SQL.
J	The analyte was positively identified; however, the associated numerical value is an estimate of the concentration of the analyte in the sample.	If the analyte is selected as a COPC, it is assumed to be present at the estimated concentration.
UJ	The analyte was not detected above the reported sample quantitation limit. However, the reported quantitation limit is an estimate and may or may not represent the actual limit of quantitation necessary to accurately and precisely measure the analyte in the sample.	If the analyte is selected as a COPC, then it is assumed to be present at one-half the SQL.
R	The sample results are rejected and are, therefore, unusable due to serious deficiencies in the ability to analyze the sample and meet quality control criteria. The presence or absence of the analyte cannot be verified.	Data were excluded from the HHRA.

2.1.3 Duplicate Results

The highest measured concentrations of duplicate sample analytical results were used as the concentration term in the HHRA. If both duplicate samples are non-detect, then one-half of the lower reporting limit was adopted as the proxy sample point concentration for the purpose of calculating exposure point concentrations (EPCs).

2.1.4 Data Tabulation

To facilitate the data evaluation process, the analytical results were tabulated as follows:

- The analytical data were divided into groups by sample location identification numbers, sample collection dates, sampling zone, sampling areas, and environmental media of concern.
- Analytical results were reported in the text, tables and figures using a consistent and conventional unit of measurement such as microgram per liter (μg/L) for groundwater and surface water analyses, milligrams per kilogram (mg/kg) for soil and sediment analyses, and milligrams per cubic meter (mg/m^3) for air analyses.

Summary tables were prepared in accordance with the format recommended in RAGS, Part D (USEPA 2001a), to present relevant statistical data, such as the frequency of detection, the detection limits, the range of detected concentrations, the distribution of data and the source term concentrations to be used in the HHRA. However, RAGS Part D formatted tables provided by USEPA were not used to present this information.

2.2 ANALYTICAL DATA USED TO EVALUATE RISK

Although there has been a considerable amount of data collected at the Site, not all data collected were considered appropriate for evaluating human health risk. The sections below summarize the data selected for this HHRA.

2.2.1 Soil

Both surface and subsurface soil from several historical sampling events were evaluated in this HHRA. Data from sampling events completed between 1994 and 2005 were evaluated for inclusion in the HHRA. In general, all data from the previous investigations were used in the HHRA. However, a separate evaluation was performed by excluding chemical concentrations exceeding the soil saturation limit (Csat) in the derivation of concentration terms. This evaluation was prepared in response to review comments on the draft HHRA report. Information regarding this evaluation is presented in **Attachment H**.

Attachment H1	Calculation of Chemical-specific Csat Values
Attachment H2	Exposure Point Concentration Summary

Attachment H3 ProUCL Output Tables
Attachment H4 Risk Calculations

Surface soil is defined as soil from 0 to 1 foot below ground surface (bgs). Subsurface soil is defined as soil between 1 and 10 feet bgs. For this Site, 10 feet was selected as the limit to which construction activities may occur. Ten feet was selected based on the future recreational land use of the Site. It was assumed that 10 feet was the maximum depth at which utilities would be installed.

Tables 1 to **5** present the surface and subsurface soil sample locations used for this evaluation by receptor. These tables also define the source of each data point used in the evaluation. **Table 2-6** of the RI report identifies the analytical parameters completed for soil. **Tables 4-8A** and **4-8B** of the RI report provides a summary of the analytical results for surface and subsurface soil. **Figure 5** graphically presents the sample locations selected to evaluate human receptors at the Site.

2.2.2 Sediment

The sediment data used to evaluate human receptors was selected based on those areas in Chequamegon Bay that are associated with human activity and are at depths that are likely to be contacted. Waders are typically assumed to come in contact with surface sediments only when evaluating exposures associated with a wading scenario. For this HHRA, sediment data between 0 to 2 foot in depth and with 4 feet or less of surface water cover were used in response to review comments on the draft HHRA Report.

Presented below is a list of sediment locations evaluated in the HHRA.

2200N-1600E	2400N-2000E
2250N-1400E	2400N-2100E
2300N-1600E	2400N-2100E
2300N-1700E	2400N-2200E
2350N-1400E	2400N-2300E
2400N-1200E	2500N-2300E
2400N-1300E	2600N-2400E

These data were selected based on a conservative assumption that waders may come in contact with affected sediments at depths of up to 4 feet when collecting wood. In addition, the sampling locations selected to evaluate risk were also chosen to reflect the estimated 2 feet drop in Lake Superior water levels observed in 2007.

In addition, it was also assumed that sediment exposures could occur during surface water exposures. In this instance chemicals that are adsorbed on suspended sediment particles are assumed to be available for contact. However, there are no measured concentrations for this data set. Instead, a contact rate was developed based on the total suspended solids measurement of surface water using the equation below.

$$\text{Sediment Ingestion Rate } {}^{mg}/_{hour} = \text{Surface Water Ingestion Rate } \left({}^{mL}/_{hour}\right) \times \textit{Total} \text{ Solids } {}^{mg}/_{mL}$$

Table 6 presents the sediment data used for this HHRA. **Table 2-6** of the RI report identifies the analytical parameters completed for sediment. **Table 4-9** of the RI report provides a summary of the analytical results for sediment. **Figure 5** outlines those locations that were selected to evaluate human receptors at the Site.

2.2.3 Surface Water

It was assumed that all surface water within affected areas of Chequamegon Bay could be accessed during recreational activities; therefore, analytical data collected in 1998 (by SEH) and 2005 (as part of the RI) were used in the HHRA.

Evaluating exposures to contaminated surface water has been challenging at the site due to a limited number of samples collected when natural factors caused the release of tar slicks. On November 15, 2005, during RI sampling activities, surface water samples were collected shortly after a tar slick was reported and photographed by a citizen, however, no slicks were observed by sample collectors and the subsequent data does not indicate notable surface water impacts. However, based on a single surface water sample collected on January 14 and 15, 1998, the 1998 SEH report calculated unacceptable levels of current and future health risks for workers, trespassers, and people engaged in recreational activities on the site. Since this exposure pathway poses one of the greatest potential health risks at the site, the revised HHRA report includes an evaluation of exposures to "oil slicks" in surface water. Because no samples of the "oil slick" have been collected, this exposure medium and associated pathways were evaluated using:

- Laboratory analytical data of the dense non-aqueous phase liquid (DNAPL) samples collected from the product stream recovered from the active free product recovery system for the Copper Falls aquifer (**Attachment I1**)

- Chemical-specific solubility values of chemicals detected in the DNAPL sample (**Attachment I2**).

The use of this alternative data in evaluating the surface water exposure pathway has limitations and uncertainties, and is very conservative. A discussion of these limitations and uncertainties associated with the use of this data is provided in **Section 6**.

Table 7 identifies those sample data by sampling event that were used to evaluate exposure to surface water. **Table 2-6** of the RI report identifies the analytical parameters completed for surface water. **Table 4-11** of the RI report provides a summary of the analytical results for surface water. **Figure 5** shows those surface water locations that were selected to evaluate human receptors at the Site.

2.2.4 Air

2.2.4.1 Soil Vapor

Soil vapor samples were collected from soil vapor probes installed in the uppermost water-bearing unit in the vicinity of the former MGP facility. These samples were collected to provide data that were used to evaluate potential vapor migration and to ensure that soil vapors are not migrating off-site through subsurface soil towards adjacent private properties and into residential structures.

Table 8 presents the soil vapor data used for the HHRA. **Table 2-7** of the RI report identifies the analytical parameters completed for soil vapor. **Table 4-12** of the RI report provides a summary of the analytical results for soil vapor. **Figure 5** presents locations selected to evaluate human receptors at the Site.

2.2.4.2 Indoor Air Vapor Investigation

An indoor air sample was collected to evaluate the potential for vapor migration into the existing NSPW Service Center building, which overlies impacted soil in the backfilled ravine. The indoor air investigation was designed to evaluate the chemicals present in indoor air and sub-slab soil vapors to determine if this area is being impacted by soil vapor migration and intrusion. **Table 8** presents the indoor air data used for the HHRA. **Table 2-7** of the RI report identifies the analytical parameters completed for indoor air. **Figure 5** presents those locations selected to evaluate human receptors at the Site.

2.2.4.3 Trench Air

Construction worker exposures to VOCs in trench air were estimated using the maximum detected concentrations in groundwater for Kreher Park, the Upper Bluff, and the Filled Ravine areas of the Site.

Table 4-7 of the 2006 RI report presents the groundwater data used for the HHRA. **Figure 2-1** of the same report shows the locations where groundwater samples were collected.

2.2.5 Biota

Several species of fin fish were collected at the Site. However, for the HHRA only the following three were assumed to be consumed on a consistent basis. These fin fish include:

- Shorthead Redhorse (*Moxostoma macrolepidotum*)
- Walleye (*Stizostedion vitreum*)
- Rainbow Smelt (*Osmerus mordax*)

Although samples were prepared and analyzed as either whole fish or fillets, only data associated with the edible portion were used in the HHRA. It was assumed that the sample as prepared for sampling corresponded to the edible portion of the fish. Fish were prepared as indicated below.

- Eight whole fish composite samples of smelt were collected from the Site and prepared as if for frying, i.e. their heads and entrails removed.
- Walleye were filleted (the skin was removed)

- Shorthead redhorse were processed as for smoking or pickling, i.e., only the head and entrails were removed.

Table 9 lists the fish samples used for this HHRA. **Table 2-6** of the RI report identifies the analytical parameters completed for fish tissue. **Figure 6** illustrates the locations selected to evaluate human receptors at the Site.

2.2.6 Exposure to Shallow Uncovered Groundwater

While groundwater at the site is not currently used for drinking water, it is plausible that future construction workers digging trenches in Kreher Park could be exposed to COPCs in shallow groundwater via dermal contact, inhalation, and incidental ingestion. Because oily materials in groundwater at Kreher Park were not sampled during the RI, the COPCs and related concentrations used for evaluating this exposure pathway were derived from the laboratory analytical data of the DNAPL samples collected from the product stream recovered from the active free product recovery system for the Copper Falls aquifer (Appendix D6 of the RI report) and chemical-specific solubility values of chemicals detected in the DNAPL sample.

2.3 IDENTIFICATION OF CHEMICALS OF POTENTIAL CONCERN

The procedures used for selecting COPCs evaluated in the baseline HHRA are summarized in the following sections.

2.3.1 Comparison with Background Concentrations

USEPA provides guidance indicating that an inorganic chemical can be excluded from further consideration in the HHRA if the detected concentrations are within the range of naturally occurring background levels (USEPA, 1989). Although background levels were identified in the RI/FS Work Plan as one of the screening criteria for identifying COPCs, no inorganic chemicals were excluded from the HHRA based on background comparison due to the lack of relevant medium-specific background levels.

2.3.2 Risk-Based Screening Approach

Although the presence of many chemicals may be identified in the environmental samples collected during site investigative activities, the results of a baseline HHRA are typically driven

by a few chemicals and exposure pathways. To streamline the HHRA process and focus efforts on important issues, several methods have been developed by the regulatory agencies and the scientific community for the identification of chemicals and pathways that contribute significantly to the total risks posed by a site. A tiered, risk-based approach was used for the selection of COPCs to be further evaluated in the detailed HHRA for the Site. This approach is based on USEPA-developed methodology and follows standard HHRA procedures.

The maximum detected concentration of a chemical was compared with chemical- and medium-specific risk-based screening concentrations (RBSCs), defined as concentrations that are not expected to result in any adverse impact based on exposure conditions which served as the basis for the calculation. A chemical was selected as a COPC if its maximum detected concentration value exceeds the RBSC.

However, because there were no data collected that is representative of the oily materials in groundwater and surface water, laboratory analytical data collected from the product stream recovered from the active free product recovery system for the Copper Falls aquifer (Attachments I1 and I2) were used to evaluate risks to the construction worker, recreational swimmer and recreational wader receptors. Because there are no readily available risk-based screening values for oily materials, all chemicals that were detected in the product stream were selected as COPCs.

For the evaluation of construction worker dermal and inhalation exposures to VOCs in trench, the maximum detected groundwater concentration at three domains (Kreher Park, Upper Bluff, Filled Ravine) was used to estimate risk. All chemicals detected in groundwater were identified as COPCs. The groundwater data was not screened against RBSCs concentration prior to risk characterization. This approach potentially overestimates risks to construction worker receptors as not all chemicals detected were present at concentrations greater than their RBSC.

For purposes of this project, the preliminary remediation goals (PRGs) derived by the USEPA Region 9 (USEPA, 2004b) were adopted as the primary source of RBSCs because they are based on conservative assumptions of exposure scenarios. In addition, the use of these PRGs for screening purposes is considered to be common practice by USEPA Region 5.

For those chemicals lacking an RBSC (i.e., PRG or risk-based concentration [RBC]) the standard practice of selecting surrogate chemicals based on similarities in structure was used to determine

if a chemical should be included as a COPC. The surrogates used are identified in **Tables 10** to **18.**

It should also be noted that RBSCs that are protective of noncarcinogenic effects were adjusted by a factor of 0.1 (i.e., divided by a factor of 10) to account for possible additive effects of multiple chemicals. All RBSCs for the protection of carcinogenic effects are based on a target cancer risk of 1E-06.

Sources of the RBSCs used for this project are presented below by media of concern.

	PRG				RBC	AWQC	VI
	Industrial Soil	Residential Soil	Tap Water	Ambient Air	Fish Tissue	Surface Water Ingestion	Target Indoor Air Concentration
Chemicals in Soil	×	×					
Chemicals in Indoor Air/Soil Gas							×
Chemicals in Trench Air				x			
Chemicals in Surface Water						×	
Chemicals in Sediment		×					
Chemicals in Fish Tissue					×		
Chemicals in Groundwater			x				

PRG – USEPA Region 9 Preliminary Remediation Goal (October 2004) (USEPA, 2004b).
RBC – USEPA Region 3 Risk-based Concentrations (October 2005) (USEPA, 2005a)
AWQC – USEPA National Recommended Water Quality Criteria (20026) for human health (water and organism) (USEPA, 2006a).
VI – Evaluating The Vapor Intrusion To Indoor Air Pathway From Groundwater and Soils (USEPA 2001b).

2.3.3 COPC Summary

The COPCs identified for this are primarily metals, SVOCs, and limited VOCs. A summary of the COPCs by receptor and medium is presented below. **Tables 10 to 19** present the detail screening summary tables by receptor and medium.

Summary of the COPCs by Receptor and Medium

Chemical[a]	Residential				Commercial/Industrial			Construction Worker	Recreational			
					General Industrial Worker		Maintenance Worker		Recreational User	Swimmer Wader		Fisherman
	SS & SB	SS	S:0-3	IA	SS	IA	SS	SS & SB & OA & GW	SS	SD	SW	Fish
Inorganics												
Antimony												
Arsenic	×	×	×		×		×	×	×			
Cadmium									×			
Iron										×		
Lead	×	×	×				×	×	×			
Manganese										×		
Thallium	×								×			
Vanadium										×		
SVOCs												
1-Methylnaphthalene	×		×					×		×		×
2-Methylnaphthalene	×							×		×		
Acenaphthene	×							×		×		
Benzo(a)anthracene	×		×				×	×	×	×	×	×
Benzo(a)pyrene	×	×	×		×		×	×	×	×	×	×
Benzo(e)pyrene												×
Benzo(b)fluoranthene	×		×				×	×	×	×	×	×
Benzo(k)fluoranthene	×		×					×	×	×	×	
Chrysene	×							×		×	×	
Dibenzo(a,h)anthracene	×		×					×		×	×	×
Dibenzofuran	×							×				×
Fluoranthene	×		×					×		×		
Fluorene	×							×		×		
Indeno(1,2,3-cd)pyrene	×		×				×	×	×	×	×	
Naphthalene	×		×					×		×		
Phenanthrene	×											
Pyrene	×		×					×		×		

Chemical[a]	Residential				Commercial/Industrial			Construction Worker	Recreational			
					General Industrial Worker		Maintenance Worker		Recreational User	Swimmer Wader		Fisherman
	SS & SB	SS	S:0-3	IA	SS	IA	SS	SS & SB & OA & GW	SS	SD	SW	Fish
VOCs												
1,2,4-Trichlorobenzene	×							×				
1,2,4-Trimethylbenzene	×		×			×		×				
1,3,5-Trimethylbenzene	×		×					×				
1,4-Dichlorobenzene						×						
Benzene	×		×			×		×				
Ethylbenzene								×				
n-Butyl benzene								×				
sec-Butyl benzene								×				
Carbon tetrachloride						×						
Methylene chloride										×		
Toluene								×				
Trichloroethylene						×						
Xylenes (total)	×							×		×		

Notes:
SS – surface soil
SB – subsurface soil
S:0-3 – soil (0 to 3 foot depth)
GW – shallow groundwater
IA – indoor air
OA – outdoor air
SD – sediment
SW – surface water

Exposure assessment involves the identification of the potential human exposure pathways at the Site for present and potential future use scenarios. Present conditions are as they exist today and future conditions are based on potential future land uses of the Site should there be no cleanup. Potential release and transport mechanisms were identified for contaminated source media. Exposure pathways identified in the WDNR HHRA (SEH, 1998) were finalized by assessing additional information gathered during this RI.

The exposure pathway links the sources, types of environmental releases, and environmental fate with receptor locations and activity patterns. Generally, an exposure pathway is considered complete if it consists of the following four elements:

- A source and mechanism of release;
- A transport medium;
- An exposure point (i.e., point of potential contact with an impacted medium); and
- An exposure route (e.g., ingestion) at the exposure point.

All present and potential future use scenarios presented in the RI/FS Work Plan (URS, 2005) were evaluated. However, additional site-specific information gathered during the implementation of the work plan resulted in the deletion of some exposure scenarios for quantitative analysis. The rationale for exclusion of these exposure scenarios is discussed in Section 3.1.4.

3.1 HUMAN HEALTH CONCEPTUAL SITE MODEL

A conceptual site model (CSM) for the Site has been developed to identify the focus of the HHRA. A schematic presentation of the CSM is included as **Figure 7**. The CSM integrates historical information to preliminarily define source areas, release and transport processes, points of contact with affected media, complete and incomplete exposure routes, and potentially exposed populations for current and expected future Site uses. The CSM was refined based on Site-specific information gathered during the implementation of the work plan.

3.1.1 Known and Suspected Sources of Chemical Impacts and Release Mechanisms

Based on information with respect to the history of the Site and the results of previous investigations, the potential primary sources of impact are likely associated with past industrial operations; e.g., past releases from the former MGP, releases of petroleum-based products from

railcar off loading, releases from the construction and operation of the former WWTP, releases from filling activities at the Lakefront, or a combination of these possible sources. Surface and subsurface soil and groundwater that have been impacted may act as secondary sources of impact through mechanisms such as leaching of chemicals from soil, groundwater recharge to surface water and wind and mechanical erosion of chemicals in soil.

3.1.2 Retention or Transport Media

The medium directly impacted by past industrial activities is soil. Dust is considered a potential transport medium, because chemicals in soil may become entrained in fugitive dust. Surface runoff is considered a transport medium, because storm events may have generated episodic overland flow and carried chemicals away from disposal or spill areas.

3.1.3 Transport Pathway

Release mechanisms and transport pathways were evaluated for the Site. Listed below are potential cross-media transfer mechanisms of chemicals:

- Chemicals in subsurface soil may enter groundwater through infiltration/percolation;
- Chemicals in surface soil may be transported to surface water and sediments through surface runoff;
- Chemicals in groundwater may be transported to surface water and sediments through groundwater discharge;
- Chemicals in groundwater may become uncovered as surface water when a trench is excavated in Kreher park soils;
- Chemicals in surface soil may be transported to the atmosphere via volatilization or fugitive dust emission;
- Chemicals in soil or groundwater may be transported to the atmosphere or indoor air through volatilization;
- Chemicals in surface water and sediments may be transported to fish tissue through bioconcentration; and
- Chemicals in sediments may be released to surface water when agitated.

3.1.4 Receptors and Exposure Scenario

Presented below is an overview of populations of potential concern selected for further evaluation in this HHRA. Potential receptors are discussed based on medium of interest (i.e., soil, groundwater, sediment, surface water, biota, and air). Updates to the receptor populations identified in the Final RI/FS Work Plan (URS, 2005) are discussed as necessary.

3.1.4.1 Exposure to COPCs in Soil

Residential Land Use Scenario: **Child and Adult Residents**

Upper Bluff - There is a residential area located up gradient from the Kreher Park area of the Site on the upper bluff area near the former ravine. Described below were three exposure scenarios assumed in this HHRA for the residential receptors:

- **Exposure to surface (0-1 ft) and subsurface soil (1-10 feet bgs)** This assumption was made because new construction would involve excavation of soil for the construction of basements. Therefore, subsurface soil would be brought to the surface resulting in a potential exposure pathway for residential receptors. This scenario represents the worst case for residential receptors, but is not likely to be the actual scenario associated with the Site.
- **Exposure to surface soil** The residential neighborhoods adjacent to the Site are established neighborhoods and are expected to remain in the future. According to the Ashland Wisconsin Waterfront Development Plan, the future use of the Kreher Park portion of the Site does not include a residential scenario. In an established residential setting and without intrusive activities, receptors would most likely be exposed to surface soil only.
- **Exposure to soil in 0-3 ft bgs** For informational purposes, COPCs in soil between 0 and 3 ft bgs were also considered for residential receptors based on the assumption that receptors could potentially be exposed to soil in 0-3 ft bgs when performing landscaping or gardening activities.

For the purpose of this HHRA, child and adult residents are assumed to be exposed to COPCs in soil via incidental ingestion, inhalation (of soil-borne vapor and particulates) and dermal contact pathways.

Recreational Use Scenario: Child, Adolescent and Adult Visitors

Kreher Park is now comprised of City parkland. Child, adolescent and adult visitors are assumed to be exposed to COPCs in surface soil via incidental ingestion, inhalation (of soil-borne vapor and particulates) and dermal contact pathways.

Industrial/Commercial Land Use Scenario: Maintenance Workers

Although the Final RI/FS Work Plan (URS, 2005) indicated maintenance workers currently access the Site, additional information collected during the implementation of the RI/FS Work Plan indicates that City workers and utility maintenance personnel do not access the Site. However, the City may develop the existing marina and expand it into the affected area for recreational use. Therefore, a potential future maintenance worker was considered a receptor to surface soil at Kreher Park and the unpaved portions of the Upper Bluff area. It is conservatively assumed that maintenance workers may be exposed to COPCs in surface soil via incidental ingestion, inhalation (of soil-borne vapor and particulates) and dermal contact pathways.

Industrial/Commercial Land Use Scenario: General Industrial Workers

Except for the NSPW facility, no other industrial/commercial facilities exist within the Site. For this HHRA, general workers are defined as NSPW employees involved with non-intrusive, operational activities. Current and potential future general workers are not likely to be subject to significant exposure to environmental media in the normal course of their daily work. Although the potential for exposure to occur is expected to be low, general workers are assumed to be exposed to COPCs in surface soil via incidental ingestion, inhalation (of soil-borne vapor and particulates) and dermal contact pathways.

Industrial/Commercial Land Use Scenario: Construction Workers

Upper Bluff and Kreher Park - It is conservatively assumed that construction activities could take place at every area included in this evaluation and it is possible for construction workers to be exposed to COPCs detected in surface and subsurface soil samples collected from the Site via incidental ingestion, inhalation (of soil-borne vapor and particulates) and dermal contact pathways. For this HHRA subsurface soil is defined as a depth of 10 feet or less, which is a conservative estimate of the limit to which construction activities may occur based on the current and proposed future land use at the Site.

For informational purposes, a hot spot analysis was performed for construction worker using soil data collected from the Former Coal Tar Dump. The results of this analysis are presented in Section 6.6.

3.1.4.2 Exposure to COPCs in Indoor Air – Residents and Industrial Workers

Upper Bluff - There is a residential area located up gradient from the Kreher Park area of the Site on the upper bluff area, near the former ravine. For the purpose of this HHRA child and adult residents are assumed to be potentially exposed to COPCs volatilizing from soil and groundwater and entering the residences located near the ravine. In addition, potential exposures to COPCs in indoor air were also evaluated for industrial workers who may enter the NSPW service center/vehicle maintenance building periodically.

3.1.4.3 Exposure to COPCs in Groundwater

Trespassing Land Use Scenario: **Trespassers**

The RI/FS Work Plan indicated that groundwater in the seep area was a potential exposure point for trespassers. However, this exposure point has been eliminated because highly impacted soil was removed from the former seep area and the area was capped as part of the 2002 interim action response (URS, 2002). Therefore, this exposure pathway is no longer complete and was not quantitatively evaluated in the HHRA.

Another potential point of exposure described in the RI/FS Work Plan was COPCs in groundwater in the former WWTP building where affected groundwater has infiltrated into the basement. The building is locked and the perimeter is partially fenced. A quantitative evaluation for the potential trespasser exposures to the indoor air and water inside the former WWTP building was not performed due to the lack of data. No water samples were collected from the building. In 2002, a consultant for the City of Ashland inspected the inside of the WWTP building and collected a single round of indoor air samples to address potential inhalation exposure to City of Ashland workers. Samples were only analyzed for limited chemicals (selected PAHs, trimethylbenzene and acetic acid). The results of this sampling indicated that Site-related compounds are probably in the indoor air of the former WWTP building, and a thorough indoor air investigation was recommended before final re-use decisions (WDHFS, 2003).

Although access to the WWTP remains unrestricted, the potential for dermal, inhalation, and incidental ingestion exposure to COPCs in groundwater seepage inside the WWTP building is considered low because the building is locked and the perimeter is partially fenced. If, however, it is deemed necessary to quantitatively evaluate trespasser exposure to COPCs in indoor air from groundwater seepage inside the building, indoor air and water samples should be collected for laboratory analyses for Site-related COPCs to support the development of a quantitative evaluation.

Residential and Industrial/Commercial Land Use Scenarios

Groundwater contamination is present in both a shallow aquifer and a confined deep aquifer. Currently the shallow groundwater is not used as a potable water source. There are two artesian wells in the Site vicinity—one located near Prentice Avenue on the eastern boundary of the Site and the other located near the marina on the western boundary. Both wells draw water from the Copper Falls aquifer, which is a deep aquifer separated from the shallow groundwater by the Miller Creek Formation (URS, 2005; ATSDR, 2003). As precautionary measure, the City of Ashland temporarily closed these wells for public use in August 2004. To date water from these wells have met all federal and state safe drinking water standards. Water from these artesian wells is considered safe to drink as Site-related chemicals have not been detected in these wells at levels of concern (ATSDR, 2003).

Except for the two artesian wells at Kreher Park, the Copper Falls aquifer is not used for drinking water and is not considered a source of human exposure. Shallow groundwater at the Site is not a drinking water source for the City of Ashland. Drinking water at the Site is provided by the City of Ashland that draws its water from intakes in Lake Superior, located approximately one mile northeast of the Site and is outside the known extent of surface water contamination. Therefore, there are no known ingestion receptors to shallow groundwater beneath the Site.

Industrial/Commercial Land Use Scenario: Construction Workers

Kreher Park - It is conservatively assumed that in the future construction activities may take place within Kreher Park and it is possible for construction workers to be exposed to oily materials in groundwater via the dermal contact, inhalation, and incidental ingestion pathways when performing excavation activities below the water table. Because oily materials in groundwater were not sampled during the RI, concentrations of chemicals in "oily water" were based on a derived concentration using the laboratory analytical data of the dense non-aqueous

phase liquid (DNAPL) samples collected from the product stream recovered from the active free product recovery system for the Copper Falls aquifer (Appendix D6 of the RI report) and chemical-specific solubility values of chemicals detected in the DNAPL sample.

Kreher Park, Upper Bluff, Filled Ravine – It is conservatively assumed that trenching activities could take place within Kreher Park, the Upper Bluff, and the Filled Ravine resulting in construction/excavation worker exposure to COPCs in trench air. Because there is no data which measures the concentrations of VOCs in trench air, the maximum detected groundwater concentration within each domain was used to model a trench air concentration to which construction/excavation workers might be exposed. Attachment J of the HHRA, presents the proposed trench air concentrations.

3.1.4.4 Exposure to COPCs in Surface Water and Sediments

Recreational Use Scenario: **Adolescent and Adult Visitors**

Kreher Park and Chequamegon Bay Sediments – The Site is located and surrounded by facilities that draw the public to the lakefront – a City marina, public swimming beach, a boat ramp and an RV park and campground. Adolescent and adult visitors are assumed to be exposed to COPCs in surface water and sediments via incidental ingestion and dermal contact pathways while swimming, wading, fishing, or boating. However, only risks associated with swimming and wading activities were quantified in the HHRA. This is because they represent activities that have the greatest contact with impacted media and are considered more conservative than exposures associated with fishing and boating.

Evaluating exposures to contaminated surface water has been challenging at the site due to a limited number of samples collected when natural factors caused the release of tar slicks. On November 15, 2005, during RI sampling activities, surface water samples were collected shortly after a tar slick was reported and photographed by a citizen, however, no slicks were observed by sample collectors and the subsequent data does not indicate notable surface water impacts. However, based on a single surface water sample collected in January 1998, the 1998 SEH report calculated unacceptable levels of current and future health risks for workers, trespassers, and people engaged in recreational activities on the site. Since this exposure pathway poses one of the greatest potential health risks at the site, the revised HHRA report includes an evaluation of exposures to "oil slicks" in surface water as well as an evaluation of exposures to surface water using the 1998 SEH data. Because no samples of the "oil slick" have been collected, this exposure medium and associated pathways were evaluated using:

- Laboratory analytical data of the DNAPL samples collected from the product stream recovered from the active free product recovery system for the Copper Falls aquifer (**Attachment I1**)
- Chemical-specific solubility values of chemicals detected in the DNAPL sample (**Attachment I2**).

The use of this alternative data in evaluating exposure to COPCs in surface water has limitations and uncertainties, and is very conservative. A discussion of the limitations and uncertainties associated with the use of this data is provided in **Section 6**.

3.1.4.5 Exposure to COPCs in Fish Tissue

Subsistence Fishing Scenario: **Adult Subsistence Fisher**

Impacted Sediment Areas – Adult subsistence fishers were selected as the fishing receptors because there are two Chippewa Bands (the Bad River Band and the Red Cliff Band of Lake Superior Chippewa) who may use Chequamegon Bay as their source of fish. For this HHRA it is conservatively assumed that adult subsistence fishers may be exposed to COPCs via ingestion of locally-caught fish. Although this scenario was selected based on the presence of the two Chippewa Bands, this exposure scenario and the selected exposure parameters are applicable to any subsistence fisher ingesting fish from Chequamegon Bay. **Attachment A** provides detailed information regarding the exposure parameters used and their sources.

Presented below is an overview of receptors of potential concern selected for further evaluation in this HHRA. Potential receptors are discussed based on medium of interest (i.e., soil, sediment, surface water, biota, and air). A detailed discussion of the risks associated with each receptor population is presented in **Section 5.1.**

SUMMARY OF PATHWAYS EVALUATED IN HHRA							
Receptor Pathway	**Media of Interest[a]**						
	Surface Soil	**Subsurface Soil**	**Sediment**	**Surface Water**	**Indoor Air**	**Groundwater**	**Biota**
Industrial/Commercial Exposure Scenario/General Industrial Workers:							
Inhalation of airborne COPCs	FMGP				SCB		
Incidental ingestion of COPCs	FMGP						
Dermal contact with COPCs	FMGP						
Industrial/Commercial Exposure Scenario/ Construction Worker:							
Inhalation of airborne COPCs	KP FMGP	KP FMGP					
Incidental ingestion of COPCs	KP FMGP	KP FMGP					
Dermal contact with COPCs	KP FMGP	KP FMGP					
Dermal contact with COPCs in "oily water"						KP	
Incidental ingestion of COPCs in "oily water"						KP	
Inhalation of COPCs in Trench Air[b]						KP UB FR	
Industrial/Commercial Worker Exposure Scenario/Maintenance Worker:							
Inhalation of airborne COPCs	KP UB	KP UB					
Incidental ingestion of COPCs	KP UB	KP UB					
Dermal contact with COPCs	KP UB	KP UB					
WWTP Trespassing Land Use Scenario							
Inhalation of airborne COPCs						KP	
Incidental ingestion of COPCs						KP	
Dermal contact with COPCs						KP	
Recreational Exposure Scenario/Children:							
Inhalation of airborne COPCs	KP						
Incidental ingestion of COPCs	KP						
Dermal contact with COPCs	KP						
Recreational Exposure Scenario/Adolescents:							
Inhalation of airborne COPCs	KP						
Incidental ingestion of COPCs	KP						
Dermal contact with COPCs	KP						
Recreational Exposure Scenario/Adults:							
Inhalation of airborne COPCs	KP						
Incidental ingestion of COPCs	KP						
Dermal contact with COPCs	KP						
Recreational Exposure Scenario/Swimmer & Wader/Adults:							
Incidental ingestion of COPCs			CB	CB			
Dermal contact with COPCs			CB	CB			
Incidental ingestion of COPCs in oil slicks"				CB			

SUMMARY OF PATHWAYS EVALUATED IN HHRA							
Receptor Pathway	**Media of Interest[a]**						
	Surface Soil	**Subsurface Soil**	**Sediment**	**Surface Water**	**Indoor Air**	**Groundwater**	**Biota**
Dermal contact with "oil slicks"				CB			
Recreational Exposure Scenario/Swimmer & Wader/Adolescents :							
Incidental ingestion of COPCs			CB	CB			
Inhalation of airborne COPCs					UB		
Dermal contact with COPCs			CB	CB			
Incidental ingestion of COPCs in oil slicks"				CB			
Dermal contact with "oil slicks"				CB			
Subsistence Fisher Exposure Scenario:							
Ingestion of COPCs in fish							CB
Off-site Residential Exposure Scenario:							
Inhalation of airborne COPCs	UB	UB			UB		
Incidental ingestion of COPCs	UB	UB					
Dermal contact with COPCs	UB	UB					

[a]The data set used to estimate risk for each receptor is defined as indicated below:

- FMGP – Former Manufactured Gas Plant
- KP – Kreher Park
- UB – Upper Bluff
- SCB – Service Center Building
- CB – Chequamegon Bay
- FR – Filled Ravine

[b] For the exposure to COPCs in trench air, it is assumed that workers may inhale COPCs volatilizing from groundwater encountered in the excavated trench (Attachment J).

3.2 QUANTIFICATION OF CHEMICAL INTAKES

Integration of data gathered in the exposure assessment (i.e., the extent, frequency, and duration of exposure for the populations and pathways of concern) into a quantitative expression of chemical-specific intake is necessary to perform a quantitative risk characterization.

The potential for human receptors to be exposed to impacted media through relevant routes of exposure (e.g., inhalation, ingestion and dermal contact) were evaluated. Exposure pathways considered not applicable, based on site-specific information, were excluded from the quantitative evaluation in the baseline HHRA. Rationale for the elimination of exposure pathways is provided in respective sections.

Estimates of intake of COPCs are required for quantitative risk characterization. Described below is the basic equation used to calculate the human intake of COPCs (USEPA, 1989):

$$I = C \times \frac{IR \times EF \times ED}{BW \times AT}$$

Where:

I = Daily intake (mg of chemical per kg of body weight per day)
C = Concentration of COPC (e.g., mg/kg in soil or fish, mg/L in water or mg/m^3 in air)
IR = Intake rate; the amount of contaminated medium contacted over the exposure period (e.g., mg/day for soil and fish, L/day for water and m^3/day for air)
EF = Exposure frequency; describes how often exposure occurs (days/year).
ED = Exposure duration; describes how long exposure occurs (years).
BW = Body weight; the average body weight over the exposure period (kg)
AT = Averaging time; period over which exposure is averaged (days)

Each of the intake variables in the above equation consists of a range of values in the literature. To account for uncertainties associated with parameter values, two separate exposure scenarios were evaluated in this HHRA: a reasonable maximum exposure (RME) scenario and an average case (i.e., central tendency evaluation [CTE]). The RME represents the maximum exposure that is reasonably likely to occur while the CTE is representative of average exposure. The RME scenario was calculated using the 95% upper confidence limit of the arithmetic mean (95% UCLs) concentration and a combination of the mean and upper-bound exposure parameter values. The CTE scenario was calculated using the arithmetic mean concentration as the EPC and the mean exposure parameter values.

General information regarding the formulae and parameter values for pathways evaluated in this HHRA is provided in **Attachment A, Tables 1 - 11** for both the RME and CTE scenarios.

3.3 DISTRIBUTION TESTING AND CALCULATION OF 95% UPPER CONFIDENCE LIMITS

The RI/FS Work Plan (URS, 2005) for the Site provided extensive detail outlining the methodology to be used to test the distribution of each data set and subsequent calculation of the 95% UCLs. For the HHRA, the USEPA guidance "Calculating the Upper Confidence Limits for Exposure Point Concentrations at Hazardous Waste Sites" (USEPA, 2002b) and the accompanying ProUCL software (USEPA, 2004c) was used to estimate UCLs. Although the RI/FS Work Plan approach was in compliance with USEPA guidance, it did not indicate that USEPA software would be used to estimate UCLs for the Site, which is the preferred method for

estimating 95% UCLs. **Attachment B1** provides summary tables which includes RME EPCs for each receptor data set evaluated. RME output from the ProUCL software (USEPA, 2004c) is presented in **Attachment B2**. A summary of the EPCs used for the CTE scenario are presented in **Attachments E**. A summary of the EPCs and associated ProUCL output tables for evaluations discussed in the Uncertainty Analysis (Section 6) is presented in **Attachments F1** and **F2**, respectively.

For this HHRA, distribution testing and UCL calculations were attempted when the sample population was greater than five and the percentage of nondetects was 15% or less. For data sets not meeting these criteria, the maximum detected concentration was selected as the EPC. For evaluating health impacts potentially associated with exposures to lead using either the Integrated Exposure Uptake Biokinetic Model (IEUBK) for Lead (USEPA, 1994; USEPA, 2005b) or the Adult Lead Model (ALM) (USEPA, 2003a), the average concentration of lead was used, in accordance with the USEPA guidance.

3.4 GROUNDWATER AND SURFACE WATER CONCENTRATION TERMS

Oily materials in groundwater or slicks in surface water were not sampled during the RI. For the purpose of this evaluation, concentrations of chemicals in "oily water" were based on the following:

- Laboratory analytical data of the dense non-aqueous phase liquid (DNAPL) samples collected from the product stream recovered from the active free product recovery system for the Copper Falls aquifer (**Attachment I1**).
- Chemical-specific solubility values of chemicals detected in the DNAPL sample (**Attachment I2**).

3.5 TRENCH AIR CONCENTRATION TERMS

No data is available for assessing risk to construction/excavation workers exposed to VOCs in trench air at Kreher Park, the Upper Bluff, or the Filled Ravine. Therefore, the maximum detected concentrations in groundwater for each of these domains was used to model a concentration in trench air using equations presented as part of the Virginia Department of Environmental Quality risk assessment guidance (VADEQ, 2006).

The toxicity assessment provides a framework for characterizing the relationship between the magnitude of exposure to a chemical and the nature and likelihood of adverse health effects that may result from such exposure. In an HHRA, chemical toxicity is typically divided into two categories: carcinogenic and noncarcinogenic effects of concern. Potential health effects are evaluated separately for these two categories, because their toxicity criteria are based on different mechanistic assumptions and associated risks are expressed in different units. Provided in this subsection is an overview of the methodology used to develop a toxicity assessment as part of the HHRA for the Site.

4.1 SOURCES OF TOXICITY INFORMATION

Pertinent toxicological and dose-response information for chemicals were selected from the following sources, in accordance with USEPA guidance (USEPA, 2003b):

- Tier 1 – Integrated Risk Information System (IRIS), available on-line (USEPA, 2006)
- Tier 2 – USEPA's Provisional Peer-Reviewed Toxicity Values (PPRTVs)
- Tier 3 – Other toxicity values (e.g., California Environmental Protection Agency, the

Agency for Toxic Substances and Disease Registry (ATSDR), and USEPA's Health Effects Assessment Summary Tables (HEAST) (USEPA, 1997b).

4.2 METHODOLOGY FOR EVALUATING CARCINOGENIC EFFECTS

For purposes of assessing risks associated with potential carcinogens, the USEPA has adopted the science policy position of "no-threshold;" i.e., there is essentially no level of exposure to a carcinogen which will not result in some finite possibility of tumor formation. This approach requires the development of dose-response curves correlating risks associated with given levels of exposure. Linear dose-risk response curves are generally assumed.

Carcinogenic risks associated with a given level of exposure to potential carcinogens are typically extrapolated based on slope factors (SFs) or unit risks. SFs are the upper 95% confidence limit of the slope of the dose-response curve, expressed in terms of risk per unit dose [given in $(mg/kg\text{-}day)^{-1}$]. Unit risks relate the risk of cancer development with the concentration of carcinogen in the given medium, expressed as either risk per unit concentration in air [given in $(\mu g/m^3)^{-1}$] or drinking water [given in $(\mu g/L)^{-1}$].

Current USEPA Superfund guidance for calculating a dermal SF is to adjust the oral SF with an oral absorption factor specific for that chemical. It should be noted that the oral absorption factor used in the calculation refers to absorption of the chemicals in the species upon which the SF is based; i.e., generally not absorption data in humans.

The equation for extrapolation of a default dermal SF is as follows:

$$\text{Default Dermal SF}\left[(\text{mg/kg - day})^{-1}\right] = \text{Oral SF}\left[(\text{mg/kg - day})^{-1}\right] \div \text{Oral Absorption Factor (\%)}$$

4.3 METHODOLOGY FOR EVALUATING NON-CARCINOGENIC EFFECTS

The USEPA has adopted the science policy position that protective mechanisms (such as repair, detoxification, and compensation) must be overcome before the adverse systemic health effect is manifested. Therefore, a range of exposures exists from zero to some finite value that can be tolerated by the organism without appreciable risk of expressing adverse effects.

The approach used by the USEPA to gauge the potential non-carcinogenic effects is to identify the upper boundary of the tolerance range (threshold) for each chemical and to derive an estimate of the exposure below which adverse health effects are not expected to occur. Such an estimate calculated for the oral route of exposure is an oral reference dose (RfD), and for the inhalation route of exposure is an inhalation reference concentration (RfC). The oral RfD is typically expressed as mg chemical per kg body weight per day, and the inhalation RfC is usually expressed in terms of concentration in the air (i.e., mg chemical per m^3 of air). However, for purposes of baseline HHRAs, inhalation RfC values can be converted to units of dose by multiplying by the inhalation rate (20 m^3/day, an upper-bound estimate for combined indoor-outdoor activity) and dividing by the body weight (70 kg, average body weight), as detailed in the following equation:

$$\textit{Inhalation}\ \text{RfD}\,(\text{mg/kg - day}) = \text{RfC}\,(\text{mg/m}^3) \times 20\,{}^{m^3}\!/_{day} \div 70\,\text{kg}$$

Currently, two types of oral RfDs/inhalation RfCs are available from the USEPA, depending on the length of exposure being evaluated (chronic or subchronic). Chronic oral RfDs/inhalation RfCs are specifically developed to be protective for long-term exposure to a compound, and are generally used to evaluate the non-carcinogenic effects associated with exposure periods between seven years (approximately 10% of an average lifespan) and a lifetime. Subchronic oral

RfDs/inhalation RfCs are useful for characterizing potential non-carcinogenic effects associated with shorter-term exposures. Current guideline for Superfund program risk assessment requires that subchronic oral RfDs/inhalation RfCs be used to evaluate the potential non-carcinogenic effects of exposure periods between two weeks and seven years.

Toxicological criteria specifically derived for gauging potential human health concerns associated with the dermal route of exposure has not been developed by USEPA. For purposes of this HHRA, default dermal RfD values were extrapolated from oral RfDs (USEPA 1989), if:

- Health effects following exposure are not route-specific.
- Portal-of-entry effects (e.g., dermatitis associated with dermal exposure and respiratory effects associated with inhalation exposure) are not the principal effects of concern.

Exposures with the dermal route are generally calculated as absorbed doses, while oral RfDs are expressed as administered doses. Current USEPA Superfund guidance is to adjust the oral RfD with an oral absorption factor (i.e., percent chemical that is absorbed) to extrapolate a default dermal RfD, which is expressed in terms of absorbed dose. It should be noted that the oral absorption factor used in the calculation refers to absorption of the chemicals in the species upon which the RfD is based (i.e., generally not absorption data in humans).

The equation for extrapolation of a default dermal RfD is as follows:

$$\textit{Default}\ \text{Dermal RfD}\ (\text{mg/kg - day}) = \text{Oral RfD}\ (\text{mg/kg - day}) \times \text{Oral Absorption Factor}\ (\%)$$

Toxicity values (both SFs and RfDs) used in this HHRA are provided in **Attachment A, Tables 12a** and **12b**.

4.4 TOXICOLOGICAL PROFILE FOR COPCS

Toxicological profiles are included for all selected COPCs. Toxicological profiles prepared by the ORNL and available through the online Risk Assessment Information System (RAIS) are presented in **Attachment C** on compact discs. For those chemicals for which an ORNL toxicological profile is unavailable on RAIS, an ATSDR toxicological profile was included. For chemicals without either an ORNL or an ATSDR toxicological profile, information from the National Library of Medicine's Hazardous Substance Data Bank is provided.

4.5 EVALUATING EXPOSURES TO LEAD

Because most human health effects data for lead are correlated with concentrations in the blood rather than an external dose, the traditional approach for evaluating health effects cannot be applied to lead. Lead is therefore evaluated separately from carcinogens and noncarcinogens.

USEPA has developed a model for predicting the effect of lead exposure on blood lead concentrations in children exposed to lead – the IEUBK model (IEUBK Windows v1.0 build 261, [December 2005b]). The IEUBK Model is used to predict the risk of elevated blood lead levels in children (under age seven) that are exposed to environmental lead from many sources. The model estimates the risk that a typical child, exposed to specified media lead concentrations, will exceed a certain level of concern (10 micrograms per deciliter [µg/dL]) (USEPA, December 2005b). The target criterion for lead risk is 5% or less of child residents with an estimated blood lead level in excess of 10 µg/dL. The 10 µg/dL value is the "concern threshold" recommended by the U.S. Centers for Disease Control and Prevention (CDC) (ATSDR, July 1999).

The IEUBK model was run using site-specific lead concentrations in soil and default values for all other parameters (**Attachment A, Table 13**).

USEPA has also developed an ALM (version 05/19/2003) that can be applied to adult worker receptors. The ALM is currently the accepted and standard model to assess adult non-residential exposures to lead in soil and indoor dust. The model uses a simplified representation of lead biokinetics to predict quasi steady-state blood lead concentrations among adults who have relatively steady patterns of site exposures. The methodology focuses on estimating fetal blood lead concentrations in female workers. All the equations in the model are used to calculate target concentrations based on the probability of exceeding a blood lead level of 10 µg/dL for a fetus. Lead risks are considered unacceptable for a non-residential (worker) receptor if the fetal blood lead level for more than 5% of fetuses of adult female workers is estimated to equal or exceed the CDC concern threshold of 10 µg/dL. The ALM model was run using site-specific lead concentrations in soil and default values for all other parameter (**Attachment A, Table 14**).

The ALM is used to evaluate risks of lead exposure to the fetus of pregnant female industrial workers, construction workers, and other workers that are identified as relevant receptors at a site. Other worker standards or guidelines are cited for comparative purposes (ATSDR, July 1999). The Occupational Safety and Health Administration (OSHA) blood lead level of concern in adult workers (all occupations) is 30 µg/dL; the OSHA permissible standard is 40 µg/dL for

all workers. OSHA established medical removal criteria for workers of 50 μg/dL, with reentry into the workplace allowed at 40 μg/dL. The American Conference of Governmental Industrial Hygienists (ACGIH) also established a blood lead level of concern of 30 μg/dL in workers.

5.1 RISK CHARACTERIZATION

In this section of the HHRA, toxicity and exposure assessments were integrated into quantitative and qualitative expressions of carcinogenic and noncarcinogenic risks. The detailed estimates of risks are presented numerically in **Attachment D** and are summarized in **Sections 5.1** and **5.2**.

Carcinogenic risks are estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to a potential carcinogen. In accordance with guidance provided in RAGS, Part A (USEPA 1989), incremental risk of an individual developing cancer can be estimated by multiplying the calculated daily intakes, that are averaged over a lifetime of exposure, by the SFs. This carcinogenic risk estimate represents an upper-bound value since the SF is often an upper 95% confidence limit of probability of response that is extrapolated from experimental animal data using a multistage model.

The potential for noncarcinogenic effects was evaluated by comparing an exposure level over a specified time period with an RfD derived for a similar exposure period. This ratio of exposure to toxicity is referred to as a hazard quotient (HQ). This HQ assumes there is a level of exposure below which it is unlikely even for sensitive populations to experience adverse health effects. If the HQ exceeds one, there may be concern for potential noncancer effects; however, this value should not be interpreted as a probability.

Carcinogenic and noncarcinogenic risk estimates were combined across pathways, as appropriate, to account for potential additive effects. The sum of HQs is termed a hazard index (HI). In general, USEPA recommends a target value or risk range (i.e., HI = 1 or cancer risk [CR] = 10^{-4} to 10^{-6}) as threshold values for potential human health impacts. The WDNR and WDHFS recommend a target value or cancer risk threshold of 10^{-5} and noncancer risk threshold of 1 for potential human health. For the HHRA, risks are compared to both the USEPA, WDNR, and WDHFS target risk values.

When the HI exceeds unity, then the HQs will be segregated based on similarities in target organ effects. Information regarding target organs following exposures to COPCs was retrieved from the following sources:

- Risk Integrated System for Closure. Indiana Department of Environmental Quality.
- Tiered Approach to Corrective Action Objectives. Illinois Environmental Protection Agency.

- *Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites.* March 2002. OSWER 9355.4-24 (USEPA, 2002a).

The risk characterization results presented in **Attachment D** for the RME scenario were compared to these target levels and are presented below for all media evaluated. These levels aid in determining the objectives of the baseline HHRA, which include determining whether additional response action is necessary at the Site. These levels provide a basis for determining residual chemical levels that are adequately protective of human health, provide a basis for comparing potential health impacts of various remedial alternatives, and help support selection of the no-action remedial alternative, where appropriate.

5.2 RISK CHARACTERIZATION RESULTS

Risks were compared to both USEPA target risk ranges (CR=10^{-4} to 10^{-6} and HI =1) as well as the target risk thresholds for WDNR. Where the calculated carcinogenic and noncarcinogenic risk exceed the either threshold, it is noted in the text discussion below. **Attachment D** provides a detailed presentation of the carcinogenic and noncarcinogenic risk calculations.

5.2.1 Risk Summary for the Residential Scenario

Risks associated with exposure to surface and subsurface soil for residents are a CR of 5×10^{-4} and an HI of 15 for samples collected within the filled ravine of former MGP. Both the cancer and noncancer risk exceed the USEPA target risk range of 10^{-4} to 10^{-6} and the WDNR threshold of 10^{-5} for cancer and an HI of 1 for noncancer endpoints, respectively. The resulting cancer risk of 5×10^{-4} is primarily attributed to benzo(a)pyrene (65%) and dibenzo(a,h)anthracene (10%). Upon review of the data gathered for benzo(a)pyrene, 10 sampling locations (located in both the filled ravine and the Upper Bluff) with detectable concentrations ranging from 22 to 340 mg/kg at intervals between 1 to 8 feet bgs are the main contributors to the benzo(a)pyrene cancer risk. In addition, one sample location for dibenzo(a,h)anthracene (CP110) with a reported concentration of 3.8 mg/kg (1 to 3 feet bgs) is the main contributor to the dibenzo(a,h)anthracene cancer risk.

The resulting HI of 15 is primarily attributed to naphthalene (with an HI of 11). Detailed calculations of cancer and noncancer risk are presented in **Attachment D, Tables 1** through **3**. Based on the results of the IEUBK model inputting an average lead concentration of 90.5 mg/kg,, the percentage of children predicted to have a blood lead concentration greater than 10

μg/dL is 0.11, which is within USEPA's target criteria of no more than 5% above the concern threshold of 10 μg/dL concentration. The results of the IEUBK are presented in Attachment D, Table 3f. While one location (GP-110 (1-3')) had a highly elevated lead concentration of 4000 mg/kg, only one other sample (GP-115 (1-3') had a concentration (480 mg/kg) that exceeded the screening level of 400 mg/kg. Thus, while there are elevated concentrations are in the loading dock area of the NSPW, the average concentration is below the screening level.

Based on the results of the IEUBK model, the percentage of children predicted to have a blood lead concentration greater than 10 μg/dL is 0.11, which is within USEPA's target criteria of no more than 5% above the concern threshold of 10 μg/dL concentration. The results of the IEUBK are presented in **Attachment D, Table 3f**.

5.2.1.1 Indoor Air Pathway

Measured concentrations in soil vapor samples collected from subsurface soil within the filled ravine area of the Site did not exceed the USEPA's risk target shallow soil vapor screening concentrations at a target risk level of 10^{-5} (**Table 17**) indicating that subsurface vapors are not migrating off-site towards the residential area at St. Claire Street and Prentice Avenue.

Summary of RME Carcinogenic and Noncarcinogenic Risks[a]

Receptor	Table	Soil		Oily Materials in Surface Water[d]		Sediment		Oily Materials in Groundwater[d]		Biota		Indoor Air[b, c]	
		CR	HI	CR	HI	CR	HI	CR	HI	CR	HI	CR	HI
Resident	20	**5×10^{-4}**	**15**	–	–	–	–	–	–	–	–		
Recreational Adult	21	4×10^{-6}	0.002	–	–	–	–	–	–	–	–	–	–
Recreational Adolescent	22	2×10^{-6}	0.003	–	–	–	–	–	–	–	–	–	–
Recreational Child	23	1×10^{-5}	0.04	–	–	–	–	–	–	–	–	–	–
Adult Swimmer	24	–	–	**9×10^{-2}**	**6**	4×10^{-5}	0.05	–	–	–	–	–	–
Adolescent Swimmer	25	–	–	–	–	2×10^{-5}	0.05	–	–	–	–	–	–
Adult Wader	26	–	–	**5×10^{-2}**	**4**	4×10^{-5}	0.05	–	–	–	–	–	–
Adolescent Wader	27	–	–	–	–	2×10^{-5}	0.05	–	–	–	–	–	–
Industrial Worker	28 29	6×10^{-6}	0.007	–	–	–	–	–	–	–	–	**8×10^{-5}**	**3**
Maintenance Worker	30	1×10^{-6}	0.001	–	–	–	–	–	–	–	–	–	–
Construction Worker[c]	31	**1×10^{-4}**	**38**	–	–	–	–	**7×10^{-3}**	**59.5**	–	–	**8.34E-03 (KP) 2.14E-05 (UB) 3.29E-02 (FR)**	**17152 (KP) 228 (UB) 646601 (FR)**
Subsistence Fisher	32	–	–	–	–	–	–	–	–	**1×10^{-4}**	0.01	–	–

[a] No COPCs were identified for soil gas and surface water. Risks based on exposure to these media were not quantified.
[b] For the industrial worker, the air risks were estimated using indoor air data from sample locations NS-GSINDOOR-0405 and NS-GSINDOOR-0705.
[c] For the construction worker, the groundwater risks were calculated using a derived concentration of "oily materials" in groundwater estimated using the laboratory analytical data of the DNAPL samples collected from the product stream recovered from the active free product recovery system for the Copper Falls aquifer.
[d] Represents the linear low dose risks calculated for both the recreational and construction receptors. The non-linear low dose risks are presented in Attachments I1 and I2. Although calculations of the surface water risks associated with exposures to the 1998 SEH data were completed (Attachment K), only the oily slicks risk results are presented since they represent the most conservative approach.

Risks in bold are greater than the USEPA range for acceptable risk (1×10^{-4} to 1×10^{-6}). Cancer risks that are underlined are greater than the Wisconsin Department of Public Health threshold of 1×10^{-5}.

KP – Calculated using the Virginia Department of Environmental Quality Equation 3-8: Exposure of Workers to Volatiles in a Construction/Utility Trench (Groundwater less than 15 feet deep). Maximum detected concentrations in groundwater from Kreher Park were used as the exposure point concentration. Detailed calculations for this exposure pathway are presented in Attachment J.

UB – Calculated using the Virginia Department of Environmental Quality Equation 3-8: Exposure of Workers to Volatiles in a Construction/Utility Trench (Groundwater less than 15 feet deep). Maximum detected concentrations in groundwater from the Upper Bluff were used as the exposure point concentration. Detailed calculations for this exposure pathway are presented in Attachment J.

FR – Calculated using the Virginia Department of Environmental Quality Equation 3-8: Exposure of Workers to Volatiles in a Construction/Utility Trench (Groundwater less than 15 feet deep). Maximum detected concentrations in groundwater from the Filled Ravine were used as the exposure point concentration. Detailed calculations for this exposure pathway are presented in Attachment J.

5.2.1.2 Residential Risk Discussion

PAHs appear to be the primary risk drivers for the residential receptor within the filled ravine area of the former MGP. The highest concentrations of PAHs, and thus the highest risks, are associated with PAHs detected at depths of 0 to 3 feet bgs. However, residents are not currently located in this area of the Site and residential areas are not likely to be established at this part of the Site in the future.

For this HHRA, it was conservatively assumed that the residential receptors would be exposed to both surface and subsurface soil. This assumption was made because new construction would involve excavation of soil for the construction of basements or foundations. Therefore, soil with high chemical concentrations would be brought to the surface resulting in a potential exposure pathway for residential receptors. This scenario represents the worst case for residential receptors, but is not likely to be the actual scenario associated with the Site. The residential neighborhoods adjacent to the Site are established neighborhoods and are expected to remain in the future. According to the Ashland Wisconsin Waterfront Development Plan, the future use of the Kreher Park portion of the Site does not include a residential scenario. Therefore, residential receptors would only be exposed to surface soil. If it is assumed that residential receptors adjacent to the Site tend gardens, then it is possible that the first three feet of soil will represent the most likely exposure point.

Re-evaluating the residential receptor using EPCs derived based on the exposure to surface soil and soil to a depth of 3 feet indicates that carcinogenic and noncarcinogenic risks within USEPA's target risk range of 10^{-4} to 10^{-6} for cancer endpoints and an HI of 1 for noncancer endpoints. However, the estimated cancer risk for surface soil remains above the WDNR threshold of 10^{-5}.

Receptor	Table	Soil	
		CR	HI
Resident (Surface Soil only)	33	1×10^{-5}	0.2
Residential (0-3 feet bgs)	34	3×10^{-4}	0.9

The resulting CR of 1×10^{-5} for exposure to surface soil only is primarily attributed to arsenic (76%). Upon review of the data, one sampling location (lSS19) with a reported concentration of 8.5 mg/kg is the main contributor to arsenic cancer risk. **Attachment F1, Tables 1** through **5,** in **Appendix H** provides a detailed presentation of these calculations.

Seventy eight percent of the resulting CR of 3×10^{-4} (exposure to soil between 0 and 3 feet bgs) is attributed to benzo(a)pyrene. Upon review of the data, 12 sampling locations within the filled ravine area with reported concentrations ranging from 0.19 to 220 mg/kg (at depths greater than 1 foot bgs) are the main contributors to cancer risk. **Attachment F2, Tables 6** through **10** in **Appendix H** provide a detailed presentation of these calculations.

5.2.2 Risk Summary for the Recreational Scenario

The following pathways were considered for the recreational scenarios:

- Recreational adults, adolescent, and children exposed to surface soil
- Recreational adult and adolescent swimmers exposed to surface water
- Recreational adult and adolescent waders exposed to sediment and surface water

In general, risks associated with COPC exposures to surface soils by recreational users were estimated to be with CRs ranging between 1×10^{-5} and 1×10^{-6}, and HIs ranging between 0.002 and 0.04. Risk associated with swimmer and wader exposures to COPCs in sediments were estimated to be with CRs between $1x10^{-5}$ and $3x10^{-9}$, and His between 0.002 and 0.00002. For adult swimmer and wader exposure to oily materials in surface water, CR was $9x10^{-2}$ and $5x10^{-2}$, and HI was 6 and 4, respectively. Risks associated with each medium and recreational receptor are discussed below.

5.2.2.1 Risk Summary for Recreational Users Exposed to Surface Soil

Only limited metals and carcinogenic PAHs were identified as COPCs for recreational user exposure to surface soil. Cancer and noncancer risks to recreational adults and adolescents exposed to surface soil are generally a CR between 1×10^{-6} and 1×10^{-4} and less than an HI of 1. Risks to a recreational child exposed to surface soil are estimated to be a CR of 1×10^{-4}, and an HI of less than 1. The primary risk driver for the recreational adult, adolescent and child is benzo(a)pyrene.

A summary of the risks to the recreational adult, adolescent, and child are provided in **Tables 21**, **22**, and **23**. A detailed presentation of the risk calculations for the recreational adult, adolescent, and child are provided in **Attachment D**, **Tables 4** to **12**.

Recreational Adults

Risks associated with exposure to surface soil for recreational adults are a CR of 4×10^{-6} and an HI of 0.002 for samples collected within Kreher Park. Both the cancer and noncancer risks are within the USEPA target risk range of 10^{-4} to 10^{-6} for cancer and an HI of 1 for noncancer endpoints, respectively. These calculated risks are below the carcinogenic and noncarcinogenic WDNR thresholds (i.e., a CR of 10^{-5} and an HI of 1). Approximately 76% of the resulting CR of 3×10^{-6} is attributed to benzo(a)pyrene. Upon review of the data gathered for benzo(a)pyrene for the Site, four sampling locations (located in Kreher Park, one of which is located within the Former Coal Tar Dump, sample TP-118) with detectable concentrations ranging from 7.4 to 68 mg/kg at intervals between 0 to 1 foot bgs are the main contributors to the benzo(a)pyrene cancer risk.

Detailed calculations of the risks to recreational adults are presented in **Attachment D, Tables 4** to **6**.

Recreational Adolescents

Risks associated with exposure to surface soil for recreational adolescents are a CR of 2×10^{-6} and an HI of 0.003 for samples collected within Kreher Park. Both the cancer and noncancer risk are within the USEPA target CR of 10^{-4} to 10^{-6} for cancer and an HI of 1 for noncancer endpoints, respectively. These calculated risks are below the carcinogenic and noncarcinogenic WDNR thresholds.

Approximately 76% of the resulting cancer risk is attributable to benzo(a)pyrene. Upon review of the data gathered for benzo(a)pyrene for the Site, four sampling locations (located in Kreher Park, one of which is located within the Former Coal Tar Dump, sample TP-118) with detectable concentrations ranging from 7.4 to 68 mg/kg at intervals between 0 to 1 foot bgs are the main contributors to the benzo(a)pyrene cancer risk.

Detailed calculations of the risks to recreational adolescents are presented in **Attachment D, Tables 7** to **9**.

Recreational Children

Risks associated with exposure to surface soil for recreational children are a CR of 1×10^{-5} and an HI of 0.04 for samples collected within Kreher Park. Both the cancer and noncancer risks are within the USEPA target CR range of 10^{-4} to 10^{-6} for cancer and an HI of 1 for noncancer endpoints, respectively. The calculated carcinogenic risk is equal to the carcinogenic WDNR threshold, but less than the noncarcinogenic WDNR threshold. Approximately 74% of the resulting cancer risk is attributed to benzo(a)pyrene. Upon review of the data gathered for benzo(a)pyrene for the Site, four sampling locations (located in Kreher Park, one of which is located within the Former Coal Tar Dump, sample TP-118) with detectable concentrations ranging from 7.4 to 68 mg/kg at intervals between 0 to 1 foot bgs are the main contributors to the benzo(a)pyrene cancer risk.

Detailed calculations of the risks to recreational children are presented in **Attachment D, Tables 10** to **12**.

5.2.2.2 Risk Summary for Recreational Swimmers Exposed to Sediment and Surface Water

Surface water in Chequamegon Bay has a number of issues associated with the existing data set. First, the 2005 surface water data does not confirm the 1998 SEH sampling data which indicates that carcinogenic PAHs are present at concentrations greater than screening levels. Second, oil slicks have been visually observed within Chequamegon Bay. No analytical data is available which measures the levels of chemicals which might be present in oil slick surface water. Therefore, surface water exposures were evaluated using both the 1998 SEH data and analytical data collected from the product stream from the active free product recovery system for the Copper Falls aquifer or chemical-specific solubility values detected in the DNAPL sample. This approach was used to provide a range of risks associated with the 1998 SEH sampling data and the "oil slicks."

Adult and Adolescent Swimmers Exposed to Surface Water

WHFS calculated the risks associated with exposures to the 1998 surface water data. Because no COPCs were identified in the 2005 RI data set, only the 1998 data were used for estimating risks. Detailed calculations using 1998 surface water data and exposure parameters consistent with the Site are presented in **Attachment K, Tables 1** to **6** and are summarized below for the recreational adult and adolescent swimmers.

Receptor	Calculated Risks Using 1998 SEH Surface Water Data	
	Cancer Risk	Noncancer Risk
Adult Swimmer	6×10^{-5}	NE
Adolescent Swimmer	3×10^{-5}	NE

NE – Not evaluated. Only carcinogenic PAHs were present in surface water at concentrations greater than the RBSC.

Adult Swimmers Exposed to Oil Slicks in Surface Water

Risks associated with exposures to oil slicks in surface water were evaluated. This pathway was evaluated because a tar slick was reported and photographed by a citizen. Although, no slicks were observed by sample collectors and the subsequent data do not indicate notable surface water impacts, the 1998 SEH report calculated unacceptable levels of current and future health risks for workers, trespassers, and people engaged in recreational activities on the site. Since this exposure pathway poses one of the greatest potential health risks at the site, the revised HHRA report includes an evaluation of exposures to "oil slicks" in surface water in addition to the evaluation of the 1998 SEH data.

Risks associated with exposures to oil slicks in surface water were estimated for the recreational swimmers using concentrations of DNAPLs collected from the product stream recovered from the active free product recovery system for the Copper Falls aquifer. Risks associated with exposure to oil slicks in surface water are a CR of 9×10^{-2} and an HI of 6. The primary carcinogenic risk drivers are benzo(a)pyrene (62%) and dibenzo(a,h)anthracene (29%). The primary noncarcinogenic risk drivers are 2-methylnaphthalene (54%), naphthalene (12%) and benzene (16%).

Detailed calculations of the risks to adult swimmers exposed to oil slicks are presented in **Attachment I1**, **Tables 1** to **6**. **Attachment I2** provides detailed calculations using the chemical-specific solubility values.

Adult Swimmers Exposed to Sediment

Risks associated with exposure to sediment for adult swimmers are a CR of 4×10^{-5} and an HI of 0.05 for samples collected within Chequamegon Bay. Both the cancer and noncancer risk are within the USEPA target risk range of 10^{-4} to 10^{-6} for cancer and an HI of 1 for noncancer

endpoints, respectively. However, the cancer risk is greater than the WDNR target risk of 1×10^{-5}.

Detailed calculations of the risks to adult swimmers are presented in **Attachment D, Tables 13** and **14**.

Adolescent Swimmers Exposed to Oil Slicks in Surface Water

Risks associated with exposures to oil slicks in surface water were evaluated. This pathway was evaluated because a tar slick was reported and photographed by a citizen. Although, no slicks were observed by sample collectors and the subsequent data does not indicate notable surface water impacts, the s SEH report calculated unacceptable levels of current and future health risks for workers, trespassers, and people engaged in recreational activities on the site. Since this exposure pathway poses one of the greatest potential health risks at the site, the revised HHRA report includes an evaluation of exposures to "oil slicks" in surface water in addition to the evaluation of the 1998 SEH data.

Risks associated with exposures to oil slicks in surface water were estimated for the recreational swimmers using concentrations of DNAPLs collected from the product stream recovered from the active free product recovery system for the Copper Falls aquifer. Risks associated with exposure to oil slicks in surface water are a CR of 9×10^{-2} and an HI of 6. The primary carcinogenic risk drivers are benzo(a)pyrene (62%) and dibenzo(a,h)anthracene (29%). The primary noncarcinogenic risk drivers are 2-methylnaphthalene (54%), naphthalene (12%) and benzene (16%).

Detailed calculations of the risks to adolescent swimmers exposed to oil slicks are presented in **Attachment I1, Tables 7** to **12. Attachment I2** provides detailed calculations using the chemical-specific solubility values.

Adolescent Swimmers Exposed to Sediment

Risks associated with exposure to sediment for adolescent swimmers are a CR of 2×10^{-5} and an HI of 0.05 for samples collected within Chequamegon Bay. Both the cancer and noncancer risk are within the USEPA target risk range of 10^{-4} to 10^{-6} for cancer and an HI of 1 for noncancer endpoints, respectively. However, the cancer risk is greater than the WDNR target risk of 1×10^{-5}.

Detailed calculations of the risks to adolescent swimmers are presented in **Attachment D, Tables 15** and **16**.

5.2.2.3 Risk Summary for Recreational Waders Exposed to Sediment and Surface Water

Surface water in Chequamegon Bay has a number of issues associated with the existing data set. First, the 2005 surface water data does not confirm the 1998 SEH sampling data which indicates that carcinogenic PAHs are present at concentrations greater than screening levels. Second, oil slicks have been visually observed within Chequamegon Bay. No analytical data is available which measures the levels of chemicals which might be present in oil slick surface water. Therefore, surface water exposures were evaluated using both the 1998 SEH data and analytical data collected from the product stream from the active free product recovery system for the Copper Falls aquifer or chemical-specific solubility values detected in the dense non-aqueous phase liquid (DNAPL) sample. This approach was used to provide a range of risks associated with the 1998 SEH sampling data and the "oil slicks."

Adult and Adolescent Waders Exposed to Surface Water

WDNR calculated the risks associated with exposures to the 1998 surface water data. Because no COPCs were identified in the 2005 RI data set, only the 1998 data were used for estimating risks. Detailed calculations using 1998 surface water data and exposure parameters consistent with the Site are presented in **Attachment K, Tables 1** to **6** and are summarized below for the recreational adult and adolescent waders.

Receptor	Calculated Risks Using 1998 SEH Surface Water Data	
	Cancer Risk	Noncancer Risk
Adult Wader	4×10^{-5}	NE
Adolescent Wader	2×10^{-5}	NE

NE – Not evaluated. Only carcinogenic PAHs were present in surface water at concentrations greater than the RBSC.

Adult Waders Exposed to Oily Slicks in Surface Water

Risks associated with exposures to oil slicks in surface water were estimated for the adult waders using concentrations of DNAPLs collected from the product stream recovered from the active

free product recovery system for the Copper Falls aquifer. Risks associated with exposure to oil slicks in surface water are a CR of 5×10^{-2} and an HI of 4. The primary carcinogenic risk drivers are benzo(a)pyrene (62%) and dibenzo(a,h)anthracene (29%). The primary noncarcinogenic risk drivers are 2-methylnaphthalene (54%), naphthalene (12%) and benzene (16%).

Detailed calculations of the risks to adult swimmers exposed to oil slicks are presented in **Attachment I1, Tables 13** to **18**. **Attachment I2** provides detailed calculations using the chemical-specific solubility values.

Adult Waders Exposed to Sediment

Risks associated with exposure to sediment for adult waders are a CR of 4×10^{-5} and an HI of 0.05 for samples collected within Chequamegon Bay. The cancer risk is within the USEPA target risk range of 10^{-4} to 10^{-6} for cancer and noncancer risk is less than the target HI of 1 for noncancer endpoints. However, the cancer risk is greater than the WDNR target risk of 1×10^{-5}.

Approximately 82% of the resulting cancer risk is attributable to benzo(a)pyrene. Upon review of the data gathered for benzo(a)pyrene for the site, three sampling locations (2200N-1600E, 2250N-1400E, 2400N-1200E) with detectable concentrations ranging from 10.5 to 26 mg/kg at intervals between 0 to 2 feet bgs are the main contributors to the benzo(a)pyrene cancer risk. Detailed calculations of the risks to adult waders are presented in **Attachment D, Tables 17** and **18**.

Adolescent Waders Exposed to Oil Slicks in Surface Water

Risks associated with exposures to oil slicks in surface water were estimated for the adult waders using concentrations of DNAPLs collected from the product stream recovered from the active free product recovery system for the Copper Falls aquifer. Risks associated with exposure to oil slicks in surface water are a CR of 2×10^{-2} and an HI of 4. The primary carcinogenic risk drivers are benzo(a)pyrene (62%) and dibenzo(a,h)anthracene (29%). The primary noncarcinogenic risk drivers are 2-methylnaphthalene (54%), naphthalene (12%) and benzene (16%).

Detailed calculations of the risks to adult swimmers exposed to oil slicks are presented in **Attachment I1, Tables 19** to **24**. **Attachment I2** provides detailed calculations using the chemical-specific solubility values.

Adolescent Waders Exposed to Sediment

Risks associated with exposure to sediment for adolescent waders are a CR of 2×10^{-5} and an HI of 0.05 for samples collected within Chequamegon Bay. The cancer risk is within the USEPA target risk range of 10^{-4} to 10^{-6} for cancer and an HI of 1 for noncancer endpoints. However, the cancer risk is greater than the WDNR target risk of 1×10^{-5}.

Approximately 82% of the resulting cancer risk is attributable to benzo(a)pyrene. Upon review of the data gathered for benzo(a)pyrene for the Site, three sampling locations (2200N-1600E, 2250N-1400E, 2400N-1200E) with detectable concentrations ranging from 10.5 to 26 mg/kg at intervals between 0 to 2 feet bgs are the main contributors to the benzo(a)pyrene cancer risk.

Detailed calculations of the risks to adolescent waders are presented in **Attachment D, Tables 19** and **20**.

5.2.3 Risk Summary for the Construction Worker Scenario

Soil Exposures

PAHs appear to be the primary cancer risk drivers for the construction scenario within the Kreher Park area of the Site. Of the calculated CR of 1×10^{-4}, approximately 71% is attributable to benzo(a)pyrene and 11% is attributable to dibenzo(a,h)anthracene. Upon review of the data, 27 sampling locations (located in both the filled ravine and Kreher Park) with detectable concentrations ranging from 205 to 3,000 mg/kg at intervals between 1 to 8 feet bgs are the main contributors to the benzo(a)pyrene cancer risk. In addition, 24 sample locations for dibenzo(a,h)anthracene (located in Kreher Park) with an detectable concentrations ranging from 28 to 250 mg/kg (2 to 8 feet bgs) are the main contributors to the dibenzo(a,h)anthracene cancer risk. Detailed calculations of the construction scenario cancer risks are provided in **Attachment D, Tables 21** to **23**.

The resulting HI of 38 is primarily attributed to naphthalene (with an HI of 31) and 2-methylnaphthalene (with an HI of 1). Because the HI exceeds 1, the noncancer risk for this receptor was re-calculated based on target organs affected by each chemical. **Table 31** shows that target organ-specific HI is greater than 1 for respiratory and systemic target organ effects. Detailed calculation of the construction scenario noncancer risks are provided in **Attachment D, Tables 21** to **23**.

Based on the results of the ALM, the percentage of developing fetuses predicted to have a blood lead concentration greater than 10 µg/dL is 1.5, which is within USEPA's target criteria of no more than 5% of fetuses of adult female workers above the concern threshold of 10 µg/dL. The results of the ALM are presented in **Attachment D, Table 3f.**

Based on the results of the ALM inputting an average lead concentration of 88.7 mg/kg, the percentage of developing fetuses predicted to have a blood lead concentration greater than 10 µg/dL is 1.5, which is within USEPA's target criteria of no more than 5% of fetuses of adult female workers above the concern threshold of 10 µg/dL. The results of the ALM are presented in Attachment D, Table 3f. While one location (GP-110 (1-3')) had a highly elevated lead concentration of 4000 mg/kg, only one other sample (GP-115 (1-3') had a concentration (480 mg/kg) that exceeded the screening level of 400 mg/kg. Thus, while there are elevated concentrations are in the loading dock area of the NSPW, the average concentration is below the screening level.

For this HHRA, it was assumed that the construction receptors would be exposed to both surface and subsurface soil. This assumption was made based on the definition of the construction scenario (USEPA, 2002a), which would involve the construction of residential or commercial structures at the Site. This represents the worst case scenario and is not likely to occur at the Site given both its current and future land use. Kreher Park is an established park and is expected to remain in the future. Any expansion to the recreational areas of the Site would likely be associated with activities such as the installation of landscaping, sidewalks, and parking lots all of which do not involve excavation to significant depths (USEPA, 2002a). Therefore, construction receptors would most likely be exposed to shallow soils.

A hot spot analysis was performed for the construction scenario using data collected from the following locations near the Former Coal Tar Dump. The results of this analysis are presented in **Section 6.6**.

Location	Sample ID	Depth (feet)
TP-4	1040	4-6
TP-4	933	4-6
TP112	NS-GWTP112-0605	4.5-5
TP112	NS-SOTP112-0-1-061405	0-1
TP112	NS-SOTP112-5	4.5-5
TP112	NS-SOTP112.5-AD	4.5-5
TP113	NS-SOTP113-0-1-061405	0-1
TP113	NS-SOTP113-4	3.5-4
TP115	NS-SOTP115-0-1-061305	0-1
TP115	NS-SOTP115-4	3.5-4
TP115	NS-SOTP115-4-AD	3.5-4
TP116	NS-SOTP116-0-1-061305	0-1
TP116	NS-SOTP116-3	2.5-3
TP118	NS-GWTP118-0605	3.5-4
TP118	NS-SOTP118-3	0-1
TP118	NS-SOTP118-3	3.5-4
TP119	NS-SOTP119-0-1-061305	0-1
TP119	NS-SOTP119-5	4.5-5

Groundwater Exposures

Cancer and noncancer risks associated with the exposure to "oily materials" in groundwater are 7×10^{-3} and 60, respectively. Benzo(a)pyrene (64 percent) and dibenzo(a,h)anthracene (27%) are the primary carcinogenic risk drivers. The primary noncarcinogenic risk drivers are 2-methylnaphthalene (54%), naphthalene (12%), and benzene (16%).

Detailed calculations for this receptor are provided in **Attachment I1**, **Tables 25** to **31**. **Attachment I2** provides detailed calculations using the chemical-specific solubility values.

Trench Air

Cancer and noncancer risks associated with exposure to VOCs in trench air are presented below.

Domain	Trench Air	
	CR	HI
Kreher Park	8.34×10^{-3}	17152
Upper Bluff	2.14×10^{-5}	228
Filled Ravine	3.29×10^{-2}	646601

The primary cancer risk drivers at Kreher Park are benzene (77%) and benzo(a)pyrene (23%). The primary risk driver at the Upper Bluff is benzene (100%). The primary risk driver at the Filled Ravine are benzene (47%) and benzo(a)pyrene (53%).

Detailed calculations for this receptor are provided in **Attachment J, Tables 1** to **2(A-C)**.

5.2.4 Risk Summary for the General Industrial Worker

For the industrial worker, samples collected within a 0-2 foot depth interval should be included in the 0-1 ft dataset, as the average sample depth was 1 foot (i.e., , GP-137, GP-131, GP-120). An conservative evaluation of the risks was performed using the average concentration of benzo(a)pyrene at these locations as the EPC since the concentrations of these samples were greater than maximum detected concentration within the industrial worker dataset. Risks from ingestion and dermal contact exposure were calculated. . Cancer and noncancer risks associated Cancer and noncancer risks associated with the exposure to surface soil for the general industrial worker receptor are a CR of 1×10^{-6} and an HI of 0.007. Cancer and noncancer risks associated with exposure to indoor air are a CR of 8×10^{-5} and an HI of 3, respectively. The primary cancer risk drivers are trichloroethylene (44%) and benzene (3%). The resulting HI of 3 is primarily attributed to 1,2,4-trimethylbenzene with an HI of 2.

The results of these evaluations are summarized in **Tables 28** and **29**. Detailed calculations for this receptor are provided in **Attachment D, Tables 24 - 27**.

5.2.5 Risk Summary for the Maintenance Worker

Cancer and noncancer risks associated with the exposure to surface soil for the maintenance worker receptor are a CR of 1×10^{-6} and an HI of 0.001. Risks for this receptor are within the target risk levels. The results of this evaluation are summarized in **Attachment D, Tables 28 – 30**.

Based on the results of the ALM, the percentage of developing fetuses predicted to have a blood lead concentration greater than 10 μg/dL is 1.6, which is within USEPA's target criteria of no more than 5% of fetuses of adult female workers above the concern threshold of 10 μg/dL. A detailed presentation of the ALM for the maintenance worker is provided in **Attachment D, Table 30f**.

5.2.6 Risk Summary for the Subsistence Fisherman

Risks associated with the ingestion of locally-caught fish from Chequamegon Bay is a CR of 1×10^{-4}, which is just within the USEPA target cancer risk range of 10^{-4} to 10^{-6} for cancer

endpoints, but greater than the WDNR threshold of 10^{-5}. Although the primary risk drivers for this scenario are the carcinogenic PAHs (benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[e]pyrene, dibenzo[a,h]anthracene, and indeno[1,2,3-cd]pyrene), individual cancer risks for each detected carcinogenic PAH is between 1×10^{-5} and 1×10^{-6}. The results of this evaluation are summarized in **Table 32**. Detailed calculations for this receptor are provided in **Attachment D, Tables 31a** and **31b**.

5.3 CENTRAL TENDENCY EVALUATION

Quantitative measures of uncertainty involve the calculation of CTE risk estimates. The CTE calculation involves the use of 50th percentile input parameters in carcinogenic and noncarcinogenic risk estimates as opposed to upper-bound values for parameters used in the RME calculations. The 50th percentile parameters are considered representative of the general receptor population. The chemicals driving the RME risk were evaluated using these average exposure assumptions and the arithmetic mean concentration to derive risk for the CTE scenario rather than the upper-bound and 95% UCL concentrations used for the RME scenario. The CTE scenario was only calculated for pathways in which RME risks exceed the target risk goals (i.e., USEPA carcinogenic risks greater than 10^{-4} and an HI greater than1 and WDNR carcinogenic risk of 10^{-5} and an HI greater than 1).

The results of this evaluation are summarized below. Detailed CTE calculations are provided in **Attachment E, Tables 1** through **6** for residential receptors, **Tables 7** through **9** for construction workers, **Table 10** for the industrial worker and **Table 11** for the subsistence fisherman.

Receptor	Table	Soil	
		CR	HI
Resident (0-10 foot soil depth)	35	2×10^{-4}	8
Resident (0-3 foot soil depth)	36	5×10^{-5}	0.3
Resident (0-1 foot soil depth)	37	5×10^{-6}	0.1
Construction Worker	38	2×10^{-5}	26
Industrial Worker (indoor air)	39	2×10^{-5}	1
Subsistence Fisherman	40	3×10^{-6}	0.0003

5.3.1 *Residents (0-10 foot soil depth)*

Approximately 70% of the resulting CR of 1×10^{-4} for residents exposed to soil between 0 and 10 feet is attributable to benzo(a)pyrene. Upon review of the data , 12 sampling locations (located in both the filled ravine and the Upper Bluff) with detectable concentrations ranging from 16 to 340 mg/kg at intervals between 1 to 8 feet bgs are the main contributors to the benzo(a)pyrene cancer risk. The resulting HI of 5 is primarily attributed to naphthalene (with an HI of 3).

5.3.2 Residents (0-3 foot soil depth)

The resulting cancer risk of 5×10^{-5} for residents exposed to 0 to 3 feet of soils is primarily attributed to benzo(a)pyrene (71%). Upon review of the data, three sampling locations (GP-110, GP-113, and GP-115) with detectable concentrations ranging from 7.8 to 220 mg/kg at intervals between 1 to 3 feet bgs are the main contributors to the benzo(a)pyrene cancer risk. The resulting HI of 0.3 is below the target criterion for the HI of 1.

5.3.3 Residents (0-1 foot soil depth)

The resulting cancer risk of 5×10^{-6} for residents exposed to 0 to 1 feet of soil is primarily attributed to arsenic (79%). Upon review of the data, three sampling locations (SS19, SS15, and SS18) with detectable concentrations ranging from 3.5 to 8.5 mg/kg at in surface soil are the main contributors to the arsenic cancer risk. The resulting HI of 0.1 is below the target criterion for the HI of 1.

5.3.4 Construction Worker

The resulting CR of 2×10^{-5} is primarily attributed to benzo(a)pyrene and dibenz(a,h)anthracene. Approximately 82% of the resulting cancer risk is attributable to benzo(a)pyrene (71%) and to dibenzo(a,h)anthracene (11%). Upon review of the data, 30 sampling locations (located in the filled ravine, the Upper Bluff, and Kreher Park) with detectable concentrations ranging from 130 to 3,000 mg/kg at intervals between 1 to 8 feet bgs are the main contributors to the benzo(a)pyrene cancer risk. In addition, 23 sample locations for dibenzo(a,h)anthracene (located in Kreher Park) with an detectable concentrations ranging from of 28 to 250 mg/kg (2 to 8 feet bgs) are the main contributors to the dibenzo(a,h)anthracene cancer risk. The resulting HI of 26 is primarily attributed to naphthalene with an HI of 23.

5.3.4. Industrial Worker

Cancer and noncancer risks associated with exposure to indoor air for industrial workers are a CR of 2×10^{-5} and an HI of 1, respectively. Both the cancer and noncancer risks are within USEPA target levels of 10^{-4} to 10^{-6} for cancer risk and an HI of 1, but greater than the WDNR cancer threshold of 10^{-5}.

Attachment F2, **Table 10a** and **10b** provide detailed calculations for cancer and noncancer risks. **Table 39** summarizes the CTE for this receptor.

5.3.5 Subsistence Fisherman

Cancer and noncancer risks associated with ingestion of locally-caught fish by a subsistence fisher are a CR of 3×10^{-6} and an HI of 0.0003, respectively. Both the cancer and noncancer risks are within USEPA's target risk levels of 10^{-4} to 10^{-6} for cancer risk and an HI of 1 and less than the WDNR cancer threshold of 10^{-5} and noncancer threshold of 1. The primary risks driver is benzo(a)pyrene with a cancer risk of 2×10^{-6}.

Attachment F2, **Tables 11a** and **11b** provide detailed calculations for cancer and noncancer risks. **Table 40** summarizes the risks estimated for this receptor.

6.1 OVERVIEW

In any HHRA, estimates of potential carcinogenic risk and noncarcinogenic health effects have numerous associated uncertainties. The primary areas of uncertainty and limitations are qualitatively discussed. Areas of uncertainty that are discussed in the RI report include, but are not limited, the following:

- Data collection and evaluation;
- Assumptions regarding exposure scenarios;
- Applicability and assumptions of models selected to predict the fate and transport of COPCs in the environment; and
- Parameter values for estimating intake of COPCs.

Each type of uncertainty is discussed in the sections that follow.

6.2 DATA COLLECTION AND EVALUATION

6.2.1 Residential Scenario Evaluation

For this risk assessment it was assumed that the residential receptors would be exposed to both surface and subsurface soil. This assumption was made because new construction would involve excavation of soil for the construction of basements. Therefore, soil with high chemical concentrations would be brought to the surface resulting in a potential exposure pathway for residential receptors. This scenario represents the worst case for residential receptors, but is not likely to be the actual scenario associated with the Site. The residential neighborhoods adjacent to the Site are established neighborhoods and are expected to remain so in the future. According to the Ashland Wisconsin Waterfront Development Plan, the future use of the Kreher Park portion of the Site does not include a residential scenario. Therefore, residential receptors would only be exposed to surface soil. If it is assumed that residential receptors adjacent to the Site tend gardens, then it is possible that the first three feet of soil will represent the most likely exposure point.

Re-evaluating the residential receptor using EPCs derived based on the exposure to surface soil or soil to a depth to three feet indicates that carcinogenic and noncarcinogenic risks are as presented below.

Receptor	RME Table	RME		CTE Table	CTE	
		CR	HI		CR	HI
Resident (0 – 10 feet soil depth)	20	5×10^{-4}	15	35	1×10^{-4}	5
Resident (0-1 foot soil depth)	33	1×10^{-5}	0.2	36	5×10^{-6}	0.1
Resident (0 – 3 feet soil depth)	34	3×10^{-4}	0.9	37	5×10^{-5}	0.3

An examination of the analytical data used to derive the carcinogenic and noncarcinogenic risks to residents exposed to surface and subsurface soil to a depth of 3 feet shows that the risks are highest in samples collected between 1 and 3 feet bgs for the samples collected in the courtyard area of the former MGP. Locations GP110 and GP115 had the highest detections of all chemicals identified as COPCs at the 1 to 3 feet depth. An examination of the risks associated with sample location SS-24, which is located between the residence on Lakeshore Drive and the former MGP, shows that both carcinogenic and noncarcinogenic risks are 7×10^{-6} and 0.1, respectively.

Based on this re-evaluation of the data, the risks associated with the residential receptor are most likely overestimated based on the assumptions used to obtain the dataset used to evaluate risk. Based on the current configuration of residential areas adjacent to the Site and the future land use presented in the Ashland Wisconsin Waterfront Development Plan, risks to residential receptors would only be associated with surface soil exposures (i.e., 0-1 foot). Surface soil carcinogenic and noncarcinogenic risks are estimated to be within both the USEPA and the WDNR target risk range.

6.2.2 Indoor Air Evaluation

NSPW Former MGP Facility

Based on the data collected, the indoor air concentrations were as much as an order of magnitude higher than the air concentrations detected in ambient air or soil gas samples. This suggests that vapor intrusion may not be primary source of VOCs detected in the indoor samples. However, because of the nature of the chemicals detected in indoor air samples, ambient air, and soil gas samples, the chemicals detected are somewhat dissimilar **(Table 19)**. The chemicals detected in indoor air samples include chemicals that may be associated with solvents rather than chemicals that have been associated with historic activities at the site. There is the possibility that there may be other sources of VOCs (e.g., benzene) within the former MGP facility buildings that may have contributed to the types of chemicals detected in indoor samples. As a conservative measure, all chemicals detected in the indoor air samples were included in the quantitative

evaluation and the results of the evaluation suggest that risks to residents are within acceptable USEPA limits.

An HI of 3 was calculated for the worker exposure to indoor air pathway under the RME scenario. This risk level is likely to be an over-estimate because:

- It was estimated using the maximum detected concentrations as the concentrations at points of exposure.
- It was calculated based on the exposure parameters for the industrial/commercial workers (i.e., an individual works at the Site for 8 hours per day, 5 days per week, 50 weeks per year for a total of 25 years). The NSPW Service Center where the indoor air samples were collected, is used as a warehouse; there is an office space inside the building, but used only on a part-time basis.

Former WWTP

Although the approved RI Work Plan (URS, 2005) described that the trespasser scenario to the existing WWTP would be evaluated, a quantitative evaluation for the potential trespasser exposures to indoor air and groundwater seepage inside the WWTP was not performed because no water or indoor air samples were collected from the building during the RI sampling program because of access restrictions. In 2002, a consultant for the City of Ashland inspected the inside of the WWTP building and collected a single round of indoor air samples to address potential inhalation exposure to City of Ashland workers. Samples were only analyzed for limited chemicals (selected PAHs, trimethylbenzene and acetic acid). The results of this sampling suggested that Site-related compounds are probably in the indoor air of the former WWTP building, and a thorough indoor air investigation was recommended before final re-use decisions (WDHFS, 2003). Therefore the risks associated with this exposure pathway are unknown.

6.2.3 Surface Water Evaluation

All surface water data (1998 SEH and 2005 RI) were used to estimate risks to recreational receptors. However, a review of the 1998 SEH data and the 2005 (high-energy and low-energy) data indicates that the 2005 (both high- and low-energy data) did not confirm the presence of site-related chemicals in surface water at concentrations greater than the RBSCs. Because the more recent data collected during the RI do not confirm the 1998 surface water data collected as

part of the SEH HHRA, the risks associated with the use of these data likely overestimates potential risks associated with this pathway.

6.3 EXPOSURE ASSESSMENT

6.3.1 Exposure Scenario Assumptions

The assumptions used to identify the exposure scenarios evaluated in the HHRA were based on USEPA guidance, Site history, current land use, and limited information concerning future use of the Site. It is assumed that the primary exposure scenario is recreational for Kreher Park. Based on this land use, other scenarios (maintenance and construction) and pathways were developed. If the City of Ashland changes its decision to expand the recreational areas in the future, the HHRA may need to be revisited to determine the risks associated with the future land use.

6.3.2 Fate and Transport Assumptions

6.3.2.1 Volatilization Factors

Site-specific values needed for calculating volatilization factors (VFs) were unavailable. Therefore, chemical and physical parameters were selected from default values recommended in known literature sources based on the predominant soil type of silty clay. Using this approach to calculate Site-specific VFs may potentially result in an over- or under-estimate of risks if the actual Site-specific chemical and physical parameters are significantly different from default values selected based on the silty clay soil type.

6.3.2.2 Particulate Emission Factors

For the general industrial worker and residential scenarios, it was assumed that the inhalation of fugitive dusts generated by wind erosion was of concern. To estimate risks to this pathway, a particulate emission factor (PEF) is needed to relate the chemical concentration in soil to the concentration of dust particles in the air. For this HHRA, Site-specific values for the wind erosion dispersion factor and non-erodible surface cover were used for the residential and commercial/industrial scenarios. Because the non-erodible surface cover is based on current conditions, the risks estimated may not be representative of conditions with greater or lesser surface cover after the Site is developed for re-use.

For the construction scenario, the PEF was estimated using a combination of default and Site-specific information. USEPA's Supplemental SSL Guidance (USEPA, 2002a) was followed to estimate a PEF for both fugitive dusts associated with vehicular traffic on unpaved roads and for any other construction related activities (e.g., grading, dozing, tilling, wind erosion). Although it is assumed that future construction work will be limited to expansion of the Site as a recreational area, currently there are no plans in place for this work. Therefore, little Site-specific information exists concerning the actual construction activities that may occur. As such, a representative PEF for the Site could not be calculated and the actual PEF could be greater than or less than the estimated value.

Attachment G, Tables 1 through **14** present the PEF calculations for the commercial/industrial, residential, and construction scenarios. **Attachment G** also provides a detailed presentation of the default and limited Site-specific values used for the derivation of PEF values.

6.3.3 Extrapolation of Vapor Concentrations from Surface Water

There is no methodology available for quantifying concentrations of vapor from surface water available in USEPA guidance. Therefore, risks to recreational receptors exposed to VOCs in surface water were not evaluated quantitatively.

Potential risks associated with exposures to oily slicks in surface water were quantified for the adult swimmer and wader exposure to COPCs (using DNAPL data due to the lack of oily slick data) via the incidental ingestion and dermal contact pathways. Although risks via inhalation of chemicals volatilizing from surface water were not quantified for these exposure scenarios, risks posed by the inhalation pathway is expected to be significantly lower than those associated with the ingestion and dermal pathways.

6.3.4 Receptor Exposure Parameter Values

Although there are future plans for expanding the recreational areas, specific information regarding construction and excavation activities that might occur is unknown. Therefore, risks to construction worker receptors based on the assumptions used in this HHRA may over- or under-estimate risks to this receptor population.

Additionally, little information is available concerning the maintenance work that is completed at the Site currently and none is available for future maintenance activities. The assumptions regarding the exposure frequency for maintenance workers is a based on seasonal weather patterns. The actual risks to this receptor are unknown but the estimates presented in this HHRA are based on conservative assumptions.

6.3.5 Exposure Point Concentrations

6.3.5.1 Indoor Air

In general, EPCs used in the RME were based on statistically-derived concentrations calculated using USEPA's ProUCL software with two notable exceptions. For indoor air, two samples were collected for the purpose of evaluating risk to potential receptors. Because a UCL could not be calculated with only two samples, the maximum concentration was used as the EPC. Use of the maximum detected concentration may potentially overestimate risk associated with exposure to indoor air. However, the true risk is unknown.

6.3.5.2 Oily Material and Oil Slicks

Information regarding chemical-specific concentrations in oily water is unavailable because oily water (groundwater or surface water containing slicks) was not sampled during previous investigations. To complete a quantitative evaluation of health risks potentially posed by oily material, concentration terms used in this evaluation included the analytical data of the DNAPL and chemical-specific solubility values. The use of these concentrations may result in an overestimate of risks calculated for the oily material.

Oil sheens are typically the lighter fraction of Site hydrocarbons, i.e., short chain alkenes, VOCs, and perhaps sole low molecular weight polycyclic aromatic hydrocarbons (PAHs). Since high molecular weight PAHs are too insoluble and/or are crystalline in nature they are probably not part of the sheens observed. While sheens are visually obvious, the concentrations of the hydrocarbons in sheen are not necessarily high.

Appearance of Oil on Water	Estimated Hydrocarbon Concentration (mg/L)
Barely visible	0.05
Silver sheen	0.1
First trace of color	0.2
Bright bands of color, iridescent	0.4
Colours tending to be dull	1.2
Colours fairly dark, rainbow tints	2.4
Brown or black	12
Brown / dark brown	120

As indicated by the above table, concentration terms used in this evaluation (DNAPL data or chemical-specific solubility) are significantly higher than estimated levels of total hydrocarbon concentrations in the 0.2 to 2.4 mg/L range, based on colors of sheens observed (Doerffer, 1992).. Therefore, estimated risk levels for potential exposures to oily materials in groundwater or slicks in surface water represent conservative overestimates and should not be used as the basis for deriving remedial action objectives.

6.3.5.3 Trench Air

Information regarding chemical-specific concentrations in trench air at Kreher Park, the Upper Bluff and the Filled Ravine is unavailable because air samples were not collected during was not sampled during previous investigations. To complete a quantitative evaluation of health risks potentially posed by chemicals volatilizing from groundwater into trench air, the maximum detected groundwater concentration was used to model a trench air concentration using models presented as part of the Virginia Department of Environmental Quality risk assessment guidance (VADEQ, 2006). The use of the maximum detected concentration within each exposure area potentially overestimates risks since groundwater concentrations are not likely to remain static and are subject to mixing within each zone.

6.3.6 Evaluation of Concentrations Exceeding C_{sat}

A separate evaluation was performed by characterizing risks using EPCs that were derived by excluding chemical concentrations in soil that exceeded the chemical-specific C_{sat}. This evaluation was prepared in response to review comments on the draft HHRA report.

For the purpose of this evaluation, C_{sat} values were calculated for chemicals that are in liquid form at the ambient soil temperature (55 degrees Fahrenheit). Chemical concentrations were

compared to the C_{sat} values and EPCs were derived by excluding concentrations that exceeded $C_{sa}t$ values. Cumulative risks calculated using these EPCs are presented on **Tables 41** through **45**. Presented below is a comparison of the results of this evaluation to the risk evaluation using the entire soil dataset.

Scenario	EPCs Derived Based on the Entire Data Set		EPCs Derived by Excluding Concentrations > Csat	
	CR	HI	CR	HI
Residents (0-10 feet)/RME	5×10^{-4}	15	5×10^{-4}	14
Construction Worker (0-10 feet)/RME	1×10^{-4}	38	1×10^{-4}	33
Residents (0-10 feet)/CTE	2×10^{-4}	8	1×10^{-4}	4
Construction Worker (0-10 feet)/CTE	3×10^{-5}	13	2×10^{-5}	9
Residents (0-3 feet)/RME	5×10^{-5}	0.3	3×10^{-4}	0.9

As indicated by this comparison, similar risk levels were calculated using EPCs derived based on all soil data in the relevant data sets or data that excluded concentrations exceeding C_{sat}.

6.3.7 Lack of Established Methodology

The methodology that was developed by the USEPA for quantifying dermal absorption of chemicals in aqueous media and presented in RAGS Part E was used in calculating risks following exposure to "oily material" in groundwater because of a lack of equations and mathematical models developed specifically for oily materials. The use of this approach is likely to introduce uncertainties into the estimated risk values.

6.4 TOXICITY ASSESSMENT

6.4.1 Use of Unverified Toxicity Values

There were several chemicals (as presented in **Attachment A, Tables 11a and 11b**) detected at this site for which there are only provisional toxicity values. The USEPA process for developing provisional toxicity values is inherently conservative and is not subject to the same vigorous review process as toxicity criteria that have been verified. For this HHRA, 2-methylnaphthalene is a risk driver based on its provisional toxicity value. Because the toxicity values are based on

limited animal and human data, the true risks associated with these chemicals is not completely known.

6.4.2 Lack of Toxicity Values for Detected Chemicals

There were several chemicals (1-methylnaphthalene, acenaphthylene, benzo[e]pyrene, benzo[g,h,i]perylene, phenanthrene, 1,2,3-trimethylbenzene, p-isopropyltoluene) that were detected at the Site and for which there are no toxicity values. Because of the lack of information available for these chemicals, the true risk to potential receptors at the Site is unknown. However, because these chemicals were detected in areas where primary risk drivers are located, it is likely that if any remediation based on known risk drivers will address chemicals for which there is a lack of toxicity data.

6.5 COMPARISON TO 1998 SEH BASELINE HHRA

In 1998, SEH completed a baseline HHRA for the Site and adjacent near-shore sediments for the WDNR to evaluate the potential existing and future adverse health effects caused by hazardous substance releases from the Site in the absence of any actions to control or mitigate the releases. The current HHRA was completed as part of the requirements for the investigation of a Superfund site. A comparison of the two HHRAs was completed to determine if the collection of additional data during the RI affects the conclusions of the HHRA for the Site. However, it is important to note that the two HHRAs were prepared in accordance with different regulatory framework (NR 700 for the HHRA prepared by SEH and the NCP for the current HHRA), and slightly different receptors, areas of interest, and media were evaluated. Therefore, a point-by-point comparison cannot be completed using the information from the SEH HHRA as it was presented in the 1998 document. Instead, when this occurs specific information, which will allow the end user to determine how the comparisons were made, will be included in the discussion.

6.5.1 Comparison of Media of Interest

The 1998 SEH HHRA identifies groundwater, seep water, surface water, surface soil, subsurface soil, sediment and fish tissue as the media of interest for receptors contacting impacted media at the Site. Since the completion of the 1998 SEH baseline HHRA, two activities have impacted the media of interest for the Site. The results of these activities yielded the following changes to the media of interest for the Site:

- NSPW implemented interim removal actions in 2000 and 2002 to mitigate exposure risks to contaminants and to recover free-product from the deep aquifer. A low-flow pumping system currently extracts free-product from the deep aquifer, treating the entrained groundwater before discharging it to the City of Ashland's sanitary sewer.
- Discharge through the buried pipe in the former filled ravine was the source of the seep at Kreher Park. An extraction well, installed by NSPW at the base of the filled ravine, was part of a larger interim action that included excavation of contaminated materials at the former seep area and placement of a low-permeability cap to eliminate the intermittent seep discharge and mitigate environmental exposure of the associated contaminants.

Therefore, the exposure pathways associated with seep water (ingestion, inhalation and dermal absorption) identified in the 1998 SEH baseline HHRA are no longer complete and were not evaluated for the current HHRA. The media of interest for the current HHRA include groundwater, surface water, sediment, surface soil, subsurface soil and fish. The primary differences between the media evaluated in both reports are associated with the evaluation of the groundwater and surface water.

6.5.1.1 Groundwater

For the SEH HHRA, groundwater was evaluated for receptors exposed to impacted groundwater at the seep area and the utility trench. Data were available to complete a quantitative estimate of risk for groundwater at these locations.

For the current HHRA, "oily materials" in groundwater were evaluated for the construction worker receptor. However, no data, which measures the concentrations of Site-related chemicals, is available to complete a quantitative estimate of risk. In lieu of suitable data, laboratory analytical data of the DNAPL samples collected from the product stream recovered from the active free product recovery system for the Copper Falls aquifer were used. Use of this data is highly conservative and is likely to overestimate risk.

6.5.1.2 Surface Water

For both the SEH HHRA and the current HHRA, measured surface water concentrations were used to evaluate risks to recreational receptors. The 2005 data identifies no COPCs in surface water, while the 1998 data indicates that carcinogenic PAHs are present concentrations greater

than RBSCs. In addition, for the current HHRA, recreational adult swimmers and waders were also evaluated for exposures to "oil slicks" in surface water. Because there are no 2005 data available for this evaluation, chemical-specific solubility values of chemicals detected in the DNAPL sample were used to estimate risk. Use of solubility values is highly conservative and is likely to overestimate actual risk.

6.5.1.3 Fish

The SEH HHRA did not evaluate the ingestion of fish pathway using tissue data. Instead, a fish tissue concentration was modeled based on detections of chemicals in the water column. Because only metals were detected in surface water, no organic chemicals were modeled. The current HHRA uses three fish species to determine risks to subsistence fisherman ingesting fish caught in Chequamegon Bay. The current HHRA indicates that risks to subsistence fishermen based on detections of both organic and inorganic chemicals in fish tissue and more accurately represents risk to subsistence fishermen.

6.5.2 Comparison of Exposure Areas

Both the 1998 SEH and the current HHRA divided the Site into subunits in order to group the data and more accurately assess the contaminants to which various populations may be exposed. However, the 1998 SEH HHRA did not address contamination associated with the former filled ravine, the location where some of the highest concentrations of Site-related chemicals have been observed in soil. According to Section 1.2 of the SEH HHRA, the area of evaluation is the "*Ashland Lakefront Property (Kreher Park) and adjacent offshore sediment in Ashland, Wisconsin.*" Additionally the last paragraph of Section 1.2 of the SEH HHRA states:

The HHRA is limited to the 20 acre area described above and is further limited to considering only the upper shallow groundwater table, site soils (both surface and subsurface), and nearshore lake water and shallow sediments. The baseline HHRA does not include evaluation of contamination located in the ravine up gradient to the Ashland Lakefront Property or contaminants located in the lower Copper Falls aquifer.

Therefore, the 1998 SEH baseline HHRA exposure areas were limited to what is now identified as Kreher Park and the near shore area of Chequamegon Bay. Based on the Figure 2 of the SEH HHRA, this 20 acre parcel of land was divided into 4 subunits for evaluation. These subunits include:

- Kreher Park (Site in General)
- Near Shore Lake Area
- Current Utility Trench
- Seep Area

The SEH HHRA did not include the NSPW Garage, Main Office, Storage Yard or the residential area north of the NSPW Garage
The current HHRA domains include:

- Kreher Park
- Chequamegon Bay
- Filled Ravine
- Upper Bluff

The current HHRA does not specifically address a utility trench area for its worker population; however, it does include this area as part of the overall exposure area for workers. Because there are no definite re-use plans that have been developed for the Site, it was assumed in the current HHRA that worker receptors may potentially be exposed to soil throughout the entire impacted area. Because the future land use is unknown, this approach is more conservative than the approach used in the 1998 SEH HHRA

6.5.3 Comparison of Receptors

In general, each HHRA evaluated similar receptors. Except for the trespassing scenario, which was not evaluated quantitatively, the current HHRA is more comprehensive than the 1998 SEH HHRA as it includes more task-specific receptors.

SEH HHRA receptors identified for quantitative risk assessment in both current and future scenarios include:

- Occupational city workers exposed to soil and groundwater
- Recreational adults, children and adolescents exposed to surface soil, surface water, and fish
- Adolescent trespassers exposed to surface soil and groundwater

Current HHRA receptors identified for quantitative risk assessment in current and future land use scenarios include:

- General industrial workers exposed to indoor air
- Maintenance workers exposed to surface soil
- Construction workers exposed to surface soil, subsurface soil and oily material in groundwater
- Recreational adults, children, and adolescents exposed to surface soil, surface water, sediment and "oil slicks" in surface water
- Subsistence fishermen
- Site residents exposed to surface soil, soil (0-3 feet bgs) and surface and subsurface soil (0-10 feet bgs)

The list generated for the current HHRA is more comprehensive than the SEH HHRA because receptors were based on all possible receptors that could potentially be associated with current and future land uses for the impacted area. Specifically, receptor exposures for indoor, oily material in groundwater and oil slicks in surface water were incorporated in to the current report. These pathways, although highly uncertain, provide another measurement of risk for the Site.

6.5.4 COPCs

In general, the classes of COPCs selected for both the SEH HHRA and the current HHRA are similar for soils. The COPCs selected for both HHRAs is limited primarily to carcinogenic and noncarcinogenic PAHs, VOCs, and limited metals in both surface and sediment. It is important to note that the list of COPCs identified for surface soils at the Site in General (Kreher Park) for the SEH HHRA is much shorter than that for the current HHRA. The current COPC list includes at least nine COPCs (4 metals and 5 PAHs) but the SEH HHRA identifies only three VOCs and two metals (Table 2). It is not likely that the minor changes in the COPCs selected make an significant impact of the risk values calculated for the receptors evaluated.

It appears that although, the SEH HHRA identifies COPCs for surface water, these COPCs are limited to metals (copper, iron, and zinc) (Table A-14 of the 1998 SEH HHRA). Although the current HHRA does not identify any surface water COPCs, surface water was conservatively evaluated for exposure to carcinogenic PAHs in selected surface water samples from the 1998 SEH report (**Table 16**) and Site-related chemicals in "oil slicks." Therefore, the approach used

for the surface water exposures for the current HHRA is overly conservative as it uses analytical data associated with the active free product recovery system for the Copper Falls aquifer.

It appears that for the SEH HHRA, background values were used to eliminate detections of metals in various media. This approach was not used for the current HHRA. It is important to note that in Tables A-2 and A-3 which present the summary of detected chemicals in the background samples for subsurface soil and surface soil, Site-related organic compounds were detected indicating that the sample locations selected for background may be impacted. Both approaches may potentially underestimate actual site risks in that metals impacting risk are ignored as background when they might be associated with Site-related conditions.

6.5.5 Toxicity Assessment

Since the time the SEH HHRA was completed, toxicity values have either changed or chemicals were added to the database of toxicity values presented as part of USEPA's IRIS. In addition, provisional values from the Superfund Technical Information Center were also made available for use. Specific changes in the toxicity values are presented below:

- Toxicity values were added to the IRIS database. Prior to 2003, no toxicity values were available for 2-methylnaphthalene. For the SEH HHRA, this chemical was selected as a COPC but risks were not estimated.
- No toxicity values were listed for phenol, n-propylbenzene, n-butylbenzene for the SEH HHRA. However, values are now available for phenol on IRIS and provisional values are available for both n-propylbenzene and n-butylbenzene.
- The toxicity values for total xylenes were updated in 2003.
- The toxicity values for naphthalene were updated in 1998.
- The toxicity values for toluene were updated in 2005.
- The toxicity values for benzene were updated in 1998, 2000, and 2003.

The lack of a toxicity value for 2-methylnaphthalene in the 1998 SEH HHRA, most likely resulted in lower calculation of noncarcinogenic risk. The remaining changes to the toxicity values did effect the overall risk, but not as significantly as adding the risks associated with exposures to 2-methylnaphthalene.

Because no toxicity values are available for lead and no models were available for assessing risk to adult receptors exposed to lead, the SEH HHRA only looked at a qualitative review of this metal. However, for the current HHRA the USEPA Adult Lead Model was used to identify if risks associated with occupational lead exposures were unacceptable. In addition, residential child exposures to lead were also evaluated using the Integrated Exposure Uptake Biokinetic model to determine if child exposures to lead were unacceptable.

The differences in the toxicity values presented in the SEH and current HHRAs indicates that the current HHRA includes a more comprehensive quantitative discussion of risk than the SEH HHRA.

6.5.6 Comparison of the Data Sets Used for Evaluation

The number of samples used in the HHRAs differs significantly in that the number of soil samples upon which the current HHRA is based was greater. For the SEH HHRA, the soil data sets (Table 1) indicate that except for subsurface soil at Kreher Park, the data sets were less than 10. This generally resulted in the use of the maximum detected concentration as the EPC used to estimate risk. The use of the maximum detected concentration, although not unacceptable for estimating risk, it results in a high degree of uncertainty in that the actual concentration to which a receptor might be exposed is unknown and the EPC used could either over- or underestimate risk.

The number of samples used for sediment and surface water are similar.

6.5.7 Comparison of Calculated Cancer and Noncancer Risk

In order to compare risks calculated for each HHRA, it is necessary to look at risks using a receptor and exposure scheme that is similar for both HHRAs. For this comparison, the comparison was completed using the receptors and exposure pathways identified in the RI/FS Work Plan (URS, 2005).

The table presented below shows that generally cancer and noncancer risks are within the USEPA target goals of CRs from 10^{-4} to 10^{-6} for cancer risks and an HI of 1 for noncancer risks. When there are calculated risks above USEPA target levels, they were generally for similar receptors (City worker exposed to subsurface soil and construction worker).
There are distinct differences between both HHRAs. These differences include:

- Residential receptors were not evaluated in the 1998 SEH baseline HHRA. No comparisons can be made for this land use scenario.

- Although evaluated for the 1998 SEH baseline HHRA, the former seep area no longer represents a complete exposure pathway because impacted soil was removed prior to the area being capped in 2002. Therefore, the risks estimated are no longer valid. With the elimination of this exposure medium, the differences in the cancer and noncancer risks for recreational receptors exposed to media at Kreher Park, the comparison demonstrates that risks estimated in both HHRAs are similar and are within the USEPA target range for cancer and noncancer risk.

- Although surface water was evaluated for the 1998 SEH baseline HHRA, there were no surface water COPCs identified using the current surface water dataset. The SEH HHRA surface water risks estimated for the swimmers and waders were less than the USEPA target HI less of 1. Surface water risks estimated using the one sample where carcinogenic PAHs were detected shows that the cancer risks were within USEPA's target risk range, but greater than the WDNR target risk level of 1×10^{-5}. It is important to note that the current data set consists of high energy events (i.e., events likely to cause chemicals in the underlying sediment to resuspend Site-related chemicals to surface water) and low energy events (i.e., calm water) that were collected to verify the presence or absence of surface water contamination.

- The differences between the risks estimated for ingestion of fish are most likely because the 1998 SEH baseline HHRA used modeling to develop fish tissue EPCs using surface water data. The current HHRA uses actual fish tissue data to estimate risk and is more representative of Site conditions.

- The worker populations are different between the two HHRAs; therefore, comparisons between the general industrial worker and maintenance worker cannot be completed because they were not evaluated. However, the SEH HHRA utility worker can be compared to the current HHRA construction worker. The notable exposure parameter differences between the two receptors are note below:

Parameter	SEH HHRA Value	Current HHRA Value
Exposure Frequency	30 days/year	250 days/year
Skin Surface Area	1,311 -2,199 cm^2	1,930 cm
Soil Ingestion Rate	160 mg/day	330 mg/day
Inhalation Rate	3.3 m^3/hour	1.5 m^3/hour
Exposure Time	1 hour/day	8 hours/day

In general, the exposure parameter values used for the current HHRA construction worker are more conservative. Therefore, the risks calculated for the current HHRA are inherently more conservative.

- In general, the recreational receptor exposures are similar. The one notable difference is that the SEH HHRA assumes that recreational receptors will ingest fish from the impacted area. The current HHRA evaluates this pathway using a subsistence fisherman to evaluate this exposure pathway. Otherwise the exposure parameters used to estimate risk to recreational receptors are similar and will not affect the overall risk values obtained for the Site.

6.6 HOT SPOT ANALYSIS

A hot spot analysis was performed for the construction worker scenario using data collected near the former tar pit (TP-4, TP113, TP115, TP116, TP118, and TP119). The resulting cancer risk of 4E-06 is primarily attributed to benzo(a)pyrene (72%). Upon review of the data gathered for benzo(a)pyrene for the former tar pit, 8 samples with detectable concentrations ranging from 1,400 – 2,600 mg/kg between 2.5 and 5 feet bgs are the main contributors to the benzo(a)pyrene cancer risk. As a upperbound estimate of risks to a construction worker, the maximum detected concentrations of benzo(a)pyrene (3000 mg/kg) and naphthalene (37000 mg/kg) were also used to evaluate hot spot risk. The risks from ingestion and dermal contact with benzo(a)pyrene was 1.3E-03; the hazard index from ingestion, dermal contact, and inhalation of naphthalene was 972.

A hot spot analysis was performed for the construction scenario using data collected in the vicinity of the Former Coal Tar Dump in Kreher Park (TP-4, TP113, TP115, TP116, TP118, and TP119). This evaluation was completed as a worse case evaluation of potential risks

following exposures to elevated concentrations over a short duration when receptors are engaging in activities that may result in greater contact with soil.

The resulting cancer risk of 4×10^{-6} is primarily attributed to benzo(a)pyrene (72%). Upon review of the data gathered for benzo(a)pyrene for the site, 8 samples with reported concentrations ranging from 1,400 to 2,600 mg/kg between 2.5 and 5 feet bgs are the main contributors to the benzo(a)pyrene cancer risk.

6.7 QUANTIFICATION OF DERMAL EXPOSURE TO PAHs

There are no published dermal SFs available for any chemicals in any USEPA database. As indicated in Sections **4.2** and **4.3** of this HHRA, current USEPA guidance recommends converting oral SFs (an administered dose) using an gastrointestinal absorption factor to a dermal SF (an absorbed dose), if a chemical does not cause toxicological effects at the point of contact. However, based on literature evidence, PAHs have been shown to induce systemic toxicity and tumors at distant organs as well as point of contact. For this reason, the current default approach for extrapolating dermal

	1998 SEH Baseline HHRA								2007 HHRA											
	SS		SD		SW		FISH		SS		GW		SD		SW		FISH		Indoor Air	
Receptor[a, b]	CR	HI	CR	HI	CR	HI	CR	HI	CR	HI	CR	HI	CR	HI	CR	HI	CR	HI	CR	HI
Resident	NE	NE	NE	NE	NE	NE	NE	NE	**5×10^{-4}**	**15**	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Recreational Adult[d]	-	0.0006	NA	NA	NA	NA	NA	NA	4×10^{-6}	0.002	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Recreational Adolescent[d]	-	0.02	NA	NA	NA	NA	NA	NA	2×10^{-6}	0.003	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Recreational Child[d]	-	0.06	NA	NA	-	5×10^{-5}	NA	NA	1×10^{-5}	0.04	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Adult Swimmer	NA	NA	3×10^{-5}	0.1	6×10^{-5}	2×10^{-5}	NA	NA	NA	NA	NA	NA	4×10^{-5}	0.05	**9×10^{-2}**	**6**	NA	NA	NA	NA
Adolescent Swimmer	NA	NA	2×10^{-5}	0.3	3×10^{-5}	4×10^{-5}	NA	NA	NA	NA	NA	NA	2×10^{-5}	0.05	3×10^{-9}	2×10^{-5}	NA	NA	NA	NA
Child Swimmer	NA	NA	3×10^{-5}	0.7	-	2×10^{-4}	NA	NA	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE	NE
Adult Wader[d]	NA	NA	3×10^{-5}	0.1	4×10^{-5}	NE	NA	NA	NA	NA	NA	NA	4×10^{-5}	0.05	**5×10^{-2}**	**4**	NA	NA	NA	NA
Adolescent Wader[d]	NA	NA	2×10^{-5}	0.3	2×10^{-5}	NE	NA	NA	NA	NA	NA	NA	2×10^{-5}	0.05	NA	NA	NA	NA	NA	NA
Industrial Worker[e]	NA	1.2×10^{-4}	NA	NA	NA	NA	NA	NA	6×10^{-6}	0.007	NA	NA	NA	NA	NA	NA	NE	NE	8×10^{-5}	**3**
Maintenance Worker	NE	NE	NA	NA	NA	NA	NA	NA	1×10^{-6}	0.001	NA	NA	NA	NA	NA	NA	NE	NE	NE	NE
Construction Worker[c]	**2×10^{-4}**	0.5	NA	NA	NA	NA	NA	NA	**1×10^{-4}**	**38**	7×10^{-3}	**60**	NA	NA	NA	NA	NE	NE	8.34E-03 (KP) 2.14E-05 (UB) 3.29E-02 (FR)	17152 (KP) 228 (UB) 646601 (FR)
Subsistence Fisher - Adult	NA	NA	NA	NA	NA	NA	-	0.05	NA	NA	NA	NA	NA	NA	NA	NA	**1×10^{-4}**	0.01	NA	NA
Subsistence Fisher – Adol.	NA	NA	NA	NA	NA	NA	-	0.08	NE	NE	NE	NE	NE	NE	NA	NA	NE	NE	NE	NE
Subsistence Fisher - Child	NA	NA	NA	NA	NA	NA	-	0.04	NE	NE	NE	NE	NE	NE	NA	NA	NE	NE	NE	NE

[a] Because each HHRA evaluated different receptor populations, the risks presented above are for receptor populations that were standardized based on the approved RI/FS Work Plan.
[b] Because the seep was capped during the 2002 interim response action, exposures to this medium are no longer complete and were not used in this comparison.
[c] Construction workers were not evaluated in the 1998 SEH baseline HHRA. The values presented for this worker represent the subsurface soil risks for City Workers at the Utility Trench.
[d] Recreational receptor comparison uses only the current risk calculations provided in Attachment K Tables 1 and 6.
[e] Industrial worker comparison uses only the current risk calculations provided in Tables D-5 and D-19 of the SEH HHRA.
SS – soil
SD – sediment
SW – surface water
NA – Not applicable. This is not a relevant exposure medium for this receptor. Refer to **Section 3.1.4** for a discussion of the receptors evaluated for the Site.
NE – Not evaluated. This receptor or exposure medium was not evaluated. A suitable equivalent population could not be determined.
Items in **bold** represent cancer and noncancer risks that are greater than USEPA target levels of 10^{-4} for cancer risks and 1 for noncancer risks. Cancer risks that are underlined are greater than the WDNR threshold of 1×10^{-5}.

SF values are not applicable to PAHs. Therefore, RAGS Part A (USEPA, 1989) and Part E (USEPA, 2004), only recommend a qualitative evaluation of the carcinogenic effects of PAHs. Although a quantitative evaluation for this pathway was completed in this HHRA, as requested by Agencies, the actual cancer risks associated with dermal exposure to PAHs are unknown.

The results of the HHRA indicate that five exposure pathways result in estimated risks that exceed USEPA's target risk levels and seven exposure pathways result in estimated risks that are either equivalent to or exceed the WDNR threshold of 1×10^{-5}. These exceedances are indicated below.

Exceeds USEPA Threshold (CR $\geq 1\times10^{-4}$ or HI >1)	**Exceeds Wisconsin Threshold** (CR$\geq1\times10^{-5}$ or HI >1)
Residents (Soil[0-3 feet and all soil depths] - Cancer)	Residents (Soil[0-3 feet and all soil depths] - Cancer)
–	Residential Child (Soil – Noncancer)
Construction Worker (Soil [0-10 feet bgs]/Groundwater)	Construction Worker (Soil [0-10 feet bgs]/Groundwater)
Construction Worker (Trench Air)	Construction Worker (Trench Air)
Adult Swimmer (Surface Water)	Adult Swimmer (Surface Water)
Adult Wader (Surface Water/Oil slicks)	Adult Wader (Surface Water/oil Slicks/Sediment)
Industrial Worker (Indoor Air)	Industrial Worker (Indoor Air)
Subsistence Fisher (Biota)	Subsistence Fisher (Biota)

These include estimates for the RME scenarios for potential cancer risks and non-cancer risks. These conclusions are based on assumed exposures to soil in the filled ravine area (for residential receptors) and the filled ravine, upper bluff and Kreher Park area (for construction worker receptors), and to indoor air samples collected at NSPW Service Center. Carcinogenic risks based on CTE scenarios indicate that only the residential receptor exposure to soil (all soil depths to 10 feet bgs) are estimated to be at 1×10^{-4}, the upper-end of the USEPA target risk range or greater than the WDNR threshold. Carcinogenic risks based on the RME scenarios for residential receptor exposure to soils for all depths exceed 1×10^{-4}; i.e., the upper-end of the USEPA target risk range. Noncarcinogenic risks for the residential receptor (for soil depths 0-1 foot and 0-3 feet bgs) and risks associated with the construction scenario are within acceptable levels. However, residential receptor exposure to subsurface soil is not expected, given the current and potential future land use of the Site. For this Site, residential risks associated with CTE exposures to surface soil (0 to 1 foot bgs) are within the target risk ranges, but the RME exposures exceed the target risk range.

Although the results of the HHRA indicate risks for the construction workers under the RME conditions exceed USEPA's target risk levels, the assumptions used to estimate risks to this receptor were conservative and assumed the worst case. Given both the current and future land

use of the Site, it is unlikely that construction workers would be exposed to soil in the filled ravine and Upper Bluff. The most likely scenario for the future construction worker is exposure to soil within 0 to 4 feet bgs in Kreher Park (a typical depth for the installation of underground utility corridors), as most activities associated with the implementation of the future land use would be associated with regrading, landscaping, and road or parking lot construction. However, the depth to groundwater in Kreher Park is relatively shallow due to the lake-filled material comprising most of the park. Consequently, it is possible that construction workers excavating and installing utilities in such underground corridors in certain portions of Kreher Park may encounter COPC impacted sub-surface soils and NAPLs in groundwater.

An HI of 3 was calculated for the general industrial worker exposure to indoor air pathway under the RME conditions. This risk level is likely to be an overestimate because:

- It was estimated using the maximum detected concentrations as the concentrations at points of exposure.
- It was calculated based on USEPA default exposure parameters for the industrial /commercial workers (i.e., an individual works at the Site for 8 hours per day, 5 days per week, 50 weeks per year for a total of 25 years). The NSPW Service Center is used as a warehouse; there is an office space inside the building, but used only on a part-time basis.

Cancer risks to subsistence fisher (finfish) are equivalent to 1×10^{-4}, the upper-end of the USEPA target risk range, and greater than the WDNR threshold of 1×10^{-5}. Noncarcinogenic risk is within acceptable limits for both USEPA and WDNR.

Risks to recreational children (surface soil) are equivalent to 1×10^{-5}, which is the WDNR cancer risk threshold. However, risks to adolescent and adult receptors exposed to surface soil are below the USEPA acceptable risk range and below the WDNR risk threshold.

Risks to waders and swimmers (sediments), industrial workers (surface soil), and maintenance workers (surface soil) are all within USEPA's target risk range of 10^{-4} to 10^{-6} for lifetime cancer risk and a target HI of less than or equal to 1 for non-cancer risk and are less than the WDNR threshold of 1×10^{-5} for lifetime cancer risk and a target HI of less than or equal to 1 for non-cancer risk.

At the request of the WDNR, risks were also estimated for construction workers exposed to "oily materials" in groundwater via dermal contact and swimmers and waders who may be exposed to

oil slicks in surface water via ingestion and dermal contact. Because no media-specific concentrations are available for either scenario, risks were estimated using analytical data collected from the product stream from the active free product recovery system for the Copper Falls aquifer or chemical-specific solubility values detected in the DNAPL sample. Risks to construction workers exposed to "oily material" in groundwater and adult swimmers and waders exposed to "oil slicks" in surface water is greater than both the USEPA upper risk range (CR 1×10^{-4} and HI of 1) and than WDNR threshold (CR 1×10^{-5} and HI of 1). However, it is important to note that there is much uncertainty associated with estimating risks to oily material in groundwater or oil slicks in surface water. The primary uncertainties are associated with the lack of:

- Established methodology for estimating this exposure pathway
- Relevant oily material data resulting in the use of DNAPL data that are expected to result in an overestimate of risk.

In accordance with the AOC, the Remedial Action Objectives were prepared to document objectives based upon human health and ecological risk assessment results. This section primarily focuses on the COPCs for each media, potential exposure pathways and receptors, and acceptable contaminant levels, or range of levels (protectiveness), at particular locations for each exposure route. A brief summary of the Ashland Lakefront Site is provided along with an outline of the remedial alternatives process

8.1 INTRODUCTION

The Site contains property owned by NSPW, a portion of Kreher Park, the former Wastewater Treatment Plant (WWTP), and a portion of the Chequamegon Bay inlet area adjacent to Kreher Park. The primary contaminant source is the former manufactured gas plant which previously occupied the NSPW property. In addition, other possible industrial operations might have contributed to the contaminant source at Kreher Park.

Site characterization began in 1989 when apparent contamination was discovered at Kreher Park. The primary contaminants at the Site are derived from tar compounds, including VOCs and PAHs. Soils, groundwater, and offshore sediments have been impacted. Additionally, free-product derived from the tars is present as a non-aqueous phase liquid (NAPL) in the upper reaches of a filled ravine on the NSPW property, at Kreher Park including the former "seep" area, in the off-shore sediments, and in the upper elevations of the deep Copper Falls aquifer. The free-product in the deep aquifer is surrounded by a dissolved phase contaminant plume that extends north from the area of the free-product in the direction of groundwater flow. Although contaminants have migrated down gradient in the underlying Copper Falls aquifer, both the vertical and lateral extent of contamination is limited by strong upward gradients that create artesian conditions at the Lakefront.

NSPW implemented interim removal actions in 2000 to mitigate exposure risks to contaminants and to recover free-product from the deep aquifer. A low-flow pumping system currently extracts free-product from the deep aquifer, treating the entrained groundwater before discharging it to the City of Ashland's sanitary sewer. Additionally, NSPW installed an extraction well at the base of the filled ravine that was the source of the seep discharge at Kreher Park. This extraction well was part of a larger interim action that included excavation of contaminated materials at the former seep area and placement of a low-permeability cap to eliminate the intermittent seep discharge and mitigate environmental exposure of the associated contaminants.

8.2 CHEMICALS OF POTENTIAL CONCERN

The primary contaminants at the NSPW Site consist of VOCs and SVOCs. Benzene is the most commonly occurring VOC. SVOCs consist predominantly of a group of PAH compounds. The most commonly occurring PAH at the Site is naphthalene. Some metals (lead, thallium and arsenic) and inorganic compounds (cyanides) have also been found, but these are sporadic are not considered significant COPCs.

The baseline revised Human Health Risk Assessment (HHRA) (URS, 2007) used a tiered, risk-based approach to evaluate COPCs for the various exposure scenarios. The results of the HHRA evaluation found the following COPCs for the Site.

In the HHRA, the toxicity assessment provides a framework for characterizing the relationship between the magnitude of exposure to a chemical and the nature and likelihood of adverse health effects that may result from such exposure. Chemical toxicity is typically divided into two categories: carcinogenic and noncarcinogenic. Potential health effects are evaluated separately for these two categories, because their toxicity criteria are based on different mechanistic assumptions and associated risks are expressed in different units. Thus, the COPC list was refined using toxicology, pathways, and exposure during the HHRA for the Site. No COPCs were identified in the HHRA for groundwater because groundwater is not used as a potable water supply, though construction worker exposure to groundwater is possible. At the former Waste Water Treatment Plant (WWTP), trespassers who enter the buildings can potentially inhale vapors and have direct dermal contact with contaminated groundwater and NAPLs that have infiltrated the flooded lower level of the facility. The COPCs identified for surface water include PAHs. The COPCs identified for sediment include metals and PAHs. PAHs were found to be COPCs in fish. Several volatile compounds were identified COPCs in indoor air.

List of COPCs Identified by the HHRA					
Surface Water	**Groundwater**	**Sediment**	**Soil**	**Fish**	**Indoor Air**
Benzo(a)anthracene	1-Methylnaphthalene	Antimony	1-Methylnaphthalene	1-Methylnaphthalene	1,2,4-Trimethylbenzene
Benzo(a)pyrene	2-Methylnaphthalene	Iron	2-Methylnaphthalene		1,4-Dichlorobenzene
Benzo(b)fluoranthene	Acenaphthene	Manganese	Acenaphthene	Benzo(a)anthracene	Benzene
Benzo(k)fluoranthene	Benzo(a)anthracene	Vanadium	Benzo(a)anthracene		Carbon tetrachloride
Chrysene	Benzo(a)pyrene	1-Methylnaphthalene	Benzo(a)pyrene	Benzo(e)pyrene	Trichloroethylene
Dibenzo(a,h)anthracene	Benzo(b)fluoranthene	2-Methylnaphthalene	Benzo(b)fluoranthene	Benzo(b)fluoranthene	
Indeno(1,2,3-cd)pyrene	Benzo(k)fluoranthene	Benzo(a)anthracene	Benzo(k)fluoranthene	Dibenzo(a,h)anthracene	
	Chrysene	Benzo(a)pyrene	Chrysene	Dibenzofuran	
	Dibenzo(a,h)anthracene	Benzo(b)fluoranthene	Dibenzo(a,h)anthracene		
	Dibenzofuran	Benzo(k)fluoranthene	Dibenzofuran		
	Fluoranthene	Indeno(1,2,3-cd)pyrene	Fluoranthene		
	Fluorene	Naphthalene	Fluorene		
	Indeno(1,2,3-cd)pyrene		Indeno(1,2,3-cd)pyrene		
	Naphthalene		Naphthalene		
	Phenanthrene		Phenanthrene		
	Pyrene		Pyrene		
	1,2,4-Trichlorobenzene		1,2,4-Trichlorobenzene		
	1,2,4-Trimethylbenzene		1,2,4-Trimethylbenzene		
	1,3,5-Trimethylbenzene		1,3,5-Trimethylbenzene		
	Benzene		Benzene		
	Ethylbenzene		Ethylbenzene		
	Toluene		Toluene		
	Total Xylenes		n-Butylbenzene		
			sec-Butylbenzene		
			Total Xylenes		
			Arsenic		
			Lead		
			Thallium		

8.3 POTENTIAL EXPOSURE PATHWAYS AND RECEPTORS

The exposure pathway links the sources, types of environmental releases, and environmental fate with receptor locations and activity patterns. Generally, an exposure pathway is considered complete if it consists of the following four elements:

- A source and mechanism of release;
- A transport medium;
- An exposure point (i.e., point of potential contact with an impacted medium); and
- An exposure route (e.g., ingestion) at the exposure point.

Release mechanisms and transport pathways were evaluated for the Site. Listed below are potential cross-media transfer mechanisms of chemicals:

- Chemicals in subsurface soil may enter groundwater through infiltration/percolation;
- Chemicals in surface soil may be transported to surface water and sediments through surface runoff and backfilling;
- Chemicals in groundwater may be transported to surface water and sediments through groundwater discharge;
- Chemicals in groundwater may be infiltrating the lower level of the former WWTP located in Kreher Park;
- Chemicals in surface soil may be transported to the atmosphere via volatilization or fugitive dust emission;
- Chemicals in soil or groundwater may be transported to the atmosphere or indoor air through volatilization;
- Chemicals in surface water and sediments may be transported to the tissues of aquatic organisms or higher trophic levels through bioaccumulation; and
- Chemicals in sediments may be released to surface water when sediments are disturbed.

8.3.1 Human Health Receptors and Exposure Scenario

Presented below is an overview of exposure pathways of potential concern selected for further evaluation in the HHRA. Potential receptors are discussed based on medium of interest (i.e., soil, groundwater, sediment, surface water, biota, and air). Updates to the receptor populations identified in the Final Work Plan (URS, 2005) are discussed as necessary.

8.3.1.1 Exposure to COPCs in Soil

Residential Land Use Scenario: Child and Adult Residents

Upper Bluff - There is a residential area located up gradient from the Kreher Park area of the Site at the upper bluff area northeast of the former ravine. Described below were three exposure scenarios assumed in the HHRA for the residential receptors:

Exposure to surface (0-1 foot) and subsurface soil (1-10 feet bgs).

This assumption was made because new construction would involve excavation of soil for the construction of footings or basements. Therefore, subsurface soil would be brought to the surface resulting in a potential exposure pathway for residential receptors. This scenario represents the worst case for residential receptors, but is not likely to be the actual scenario associated with the Site.

Exposure to surface soil.

The residential neighborhoods adjacent to the Site are established neighborhoods and are expected to remain so in the future. According to the Ashland Wisconsin Waterfront Development Plan, the future use of the Kreher Park portion of the Site does not include a residential scenario. In an established residential setting and without intrusive activities, receptors would most likely be exposed to surface soil.

Exposure to soil in 0-3 feet bgs.

For informational purposes, COPCs in soil between 0 and 3 feet bgs were also considered for residential receptors based on the assumption that receptors could potentially be exposed to soil from 0-3 feet bgs when performing landscaping or gardening activities.

For the purpose of the HHRA, child and adult residents were assumed to be exposed to COPCs in soil via incidental ingestion, inhalation (of soil-borne vapor and particulates) and dermal contact pathways.

Recreational Use Scenario: Child, Adolescent and Adult Visitors

Kreher Park is now zoned as City parkland. Child, adolescent and adult visitors are assumed to be exposed to COPCs in surface soil via incidental ingestion, inhalation (of soil-borne vapor and particulates) and dermal contact pathways.

Industrial/Commercial Land Use Scenario: Maintenance Workers

Although the final Work Plan indicated maintenance workers currently access the Site, additional information collected during the RI indicates that City workers and utility maintenance personnel do not access the Site. However, the City may develop the existing marina and expand it into the affected area for recreational use. Therefore, a potential future maintenance worker was considered a receptor to surface soil at Kreher Park and the unpaved portions of the Upper Bluff area. It is conservatively assumed that maintenance workers may be exposed to COPCs in surface soil via incidental ingestion, inhalation (of soil-borne vapor and particulates) and dermal contact pathways.

Industrial/Commercial Land Use Scenario: General Industrial Workers

Except for the NSPW facility, no other industrial/commercial facilities exist within the Site. For this HHRA, general workers are defined as NSPW employees involved with non-intrusive, operational activities. Current and potential future general workers are not likely to be subject to significant exposure to environmental media in the normal course of their daily work. Although the potential for exposure to occur is expected to be low, general workers are assumed to be exposed to COPCs in surface soil via incidental ingestion, inhalation (of soil-borne vapor and particulates) and dermal contact pathways.

Industrial/Commercial Land Use Scenario: Construction Workers

Upper Bluff and Kreher Park - It is conservatively assumed that construction activities could take place at every area included in this evaluation and it is possible for construction workers to be exposed to COPCs in surface and subsurface soil via incidental ingestion, inhalation (of soil-borne vapor and particulates) and dermal contact pathways. For this HHRA subsurface soil is defined as a depth of 10 feet or less, which is a conservative estimate of the limit to which construction activities may occur based on the current and proposed future land use at the Site.

8.3.1.2 Exposure to COPCs in Indoor Air – Residents and Industrial Workers

Upper Bluff - The residential area located up gradient from Kreher Park at the upper bluff area northeast of the former ravine was evaluated. For the purpose of the HHRA, child and adult residents were assumed to be potentially exposed to COPCs volatilizing from soil and groundwater and entering the residences located near the ravine. In addition, potential exposures to COPCs in indoor air were also evaluated for industrial workers who may enter the NSPW service center/vehicle maintenance building periodically.

Kreher Park – trespassers who enter the former WWTP can potentially inhale vapors released to contaminated groundwater and NAPLs that have infiltrated the flooded lower level of the facility. The potential health risks associated with this exposure pathway was part of the RI/FS work plan (URS, 2005), but was not quantitatively evaluated by the HHRA and is a data gap. This exposure pathway was not quantitatively evaluated because access to the interior of the plant was restricted during the RI/FS study and no samples could be collected. Additionally, earlier indoor air analyses results collected by the City of Ashland (2002) were not available for review as part of the HHRA. Despite this shortcoming, direct contact exposures to NAPL or "free-product" in groundwater may pose an unacceptable health risk.

8.3.1.3 Exposure to COPCs in Groundwater: Trespassing Land Use Scenario

The final Work Plan indicated that groundwater in the seep area was a potential exposure point for trespassers. However, this exposure point was eliminated because the seep area was capped as part of the 2002 interim action response (URS, 2002). This exposure pathway is no longer complete and was not quantitatively evaluated in the HHRA.

Another potential point of exposure to groundwater is the former WWTP building where groundwater has infiltrated into the basement. The building is locked and the perimeter is partially fenced. A quantitative evaluation for the potential trespasser exposures to the indoor air and water inside the former WWTP building was not performed due to the lack of data.

Industrial/Commercial Land Use Scenario: Construction Workers

Kreher Park - It is conservatively assumed that construction activities could take place at every area included in this evaluation and it is possible for construction workers to be exposed to COPCs in shallow groundwater at Kreher Park via incidental ingestion, inhalation of vapors, and

dermal contact pathways. For this HHRA, shallow groundwater is defined as a depth of 10 feet or less, which is a conservative estimate of the limit to which construction activities may occur based on the current and proposed future land use at the Site.

Residential and Industrial/Commercial Land Use Scenarios

Groundwater is present in both the water table aquifer and a confined deep aquifer. Currently the shallow groundwater is not used as a potable water source. There are two artesian wells in the Site vicinity—one located near Prentice Avenue on the eastern boundary of the Site and the other located near the marina on the western boundary. Both wells draw water from the Copper Falls aquifer, the confined deep aquifer that is separated from the shallow groundwater by the Miller Creek Formation (URS, 2005; ATSDR, 2003). The City of Ashland restricted public access to these wells for public use in August 2004. To date water from these wells have met all federal and state safe drinking water standards. Water from these artesian wells is considered safe to drink as Site-related chemicals have not been detected in these wells at levels of concern (ATSDR, 2003).

Except for the two artesian wells, the Copper Falls aquifer is not used for drinking water and is not considered a source of human exposure. Shallow groundwater at the Site is not a drinking water source for the City of Ashland. Drinking water at the Site is provided by the City of Ashland that draws its water from intakes in Lake Superior, located approximately one mile northeast of the Site outside the known extent of surface water contamination. Therefore, there are no known receptors to shallow groundwater beneath the Site.

8.3.1.4 Exposure to COPCs in Surface Water and Sediments

Recreational Use Scenario: Child, Adolescent and Adult Visitors to Kreher Park and Chequamegon Bay

The Site is surrounded by facilities that draw the public to the lakefront – a City marina, public swimming beach, a boat ramp and an RV park and campground. Child, adolescent and adult visitors are assumed to be exposed to COPCs in surface water and sediments via incidental ingestion, inhalation of vapors, and dermal contact pathways while swimming, wading, fishing, or boating. However, only risks associated with swimming and wading activities were quantified in the HHRA. This is because they represent activities that have the greatest contact

with impacted media and are considered more conservative than exposures associated with fishing and boating.

8.3.1.1 Exposure to COPCs in Fish Tissue

Subsistence fishers were selected as the fishing receptors because there are two Chippewa Bands (the Bad River Band and the Red Cliff Band of Lake Superior Chippewa) who may use Chequamegon Bay as their source of fish. For the HHRA it was conservatively assumed that adult subsistence fishers may be exposed to COPCs via ingestion of locally-caught fish. Although this scenario was selected based on the presence of the two Chippewa Bands, this exposure scenario and the selected exposure parameters are applicable to any subsistence fisher ingesting fish from Chequamegon Bay.

8.3.2 Ecological Receptors and Exposure Scenario

In the BERA (URS, 2006), the potential risk to ecological receptors was evaluated for benthic macroinvertebrates, fish, birds, and mammals. The potential contact points for ecological receptors include surface water, surface soil and food/prey in terrestrial habitats; and, surface water, sediment and food/prey in aquatic and wetland habitats.

Each of these contact points and their respective exposure media were addressed in the BERA.

8.3.2.1 Routes of Entry

The potential routes of entry for ecological receptors are:

- Direct contact: dermal and/or gill absorption;
- Ingestion; and,
- Inhalation.

In the exposure analysis the relationship between receptors at the Site and potential stressors (chemical, biological, or physical entities that may result in adverse effects to one or more receptors or groups of receptors) were evaluated. EPCs used to estimate exposure were calculated as the UCL95 of the exposure medium. EPCs calculated for sediment, soil, or tissue residues were based directly upon the levels of contaminants in these media. There were no COPCs for surface water.

Exposure estimates for birds and mammals were calculated using food web models. Simplified food web models were developed to calculate average daily doses (ADDs) of COPCs that representative receptors experience through exposure to sediment, and surface soil at the Site. The ADD represents the dose of a chemical that a receptor may ingest if it foraged within designated exposure units. ADDs for wildlife receptors are calculated using (1) exposure-point concentrations for prey and media developed for each, (2) COPC-specific bioaccumulation factors or bioaccumulation models for dietary items, and (3) receptor-specific exposure parameters and food chain model assumptions, (e.g., diet composition, foraging area, amount of incidental soil or sediment ingested, etc.).

8.3.2.2 Risk Characterization

Risk Characterization was the final phase of the BERA. In Risk Characterization, the information from the effects and exposure analyses were used to determine a probability of adverse effects to receptors of concern and discuss the strengths, weaknesses, and assumptions in the BERA. Risk estimates (or Hazard Quotients) were developed for each assessment endpoint based upon comparison of site-specific media concentrations and/or estimated ingested contaminant dose estimates (the latter for wildlife) to effects levels (generic criteria, benchmarks and TRVs) for the various ROCs. Finally risk was characterized for each assessment endpoint by integrating the risk estimate with the results of other lines of evidence, if available.

The results of the risk characterization indicated that there are potentially unacceptable impacts to the benthic macroinvertebrate community in aquatic portions of the Site. Two lines of evidence, bulk sediment chemistry and sediment toxicity testing, indicated that the probability of impairment at the community level was likely. Effects observed from the URS field surveys of the existing benthic community indicated effects that were less dramatic than those demonstrated in the laboratory toxicity studies, but interpretation of the field survey data is made very difficult by a high degree of variability and lack of comparability between reference and site stations.

The BERA concluded that the potential for adverse effects to ecological receptors other than benthic macroinvertebrates was not sufficient to result in significant adverse alterations to populations and communities of these ecological receptors.

8.3.3 Remedial Action Objectives

The specific goals of the remedial actions are defined by acceptable contaminant levels, or a range of levels at each location for each exposure route. The acceptable contaminant level (or protectiveness) is determined based on the findings of the HHRA and the BERA. The general goal of these objectives is to protect human health and environmental receptors at risk due to constituents at the site. These objectives are subject to the criteria evaluated in the FS, and include:

- Eliminate or reduce potential risks to human health and to aquatic and terrestrial animals and to the environment from exposure to contaminants;
- Eliminate future migration of contaminants to receptors;
- Eliminate on-site migration of contaminants;
- Eliminate or reduce contaminant migration to Chequamegon Bay;
- Remove or reduce free-product (NAPL) present at the upper bluff (filled ravine/NSPW property and the Copper Falls Aquifer);
- Remove or reduce free product (NAPL) at Kreher Park;
- Remove or reduce free product (NAPL) from the sediments in Chequamegon Bay;
- Minimize short term risk to human health and to aquatic and terrestrial animals and to the environment: from exposure to contaminants during the implementation of the remedial action.

The HHRA was based upon the protection of human health. The BERA was based upon a risk management goal of maintenance (or provision) of soil, sediment, water quality, food source, and habitat conditions capable of supporting a "functioning ecosystem" for the ecological populations inhabiting or using the Site. The HHRA was used to develop RAOs for soil, and the BERA was used to development RAOs for surface water and sediment. Although HHRA results indicate that groundwater is not currently used as a potable water supply, construction workers may encounter groundwater in a trench. RAOs for dissolved phase and free-phase (tar) groundwater contamination were also developed for groundwater. The development of RAOs is described in the following sections. RAOs for site media are summarized below.

The basis and rationale for soil remediation objectives is protection of reasonable future uses. This includes industrial, commercial and utility worker protection and protection of recreational users of Kreher Park. The basis and rationale for groundwater remediation objectives is based on

anticipated commercial/industrial and recreational land use. These objectives were developed to eliminate exposure and protect against off-site migration of contaminants. The basis and rationale for surface water remedial objectives are to minimize the potential for contaminant exposure to surface water users and reduce migration of groundwater and sediment contaminants to surface water that could result in exceedance of surface water standards. The basis and rationale for sediment remedial objectives are to protect populations of aquatic organisms, including fish, and to protect against migration of contaminants from sediments to surface water.

Remedial Action Objective Summary by Site Media		
Environmental Media	**Receptor**	**Preliminary Remedial Action Objectives**
Groundwater	Human Health	Protect human health by eliminating exposure (direct contact, ingestion, inhalation) to groundwater with COPCs in excess of regulatory or risk-based standards; reduce contaminant levels in groundwater to meet MCLs and State of Wisconsin Drinking Water Standards
	Environment Ecological Receptors)	Protect the environment by controlling the off-site migration of contaminants in groundwater to surrounding surface water bodies which would result in exceedance of ARARs for COPCs in surrounding surface waters.
		Conduct free product removal to halt or contain the discharge of a hazardous substance or to minimize the harmful effects of the discharge to the air, land or water.
Soil	Human Health	Protect human health by reducing or eliminating exposure (ingestion/direct contact/inhalation) to soil having COPCs representing an excess cancer risk greater than 10^{-6} as a point of departure (with cumulative excess cancer risks not exceeding 10^{-5}) and a hazard index (HI) greater than 1 for reasonably anticipated future land use scenarios.
		Ensure future beneficial commercial/industrial use of the site and recreational use of Kreher Park.
	Environment (Ecological Receptors)	Protect populations of ecological receptors or individuals of protected species by eliminating exposure (direct contact with or incidental ingestion of soils or prey) to soil with levels of COPCs that would pose an unacceptable risk.
		Conduct free product removal to halt or contain the discharge of a hazardous substance or to minimize the harmful effects of the discharge to the air, land or water.
		Protect the environment by minimizing/eliminating the migration of contaminants in the soil to groundwater or to surrounding surface water bodies.
Surface Water	Human Health	Protect human health by minimizing exposures (direct contact, ingestion, inhalation) to surface water that has been impacted by Site-related groundwater and sediment with concentrations of COPCs such that regulatory or risk-based surface water standards have been exceeded.
	Environment (Ecological Receptors)	Protect the environment by controlling the migration of contaminants in groundwater and in sediments to surface water which would result in exceedance of ARARs for COPCs in surface water.
		Reduce Site-related COPC levels in the surface water to meet State of Wisconsin Surface Water Quality Standards.
Sediments	Human Health	Protect human health by eliminating exposure (direct contact, ingestion, inhalation, fish ingestion) to sediment with COPCs in excess of regulatory or risk-based standards.
	Environment (Ecological Receptors)	Protect populations of ecological receptors or individuals of protected species by eliminating exposure (direct contact with incidental ingestion of sediments or of prey) to sediment with levels of COPCs that would pose an unacceptable risk.
		Conduct free product removal to halt or contain the discharge of a hazardous substance or to minimize the harmful effects of the discharge to the air, land or water.

8.3.3.1 HHRA Based Remedial Action Objectives for Soil, Surface Water and Groundwater

The results of the HHRA indicate that only residential exposure pathways (for soil depths between 0 to 3 feet or all soil depths to 10 feet bgs) and construction worker exposure pathways (for soil depths between 0 and 10 feet) are associated with unacceptable risks (Cancer Risk (CR) greater than 10-4 and Hazard Index (HI) greater than 1) based on exposures to soil in the filled ravine area for residential receptors and the Kreher Park area for construction worker receptors. However, residential receptors are not expected to be exposed to subsurface soil given the current and potential future land use of the Site. (Residential land use in Kreher Park is not anticipated, and residential land use in the upper bluff area is located outside the backfilled ravine where contamination has been identified.) For this Site, risks associated with exposures to surface soil are within acceptable risk ranges.

Although the results of the HHRA indicate risks for exposure to soils and the construction worker scenario exceed USEPA acceptable levels, the assumptions used to estimate risks to this receptor were conservative and considered the worst case. Given both the current and future land use of the Site, it is not likely that construction workers would be exposed to subsurface soil at depths beyond 4 feet bgs (a typical depth for the installation of underground utility corridors), as most activities associated with the implementation of the future land use would be associated with subsurface activities such as regrading, landscaping, and road or parking lot construction. The risk for exposure of construction workers to groundwater was based upon exposure to free product (NAPL), using data for NAPL from samples collected from the free product recovery system currently removing free product from the Copper Falls Aquifer. Although exposure of construction workers to free product with concentrations of chemicals similar to what is collected in recovery wells is highly unlikely and introduces substantial uncertainty into quantification of this exposure pathway, this analysis was conducted at EPA's request. The results of this analysis indicated a carcinogenic risk ranging between 3×10^{-5} and 7×10^{-3} and non-carcinogenic (hazard indices) risk of between 2×10^{-1} and 3×10^{3}. However, based on the above discussion, risks to this receptor population from soil and groundwater exposure are most likely overstated.

Risks to recreational users (surface soil), waders and swimmers (sediments), industrial workers (surface soil), and maintenance workers (surface soil) are all within USEPA's acceptable range of 10^{-4} to 10^{-6} (and do not exceed a cumulative risk of 10^{-5}) for CR and 1 for HI. Risks to subsistence fishers (finfish) was at 10^{-4} and risk to a wader contacting surface water ranged from 2×10^{-5} to 6×10^{-5}.

At EPA's request, an analysis of a swimmer or wader incidentally ingesting and dermally contacting oil material (sheens) in surface water was also conducted. Using the same data from the free-product recovery system as described for dermal exposure to construction workers, risks to swimmers and waders exposed to potential oil slicks in surface water were calculated. In the unlikely event a swimmer or wader contacted oily material (sheens) in surface water 12 days a year the CR would range from 4×10^{-3} to 5×10^{-2}. The non-cancer HI ranged from 4 to 7×10^{-2}. The CR to wader or swimmer for incidental ingestion of surface water ranged from 3×10^{-8} to 1×10^{-6}. The non-cancer HI ranged from 2×10^{-4} to 1×10^{-1}. All of these levels assume worst-case conditions and are associated with a high level of uncertainty.

Preliminary Remediation Goals for Soils and Surface Water

Based on the results of the Site-specific HHRA, preliminary remediation goals (PRG) were derived for the following exposure scenarios that exceeded a cumulative cancer risk of 10^{-5} or a cumulative noncancer risk of a hazard index (HI) of 1:

- Construction worker exposure to soil at Kreher Park;
- Residential exposure to soil at the Upper Bluff; and
- Recreational exposure to surface water.

PRGs were derived for chemicals identified as the primary risk drivers using exposure parameters that were used to develop the HHRA. Presented below are chemical-specific acceptable contaminant levels for these exposure scenarios based on target cancer risk goals of 10^{-4} to 10^{-6} and target noncancer risk goals of an HI of 0.1 and 1. PRGs are not developed for fish because remediation is not plausible for fish; rather, risks from consumption is controlled through consumption advisories, and fish contaminant levels will be reduced through sediment remediation. PRGs were not developed for the indoor air pathway; indoor air levels will be reduced through groundwater remediation.

Soil Preliminary Remediation Goals for Construction Workers (mg/kg)					
Chemical	Carcinogenic Effects			Noncarcinogenic Effects	
	CR = 10^{-6}	CR = 10^{-5}	CR = 10^{-4}	HI = 0.1	HI = 1.0
SVOCs					
2-Methylnaphthalene	NA	NA	NA	1.13E + 02	1.13E + 03
Benzo(a)anthracene	2.01E + 00	2.01E + 01	2.01E + 02	1.06E + 04	1.06E + 05
Benzo(a)pyrene	2.01E - 01	2.01E + 00	2.01E + 01	NA	NA
Benzo(b)fluoranthene	2.01E + 00	2.01E + 01	2.01E + 02	NA	NA
Dibenzo(a,h)anthracene	2.01E - 01	2.01E + 00	2.01E + 01	NA	NA
Indeno(1,2,2-cd)pyrene	2.01E + 00	2.01E + 01	2.01E + 02	7.06E + 03	7.06E + 04
Naphthalene	NA	NA	NA	3.81E + 00	3.81E + 01
VOCs					
Benzene	1.4E + 00	1.4E + 01	1.4E + 02	4.11E + 00	4.11E + 01

Soil Preliminary Remediation Goals for Residents (mg/kg)				
Chemical	Carcinogenic Effects		Noncarcinogenic Effects	
	CR = 10^{-5}	CR = 10^{-4}	HI = 0.1	HI = 1.0
SVOCs				
Benzo(a)anthrancene	6.21E + 00	6.21E + 01	NA	NA
Benzo(a)pyrene	6.21E - 01	6.21E + 00	NA	NA
Benzo(b)fluoranthene	6.21E + 00	6.21E + 01	NA	NA
Dibenzo(a,h)anthracene	6.21E - 01	6.21E + 00	NA	NA
Naphthalene	NA	NA	1.70E + 00	1.70E + 01
VOCs				
Benzene	7.37E + 00	7.37E + 01	1.80E + 00	1.80E + 01

Surface Water Preliminary Remediation Goals for Swimmers (mg/L)					
Chemical	Carcinogenic Effects			Noncarcinogenic Effects	
	CR = 10^{-6}	CR = 10^{-5}	CR = 10^{-4}	HI = 0.1	HI = 1.0
SVOCs					
Benzo(a)anthrancene	2.04E - 04	2.04E - 03	2.04E - 02	NA	NA
Benzo(a)pyrene	1.17E - 05	1.17E -04	1.17E - 03	NA	NA
Benzo(b)fluoranthene	1.19E - 04	1.19E - 03	1.19E - 02	NA	NA
Dibenzo(a,h)anthracene	7.72E - 06	7.72E - 05	7.72E - 04	NA	NA
Indeno(1,2,2-cd)pyrene	1.17E - 04	1.17E - 03	1.17E - 02	NA	NA

Preliminary Remediation Goals for Groundwater

No COPCs were initially identified in the HHRA for groundwater because groundwater is not used as a potable water supply. However, exposure to contaminated groundwater and accompanying NAPLs can potentially occur via the following exposure scenarios:

- Construction worker exposure to shallow groundwater infiltrating trenches at Kreher Park; and
- Trespasser exposure to groundwater infiltrating the lower level of the former WWTP.

These pathways are further discussed and the PRGs for direct contact and inhalation of vapors from affected groundwater are presented under Section A.3.3.3 (Remedial Action Objectives for Media with No Exposure Pathways).

The COPCs in sediment included five PAHs, but the cumulative risks estimated for the recreational receptor exposures to sediments were below USEPA's target risk levels.

8.3.3.3 Remedial Action Objectives for Media with No Exposure Pathways

As described in Section 8.3.1.3 above, currently groundwater is not used as a potable water supply in the vicinity of the Site. Potential exposure to shallow groundwater encountered in Kreher Park fill was eliminated when the seep area was capped in 2002. Shallow groundwater encountered in the filled ravine and groundwater in the underlying Copper Falls aquifer is not currently being used for drinking water in the vicinity of the Site3. However, construction workers in a trench may be exposed to groundwater contaminants. For any trench excavated at Kreher Park, shallow contaminated groundwater and NAPLs can infiltrate through coarse fill materials and workers who enter the trenches can be exposed through direct dermal contact and inhalation of vapors. At the former WWTP, trespassers who enter the buildings can potentially inhale vapors and have direct dermal contact with contaminated groundwater and NAPLs that have infiltrated the flooded lower level of the facility. The potential health risks associated with

[3] Although no contaminants were detected in samples collected from two artesian wells located in Kreher Park that obtain water from the Copper Falls aquifer, the City of Ashland restricted access to these wells for public use in August 2004. Additionally, the Site is located within the City limits and serviced by a municipal water supply.

these exposure pathways have not been thoroughly evaluated by the HHRA (see Section 8. 3.3.1). Direct contact exposures to NAPL or "free product" in groundwater may pose an unacceptable health risk.

Despite these data gaps, site investigation results indicate that COPCs in the shallow Kreher Park and ravine fill units and groundwater in the underlying Copper Falls aquifer exceed regulatory enforceable groundwater quality standards. PRGs for groundwater were derived primarily from Wisconsin Administrative Code (WAC) chapter NR 140 groundwater quality standards for the most frequently occurring dissolved phase organic COPCs based on historic groundwater monitoring results. The concentrations provided in the table below provide a conservative level that will be further refined in subsequent technical memoranda and the FS.

Preliminary Remediation Goals (µg/l) for Organic COPCs in Groundwater (WAC Chapter NR 140 Enforcement Standard)			
COPC – VOCs	**ES**	**COPC – SVOCs***	**ES**
Benzene	5	Anthracene (LMW)	3,000
Ethylbenzene	700	Benzo(a)Pyrene (HMW)	0.2
Styrene	100	Benzo(b)Fluoranthene (HMW)	0.2
Toluene	1,000	Chrysene (HMW)	0.2
1,2,4-Trimethylbenzene	480**	Fluoranthene (HMW)	400
1,3,5-Trimethylbenzene		Fluorene (LMW)	400
Total Xylenes	10,000	Naphthalene (LMW)	40
		Pentachlorophenol	1
		Pyrene (HMW)	250
		Phenol	6,000

* (HMW) – Heavy molecular weight PAHs; (LMW) – Low molecular weight PAHs

** Trimethylbenzene (TMB) in groundwater will be presented as the sum of 1,2,4- and 1,3,5- TMB per the WAC ch. NR 140 standard.

Inorganic COPCs (metals and cyanide) were also detected above groundwater quality standards. Acceptable contaminant levels for groundwater were derived primarily from WAC chapter NR 140 groundwater quality standards for the most frequently occurring dissolved phase inorganic COPCs based on historic groundwater monitoring results. However, iron and manganese were

detected in samples collected from up gradient wells[4] at concentrations above groundwater quality standards. Because these elevated concentrations represent background conditions, the maximum detected concentrations have been substituted as the acceptable contaminant level for COPCs that exceed groundwater quality standards in background samples. A summary of the acceptable contaminant levels for inorganic COPCs in the Miller Creek and Copper Falls aquifer follows: The concentrations provided in the table below provide a conservative level that will be further refined in subsequent technical memoranda and the FS.

Preliminary Remediation Goals (µg/l) for Inorganic COPCs in Groundwater			
Inorganics	**ES**	**Background Concentrations for Miller Creek (Well MW-11)**	**Background Concentrations for Copper Falls (Well MW-6A)**
Arsenic	6	0 – 3.2	0 – 4.4
Antimony	10	0 – 4.3	0 – 4.1
Barium	2,000	130 – 260	640 – 710
Beryllium	4	ND	ND
Cadmium	5	0 – 0.2	ND
Chromium (+3) Chromium (+6)	100*	ND	0.87 – 2.1
Cobalt	40	0 – 16	0 – 1.1
Copper	1,300	2 – 35	2.4 – 6.1
Cyanide	200	0 – 17	0 – 4
Iron	300	7.1 – 19,000	0 – 0.0046
Lead	15	0 – 3.3	0.485 – 2.6
Manganese	50	13 – 760	30 – 410
Mercury	2	ND	ND
Nickel	100	0.95 – 24	1.6 – 4.7
Selenium	50	0 – 3.3	0 – 2.8
Silver	50	0 – 1.65	0 – 0.8
Thallium	2	ND	ND
Vanadium	30	2.1 – 38	9 – 10
Zinc	5,000	0 - 59	0 – 17

* Chromium in groundwater will be presented as total chromium per the WI ch. NR 140 standard

Free phase hydrocarbons (tar) encountered in the Kreher Park fill, ravine fill, NSPW property and Copper Falls aquifer are behaving as a source for the dissolved phase plumes identified in each unit at the Site. PRGs for free-phase tar are within these units are based on WAC NR 708.13, which states the following:

[4] Samples collected from well MW-11 located outside the ravine fill represents background conditions for shallow groundwater in the upper bluff area, and samples collected from MW-6A represent background conditions for the underlying Copper Falls aquifer.

Responsible parties shall conduct free product removal whenever it is necessary to halt or contain the discharge of a hazardous substance or to minimize the harmful effects of the discharge to the air, lands or waters of the state. When required, free product removal shall be conducted, to the maximum extent practicable, in compliance with all of the following requirements:

(1) Free product removal shall be conducted in a manner that minimizes the spread of contamination into previously uncontaminated zones using recovery and disposal techniques appropriate to the hydrologic conditions at the site or facility, and that properly reuses or treats discharges of recovery byproducts in compliance with applicable state and federal laws.

(2) Free product removal systems shall be designed to abate free product migration.

(3) Any flammable products shall be handled in a safe and competent manner to prevent fires or explosions.

Using the above criteria, the removal of free-product (tar) will be further refined in subsequent technical memoranda and the FS.

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TABLES

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Table 1
Soil Sample Locations Used to Evaluate the Residential Scenario

Domain[1]	Soil Sampling Location ID (Borhole ID)	Sample ID	Sample Date	Depth [2] (feet)	Average Depth (feet)
Filled Ravine	B-13	928	26-Apr-95	0 – 7	3.5
Filled Ravine	B-13	966	26-Apr-95	0 – 7	3.5
Filled Ravine	B-13	966	26-Apr-95	0 – 7	3.5
Filled Ravine	B-13	966	26-Apr-95	0 – 7	3.5
Filled Ravine	B-23	1076	23-Sep-98	2 – 4	3
Filled Ravine	B-23	1103	23-Sep-98	2 – 4	3
Filled Ravine	B-23	1103	23-Sep-98	2 – 4	3
Filled Ravine	B-23	1103	23-Sep-98	2 – 4	3
Filled Ravine	B-24	1083	23-Sep-98	2 – 4	3
Filled Ravine	B-24	1110	23-Sep-98	2 – 4	3
Filled Ravine	B-24	1110	23-Sep-98	2 – 4	3
Filled Ravine	B-24	1110	23-Sep-98	2 – 4	3
Filled Ravine	B-25	1081	23-Sep-98	4.5 – 6.5	5.5
Filled Ravine	B-25	1107	23-Sep-98	4.5 – 6.5	5.5
Filled Ravine	B-25	1107	23-Sep-98	4.5 – 6.5	5.5
Filled Ravine	B-25	1107	23-Sep-98	4.5 – 6.5	5.5
Filled Ravine	B-25	1107	23-Sep-98	4.5 – 6.5	5.5
Filled Ravine	GP101	NS-SOGP101-2-4-041905	19-Apr-05	2 – 4	3
Filled Ravine	GP101	NS-SOGP101-2-4-041905	19-Apr-05	2 – 4	3
Filled Ravine	GP101	NS-SOGP101-2-4-041905	19-Apr-05	2 – 4	3
Filled Ravine	GP101	NS-SOGP101-6-8-041905	19-Apr-05	6 – 8	7
Filled Ravine	GP101	NS-SOGP101-6-8-041905	19-Apr-05	6 – 8	7
Filled Ravine	GP101	NS-SOGP101-6-8-041905	19-Apr-05	6 – 8	7
Filled Ravine	GP101	NS-SQDup01-0405-041905	19-Apr-05	2 – 4	3
Filled Ravine	GP101	NS-SQDup01-0405-041905	19-Apr-05	2 – 4	3
Filled Ravine	GP101	NS-SQDup01-0405-041905	19-Apr-05	2 – 4	3
Filled Ravine	GP103	NS-SOGP103-2-4-042705	27-Apr-05	2 – 4	3
Filled Ravine	GP103	NS-SOGP103-2-4-042705	27-Apr-05	2 – 4	3
Filled Ravine	GP103	NS-SOGP103-2-4-042705	27-Apr-05	2 – 4	3
Filled Ravine	GP103	NS-SOGP103-6-8-042705	27-Apr-05	6 – 8	7
Filled Ravine	GP103	NS-SOGP103-6-8-042705	27-Apr-05	6 – 8	7
Filled Ravine	GP103	NS-SOGP103-6-8-042705	27-Apr-05	6 – 8	7
Filled Ravine	GP104	NS-SOGP104-2-4-041905	19-Apr-05	2 – 4	3
Filled Ravine	GP104	NS-SOGP104-2-4-041905	19-Apr-05	2 – 4	3
Filled Ravine	GP104	NS-SOGP104-2-4-041905	19-Apr-05	2 – 4	3
Filled Ravine	GP104	NS-SOGP104-4-6-041905	19-Apr-05	4 – 6	5
Filled Ravine	GP104	NS-SOGP104-4-6-041905	19-Apr-05	4 – 6	5
Filled Ravine	GP104	NS-SOGP104-4-6-041905	19-Apr-05	4 – 6	5
Filled Ravine	GP105	NS-SOGP105-2-4-042005	20-Apr-05	2 – 4	3
Filled Ravine	GP105	NS-SOGP105-2-4-042005	20-Apr-05	2 – 4	3
Filled Ravine	GP105	NS-SOGP105-2-4-042005	20-Apr-05	2 – 4	3
Filled Ravine	GP105	NS-SQDup03-0405-042005	20-Apr-05	2 – 4	3
Filled Ravine	GP105	NS-SQDup03-0405-042005	20-Apr-05	2 – 4	3
Filled Ravine	GP105	NS-SQDup03-0405-042005	20-Apr-05	2 – 4	3
Filled Ravine	GP106	NS-SOGP106-3-4-041905	19-Apr-05	3 – 4	3.5
Filled Ravine	GP106	NS-SOGP106-3-4-041905	19-Apr-05	3 – 4	3.5
Filled Ravine	GP106	NS-SOGP106-3-4-041905	19-Apr-05	3 – 4	3.5
Filled Ravine	GP107	NS-SOGP107-1-3, MS/MSD-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP107	NS-SOGP107-1-3, MS/MSD-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP107	NS-SOGP107-1-3, MS/MSD-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP107	NS-SOGP107-6-8-042005	20-Apr-05	6 – 8	7
Filled Ravine	GP107	NS-SOGP107-6-8-042005	20-Apr-05	6 – 8	7
Filled Ravine	GP107	NS-SOGP107-6-8-042005	20-Apr-05	6 – 8	7
Filled Ravine	GP108	NS-SOGP108-1-3-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP108	NS-SOGP108-1-3-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP108	NS-SOGP108-1-3-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP108	NS-SOGP108-8-10-042005	20-Apr-05	8 – 10	9
Filled Ravine	GP108	NS-SOGP108-8-10-042005	20-Apr-05	8 – 10	9
Filled Ravine	GP108	NS-SOGP108-8-10-042005	20-Apr-05	8 – 10	9
Filled Ravine	GP109	NS-SOGP109-2-4-041905	19-Apr-05	2 – 4	3
Filled Ravine	GP109	NS-SOGP109-2-4-041905	19-Apr-05	2 – 4	3
Filled Ravine	GP109	NS-SOGP109-2-4-041905	19-Apr-05	2 – 4	3

Table 1
Soil Sample Locations Used to Evaluate the Residential Scenario

Domain[1]	Soil Sampling Location ID (Borhole ID)	Sample ID	Sample Date	Depth [2] (feet)	Average Depth (feet)
Filled Ravine	GP109	NS-SOGP109-6-8-041905	19-Apr-05	6 – 8	7
Filled Ravine	GP109	NS-SOGP109-6-8-041905	19-Apr-05	6 – 8	7
Filled Ravine	GP109	NS-SOGP109-6-8-041905	19-Apr-05	6 – 8	7
Filled Ravine	GP110	NS-SOGP110-1-3-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP110	NS-SOGP110-1-3-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP110	NS-SOGP110-1-3-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP110	NS-SOGP110-8-10-042005	20-Apr-05	8 – 10	9
Filled Ravine	GP110	NS-SOGP110-8-10-042005	20-Apr-05	8 – 10	9
Filled Ravine	GP110	NS-SOGP110-8-10-042005	20-Apr-05	8 – 10	9
Filled Ravine	GP111	NS-SOGP111-2-4-042005	20-Apr-05	2 – 4	3
Filled Ravine	GP111	NS-SOGP111-2-4-042005	20-Apr-05	2 – 4	3
Filled Ravine	GP111	NS-SOGP111-2-4-042005	20-Apr-05	2 – 4	3
Filled Ravine	GP111	NS-SOGP111-8-10-042005	20-Apr-05	8 – 10	9
Filled Ravine	GP111	NS-SOGP111-8-10-042005	20-Apr-05	8 – 10	9
Filled Ravine	GP111	NS-SOGP111-8-10-042005	20-Apr-05	8 – 10	9
Filled Ravine	GP112	NS-SOGP112-1-3-041905	19-Apr-05	1 – 3	2
Filled Ravine	GP112	NS-SOGP112-1-3-041905	19-Apr-05	1 – 3	2
Filled Ravine	GP112	NS-SOGP112-1-3-041905	19-Apr-05	1 – 3	2
Filled Ravine	GP112	NS-SOGP112-3-4-041905	19-Apr-05	3 – 4	3.5
Filled Ravine	GP112	NS-SOGP112-3-4-041905	19-Apr-05	3 – 4	3.5
Filled Ravine	GP112	NS-SOGP112-3-4-041905	19-Apr-05	3 – 4	3.5
Filled Ravine	GP113	NS-SOGP113-1-3-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP113	NS-SOGP113-1-3-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP113	NS-SOGP113-1-3-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP113	NS-SOGP113-8-10-042005	20-Apr-05	8 – 10	9
Filled Ravine	GP113	NS-SOGP113-8-10-042005	20-Apr-05	8 – 10	9
Filled Ravine	GP113	NS-SOGP113-8-10-042005	20-Apr-05	8 – 10	9
Filled Ravine	GP113	NS-SQDup02-0405-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP113	NS-SQDup02-0405-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP113	NS-SQDup02-0405-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP114	NS-SOGP114-1-3 MS/MSD-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP114	NS-SOGP114-1-3 MS/MSD-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP114	NS-SOGP114-1-3 MS/MSD-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP114	NS-SOGP114-4-6-042005	20-Apr-05	4 – 6	5
Filled Ravine	GP114	NS-SOGP114-4-6-042005	20-Apr-05	4 – 6	5
Filled Ravine	GP114	NS-SOGP114-4-6-042005	20-Apr-05	4 – 6	5
Filled Ravine	GP115	NS-SOGP115-1-3-042105	21-Apr-05	1 – 3	2
Filled Ravine	GP115	NS-SOGP115-1-3-042105	21-Apr-05	1 – 3	2
Filled Ravine	GP115	NS-SOGP115-1-3-042105	21-Apr-05	1 – 3	2
Filled Ravine	GP115	NS-SOGP115-4-6-042105	21-Apr-05	4 – 6	5
Filled Ravine	GP115	NS-SOGP115-4-6-042105	21-Apr-05	4 – 6	5
Filled Ravine	GP115	NS-SOGP115-4-6-042105	21-Apr-05	4 – 6	5
Filled Ravine	GP116	NS-SOGP116-2-4-042005	20-Apr-05	2 – 4	3
Filled Ravine	GP116	NS-SOGP116-2-4-042005	20-Apr-05	2 – 4	3
Filled Ravine	GP116	NS-SOGP116-2-4-042005	20-Apr-05	2 – 4	3
Filled Ravine	GP116	NS-SOGP116-8-10-042005	20-Apr-05	8 – 10	9
Filled Ravine	GP116	NS-SOGP116-8-10-042005	20-Apr-05	8 – 10	9
Filled Ravine	GP116	NS-SOGP116-8-10-042005	20-Apr-05	8 – 10	9
Filled Ravine	GP117	NS-SOGP117-2-4-042005	20-Apr-05	2 – 4	3
Filled Ravine	GP117	NS-SOGP117-2-4-042005	20-Apr-05	2 – 4	3
Filled Ravine	GP117	NS-SOGP117-2-4-042005	20-Apr-05	2 – 4	3
Filled Ravine	GP117	NS-SOGP117-6-8-042005	20-Apr-05	6 – 8	7
Filled Ravine	GP117	NS-SOGP117-6-8-042005	20-Apr-05	6 – 8	7
Filled Ravine	GP117	NS-SOGP117-6-8-042005	20-Apr-05	6 – 8	7
Filled Ravine	GP118	NS-SOGP118-1-3-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP118	NS-SOGP118-1-3-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP118	NS-SOGP118-1-3-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP118	NS-SOGP118-6-8-042505	25-Apr-05	6 – 8	7
Filled Ravine	GP118	NS-SOGP118-6-8-042505	25-Apr-05	6 – 8	7
Filled Ravine	GP118	NS-SOGP118-6-8-042505	25-Apr-05	6 – 8	7
Filled Ravine	GP119	NS-SOGP119-1-3-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP119	NS-SOGP119-1-3-042505	25-Apr-05	1 – 3	2

Table 1
Soil Sample Locations Used to Evaluate the Residential Scenario

Domain[1]	Soil Sampling Location ID (Borhole ID)	Sample ID	Sample Date	Depth [2] (feet)	Average Depth (feet)
Filled Ravine	GP119	NS-SOGP119-1-3-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP119	NS-SOGP119-4-6-042505	25-Apr-05	4 – 6	5
Filled Ravine	GP119	NS-SOGP119-4-6-042505	25-Apr-05	4 – 6	5
Filled Ravine	GP119	NS-SOGP119-4-6-042505	25-Apr-05	4 – 6	5
Filled Ravine	GP120	NS-SOGP120-0-2-042105	21-Apr-05	0 – 2	1
Filled Ravine	GP120	NS-SOGP120-0-2-042105	21-Apr-05	0 – 2	1
Filled Ravine	GP120	NS-SOGP120-0-2-042105	21-Apr-05	0 – 2	1
Filled Ravine	GP120	NS-SOGP120-4-6 MS/MSD-042105	21-Apr-05	4 – 6	5
Filled Ravine	GP120	NS-SOGP120-4-6 MS/MSD-042105	21-Apr-05	4 – 6	5
Filled Ravine	GP120	NS-SOGP120-4-6 MS/MSD-042105	21-Apr-05	4 – 6	5
Filled Ravine	GP121	NS-SOGP121-1-3 MS/MSD-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP121	NS-SOGP121-1-3 MS/MSD-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP121	NS-SOGP121-1-3 MS/MSD-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP121	NS-SOGP121-4-6-042505	25-Apr-05	4 – 6	5
Filled Ravine	GP121	NS-SOGP121-4-6-042505	25-Apr-05	4 – 6	5
Filled Ravine	GP121	NS-SOGP121-4-6-042505	25-Apr-05	4 – 6	5
Filled Ravine	GP121	NS-SOGP121-6-8-042505	25-Apr-05	6 – 8	7
Filled Ravine	GP121	NS-SOGP121-6-8-042505	25-Apr-05	6 – 8	7
Filled Ravine	GP121	NS-SOGP121-6-8-042505	25-Apr-05	6 – 8	7
Filled Ravine	GP122	NS-SOGP122-1-3-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP122	NS-SOGP122-1-3-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP122	NS-SOGP122-1-3-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP122	NS-SOGP122-6-8-042505	25-Apr-05	6 – 8	7
Filled Ravine	GP122	NS-SOGP122-6-8-042505	25-Apr-05	6 – 8	7
Filled Ravine	GP122	NS-SOGP122-6-8-042505	25-Apr-05	6 – 8	7
Filled Ravine	GP122	NS-SQDup08-0405-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP122	NS-SQDup08-0405-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP122	NS-SQDup08-0405-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP123	NS-SOGP123-1-3-042105	21-Apr-05	1 – 3	2
Filled Ravine	GP123	NS-SOGP123-1-3-042105	21-Apr-05	1 – 3	2
Filled Ravine	GP123	NS-SOGP123-1-3-042105	21-Apr-05	1 – 3	2
Filled Ravine	GP123	NS-SOGP123-6-8-042105	21-Apr-05	6 – 8	7
Filled Ravine	GP123	NS-SOGP123-6-8-042105	21-Apr-05	6 – 8	7
Filled Ravine	GP123	NS-SOGP123-6-8-042105	21-Apr-05	6 – 8	7
Filled Ravine	GP123	NS-SQDup05-0405-042105	21-Apr-05	6 – 8	7
Filled Ravine	GP123	NS-SQDup05-0405-042105	21-Apr-05	6 – 8	7
Filled Ravine	GP123	NS-SQDup05-0405-042105	21-Apr-05	6 – 8	7
Filled Ravine	GP124	NS-SOGP124-1-3-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP124	NS-SOGP124-1-3-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP124	NS-SOGP124-1-3-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP124	NS-SOGP124-6-8-042505	25-Apr-05	6 – 8	7
Filled Ravine	GP124	NS-SOGP124-6-8-042505	25-Apr-05	6 – 8	7
Filled Ravine	GP124	NS-SOGP124-6-8-042505	25-Apr-05	6 – 8	7
Filled Ravine	GP125	NS-SOGP125-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP125	NS-SOGP125-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP125	NS-SOGP125-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP126	NS-SOGP126-1-3-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP126	NS-SOGP126-1-3-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP126	NS-SOGP126-1-3-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP126	NS-SOGP126-6-8-042505	25-Apr-05	6 – 8	7
Filled Ravine	GP126	NS-SOGP126-6-8-042505	25-Apr-05	6 – 8	7
Filled Ravine	GP126	NS-SOGP126-6-8-042505	25-Apr-05	6 – 8	7
Filled Ravine	GP126	NS-SQDup09-0405-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP126	NS-SQDup09-0405-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP126	NS-SQDup09-0405-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP127	NS-SOGP127-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP127	NS-SOGP127-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP127	NS-SOGP127-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP128	NS-SOGP128-1-3-042105	21-Apr-05	1 – 3	2
Filled Ravine	GP128	NS-SOGP128-1-3-042105	21-Apr-05	1 – 3	2
Filled Ravine	GP128	NS-SOGP128-1-3-042105	21-Apr-05	1 – 3	2
Filled Ravine	GP128	NS-SOGP128-4-6-042105	21-Apr-05	4 – 6	5

Table 1
Soil Sample Locations Used to Evaluate the Residential Scenario

Domain[1]	Soil Sampling Location ID (Borhole ID)	Sample ID	Sample Date	Depth [2] (feet)	Average Depth (feet)
Filled Ravine	GP128	NS-SOGP128-4-6-042105	21-Apr-05	4 – 6	5
Filled Ravine	GP128	NS-SOGP128-4-6-042105	21-Apr-05	4 – 6	5
Filled Ravine	GP129	NS-SOGP129-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP129	NS-SOGP129-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP129	NS-SOGP129-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP129	NS-SOGP129-5-7-042605	26-Apr-05	5 – 7	6
Filled Ravine	GP129	NS-SOGP129-5-7-042605	26-Apr-05	5 – 7	6
Filled Ravine	GP129	NS-SOGP129-5-7-042605	26-Apr-05	5 – 7	6
Filled Ravine	GP129	NS-SQDup10-0405-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP129	NS-SQDup10-0405-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP129	NS-SQDup10-0405-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP130	NS-SOGP130-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP130	NS-SOGP130-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP130	NS-SOGP130-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP130	NS-SOGP130-4-6-042605	26-Apr-05	4 – 6	5
Filled Ravine	GP130	NS-SOGP130-4-6-042605	26-Apr-05	4 – 6	5
Filled Ravine	GP130	NS-SOGP130-4-6-042605	26-Apr-05	4 – 6	5
Filled Ravine	GP131	NS-SOGP131-0-2-042105	21-Apr-05	0 – 2	1
Filled Ravine	GP131	NS-SOGP131-0-2-042105	21-Apr-05	0 – 2	1
Filled Ravine	GP131	NS-SOGP131-0-2-042105	21-Apr-05	0 – 2	1
Filled Ravine	GP131	NS-SOGP131-6-8-042105	21-Apr-05	6 – 8	7
Filled Ravine	GP131	NS-SOGP131-6-8-042105	21-Apr-05	6 – 8	7
Filled Ravine	GP131	NS-SOGP131-6-8-042105	21-Apr-05	6 – 8	7
Filled Ravine	GP132	NS-SOGP132-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP132	NS-SOGP132-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP132	NS-SOGP132-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP132	NS-SOGP132-4-6-042605	26-Apr-05	4 – 6	5
Filled Ravine	GP132	NS-SOGP132-4-6-042605	26-Apr-05	4 – 6	5
Filled Ravine	GP132	NS-SOGP132-4-6-042605	26-Apr-05	4 – 6	5
Filled Ravine	GP133	NS-SOGP133-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP133	NS-SOGP133-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP133	NS-SOGP133-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP133	NS-SOGP133-4-6-042605	26-Apr-05	4 – 6	5
Filled Ravine	GP133	NS-SOGP133-4-6-042605	26-Apr-05	4 – 6	5
Filled Ravine	GP133	NS-SOGP133-4-6-042605	26-Apr-05	4 – 6	5
Filled Ravine	GP134	NS-SOGP134-1-3-042105	21-Apr-05	1 – 3	2
Filled Ravine	GP134	NS-SOGP134-1-3-042105	21-Apr-05	1 – 3	2
Filled Ravine	GP134	NS-SOGP134-1-3-042105	21-Apr-05	1 – 3	2
Filled Ravine	GP135	NS-SOGP135-1-3 MS/MSD-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP135	NS-SOGP135-1-3 MS/MSD-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP135	NS-SOGP135-1-3 MS/MSD-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP136	NS-SOGP136-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP136	NS-SOGP136-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP136	NS-SOGP136-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP136	NS-SOGP136-4-6-042605	26-Apr-05	4 – 6	5
Filled Ravine	GP136	NS-SOGP136-4-6-042605	26-Apr-05	4 – 6	5
Filled Ravine	GP136	NS-SOGP136-4-6-042605	26-Apr-05	4 – 6	5
Filled Ravine	GP137	NS-SOGP137-0-2-042105	21-Apr-05	0 – 2	1
Filled Ravine	GP137	NS-SOGP137-0-2-042105	21-Apr-05	0 – 2	1
Filled Ravine	GP137	NS-SOGP137-0-2-042105	21-Apr-05	0 – 2	1
Filled Ravine	GP138	NS-SOGP138-1-3-042105	21-Apr-05	1 – 3	2
Filled Ravine	GP138	NS-SOGP138-1-3-042105	21-Apr-05	1 – 3	2
Filled Ravine	GP138	NS-SOGP138-1-3-042105	21-Apr-05	1 – 3	2
Filled Ravine	GP138	NS-SOGP138-4-6-042105	21-Apr-05	4 – 6	5
Filled Ravine	GP138	NS-SOGP138-4-6-042105	21-Apr-05	4 – 6	5
Filled Ravine	GP138	NS-SOGP138-4-6-042105	21-Apr-05	4 – 6	5
Filled Ravine	GP138	NS-SQDup06-0405-042105	21-Apr-05	4 – 6	5
Filled Ravine	GP138	NS-SQDup06-0405-042105	21-Apr-05	4 – 6	5
Filled Ravine	GP138	NS-SQDup06-0405-042105	21-Apr-05	4 – 6	5
Filled Ravine	MW-12	1079	22-Sep-98	2 – 4	3
Filled Ravine	MW-12	1099	23-Sep-98	2 – 4	3
Filled Ravine	MW-12	1099	23-Sep-98	2 – 4	3

Table 1
Soil Sample Locations Used to Evaluate the Residential Scenario

Domain[1]	Soil Sampling Location ID (Borhole ID)	Sample ID	Sample Date	Depth [2] (feet)	Average Depth (feet)
Filled Ravine	MW-12	1099	23-Sep-98	2 – 4	3
Filled Ravine	MW-9	1080	22-Sep-98	4.5 – 6.5	5.5
Filled Ravine	MW-9	1102	22-Sep-98	4.5 – 6.5	5.5
Filled Ravine	MW-9	1102	22-Sep-98	4.5 – 6.5	5.5
Filled Ravine	MW-9	1102	22-Sep-98	4.5 – 6.5	5.5
Filled Ravine	MW-9	1102	22-Sep-98	4.5 – 6.5	5.5
Filled Ravine	SB-5	1019	21-Dec-94	8 – 9	8.5
Filled Ravine	SB-5	1019	21-Dec-94	8 – 9	8.5
Filled Ravine	SB-5	1019	21-Dec-94	8 – 9	8.5
Filled Ravine	SB-5	972	21-Dec-94	8 – 9	8.5
Filled Ravine	SS24	NS-SS24-0-1-042905	29-Apr-05	0 – 1	0.5
Filled Ravine	SS24	NS-SS24-0-1-042905	29-Apr-05	0 – 1	0.5
Filled Ravine	SS24	NS-SS24-0-1-042905	29-Apr-05	0 – 1	0.5
Upper Bluff	B-26	1086	23-Sep-98	4.5 – 6.5	5.5
Upper Bluff	B-26	1106	23-Sep-98	4.5 – 6.5	5.5
Upper Bluff	B-26	1106	23-Sep-98	4.5 – 6.5	5.5
Upper Bluff	B-26	1106	23-Sep-98	4.5 – 6.5	5.5
Upper Bluff	B-27	1088	23-Sep-98	2 – 4	3
Upper Bluff	B-27	1092	23-Sep-98	7 – 9	8
Upper Bluff	B-27	1113	23-Sep-98	2 – 4	3
Upper Bluff	B-27	1113	23-Sep-98	2 – 4	3
Upper Bluff	B-27	1113	23-Sep-98	2 – 4	3
Upper Bluff	B-27	1115	23-Sep-98	7 – 9	8
Upper Bluff	B-27	1115	23-Sep-98	7 – 9	8
Upper Bluff	B-27	1115	23-Sep-98	7 – 9	8
Upper Bluff	B-28	1090	23-Sep-98	2 – 4	3
Upper Bluff	B-28	1111	23-Sep-98	2 – 4	3
Upper Bluff	B-28	1111	23-Sep-98	2 – 4	3
Upper Bluff	B-28	1111	23-Sep-98	2 – 4	3
Upper Bluff	B-29	1089	22-Sep-98	2 – 4	3
Upper Bluff	B-29	1114	22-Sep-98	2 – 4	3
Upper Bluff	B-29	1114	22-Sep-98	2 – 4	3
Upper Bluff	B-29	1114	22-Sep-98	2 – 4	3
Upper Bluff	B-30	1093	22-Sep-98	2 – 4	3
Upper Bluff	B-30	1118	22-Sep-98	2 – 4	3
Upper Bluff	B-30	1118	22-Sep-98	2 – 4	3

Table 1
Soil Sample Locations Used to Evaluate the Residential Scenario

Domain[1]	Soil Sampling Location ID (Borhole ID)	Sample ID	Sample Date	Depth [2] (feet)	Average Depth (feet)
Upper Bluff	B-30	1118	22-Sep-98	2 – 4	3
Upper Bluff	B-31	1094	23-Sep-98	2 – 4	3
Upper Bluff	B-31	1095	23-Sep-98	7 – 9	8
Upper Bluff	B-31	1117	23-Sep-98	7 – 9	8
Upper Bluff	B-31	1117	23-Sep-98	7 – 9	8
Upper Bluff	B-31	1117	23-Sep-98	7 – 9	8
Upper Bluff	B-31	1122	23-Sep-98	2 – 4	3
Upper Bluff	B-31	1122	23-Sep-98	2 – 4	3
Upper Bluff	B-31	1122	23-Sep-98	2 – 4	3
Upper Bluff	B-31	1122	23-Sep-98	2 – 4	3
Upper Bluff	B-35	1014	24-Aug-98	8 – 10	9
Upper Bluff	B-35	1014	24-Aug-98	8 – 10	9
Upper Bluff	B-35	1014	24-Aug-98	8 – 10	9
Upper Bluff	B-36	1012	23-Aug-98	3 – 5	4
Upper Bluff	B-36	1012	23-Aug-98	3 – 5	4
Upper Bluff	B-36	1012	23-Aug-98	3 – 5	4
Upper Bluff	GP-43	GP-43_6/10/02_(6-8)Grab_NM	10-Jun-02	6 – 8	7
Upper Bluff	GP-43	GP-43_6/10/02_(6-8)Grab_NM	10-Jun-02	6 – 8	7
Upper Bluff	GP-43	GP-43_6/10/02_(6-8)Grab_NM	10-Jun-02	6 – 8	7
Upper Bluff	MW-11	1075	21-Sep-98	7 – 9	8
Upper Bluff	MW-11	1104	21-Sep-98	7 – 9	8
Upper Bluff	MW-11	1104	21-Sep-98	7 – 9	8
Upper Bluff	MW-11	1104	21-Sep-98	7 – 9	8
Upper Bluff	MW-15	1078	21-Sep-98	2 – 4	3
Upper Bluff	MW-15	1101	21-Sep-98	2 – 4	3
Upper Bluff	MW-15	1101	21-Sep-98	2 – 4	3
Upper Bluff	MW-15	1101	21-Sep-98	2 – 4	3
Upper Bluff	SS14	NS-SS14-0-1-060705	07-Jun-05	0 – 1	0.5
Upper Bluff	SS14	NS-SS14-0-1-060705	07-Jun-05	0 – 1	0.5
Upper Bluff	SS14	NS-SS14-0-1-060705	07-Jun-05	0 – 1	0.5
Upper Bluff	SS15	NS-SS15-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS15	NS-SS15-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS15	NS-SS15-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS16	NS-SS16-0-1-061405	14-Jun-05	0 – 1	0.5
Upper Bluff	SS16	NS-SS16-0-1-061405	14-Jun-05	0 – 1	0.5
Upper Bluff	SS16	NS-SS16-0-1-061405	14-Jun-05	0 – 1	0.5
Upper Bluff	SS17	NS-SS17-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS17	NS-SS17-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS17	NS-SS17-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS18	NS-SS18-0-1-061405	14-Jun-05	0 – 1	0.5
Upper Bluff	SS18	NS-SS18-0-1-061405	14-Jun-05	0 – 1	0.5
Upper Bluff	SS18	NS-SS18-0-1-061405	14-Jun-05	0 – 1	0.5
Upper Bluff	SS19	NS-SS19-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS19	NS-SS19-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS19	NS-SS19-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS20	NS-SS20-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS20	NS-SS20-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS20	NS-SS20-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS21	NS-SS21-0-1-060705	07-Jun-05	0 – 1	0.5
Upper Bluff	SS21	NS-SS21-0-1-060705	07-Jun-05	0 – 1	0.5
Upper Bluff	SS21	NS-SS21-0-1-060705	07-Jun-05	0 – 1	0.5

[1] The domain is the associated SWMU or domain the sample falls within. There are 4 domains that were evaluated for the Site. Figure 3 illustrates each domain.

- Chequamegon Bay
- Filled Ravine
- Kreher Park
- Upper Bluff

[2] The residential scenario assumes that soil exposures are limited to depths of 0 to 10 feet below ground surface.

Table 2
Soil Sample Locations Used to Evaluate the Recreational Scenario

Domain[1]	Soil Sampling Location ID (Borhole ID)	Sample ID	Sample Date	Depth [2] (feet)	Average Depth (feet)
Kreher Park	SS-10	922	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-10	982	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-10	982	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-10	982	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS11	NS-SS11-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	SS11	NS-SS11-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	SS11	NS-SS11-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	SS12	NS-SS12-0-1-060705	07-Jun-05	0 – 1	0.5
Kreher Park	SS12	NS-SS12-0-1-060705	07-Jun-05	0 – 1	0.5
Kreher Park	SS12	NS-SS12-0-1-060705	07-Jun-05	0 – 1	0.5
Kreher Park	SS13	NS-SS13-0-1-060705	07-Jun-05	0 – 1	0.5
Kreher Park	SS13	NS-SS13-0-1-060705	07-Jun-05	0 – 1	0.5
Kreher Park	SS13	NS-SS13-0-1-060705	07-Jun-05	0 – 1	0.5
Kreher Park	SS-2	1007	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-2	998	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-2	998	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-2	998	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-3	1008	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-3	999	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-3	999	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-3	999	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-4	1000	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-4	1000	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-4	1000	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-4	1009	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-5	1001	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-5	1001	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-5	1001	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-5	917	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-6	1002	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-6	1002	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-6	1002	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-6	918	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-7	1003	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-7	1003	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-7	1003	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-7	919	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-8	1004	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-8	1004	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-8	1004	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-8	920	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-9	1005	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-9	1005	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-9	1005	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-9	921	06-Nov-97	0 – 0.67	0.335
Kreher Park	TP100	NS-SOTP100-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP100	NS-SOTP100-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP100	NS-SOTP100-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP101	NS-SOTP101-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP101	NS-SOTP101-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP101	NS-SOTP101-0-1-060805	08-Jun-05	0 – 1	0.5

Table 2
Soil Sample Locations Used to Evaluate the Recreational Scenario

Domain[1]	Soil Sampling Location ID (Borhole ID)	Sample ID	Sample Date	Depth [2] (feet)	Average Depth (feet)
Kreher Park	TP102	NS-SOTP102-0-1 (MS/MSD)-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP102	NS-SOTP102-0-1 (MS/MSD)-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP102	NS-SOTP102-0-1 (MS/MSD)-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP103	NS-SOTP103-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP103	NS-SOTP103-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP103	NS-SOTP103-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP104	NS-SOTP104-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP104	NS-SOTP104-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP104	NS-SOTP104-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP105	NS-SOTP105-0-1-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP105	NS-SOTP105-0-1-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP105	NS-SOTP105-0-1-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP105	NS-SQDup02-0605-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP105	NS-SQDup02-0605-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP105	NS-SQDup02-0605-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP106	NS-SOTP106-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP106	NS-SOTP106-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP106	NS-SOTP106-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP107	NS-SOTP107-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP107	NS-SOTP107-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP107	NS-SOTP107-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP108	NS-SOTP108-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP108	NS-SOTP108-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP108	NS-SOTP108-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP108	NS-SQDup01-0605-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP108	NS-SQDup01-0605-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP108	NS-SQDup01-0605-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP109	NS-SOTP109-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP109	NS-SOTP109-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP109	NS-SOTP109-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP110	NS-SOTP110-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP110	NS-SOTP110-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP110	NS-SOTP110-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP111	NS-SOTP111-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP111	NS-SOTP111-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP111	NS-SOTP111-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP112	NS-SOTP112-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP112	NS-SOTP112-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP112	NS-SOTP112-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP113	NS-SOTP113-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP113	NS-SOTP113-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP113	NS-SOTP113-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP113	NS-SQDup03-0605-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP113	NS-SQDup03-0605-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP113	NS-SQDup03-0605-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP114	NS-SOTP114-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP114	NS-SOTP114-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP114	NS-SOTP114-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP115	NS-SOTP115-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP115	NS-SOTP115-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP115	NS-SOTP115-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP116	NS-SOTP116-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP116	NS-SOTP116-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP116	NS-SOTP116-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP117	NS-SOTP117-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP117	NS-SOTP117-0-1-061305	13-Jun-05	0 – 1	0.5

Table 2
Soil Sample Locations Used to Evaluate the Recreational Scenario

Domain[1]	Soil Sampling Location ID (Borhole ID)	Sample ID	Sample Date	Depth [2] (feet)	Average Depth (feet)
Kreher Park	TP117	NS-SOTP117-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP118	NS-SOTP118-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP118	NS-SOTP118-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP118	NS-SOTP118-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP119	NS-SOTP119-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP119	NS-SOTP119-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP119	NS-SOTP119-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP120	NS-SOTP120-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP120	NS-SOTP120-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP120	NS-SOTP120-0-1-061405	14-Jun-05	0 – 1	0.5

[1] The domain is the associated SWMU or domain the sample falls within. There are 4 domains that were evaluated for the Site. Figure 3 illustrates each domain.

- Chequamegon Bay
- Filled Ravine
- Kreher Park
- Upper Bluff

[2] The recreational scenario assumes that soil exposures are non-intrusive and are limited to depths of 0 to 1 feet.

Table 3
Soil Sample Locations Used to Evaluate the Industrial Worker Scenario

Domain[1]	Soil Sampling Location ID (Borhole ID)	Sample ID	Sample Date	Depth [2] (feet)	Average Depth (feet)
Filled Ravine	SS24	NS-SS24-0-1-042905	29-Apr-05	0 – 1	0.5
Filled Ravine	SS24	NS-SS24-0-1-042905	29-Apr-05	0 – 1	0.5
Upper Bluff	SS14	NS-SS14-0-1-060705	07-Jun-05	0 – 1	0.5
Upper Bluff	SS14	NS-SS14-0-1-060705	07-Jun-05	0 – 1	0.5
Upper Bluff	SS14	NS-SS14-0-1-060705	07-Jun-05	0 – 1	0.5
Upper Bluff	SS15	NS-SS15-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS15	NS-SS15-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS15	NS-SS15-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS16	NS-SS16-0-1-061405	14-Jun-05	0 – 1	0.5
Upper Bluff	SS16	NS-SS16-0-1-061405	14-Jun-05	0 – 1	0.5
Upper Bluff	SS16	NS-SS16-0-1-061405	14-Jun-05	0 – 1	0.5
Upper Bluff	SS17	NS-SS17-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS17	NS-SS17-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS17	NS-SS17-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS18	NS-SS18-0-1-061405	14-Jun-05	0 – 1	0.5
Upper Bluff	SS18	NS-SS18-0-1-061405	14-Jun-05	0 – 1	0.5
Upper Bluff	SS18	NS-SS18-0-1-061405	14-Jun-05	0 – 1	0.5
Upper Bluff	SS19	NS-SS19-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS19	NS-SS19-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS19	NS-SS19-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS20	NS-SS20-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS20	NS-SS20-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS20	NS-SS20-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS21	NS-SS21-0-1-060705	07-Jun-05	0 – 1	0.5
Upper Bluff	SS21	NS-SS21-0-1-060705	07-Jun-05	0 – 1	0.5
Upper Bluff	SS21	NS-SS21-0-1-060705	07-Jun-05	0 – 1	0.5

[1] The domain is the associated SWMU or domain the sample falls within. There are 4 domains that were evaluated for the Site. Figure 3 illustrates each domain.

- Chequamegon Bay
- Filled Ravine
- Kreher Park
- Upper Bluff

[2] The industrial worker scenario assumes that soil exposures are non-intrusive and are limited to depths of 0 to 1 feet.

Table 4
Soil Sample Locations Used to Evaluate the Maintenance Worker Scenario

Domain[1]	Soil Sampling Location ID (Borhole ID)	Sample ID	Sample Date	Depth [2] (feet)	Average Depth (feet)
Filled Ravine	SS24	NS-SS24-0-1-042905	29-Apr-05	0 – 1	0.5
Filled Ravine	SS24	NS-SS24-0-1-042905	29-Apr-05	0 – 1	0.5
Kreher Park	SS-10	922	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-10	982	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-10	982	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-10	982	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS11	NS-SS11-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	SS11	NS-SS11-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	SS11	NS-SS11-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	SS12	NS-SS12-0-1-060705	07-Jun-05	0 – 1	0.5
Kreher Park	SS12	NS-SS12-0-1-060705	07-Jun-05	0 – 1	0.5
Kreher Park	SS12	NS-SS12-0-1-060705	07-Jun-05	0 – 1	0.5
Kreher Park	SS13	NS-SS13-0-1-060705	07-Jun-05	0 – 1	0.5
Kreher Park	SS13	NS-SS13-0-1-060705	07-Jun-05	0 – 1	0.5
Kreher Park	SS13	NS-SS13-0-1-060705	07-Jun-05	0 – 1	0.5
Kreher Park	SS-2	1007	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-2	998	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-2	998	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-2	998	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-3	1008	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-3	999	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-3	999	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-3	999	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-4	1000	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-4	1000	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-4	1000	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-4	1009	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-5	1001	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-5	1001	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-5	1001	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-5	917	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-6	1002	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-6	1002	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-6	1002	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-6	918	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-7	1003	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-7	1003	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-7	1003	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-7	919	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-8	1004	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-8	1004	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-8	1004	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-8	920	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-9	1005	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-9	1005	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-9	1005	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-9	921	06-Nov-97	0 – 0.67	0.335
Kreher Park	TP100	NS-SOTP100-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP100	NS-SOTP100-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP100	NS-SOTP100-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP101	NS-SOTP101-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP101	NS-SOTP101-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP101	NS-SOTP101-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP102	NS-SOTP102-0-1 (MS/MSD)-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP102	NS-SOTP102-0-1 (MS/MSD)-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP102	NS-SOTP102-0-1 (MS/MSD)-060905	09-Jun-05	0 – 1	0.5

Table 4
Soil Sample Locations Used to Evaluate the Maintenance Worker Scenario

Domain[1]	Soil Sampling Location ID (Borhole ID)	Sample ID	Sample Date	Depth [2] (feet)	Average Depth (feet)
Kreher Park	TP103	NS-SOTP103-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP103	NS-SOTP103-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP103	NS-SOTP103-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP104	NS-SOTP104-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP104	NS-SOTP104-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP104	NS-SOTP104-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP105	NS-SOTP105-0-1-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP105	NS-SOTP105-0-1-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP105	NS-SOTP105-0-1-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP105	NS-SQDup02-0605-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP105	NS-SQDup02-0605-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP105	NS-SQDup02-0605-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP106	NS-SOTP106-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP106	NS-SOTP106-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP106	NS-SOTP106-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP107	NS-SOTP107-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP107	NS-SOTP107-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP107	NS-SOTP107-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP108	NS-SOTP108-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP108	NS-SOTP108-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP108	NS-SOTP108-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP108	NS-SQDup01-0605-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP108	NS-SQDup01-0605-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP108	NS-SQDup01-0605-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP109	NS-SOTP109-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP109	NS-SOTP109-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP109	NS-SOTP109-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP110	NS-SOTP110-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP110	NS-SOTP110-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP110	NS-SOTP110-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP111	NS-SOTP111-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP111	NS-SOTP111-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP111	NS-SOTP111-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP112	NS-SOTP112-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP112	NS-SOTP112-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP112	NS-SOTP112-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP113	NS-SOTP113-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP113	NS-SOTP113-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP113	NS-SOTP113-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP113	NS-SQDup03-0605-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP113	NS-SQDup03-0605-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP113	NS-SQDup03-0605-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP114	NS-SOTP114-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP114	NS-SOTP114-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP114	NS-SOTP114-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP115	NS-SOTP115-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP115	NS-SOTP115-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP115	NS-SOTP115-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP116	NS-SOTP116-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP116	NS-SOTP116-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP116	NS-SOTP116-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP117	NS-SOTP117-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP117	NS-SOTP117-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP117	NS-SOTP117-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP118	NS-SOTP118-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP118	NS-SOTP118-0-1-061305	13-Jun-05	0 – 1	0.5

Table 4
Soil Sample Locations Used to Evaluate the Maintenance Worker Scenario

Domain[1]	Soil Sampling Location ID (Borhole ID)	Sample ID	Sample Date	Depth [2] (feet)	Average Depth (feet)
Kreher Park	TP118	NS-SOTP118-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP119	NS-SOTP119-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP119	NS-SOTP119-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP119	NS-SOTP119-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP120	NS-SOTP120-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP120	NS-SOTP120-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP120	NS-SOTP120-0-1-061405	14-Jun-05	0 – 1	0.5
Upper Bluff	SS14	NS-SS14-0-1-060705	07-Jun-05	0 – 1	0.5
Upper Bluff	SS14	NS-SS14-0-1-060705	07-Jun-05	0 – 1	0.5
Upper Bluff	SS14	NS-SS14-0-1-060705	07-Jun-05	0 – 1	0.5
Upper Bluff	SS15	NS-SS15-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS15	NS-SS15-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS15	NS-SS15-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS16	NS-SS16-0-1-061405	14-Jun-05	0 – 1	0.5
Upper Bluff	SS16	NS-SS16-0-1-061405	14-Jun-05	0 – 1	0.5
Upper Bluff	SS16	NS-SS16-0-1-061405	14-Jun-05	0 – 1	0.5
Upper Bluff	SS17	NS-SS17-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS17	NS-SS17-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS17	NS-SS17-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS18	NS-SS18-0-1-061405	14-Jun-05	0 – 1	0.5
Upper Bluff	SS18	NS-SS18-0-1-061405	14-Jun-05	0 – 1	0.5
Upper Bluff	SS18	NS-SS18-0-1-061405	14-Jun-05	0 – 1	0.5
Upper Bluff	SS19	NS-SS19-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS19	NS-SS19-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS19	NS-SS19-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS20	NS-SS20-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS20	NS-SS20-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS20	NS-SS20-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS21	NS-SS21-0-1-060705	07-Jun-05	0 – 1	0.5
Upper Bluff	SS21	NS-SS21-0-1-060705	07-Jun-05	0 – 1	0.5
Upper Bluff	SS21	NS-SS21-0-1-060705	07-Jun-05	0 – 1	0.5

[1] The domain is the associated SWMU or domain the sample falls within. There are 4 domains that were evaluated for the Site. Figure 3 illustrates each domain.

- Chequamegon Bay
- Filled Ravine
- Kreher Park
- Upper Bluff

[2] The maintenance worker scenario assumes that soil exposures are non-intrusive and are limited to depths of 0 to 1 feet.

Table 5
Soil Sample Locations Used to Evaluate the Construction Scenario

Domain[1]	Soil Sampling Location ID (Borhole ID)	Sample ID	Sample Date	Depth [2] (feet)	Average Depth (feet)
Filled Ravine	B-13	928	26-Apr-95	0 – 7	3.5
Filled Ravine	B-13	966	26-Apr-95	0 – 7	3.5
Filled Ravine	B-23	1076	23-Sep-98	2 – 4	3
Filled Ravine	B-23	1103	23-Sep-98	2 – 4	3
Filled Ravine	B-24	1083	23-Sep-98	2 – 4	3
Filled Ravine	B-24	1110	23-Sep-98	2 – 4	3
Filled Ravine	B-25	1081	23-Sep-98	4.5 – 6.5	5.5
Filled Ravine	B-25	1107	23-Sep-98	4.5 – 6.5	5.5
Filled Ravine	B-25	1107	23-Sep-98	4.5 – 6.5	5.5
Filled Ravine	GP101	NS-SOGP101-2-4-041905	19-Apr-05	2 – 4	3
Filled Ravine	GP101	NS-SOGP101-6-8-041905	19-Apr-05	6 – 8	7
Filled Ravine	GP101	NS-SQDup01-0405-041905	19-Apr-05	2 – 4	3
Filled Ravine	GP103	NS-SOGP103-2-4-042705	27-Apr-05	2 – 4	3
Filled Ravine	GP103	NS-SOGP103-6-8-042705	27-Apr-05	6 – 8	7
Filled Ravine	GP104	NS-SOGP104-2-4-041905	19-Apr-05	2 – 4	3
Filled Ravine	GP104	NS-SOGP104-4-6-041905	19-Apr-05	4 – 6	5
Filled Ravine	GP105	NS-SOGP105-2-4-042005	20-Apr-05	2 – 4	3
Filled Ravine	GP105	NS-SQDup03-0405-042005	20-Apr-05	2 – 4	3
Filled Ravine	GP106	NS-SOGP106-3-4-041905	19-Apr-05	3 – 4	3.5
Filled Ravine	GP107	NS-SOGP107-1-3, MS/MSD-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP107	NS-SOGP107-6-8-042005	20-Apr-05	6 – 8	7
Filled Ravine	GP108	NS-SOGP108-1-3-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP108	NS-SOGP108-8-10-042005	20-Apr-05	8 – 10	9
Filled Ravine	GP109	NS-SOGP109-2-4-041905	19-Apr-05	2 – 4	3
Filled Ravine	GP109	NS-SOGP109-6-8-041905	19-Apr-05	6 – 8	7
Filled Ravine	GP110	NS-SOGP110-1-3-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP110	NS-SOGP110-8-10-042005	20-Apr-05	8 – 10	9
Filled Ravine	GP111	NS-SOGP111-2-4-042005	20-Apr-05	2 – 4	3
Filled Ravine	GP111	NS-SOGP111-8-10-042005	20-Apr-05	8 – 10	9
Filled Ravine	GP112	NS-SOGP112-1-3-041905	19-Apr-05	1 – 3	2
Filled Ravine	GP112	NS-SOGP112-3-4-041905	19-Apr-05	3 – 4	3.5
Filled Ravine	GP113	NS-SOGP113-1-3-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP113	NS-SOGP113-8-10-042005	20-Apr-05	8 – 10	9
Filled Ravine	GP113	NS-SQDup02-0405-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP114	NS-SOGP114-1-3 MS/MSD-042005	20-Apr-05	1 – 3	2
Filled Ravine	GP114	NS-SOGP114-4-6-042005	20-Apr-05	4 – 6	5
Filled Ravine	GP115	NS-SOGP115-1-3-042105	21-Apr-05	1 – 3	2
Filled Ravine	GP115	NS-SOGP115-4-6-042105	21-Apr-05	4 – 6	5
Filled Ravine	GP116	NS-SOGP116-2-4-042005	20-Apr-05	2 – 4	3
Filled Ravine	GP116	NS-SOGP116-8-10-042005	20-Apr-05	8 – 10	9
Filled Ravine	GP117	NS-SOGP117-2-4-042005	20-Apr-05	2 – 4	3
Filled Ravine	GP117	NS-SOGP117-6-8-042005	20-Apr-05	6 – 8	7
Filled Ravine	GP118	NS-SOGP118-1-3-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP118	NS-SOGP118-6-8-042505	25-Apr-05	6 – 8	7
Filled Ravine	GP119	NS-SOGP119-1-3-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP119	NS-SOGP119-4-6-042505	25-Apr-05	4 – 6	5
Filled Ravine	GP120	NS-SOGP120-0-2-042105	21-Apr-05	0 – 2	1
Filled Ravine	GP120	NS-SOGP120-4-6 MS/MSD-042105	21-Apr-05	4 – 6	5
Filled Ravine	GP121	NS-SOGP121-1-3 MS/MSD-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP121	NS-SOGP121-4-6-042505	25-Apr-05	4 – 6	5
Filled Ravine	GP121	NS-SOGP121-6-8-042505	25-Apr-05	6 – 8	7
Filled Ravine	GP122	NS-SOGP122-1-3-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP122	NS-SOGP122-6-8-042505	25-Apr-05	6 – 8	7
Filled Ravine	GP122	NS-SQDup08-0405-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP123	NS-SOGP123-1-3-042105	21-Apr-05	1 – 3	2
Filled Ravine	GP123	NS-SOGP123-6-8-042105	21-Apr-05	6 – 8	7
Filled Ravine	GP123	NS-SQDup05-0405-042105	21-Apr-05	6 – 8	7

Table 5
Soil Sample Locations Used to Evaluate the Construction Scenario

Domain[1]	Soil Sampling Location ID (Borhole ID)	Sample ID	Sample Date	Depth [2] (feet)	Average Depth (feet)
Filled Ravine	GP124	NS-SOGP124-1-3-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP124	NS-SOGP124-6-8-042505	25-Apr-05	6 – 8	7
Filled Ravine	GP125	NS-SOGP125-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP126	NS-SOGP126-1-3-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP126	NS-SOGP126-6-8-042505	25-Apr-05	6 – 8	7
Filled Ravine	GP126	NS-SQDup09-0405-042505	25-Apr-05	1 – 3	2
Filled Ravine	GP127	NS-SOGP127-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP128	NS-SOGP128-1-3-042105	21-Apr-05	1 – 3	2
Filled Ravine	GP128	NS-SOGP128-4-6-042105	21-Apr-05	4 – 6	5
Filled Ravine	GP129	NS-SOGP129-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP129	NS-SOGP129-5-7-042605	26-Apr-05	5 – 7	6
Filled Ravine	GP129	NS-SQDup10-0405-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP130	NS-SOGP130-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP130	NS-SOGP130-4-6-042605	26-Apr-05	4 – 6	5
Filled Ravine	GP131	NS-SOGP131-0-2-042105	21-Apr-05	0 – 2	1
Filled Ravine	GP131	NS-SOGP131-6-8-042105	21-Apr-05	6 – 8	7
Filled Ravine	GP132	NS-SOGP132-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP132	NS-SOGP132-4-6-042605	26-Apr-05	4 – 6	5
Filled Ravine	GP133	NS-SOGP133-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP133	NS-SOGP133-4-6-042605	26-Apr-05	4 – 6	5
Filled Ravine	GP134	NS-SOGP134-1-3-042105	21-Apr-05	1 – 3	2
Filled Ravine	GP135	NS-SOGP135-1-3 MS/MSD-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP136	NS-SOGP136-1-3-042605	26-Apr-05	1 – 3	2
Filled Ravine	GP136	NS-SOGP136-4-6-042605	26-Apr-05	4 – 6	5
Filled Ravine	GP137	NS-SOGP137-0-2-042105	21-Apr-05	0 – 2	1
Filled Ravine	GP138	NS-SOGP138-1-3-042105	21-Apr-05	1 – 3	2
Filled Ravine	GP138	NS-SOGP138-4-6-042105	21-Apr-05	4 – 6	5
Filled Ravine	GP138	NS-SQDup06-0405-042105	21-Apr-05	4 – 6	5
Filled Ravine	MW-12	1079	22-Sep-98	2 – 4	3
Filled Ravine	MW-12	1099	23-Sep-98	2 – 4	3
Filled Ravine	MW-9	1080	22-Sep-98	4.5 – 6.5	5.5
Filled Ravine	MW-9	1102	22-Sep-98	4.5 – 6.5	5.5
Filled Ravine	MW-9	1102	22-Sep-98	4.5 – 6.5	5.5
Filled Ravine	SB-5	1019	21-Dec-94	8 – 9	8.5
Filled Ravine	SB-5	972	21-Dec-94	8 – 9	8.5
Filled Ravine	SS24	NS-SS24-0-1-042905	29-Apr-05	0 – 1	0.5
Kreher Park	B-32	1097	22-Sep-98	4.5 – 6.5	5.5
Kreher Park	B-32	1119	22-Sep-98	4.5 – 6.5	5.5
Kreher Park	GP139	NS-SOGP139-0-2-042205	22-Apr-05	0 – 2	1
Kreher Park	GP140	NS-SOGP140-1-3-042205	22-Apr-05	1 – 3	2
Kreher Park	GP141	NS-SOGP141-0-2-042205	22-Apr-05	0 – 2	1
Kreher Park	GP142	NS-SOGP142-0-2-042205	22-Apr-05	0 – 2	1
Kreher Park	GP143	NS-SOGP143-0-2-042205	22-Apr-05	0 – 2	1
Kreher Park	GP143	NS-SOGP143-6-8-042205	22-Apr-05	6 – 8	7
Kreher Park	GP144	NS-SOGP144-4-6-042805	28-Apr-05	4 – 6	5
Kreher Park	GP145	NS-SOGP145-2-4-042805	28-Apr-05	2 – 4	3
Kreher Park	GP145	NS-SQDup13-0405-042805	28-Apr-05	2 – 4	3
Kreher Park	GP146	NS-SOGP146-0-3-042805	28-Apr-05	0 – 3	1.5
Kreher Park	GP147	NS-SOGP147-2-4-042705	27-Apr-05	2 – 4	3
Kreher Park	GP148	NS-SOGP148-1-3-042705	27-Apr-05	1 – 3	2
Kreher Park	GP148	NS-SQDup12-0405-042705	27-Apr-05	1 – 3	2
Kreher Park	GP149	NS-SOGP149-1-3 MS/MSD-042805	28-Apr-05	1 – 3	2
Kreher Park	GP150	NS-SOGP150-0-2-042805	28-Apr-05	0 – 2	1
Kreher Park	GP151	NS-SOGP151-1-3-042705	27-Apr-05	1 – 3	2
Kreher Park	GP152	NS-SOGP152-2-4-042705	27-Apr-05	2 – 4	3
Kreher Park	GP152	NS-SQDup11-0405-042705	27-Apr-05	2 – 4	3
Kreher Park	GP153	NS-SOGP153-1-3-042705	27-Apr-05	1 – 3	2

Table 5
Soil Sample Locations Used to Evaluate the Construction Scenario

Domain[1]	Soil Sampling Location ID (Borhole ID)	Sample ID	Sample Date	Depth [2] (feet)	Average Depth (feet)
Kreher Park	GP154	NS-SOGP154-2-4-042705	27-Apr-05	2 – 4	3
Kreher Park	GP155	NS-SOGP155-1-3-042805	28-Apr-05	1 – 3	2
Kreher Park	GP155	NS-SOGP155-5-7-042805	28-Apr-05	5 – 7	6
Kreher Park	GP156	NS-SOGP156-0-2-042805	28-Apr-05	0 – 2	1
Kreher Park	GP156	NS-SOGP156-4-6-042805	28-Apr-05	4 – 6	5
Kreher Park	GP157	NS-SOGP157-1-3-042705	27-Apr-05	1 – 3	2
Kreher Park	GP158	NS-SOGP158-0-2-042705	27-Apr-05	0 – 2	1
Kreher Park	HP-1	923	10-Dec-97	0 – 4	2
Kreher Park	HP-1	983	10-Dec-97	0 – 4	2
Kreher Park	HP-13	909	10-Dec-97	0 – 4	2
Kreher Park	HP-13	988	10-Dec-97	0 – 4	2
Kreher Park	HP-14	910	10-Dec-97	0 – 4	2
Kreher Park	HP-14	989	10-Dec-97	0 – 4	2
Kreher Park	HP-15	911	10-Dec-97	0 – 4	2
Kreher Park	HP-15	990	10-Dec-97	0 – 4	2
Kreher Park	HP-16	912	10-Dec-97	0 – 4	2
Kreher Park	HP-16	991	10-Dec-97	0 – 4	2
Kreher Park	HP-17	913	10-Dec-97	0 – 4	2
Kreher Park	HP-17	992	10-Dec-97	0 – 4	2
Kreher Park	HP-18	914	11-Dec-97	0 – 4	2
Kreher Park	HP-18	993	11-Dec-97	0 – 4	2
Kreher Park	HP-19	915	11-Dec-97	0 – 4	2
Kreher Park	HP-19	994	11-Dec-97	0 – 4	2
Kreher Park	HP-2	924	10-Dec-97	0 – 4	2
Kreher Park	HP-2	984	10-Dec-97	0 – 4	2
Kreher Park	HP-20	916	11-Dec-97	0 – 4	2
Kreher Park	HP-20	995	11-Dec-97	0 – 4	2
Kreher Park	HP-21	926	11-Dec-97	0 – 4	2
Kreher Park	HP-21	996	11-Dec-97	0 – 4	2
Kreher Park	HP-3	925	11-Dec-97	0 – 4	2
Kreher Park	HP-3	985	11-Dec-97	0 – 4	2
Kreher Park	HP-4	907	11-Dec-97	0 – 2	1
Kreher Park	HP-4	986	11-Dec-97	0 – 2	1
Kreher Park	HP-7	908	11-Dec-97	0 – 2	1
Kreher Park	HP-7	987	11-Dec-97	0 – 2	1
Kreher Park	SS-10	922	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-10	982	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS11	NS-SS11-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	SS12	NS-SS12-0-1-060705	07-Jun-05	0 – 1	0.5
Kreher Park	SS13	NS-SS13-0-1-060705	07-Jun-05	0 – 1	0.5
Kreher Park	SS-2	1007	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-2	998	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-3	1008	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-3	999	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-4	1000	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-4	1009	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-5	1001	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-5	917	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-6	1002	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-6	918	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-7	1003	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-7	919	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-8	1004	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-8	920	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-9	1005	06-Nov-97	0 – 0.67	0.335
Kreher Park	SS-9	921	06-Nov-97	0 – 0.67	0.335
Kreher Park	TP-1	1030	07-Sep-94	5 – 7	6

Table 5
Soil Sample Locations Used to Evaluate the Construction Scenario

Domain[1]	Soil Sampling Location ID (Borhole ID)	Sample ID	Sample Date	Depth [2] (feet)	Average Depth (feet)
Kreher Park	TP-1	1123	07-Sep-94	5 – 7	6
Kreher Park	TP-1	973	07-Sep-94	5 – 7	6
Kreher Park	TP100	NS-SOTP100-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP100	NS-SOTP100-5	08-Jun-05	4.5 – 5	4.75
Kreher Park	TP100	NS-SOTP100-5-AD	08-Jun-05	4.5 – 5	4.75
Kreher Park	TP101	NS-SOTP101-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP102	NS-SOTP102-0-1 (MS/MSD)-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP103	NS-GWTP103-0605	08-Jun-05	6.5 – 7	6.75
Kreher Park	TP103	NS-SOTP103-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP104	NS-SOTP104-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP105	NS-SOTP105-0-1-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP105	NS-SQDup02-0605-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP106	NS-SOTP106-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP106	NS-SOTP106-6	07-Jun-05	5.5 – 6	5.75
Kreher Park	TP107	NS-GWTP107-0605	08-Jun-05	7.5 – 8	7.75
Kreher Park	TP107	NS-SOTP107-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP107	NS-SOTP107-8	08-Jun-05	7.5 – 8	7.75
Kreher Park	TP107	NS-SOTP107-8-AD	08-Jun-05	7.5 – 8	7.75
Kreher Park	TP108	NS-SOTP108-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP108	NS-SOTP108-PIPE	07-Jun-05	4.5 – 5	4.75
Kreher Park	TP108	NS-SQDup01-0605-060905	09-Jun-05	0 – 1	0.5
Kreher Park	TP109	NS-SOTP109-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP109	NS-SOTP109-2	07-Jun-05	2.5 – 3	2.75
Kreher Park	TP109	NS-SOTP109-2-AD	07-Jun-05	2.5 – 3	2.75
Kreher Park	TP110	NS-GWTP110-0605	09-Jun-05	3.5 – 4	3.75
Kreher Park	TP110	NS-SOTP110-0-1-060805	08-Jun-05	0 – 1	0.5
Kreher Park	TP110	NS-SOTP110-4	09-Jun-05	3.5 – 4	3.75
Kreher Park	TP111	NS-SOTP111-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP111	NS-SOTP111-6	09-Jun-05	5.5 – 6	5.75
Kreher Park	TP112	NS-GWTP112-0605	15-Jun-05	4.5 – 5	4.75
Kreher Park	TP112	NS-SOTP112-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP112	NS-SOTP112-5	15-Jun-05	4.5 – 5	4.75
Kreher Park	TP112	NS-SOTP112-5-AD	15-Jun-05	4.5 – 5	4.75
Kreher Park	TP113	NS-SOTP113-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP113	NS-SOTP113-4	15-Jun-05	3.5 – 4	3.75
Kreher Park	TP113	NS-SQDup03-0605-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP114	NS-SOTP114-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP115	NS-SOTP115-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP115	NS-SOTP115-4	14-Jun-05	3.5 – 4	3.75
Kreher Park	TP115	NS-SOTP115-4-AD	14-Jun-05	3.5 – 4	3.75
Kreher Park	TP116	NS-SOTP116-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP116	NS-SOTP116-3	14-Jun-05	2.5 – 3	2.75
Kreher Park	TP117	NS-GWTP117-0605	13-Jun-05	4.5 – 5	4.75
Kreher Park	TP117	NS-SOTP117-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP118	NS-GWTP118-0605	13-Jun-05	3.5 – 4	3.75
Kreher Park	TP118	NS-SOTP118-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP118	NS-SOTP118-3	13-Jun-05	3.5 – 4	3.75
Kreher Park	TP119	NS-SOTP119-0-1-061305	13-Jun-05	0 – 1	0.5
Kreher Park	TP119	NS-SOTP119-5	14-Jun-05	4.5 – 5	4.75
Kreher Park	TP120	NS-SOTP120-0-1-061405	14-Jun-05	0 – 1	0.5
Kreher Park	TP124A	NSP-SO-TP124A-PIPE	02-Nov-05	4 – 4.5	4.25
Kreher Park	TP128	NSP-SO-TP128-PIPE	02-Nov-05	2 – 2.5	2.25
Kreher Park	TP128	NSP-SO-TP128-PIPE-AD	02-Nov-05	2 – 2.5	2.25
Kreher Park	TP130	NSP-SO-TP130-PIPE	02-Nov-05	4 – 4.5	4.25
Kreher Park	TP130	NSP-SO-TP130-PIPE-AD	02-Nov-05	4 – 4.5	4.25
Kreher Park	TP-2	1029	07-Sep-94	2 – 4	3
Kreher Park	TP-2	1061	07-Sep-94	6 – 8	7

Table 5
Soil Sample Locations Used to Evaluate the Construction Scenario

Domain[1]	Soil Sampling Location ID (Borhole ID)	Sample ID	Sample Date	Depth [2] (feet)	Average Depth (feet)
Kreher Park	TP-2	1062	07-Sep-94	2 – 4	3
Kreher Park	TP-2	930	07-Sep-94	2 – 4	3
Kreher Park	TP-2	931	07-Sep-94	6 – 8	7
Kreher Park	TP-2	974	07-Sep-94	6 – 8	7
Kreher Park	TP-3	1027	07-Sep-94	2 – 4	3
Kreher Park	TP-3	1048	07-Sep-94	2 – 4	3
Kreher Park	TP-3	932	07-Sep-94	2 – 4	3
Kreher Park	TP-4	1040	07-Sep-94	4 – 6	5
Kreher Park	TP-4	1059	07-Sep-94	4 – 6	5
Kreher Park	TP-4	933	07-Sep-94	4 – 6	5
Kreher Park	TP-5	1025	07-Sep-94	6 – 8	7
Kreher Park	TP-5	1072	07-Sep-94	6 – 8	7
Kreher Park	TP-5	934	07-Sep-94	6 – 8	7
Kreher Park	TP-6	1024	07-Sep-94	5 – 7	6
Kreher Park	TP-6	1050	07-Sep-94	5 – 7	6
Kreher Park	TP-6	935	07-Sep-94	5 – 7	6
Kreher Park	TP-7	1023	07-Sep-94	2 – 4	3
Kreher Park	TP-7	1054	07-Sep-94	2 – 4	3
Kreher Park	TP-7	936	07-Sep-94	2 – 4	3
Kreher Park	TP-8	1043	07-Sep-94	5 – 7	6
Kreher Park	TP-8	1060	07-Sep-94	5 – 7	6
Kreher Park	TP-8	937	07-Sep-94	5 – 7	6
Kreher Park	TP-9	1042	07-Sep-94	2 – 4	3
Kreher Park	TP-9	1070	07-Sep-94	2 – 4	3
Kreher Park	TP-9	938	07-Sep-94	2 – 4	3
Kreher Park	TW-1	1044	06-Sep-94	4.5 – 6.5	5.5
Kreher Park	TW-1	1068	06-Sep-94	4.5 – 6.5	5.5
Kreher Park	TW-1	939	06-Sep-94	4.5 – 6.5	5.5
Kreher Park	TW-10	1036	08-Sep-94	4.5 – 6.5	5.5
Kreher Park	TW-10	1071	08-Sep-94	4.5 – 6.5	5.5
Kreher Park	TW-10	941	08-Sep-94	4.5 – 6.5	5.5
Kreher Park	TW-12	1033	08-Sep-94	4.5 – 6.5	5.5
Kreher Park	TW-12	1066	08-Sep-94	4.5 – 6.5	5.5
Kreher Park	TW-12	944	08-Sep-94	4.5 – 6.5	5.5
Kreher Park	TW-3	1028	06-Sep-94	7 – 9	8
Kreher Park	TW-3	1056	06-Sep-94	7 – 9	8
Kreher Park	TW-3	949	06-Sep-94	7 – 9	8
Kreher Park	TW-4	1046	07-Sep-94	7 – 9	8
Kreher Park	TW-4	950	07-Sep-94	7 – 9	8
Kreher Park	TW-4	977	07-Sep-94	7 – 9	8
Kreher Park	TW-6	1058	07-Sep-94	7 – 9	8
Kreher Park	TW-6	954	07-Sep-94	7 – 9	8
Kreher Park	TW-6	980	07-Sep-94	7 – 9	8
Kreher Park	TW-8	1038	07-Sep-94	4.5 – 6.5	5.5
Kreher Park	TW-8	1045	07-Sep-94	4.5 – 6.5	5.5
Kreher Park	TW-8	958	07-Sep-94	4.5 – 6.5	5.5
Upper Bluff	B-26	1086	23-Sep-98	4.5 – 6.5	5.5
Upper Bluff	B-26	1106	23-Sep-98	4.5 – 6.5	5.5
Upper Bluff	B-27	1088	23-Sep-98	2 – 4	3
Upper Bluff	B-27	1092	23-Sep-98	7 – 9	8
Upper Bluff	B-27	1113	23-Sep-98	2 – 4	3
Upper Bluff	B-27	1115	23-Sep-98	7 – 9	8
Upper Bluff	B-28	1090	23-Sep-98	2 – 4	3
Upper Bluff	B-28	1111	23-Sep-98	2 – 4	3
Upper Bluff	B-29	1089	22-Sep-98	2 – 4	3
Upper Bluff	B-29	1114	22-Sep-98	2 – 4	3
Upper Bluff	B-30	1093	22-Sep-98	2 – 4	3

Table 5
Soil Sample Locations Used to Evaluate the Construction Scenario

Domain[1]	Soil Sampling Location ID (Borhole ID)	Sample ID	Sample Date	Depth[2] (feet)	Average Depth (feet)
Upper Bluff	B-30	1118	22-Sep-98	2 – 4	3
Upper Bluff	B-31	1094	23-Sep-98	2 – 4	3
Upper Bluff	B-31	1095	23-Sep-98	7 – 9	8
Upper Bluff	B-31	1117	23-Sep-98	7 – 9	8
Upper Bluff	B-31	1122	23-Sep-98	2 – 4	3
Upper Bluff	B-31	1122	23-Sep-98	2 – 4	3
Upper Bluff	B-35	1014	24-Aug-98	8 – 10	9
Upper Bluff	B-36	1012	23-Aug-98	3 – 5	4
Upper Bluff	GP-43	GP-43_6/10/02_(6-8)Grab_NM	10-Jun-02	6 – 8	7
Upper Bluff	MW-11	1075	21-Sep-98	7 – 9	8
Upper Bluff	MW-11	1104	21-Sep-98	7 – 9	8
Upper Bluff	MW-15	1078	21-Sep-98	2 – 4	3
Upper Bluff	MW-15	1101	21-Sep-98	2 – 4	3
Upper Bluff	SS14	NS-SS14-0-1-060705	07-Jun-05	0 – 1	0.5
Upper Bluff	SS15	NS-SS15-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS16	NS-SS16-0-1-061405	14-Jun-05	0 – 1	0.5
Upper Bluff	SS17	NS-SS17-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS18	NS-SS18-0-1-061405	14-Jun-05	0 – 1	0.5
Upper Bluff	SS19	NS-SS19-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS20	NS-SS20-0-1-060805	08-Jun-05	0 – 1	0.5
Upper Bluff	SS21	NS-SS21-0-1-060705	07-Jun-05	0 – 1	0.5

[1] *The domain is the associated SWMU or domain the sample falls within. There are 4 domains that were evaluated for the Site. Figure 3* illustrates each domain.

- Chequamegon Bay
- Filled Ravine
- Kreher Park
- Upper Bluff

[2] The construction scenario assumes that soil exposures are limited to depths of 0 to 10 feet below ground surface

Table 6
Sediment Sample Locations Used to Evaluate the Recreational Scenario

Sediment Sampling Location ID	Domain[1]	Sample ID	Surface[2] Elevation	Sample Date	Sample Depth (feet)
2200N-1600E	Chequamegon Bay	732	601.00	3/5/2001	0 – 2
2250N-1400E	Chequamegon Bay	2250N-1400E_3/26/03_(0-0.5)Grab_NM	598.11	3/26/2003	0 – 0.5
2300N-1600E	Chequamegon Bay	741	596.18	3/5/2001	0 – 2
2300N-1700E	Chequamegon Bay	738	597.35	3/5/2001	0 – 2
2350N-1400E	Chequamegon Bay	2350N-1400E_3/26/03_(0-0.5)Grab_NM	596.22	3/26/2003	0 – 0.5
2400N-1200E	Chequamegon Bay	731	597.61	3/2/2001	0 – 2
2400N-1300E	Chequamegon Bay	736	596.52	3/2/2001	0 – 2
2400N-2000E	Chequamegon Bay	743	598.43	3/7/2001	0 – 2
2400N-2100E	Chequamegon Bay	354	598.01	1/15/1998	0 – 0.5
2400N-2100E	Chequamegon Bay	712	598.01	3/7/2001	0 – 2
2400N-2200E	Chequamegon Bay	727	598.41	3/7/2001	0 – 2
2400N-2300E	Chequamegon Bay	737	599.11	3/7/2001	0 – 2
2500N-2300E	Chequamegon Bay	2500N-2300E_3/26/03_(0-0.5)Grab_NM	596.60	3/26/2003	0 – 0.5
2600N-2400E	Chequamegon Bay	687	596.77	3/8/2001	0 – 2

[1] The domain is the associated SWMU or domain the sample falls within. There are 4 domains that were evaluated for the Site. Figure 3 illustrates each domain.

- Chequamegon Bay
- Filled Ravine
- Kreher Park
- Upper Bluff

[2] Surveyed elevation of land surface at or near sample. The Lake Superior water level is approximately 600 feet.

Table 7
Surface Water Sample Locations Used to Evaluate the Recreational Scenario

Surface Water Sample Location ID	Surface Water Sample ID	Sample Date	Start Depth	End Depth	Total Depth	Reference[1] Elevation (feet)	Comment
HHRA01	NSP-SW-HHRA01-0605-UNF-061805	6/18/2005	1.7	1.7	2.4	601.5	Low Energy Event
HHRA01	NSP-SW-WQ-DUP03-0605-UNF-061805	6/18/2005	1.7	1.7	3.7	601.5	Low Energy Event
HHRA02	NSP-SW-HHRA02-0605-UNF-061805	6/18/2005	1.35	1.35	4.6	601.5	Low Energy Event
HHRA03	NSP-SW-HHRA03-0605-UNF-061805	6/18/2005	1.2	1.2	5	601.5	Low Energy Event
HHRA04	NSP-SW-HHRA04-0605-UNF-061805	6/18/2005	1.85	1.85	3.4	601.5	Low Energy Event
HHRA04	NSP-SW-WQ-DUP04-0605-UNF-061805	6/18/2005	1.85	1.85	3.1	601.5	Low Energy Event
HHRA05	NSP-SW-HHRA05-0605-UNF-061805	6/18/2005	2.3	2.3	7.6	601.5	Low Energy Event
HHRA06	NSP-SW-HHRA06-0605-UNF-061805	6/18/2005	2.5	2.5	8.8	601.5	Low Energy Event
HHRA07	NSP-SW-HHRA07-0605-UNF-061805	6/18/2005	1.7	1.7	3.4	601.5	Low Energy Event
HHRA08	NSP-SW-HHRA08-0605-UNF-061805	6/18/2005	1.55	1.55	3.7	601.5	Low Energy Event
SW-4 2600N-2100E[2]	SW-4	1/15/1998	8	8	–	601.5	Low Energy Event
SW-7 2400N-2100E[2]	SW-7	1/15/1998	4	4	–	601.5	Low Energy Event
SW-8 2500N-1800E[2]	SW-8	1/15/1998	6	6	–	601.5	Low Energy Event
SW-9 2400N-1400E[2]	SW-9	5/14/1998	7	7	–	601.5	Low Energy Event
HHRA01	NSP-SW-HHRA1-1105-UNF-111405	11/14/2005	1.5	1.5	3	601.5	High Energy Event
HHRA02	NSP-SW-DUP02-1105-UNF-111405	11/14/2005	1.4	1.4	2.7	601.5	High Energy Event
HHRA02	NSP-SW-HHRA2-1105-UNF-111405	11/14/2005	1.4	1.4	2.7	601.5	High Energy Event
HHRA03	NSP-SW-HHRA3-1105-UNF-MSMSD-111505	11/15/2005	1.3	1.3	2.5	601.5	High Energy Event
HHRA04	NSP-SW-DUP04-1105-UNF-111505	11/15/2005	1.6	1.6	3.8	601.5	High Energy Event
HHRA04	NSP-SW-HHRA4-1105-UNF-111505	11/15/2005	1.6	1.6	3.8	601.5	High Energy Event
HHRA05	NSP-SW-HHRA5-1105-UNF-111505	11/15/2005	2.2	2.2	4.3	601.5	High Energy Event
HHRA06	NSP-SW-HHRA6-1105-UNF-111505	11/15/2005	2.5	2.5	5	601.5	High Energy Event
HHRA07	NSP-SW-HHRA7-1105-UNF-111505	11/15/2005	2	2	4	601.5	High Energy Event
HHRA08	NSP-SW-HHRA8-1105-UNF-111505	11/15/2005	1.7	1.7	3.3	601.5	High Energy Event

[1] Reference elevation is the water body elevation at time of sampling (in ft-msl).
[2] These surface water samples were collected through lake ice during a prolonged period of low energy within the water column. These samples may not represent chemical concentrations within water during and after period of heavy wave action.

Table 8
Air Sample Locations Used to Evaluate the Residential and Industrial Worker Scenario

Sample ID	Vapor or Air Location ID	Collection Method	Comment
Soil Gas Samples for Residential Scenario			
NS-GSVP05-0305	VP-05	Summa	
NS-GSVP06-0305	VP-06	Summa	
NS-GSVP07-0305	VP-07	Summa	
Indoor Air Samples for Industrial Worker Scenario			
NS-GSINDOOR-0405	Indoor	Summa	Indoor 24 hour air sample
NS-GSINDOOR-0705	Indoor	Summa	Indoor 24 hour air sample
Ambient Air Samples for Industrial Worker Scenario			
NS-GSUPWIND-0305	Upwind	Summa	Off-site ambient air
NS-GSUPWIND-0405	Upwind	Summa	Off-site ambient air
NS-GSUPWIND-0705	Upwind	Summa	Off-site ambient air

Table 9
Fish Tissue Samples Used to Evaluate the Fisher Scenario

Fish Species		
Walleye (*Stizostedion vitreum*)[a]	**Shorthead Redhorse** (*Moxostoma macrolepidotum*)[b]	**Smelt** (*Osmerus mordax*)[c]
	Sample IDs	
SVITREUM-F-1-1	MMACROLEPIDOTUM-F-1-1	NS-TA-OMORDAX-F-2-1
SVITREUM-F-1-2	MMACROLEPIDOTUM-F-1-2	NS-TA-OMORDAX-F-2-2
SVITREUM-F-1-3	MMACROLEPIDOTUM-F-1-3	NS-TA-OMORDAX-F-2-3
SVITREUM-F-1-4	MMACROLEPIDOTUM-F-1-4	NS-TA-OMORDAX-F-2-4
SVITREUM-F-1-5	MMACROLEPIDOTUM-F-1-5	NS-TA-OMORDAX-F-2
SVITREUM-F-1-6	MMACROLEPIDOTUM-F-1-6	NS-TA-OMORDAX-F-1-1
SVITREUM-F-1-7	MMACROLEPIDOTUM-F-1-7	NS-TA-OMORDAX-F-1-2

All fish were collected from Chequamegon Bay.

a – Walleye were filleted (the skin was removed).
b – Shorthead redhorse were processed as for smoking or pickling, i.e. only the head and entrails were removed.
c – Whole fish composite samples of smelt were collected from Chequamegon Bay and prepared as if for frying, i.e. their heads and entrails removed.

Table 10
Selection of Chemicals of Potential Concern
Residential Receptors Exposed to Surface Soil

Analyte	Detection Range (mg/kg)	Nondetect Range (mg/kg)	Detection Frequency	Region 9 Residential Soil PRG (mg/kg)	Basis	Exceeds?	Notes
Inorganics							
Antimony	1.2 - 2	0.41 - 1.3	9 / 86	3.13E+00	nc	No	
Arsenic	0.87 - 23.4	0.325 - 2.165	87 / 103	3.90E-01	ca	***Yes***	
Beryllium	0.071 - 7.5	0.065 - 0.465	81 / 95	1.54E+01	nc	No	
Cadmium	0.29 - 0.63	0.09 - 0.195	14 / 95	3.70E+00	nc	No	
Chromium (VI)	0.091 - 2.4	0.07 - 0.105	42 / 95	3.01E+01	ca	No	
Cyanide	0.097 - 32	0.0175 - 0.615	43 / 113	1.22E+02	nc	No	
Lead	6 - 4000	1.95 - 3.15	82 / 103	4.00E+01	nc	***Yes***	
Mercury	0.083 - 0.84	0.034 - 0.055	17 / 95	2.35E+00	nc	No	
Selenium	1.2 - 2.7	0.38 - 1.2	11 / 95	3.91E+01	nc	No	
Silver	0.87 - 3.5	0.175 - 0.385	2 / 95	3.91E+01	nc	No	
Thallium	1.5 - 1.8	0.485 - 1.05	2 / 86	5.16E-01	nc	***Yes***	
Zinc	5.5 - 402	0.95 - 3.65	107 / 112	2.35E+03	nc	No	
SVOCs							
1-Methylnaphthalene	0.051 - 3800	0.016 - 0.8	38 / 86	5.59E+01	surr	***Yes***	a
2,4-Dimethylphenol	8.2 - 8.2	0.00011 - 23	1 / 15	1.22E+02	nc	No	
2-Methylnaphthalene	0.0076 - 3300	0.0155 - 0.8	47 / 102	5.59E+01	surr	***Yes***	a
Acenaphthene	0.00059 - 670	0.016 - 19.5	33 / 101	3.68E+02	nc	***Yes***	
Acenaphthylene	0.084 - 910	0.00011 - 30.5	41 / 110	3.68E+03	surr	No	b
Anthracene	0.00047 - 750	0.019 - 19.5	39 / 110	2.19E+03	nc	No	
Benzo(a)anthracene	0.044 - 520	0.00011 - 34	50 / 102	6.21E-01	ca	***Yes***	
Benzo(a)pyrene	0.06 - 340	0.00011 - 34	50 / 104	6.21E-02	ca	***Yes***	
Benzo(b)fluoranthene	0.07 - 410	0.00011 - 37.5	50 / 102	6.21E-01	ca	***Yes***	
Benzo(e)pyrene	0.059 - 260	0.019 - 38	38 / 95	–	–	No	
Benzo(g,h,i)perylene	0.061 - 190	0.00011 - 32.5	36 / 111	2.32E+03	surr	No	c
Benzo(k)fluoranthene	0.045 - 170	0.00011 - 38	35 / 102	6.21E+00	ca	***Yes***	
Chrysene	0.059 - 470	0.00011 - 35.5	47 / 102	6.21E+01	ca	***Yes***	
Dibenzo(a,h)anthracene	0.12 - 3.8	0.00011 - 42.5	6 / 102	6.21E-02	ca	***Yes***	
Dibenzofuran	0.079 - 280	0.016 - 31.5	14 / 86	1.45E+01	nc	***Yes***	
Fluoranthene	0.047 - 1400	0.00011 - 1.5	57 / 103	2.29E+02	nc	***Yes***	
Fluorene	0.0015 - 1200	0.016 - 19.5	34 / 101	2.75E+02	nc	***Yes***	
Indeno(1,2,3-cd)pyrene	0.037 - 100	0.00011 - 39	32 / 102	6.21E-01	ca	***Yes***	
Naphthalene	0.00024 - 2900	0.0065 - 0.75	62 / 123	5.59E+00	nc	***Yes***	
o-Cresol	0.05 - 0.055	0.00011 - 32.5	2 / 110	3.06E+02	nc	No	
Phenanthrene	0.0054 - 3700	0.01 - 19.5	60 / 104	2.19E+03	nc	***Yes***	
Phenol	1.2 - 1.2	0.00011 - 35	1 / 110	1.83E+03	nc	No	
Pyrene	0.048 - 2000	0.00011 - 0.355	64 / 104	2.32E+02	nc	***Yes***	
VOCs							
1,2,3-Trimethylbenzene	0.037 - 37	0.0095 - 0.19	25 / 95	5.16E+01	surr	No	d
1,2,4-Trichlorobenzene	0.0097 - 120	0.05 - 25	7 / 16	6.22E+00	nc	***Yes***	
1,2,4-Trimethylbenzene	0.02 - 100	0.0075 - 0.0365	43 / 87	5.16E+00	nc	***Yes***	
1,3,5-Trimethylbenzene	0.0079 - 560	0.008 - 12.5	37 / 103	2.13E+00	nc	***Yes***	
Benzene	0.016 - 230	0.0025 - 3.8	49 / 104	6.43E-01	ca	***Yes***	
Ethylbenzene	0.014 - 170	0.0025 - 0.0385	40 / 98	3.95E+02	sat	No	
Isopropylbenzene	0.095 - 4.3	0.0135 - 25	4 / 16	5.72E+01	nc	No	
m & p-Xylenes	0.03 - 150	0.0125 - 1.25	40 / 96	2.71E+02	sat	No	
n-Butyl benzene	0.0094 - 17	0.0135 - 50	5 / 17	2.40E+02	sat	No	
n-Propyl benzene	0.13 - 14	0.0135 - 25	4 / 16	2.40E+02	sat	No	
o-Xylene	0.017 - 78	0.0065 - 0.032	45 / 95	2.71E+02	surr	No	e
p-Isopropyltoluene	0.072 - 1.8	0.025 - 25	5 / 16	5.70E+02	surr	No	f
sec-Butyl benzene	0.041 - 65	0.0095 - 50	9 / 111	2.20E+02	sat	No	
Styrene	1.3 - 13	0.0085 - 4.3	2 / 95	1.70E+03	sat	No	

Table 10
Selection of Chemicals of Potential Concern
Residential Receptors Exposed to Surface Soil

Analyte	Detection Range (mg/kg)	Nondetect Range (mg/kg)	Detection Frequency	Region 9 Residential Soil PRG (mg/kg)	Basis	Exceeds?	Notes
Toluene	0.014 - 320	0.0025 - 2.65	85 / 113	5.20E+02	sat	No	
Xylenes (total)	0.046 - 320	0.0025 - 75	7 / 18	2.71E+01	nc	***Yes***	

Notes:

All units are milligrams per kilogram (mg/kg).

All non-detects are presented as one-half the detection limit.

PRG – Preliminary Remediation Goal

nc – PRG based on noncarcinogenic (systemic) effects

ca – PRG based on carcinogenic effects

sat – PRG based on the soil saturation concentration

SVOC – Semi-volatile organic compound

VOC – Volatile organic compound

surr – No toxicity information is available for this chemical. The value presented is a surrogate value selected based on structural similarities. The surrogates used are defined below.

a – Naphthalene used as a surrogate for 1-methylnaphthalene and 2-methylnaphthalene.

b – Acenaphthene used as a surrogate for acenaphthylene.

c – Pyrene used as a surrogate for benzo(g,h,i)perylene.

d – 1,2,4-Trimethyl benzene used as a surrogate for 1,2,3-trimethylbenzene.

e – Isopropylbenzene used as a surrogate for p-isopropyltoluene.

f – Total xylenes used as a surrogate for m-, o-, & p-xylenes.

Table 11
Selection of Chemicals of Potential Concern
Recreational Receptors Exposed to Surface Soil

Analyte	Detection Range (mg/kg)	Nondetect Range (mg/kg)	Detection Frequency	Region 9 Residential Soil PRG (mg/kg)	Basis	Exceeds?	Notes
Inorganics							
Aluminum	870 - 14000	-	24 / 24	7.61E+03	nc	*Yes*	
Antimony	1.4 - 2.1	0.6 - 1.05	3 / 24	3.13E+00	nc	No	
Arsenic	1.3 - 13.9	0.47 - 0.7	30 / 33	3.90E-01	ca	*Yes*	
Barium	10 - 420	-	24 / 24	5.37E+02	nc	No	
Beryllium	0.1 - 8.3	1.7 - 1.7	23 / 24	1.54E+01	nc	No	
Cadmium	0.145 - 12	0.095 - 0.15	18 / 33	3.70E+00	nc	*Yes*	
Chromium	2.6 - 29	-	33 / 33	2.11E+02	ca	No	
Chromium (III)	2.6 - 29	-	24 / 24	1.00E+05	max	No	
Chromium (VI)	0.11 - 0.65	0.08 - 0.095	20 / 24	3.01E+01	ca	No	
Cobalt	0.91 - 12	-	24 / 24	9.03E+02	ca	No	
Copper	2.2 - 120	-	33 / 33	3.13E+02	nc	No	
Cyanide	0.055 - 1.4	0.05 - 0.06	11 / 25	1.22E+02	nc	No	
Iron	4100 - 94000	-	33 / 33	2.35E+03	nc	*Yes*	
Lead	5.2 - 950	2.25 - 2.25	32 / 33	4.00E+01	nc	*Yes*	
Manganese	100 - 670	-	24 / 24	1.76E+02	nc	*Yes*	
Mercury	0.13 - 0.8	0.01 - 0.044	14 / 33	2.35E+00	nc	No	
Nickel	2 - 33	-	24 / 24	1.56E+02	nc	No	
Selenium	1.4 - 2.1	0.55 - 1.2	3 / 24	3.91E+01	nc	No	
Silver	0.72 - 7.6	0.19 - 0.375	6 / 24	3.91E+01	nc	No	
Thallium	2.4 - 2.4	0.7 - 1.25	1 / 24	5.16E-01	nc	*Yes*	
Vanadium	8.9 - 63	-	24 / 24	7.82E+00	nc	*Yes*	
Zinc	4.3 - 4100	-	33 / 33	2.35E+03	nc	*Yes*	
SVOCs							
Acenaphthene	0.49 - 0.49	0.016 - 3.25	1 / 24	3.68E+02	nc	No	
Acenaphthylene	0.19 - 27	0.0155 - 0.75	7 / 25	3.68E+02	surr	No	a
Anthracene	0.68 - 1.6	0.019 - 3.85	2 / 24	2.19E+03	nc	No	
Benzo(a)anthracene	0.24 - 25	0.017 - 0.85	15 / 27	6.21E-01	ca	*Yes*	
Benzo(a)pyrene	0.29 - 68	0.01 - 0.85	16 / 33	6.21E-02	ca	*Yes*	
Benzo(b)fluoranthene	0.33 - 53	0.019 - 0.95	16 / 27	6.21E-01	ca	*Yes*	
Benzo(e)pyrene	0.25 - 49	0.019 - 0.95	11 / 24	-	-	No	
Benzo(g,h,i)perylene	0.165 - 57	0.0165 - 0.8	10 / 27	2.32E+02	surr	No	b
Benzo(k)fluoranthene	0.19 - 15	0.019 - 0.95	5 / 24	6.21E+00	ca	*Yes*	
Chrysene	0.27 - 32	0.018 - 0.9	14 / 27	6.21E+01	ca	No	
Fluoranthene	0.25 - 25	0.0185 - 0.95	16 / 27	2.29E+02	nc	No	
Fluorene	0.57 - 0.57	0.016 - 3.25	1 / 24	2.75E+02	nc	No	
Indeno(1,2,3-cd)pyrene	0.155 - 33	0.0155 - 0.8	9 / 27	6.21E-01	ca	*Yes*	
Naphthalene	0.04 - 2.144	0.01 - 3.05	6 / 33	5.59E+00	nc	No	
Phenanthrene	0.41 - 6.8	0.01 - 3.65	6 / 33	2.19E+03	surr	No	c
Pyrene	0.22 - 52	0.01 - 0.9	17 / 33	2.32E+02	nc	No	
VOCs							
1,2,4-Trichlorobenzene	0.035 - 0.302	-	3 / 3	6.22E+00	nc	No	
1,2,4-Trimethylbenzene	0.019 - 0.019	0.0075 - 0.0075	1 / 24	5.16E+00	nc	No	
1,3,5-Trimethylbenzene	0.077 - 0.114	0.008 - 0.008	2 / 26	2.13E+00	nc	No	
Benzene	0.023 - 0.495	0.0025 - 0.0075	3 / 33	6.43E-01	ca	No	
Ethylbenzene	0.025 - 0.301	0.0025 - 0.0075	4 / 33	3.95E+02	sat	No	
m & p-Xylenes	0.034 - 0.051	0.0125 - 0.0125	3 / 24	2.71E+01	surr	No	d
n-Butyl benzene	0.065 - 0.259	-	3 / 3	2.40E+02	sat	No	

Table 11
Selection of Chemicals of Potential Concern
Recreational Receptors Exposed to Surface Soil

Analyte	Detection Range (mg/kg)	Nondetect Range (mg/kg)	Detection Frequency	Region 9 Residential Soil PRG (mg/kg)	Basis	Exceeds?	Notes
o-Xylene	0.016 - 0.029	0.0065 - 0.0065	3 / 24	2.71E+01	surr	No	d
p-Isopropyltoluene	0.047 - 0.075	-	2 / 2	5.70E+01	surr	No	e
Toluene	0.012 - 0.405	0.0025 - 0.0055	16 / 33	5.20E+02	sat	No	
Trichlorofluoromethane	0.038 - 0.046	-	3 / 3	3.86E+01	nc	No	
Xylenes (total)	0.029 - 1.104	0.0025 - 0.0025	6 / 9	2.71E+01	nc	No	

Notes:

All units are milligrams per kilogram (mg/kg).

All non-detects are presented as one-half the detection limit.

PRG – Preliminary Remediation Goal

nc – PRG based on noncarcinogenic (systemic) effects
ca – PRG based on carcinogenic effects
sat – PRG based on the soil saturation concentration
max – PRG based on a ceiling limit
SVOC – Semi-volatile organic compound
VOC – Volatile organic compound
surr – No toxicity information is available for this chemical. The value presented is a surrogate value selected based on structural similarities. The surrogates used are defined below.
a – Acenaphthene used as a surrogate for acenaphthylene.
b – Pyrene used as a surrogate for benzo(g,h,i)perylene.
c – Anthracene used as a surrogate for phenanthrene.
d – Total xylenes used as a surrogate for m-, o-, &p-xylenes.
e – Isopropylbenzene used as a surrogate for p-isopropyltoluene.

Table 12
Selection of Chemicals of Potential Concern
Industrial Worker Receptors Exposed to Surface Soil

Analyte	Detection Range (mg/kg)	Nondetect Range (mg/kg)	Detection Frequency	Region 9 Industrial Soil PRG (mg/kg)	Basis	Exceeds?	Notes
Inorganics							
Antimony	1.8 - 1.9	0.5 - 0.75	2 / 12	4.09E+01	nc	No	
Arsenic	1.4 - 8.5	0.55 - 0.55	11 / 12	1.59E+00	ca	***Yes***	
Cadmium	0.36 - 0.62	0.11 - 0.145	4 / 12	4.51E+01	nc	No	
Chromium (VI)	0.16 - 0.33	0.08 - 0.09	8 / 12	6.40E+01	ca	No	
Cyanide	0.12 - 0.67	0.055 - 0.06	6 / 12	1.23E+03	nc	No	
Lead	22 - 440	2.8 - 2.8	11 / 12	8.00E+02	ca	No	
Mercury	0.083 - 0.4	0.034 - 0.0475	2 / 12	3.07E+01	nc	No	
SVOCs							
1-Methylnaphthalene	0.051 - 0.089	0.016 - 0.16	2 / 12	1.88E+02	surr	No	a
2-Methylnaphthalene	0.066 - 0.19	0.0155 - 0.155	3 / 12	1.88E+02	surr	No	a
Benzo(a)anthracene	0.082 - 0.35	0.017 - 0.17	6 / 12	2.11E+00	ca	No	
Benzo(a)pyrene	0.085 - 0.39	0.017 - 0.17	6 / 12	2.11E-01	ca	***Yes***	
Benzo(b)fluoranthene	0.12 - 0.56	0.019 - 0.19	7 / 12	2.11E+00	ca	No	
Benzo(e)pyrene	0.11 - 0.33	0.019 - 0.19	5 / 12	-	-	No	
Benzo(g,h,i)perylene	0.082 - 0.09	0.0165 - 0.165	4 / 12	2.91E+04	surr	No	b
Benzo(k)fluoranthene	0.07 - 0.21	0.019 - 0.19	3 / 12	2.11E+01	ca	No	
Chrysene	0.11 - 0.41	0.018 - 0.18	6 / 12	2.11E+02	ca	No	
Fluoranthene	0.15 - 0.77	0.0185 - 0.185	8 / 12	2.20E+03	nc	No	
Indeno(1,2,3-cd)pyrene	0.05 - 0.05	0.0155 - 0.155	1 / 12	2.11E+00	ca	No	
Naphthalene	0.051 - 0.051	0.0155 - 0.155	1 / 12	1.88E+01	nc	No	
o-Cresol	0.05 - 0.05	0.013 - 0.13	1 / 12	3.08E+03	nc	No	
Phenanthrene	0.1 - 0.62	0.0185 - 0.185	7 / 12	1.00E+05	max	No	
Pyrene	0.16 - 0.77	0.018 - 0.09	9 / 12	2.91E+03	nc	No	
VOCs							
1,2,4-Trimethylbenzene	0.02 - 0.043	0.0075 - 0.0075	2 / 12	1.70E+01	nc	No	
Ethylbenzene	0.02 - 0.02	0.0075 - 0.0075	1 / 12	3.95E+02	sat	No	
m & p-Xylenes	0.034 - 0.065	0.0125 - 0.0125	3 / 12	4.20E+02	sat	No	
o-Xylene	0.018 - 0.06	0.0065 - 0.0065	4 / 12	4.20E+02	surr	No	c
Toluene	0.014 - 2.1	0.0055 - 0.0055	7 / 12	5.20E+02	sat	No	

Notes:

All units are milligrams per kilogram (mg/kg).

PRG – preliminary remediation goal

surr – No toxicity information is available for this chemical. The value presented is a surrogate value selected based on structural similarities. The surrogates used are defined below.

nc – PRG based on noncarcinogenic (systemic) effects

ca – PRG based on carcinogenic effects

sat – PRG based on the soil saturation concentration

max – PRG based on a ceiling limit

a – Naphthalene used as a surrogate for 1-methylnaphthalene and 2-methylnaphthalene.

b – Pyrene used as a surrogate for benzo(g,h,i)perylene.

c – Total xylenes used as a surrogate for m-, o-, &p-xylenes.

Table 13
Selection of Chemicals of Potential Concern
Maintenance Worker Receptors Exposed to Surface Soil

Analyte	Detection Range (mg/kg)	Nondetect Range (mg/kg)	Detection Frequency	Region 9 Industrial Soil PRG (mg/kg)	Basis	Exceeds?	Notes
Inorganics							
Aluminum	870 - 14000	-	33 / 33	1.00E+05	max	No	
Antimony	0.7 - 2.1	0.5 - 1.05	6 / 33	4.09E+01	nc	No	
Arsenic	1.3 - 13.9	0.47 - 0.7	38 / 42	1.59E+00	ca	***Yes***	
Barium	10 - 420	-	33 / 33	6.66E+03	nc	No	
Beryllium	0.1 - 8.3	1.7 - 1.7	32 / 33	1.94E+03	ca	No	
Cadmium	0.145 - 12	0.095 - 0.15	21 / 42	4.51E+01	nc	No	
Chromium	2.6 - 29	-	42 / 42	4.48E+02	ca	No	
Chromium (III)	2.6 - 29	-	33 / 33	1.00E+05	max	No	
Chromium (VI)	0.095 - 0.65	0.08 - 0.09	28 / 33	6.40E+01	ca	No	
Cobalt	0.91 - 12	-	33 / 33	1.92E+03	ca	No	
Copper	2.2 - 120	-	42 / 42	4.09E+03	nc	No	
Cyanide	0.055 - 1.4	0.05 - 0.06	15 / 34	1.23E+03	nc	No	
Iron	4100 - 94000	-	42 / 42	1.00E+05	max	No	
Lead	5.2 - 950	2.25 - 2.8	40 / 42	8.00E+01	nc	***Yes***	
Manganese	100 - 670	-	33 / 33	1.95E+03	nc	No	
Mercury	0.0385 - 0.8	0.01 - 0.044	16 / 42	3.07E+01	nc	No	
Nickel	2 - 33	-	33 / 33	2.04E+03	nc	No	
Selenium	1.4 - 2.1	0.47 - 1.2	3 / 33	5.11E+02	nc	No	
Silver	0.72 - 7.6	0.19 - 0.375	6 / 33	5.11E+02	nc	No	
Thallium	2.4 - 2.4	0.6 - 1.25	1 / 33	6.75E+00	nc	No	
Vanadium	8.9 - 63	-	33 / 33	1.02E+02	nc	No	
Zinc	4.3 - 4100	-	42 / 42	1.00E+05	max	No	
SVOCs							
1-Methylnaphthalene	0.051 - 0.051	0.016 - 3.15	1 / 33	3.08E+03	surr	No	a
2-Methylnaphthalene	0.066 - 0.19	0.0155 - 3.1	2 / 33	3.08E+03	surr	No	a
Acenaphthene	0.49 - 0.49	0.016 - 3.25	1 / 33	2.92E+03	nc	No	
Acenaphthylene	0.19 - 27	0.0155 - 0.75	7 / 34	2.92E+03	nc	No	
Anthracene	0.68 - 1.6	0.019 - 3.85	2 / 33	1.00E+05	max	No	
Benzo(a)anthracene	0.16 - 25	0.017 - 0.85	18 / 36	2.11E+00	ca	***Yes***	
Benzo(a)pyrene	0.17 - 68	0.01 - 0.85	19 / 42	2.11E-01	ca	***Yes***	
Benzo(b)fluoranthene	0.23 - 53	0.019 - 0.95	20 / 36	2.11E+00	ca	***Yes***	
Benzo(e)pyrene	0.14 - 49	0.019 - 0.95	14 / 33	-	-	No	
Benzo(g,h,i)perylene	0.082 - 57	0.0165 - 0.8	12 / 36	3.08E+03	surr	No	b
Benzo(k)fluoranthene	0.07 - 15	0.019 - 0.95	8 / 33	2.11E+01	ca	No	
Chrysene	0.18 - 32	0.018 - 0.9	17 / 36	2.11E+02	ca	No	
Fluoranthene	0.25 - 25	0.0185 - 0.95	21 / 36	2.20E+03	nc	No	
Fluorene	0.57 - 0.57	0.016 - 3.25	1 / 33	2.63E+03	nc	No	
Indeno(1,2,3-cd)pyrene	0.05 - 33	0.0155 - 0.8	10 / 36	2.11E+00	ca	***Yes***	
Naphthalene	0.04 - 2.144	0.01 - 3.05	7 / 42	1.88E+01	nc	No	
o-Cresol	0.05 - 0.05	0.013 - 2.6	1 / 33	3.08E+03	nc	No	
Phenanthrene	0.1 - 6.8	0.01 - 3.65	10 / 42	1.00E+05	surr	No	c
Pyrene	0.22 - 52	0.01 - 0.9	23 / 42	2.91E+03	nc	No	
VOCs							
1,2,4-Trichlorobenzene	0.035 - 0.302	-	3 / 3	2.16E+01	nc	No	
1,2,4-Trimethylbenzene	0.019 - 0.043	0.0075 - 0.0075	3 / 33	1.70E+01	nc	No	
1,3,5-Trimethylbenzene	0.077 - 0.114	0.008 - 0.008	2 / 35	6.97E+00	nc	No	

Table 13
Selection of Chemicals of Potential Concern
Maintenance Worker Receptors Exposed to Surface Soil

Analyte	Detection Range (mg/kg)	Nondetect Range (mg/kg)	Detection Frequency	Region 9 Industrial Soil PRG (mg/kg)	Basis	Exceeds?	Notes
Benzene	0.023 - 0.495	0.0025 - 0.0075	3 / 42	1.41E+00	ca*	No	
Ethylbenzene	0.02 - 0.301	0.0025 - 0.0075	5 / 42	3.95E+02	sat	No	
m & p-Xylenes	0.034 - 0.065	0.0125 - 0.0125	5 / 33	1.41E+00	surr	No	d
n-Butyl benzene	0.065 - 0.259	-	3 / 3	2.40E+02	sat	No	
o-Xylene	0.016 - 0.06	0.0065 - 0.0065	5 / 33	3.95E+02	surr	No	d
p-Isopropyltoluene	0.047 - 0.075	-	2 / 2	1.98E+02	surr	No	e
Toluene	0.012 - 0.405	0.0025 - 0.0055	20 / 42	5.20E+02	sat	No	
Trichlorofluoromethane	0.038 - 0.046	-	3 / 3	2.00E+03	sat	No	
Xylenes (total)	0.029 - 1.104	0.0025 - 0.0025	6 / 9	4.20E+02	sat	No	

Notes:

All units are milligrams per kilogram (mg/kg).
All non-detects are presented as one-half the detection limit.

PRG – Preliminary Remediation Goal

nc – PRG based on noncarcinogenic (systemic) effects
ca – PRG based on carcinogenic effects
sat – PRG based on the soil saturation concentration
max – PRG based on a ceiling limit
SVOC – Semi-volatile organic compound
VOC – Volatile organic compound
surr – No toxicity information is available for this chemical. The value presented is a surrogate value selected based on structural similarities. The surrogates used are defined below.
a – Naphthalene used as a surrogate for 1-methylnaphthalene and 2-methylnaphthalene.
b – Pyrene used as a surrogate for benzo(g,h,i)perylene.
c – Anthracene used as a surrogate for phenanthrene.
c – Total xylenes used as a surrogate for m-, o-, &p-xylenes.
d – Isopropylbenzene used as a surrogate for p-isopropyltoluene.

Table 14
Selection of Chemicals of Potential Concern
Construction Worker Receptors Exposed to Surface and Subsurface Soil

Analyte	Detection Range (mg/kg)	Nondetect Range (mg/kg)	Detection Frequency	Region 9 Industrial Soil PRG (mg/kg)	Basis	Exceeds?	Notes
Inorganics							
Aluminum	750 - 21000	-	124 / 124	1.00E+05	max	No	
Antimony	0.5 - 2.1	0.41 - 1.05	14 / 116	4.09E+01	nc	No	
Arsenic	0.83 - 23.4	0.325 - 2.165	126 / 165	1.59E+00	ca	*Yes*	
Barium	10 - 420	-	126 / 126	6.66E+03	nc	No	
Beryllium	0.071 - 11	0.07 - 1.7	110 / 124	1.94E+03	ca	No	
Cadmium	0.145 - 24.8	0.095 - 0.195	43 / 142	4.51E+01	nc	No	
Chromium	2.4 - 42	-	153 / 153	4.48E+02	ca	No	
Chromium (III)	2.1 - 38	-	124 / 124	1.00E+05	max	No	
Chromium (VI)	0.075 - 2.4	0.07 - 0.105	71 / 124	6.40E+01	ca	No	
Cobalt	0.91 - 14	-	124 / 124	1.92E+03	ca	No	
Copper	2.2 - 190	0.5 - 0.5	151 / 165	4.09E+03	nc	No	
Cyanide	0.055 - 25	0.0175 - 0.615	53 / 144	1.23E+03	nc	No	
Iron	3800 - 94000	-	151 / 151	1.00E+05	max	No	
Lead	3.32 - 4000	0.5 - 3.15	151 / 182	8.00E+01	nc	*Yes*	
Manganese	12 - 2000	-	124 / 124	1.95E+03	nc	*Yes*	
Mercury	0.0239 - 1.1	0.01 - 0.06	34 / 147	3.07E+01	nc	No	
Nickel	1.5 - 52	-	142 / 142	2.04E+03	nc	No	
Selenium	1.4 - 16.5	0.38 - 1.2	14 / 125	5.11E+02	nc	No	
Silver	0.245 - 7.6	0.185 - 0.385	12 / 124	5.11E+02	nc	No	
Thallium	1.5 - 2.4	0.485 - 1.25	3 / 124	6.75E+00	nc	No	
Vanadium	8.3 - 110	-	124 / 124	1.02E+02	nc	*Yes*	
Zinc	4.3 - 4100	0.5 - 3.65	146 / 165	1.00E+05	max	No	
SVOCs							
1-Methylnaphthalene	0.04 - 21000	0.016 - 3.15	63 / 150	1.88E+01	surr	*Yes*	a
2,3,5-Trimethylnaphthalene	0.057 - 1600	-	26 / 26	-		No	
2,4-Dimethylphenol	8.2 - 8.2	0.00011 - 23	1 / 16	1.23E+03	nc	No	
2,6-Dimethylnaphthalene	0.09 - 5600	-	26 / 26	-		No	
2-Methylnaphthalene	0.0076 - 30000	0.0155 - 3.1	85 / 178	1.88E+01	surr	*Yes*	a
Acenaphthene	0.00059 - 8200	0.016 - 19.5	76 / 180	2.92E+03	nc	*Yes*	
Acenaphthylene	0.045 - 9700	0.00011 - 30.5	89 / 180	2.92E+03	surr	*Yes*	b
Anthracene	0.00047 - 4100	0.019 - 19.5	83 / 180	1.00E+05	max	No	
Benzo(a)anthracene	0.0173 - 2400	0.00011 - 34	116 / 193	2.11E+00	ca	*Yes*	
Benzo(a)fluoranthene	0.55 - 640	-	26 / 26	-		No	
Benzo(a)pyrene	0.0219 - 1800	0.00011 - 34	118 / 209	2.11E-01	ca	*Yes*	
Benzo(b)fluoranthene	0.0149 - 1200	0.00011 - 37.5	118 / 192	2.11E+00	ca	*Yes*	
Benzo(b)fluorene	0.34 - 1100	-	26 / 26	-		No	
Benzo(e)pyrene	0.054 - 1200	0.019 - 38	77 / 150	-		No	
Benzo(g,h,i)perylene	0.0099 - 1200	0.00011 - 32.5	93 / 190	2.91E+03	surr	No	c
Benzo(k)fluoranthene	0.00669 - 1500	0.00011 - 38	87 / 185	2.11E+01	ca	*Yes*	
Biphenyl	0.038 - 1700	-	26 / 26	2.33E+03	nc	No	
Chrysene	0.0118 - 2300	0.00011 - 35.5	108 / 188	2.11E+02	ca	*Yes*	
Dibenzo(a,h)anthracene	0.12 - 290	0.00011 - 42.5	37 / 172	2.11E-01	ca	*Yes*	
Dibenzofuran	0.042 - 1400	0.016 - 31.5	41 / 150	1.56E+02	nc	*Yes*	
Fluoranthene	0.047 - 5400	0.00011 - 1.5	126 / 196	2.20E+03	nc	*Yes*	
Fluorene	0.0015 - 4600	0.016 - 19.5	80 / 183	2.63E+03	nc	*Yes*	
Hopane (T19)	0.18 - 6.8	0.15 - 1.85	15 / 26	-	surr	No	d
Indeno(1,2,3-cd)pyrene	0.0113 - 1200	0.00011 - 39	89 / 189	2.11E+00	ca	*Yes*	
m & p-cresols	0.045 - 47	0.0275 - 70	18 / 150	3.08E+04	surr	No	e
Naphthalene	0.00024 - 37000	0.0065 - 3.05	130 / 246	1.88E+01	nc	*Yes*	
o-Cresol	0.0076 - 22	0.00011 - 32.5	20 / 166	3.08E+03	nc	No	
Pentachlorophenol	0.011 - 1.8	0.016 - 1.4	11 / 26	9.00E+00	ca	No	
Phenanthrene	0.0054 - 14000	0.01 - 19.5	119 / 209	1.00E+05	surr	No	f
Phenol	0.022 - 9.8	0.00011 - 35	26 / 166	1.00E+05	max	No	
Pyrene	0.042 - 7600	0.00011 - 0.9	137 / 209	2.91E+03	nc	*Yes*	
Retene	0.16 - 12	0.105 - 1.8	12 / 26	-	surr	No	d
VOCs							
1,2,3-Trimethylbenzene	0.037 - 37	0.0095 - 0.19	23 / 124	1.70E+01	surr	*Yes*	g
1,2,4-Trichlorobenzene	0.0097 - 2994	0.05 - 25	21 / 31	2.16E+01	nc	*Yes*	

Table 14
Selection of Chemicals of Potential Concern
Construction Worker Receptors Exposed to Surface and Subsurface Soil

Analyte	Detection Range (mg/kg)	Nondetect Range (mg/kg)	Detection Frequency	Region 9 Industrial Soil PRG (mg/kg)	Basis	Exceeds?	Notes
1,2,4-Trimethylbenzene	0.019 - 100	0.0075 - 0.0365	47 / 125	1.70E+01	nc	***Yes***	
1,3,5-Trimethylbenzene	0.0079 - 742	0.008 - 12.5	47 / 152	6.97E+00	nc	***Yes***	
Acetone	0.014 - 0.014	-	1 / 1	5.43E+03	nc	No	
Benzene	0.005 - 645	0.0025 - 3.8	68 / 183	1.41E+00	ca	***Yes***	
Ethylbenzene	0.014 - 2973	0.0025 - 0.0385	54 / 167	3.95E+02	sat	***Yes***	
Isopropylbenzene	0.029 - 190	0.0135 - 25	10 / 23	1.98E+02	nc	No	
m & p-Xylenes	0.028 - 150	0.0125 - 1.25	43 / 125	4.20E+02	surr	No	h
Methylene chloride	0.005 - 0.102	-	2 / 2	2.05E+01	ca	No	
n-Butyl benzene	0.0094 - 648	0.0135 - 50	18 / 31	2.40E+02	sat	***Yes***	
n-Propyl benzene	0.053 - 101	0.0135 - 25	11 / 24	2.40E+02	sat	No	
o-Xylene	0.016 - 78	0.0065 - 0.032	48 / 124	4.20E+02	surr	No	h
p-Isopropyltoluene	0.019 - 213	0.025 - 25	20 / 32	1.98E+02	surr	***Yes***	i
sec-Butyl benzene	0.03 - 2688	0.0095 - 50	10 / 143	2.20E+02	sat	***Yes***	
Styrene	0.023 - 13	0.0085 - 4.3	3 / 124	1.70E+03	sat	No	
Toluene	0.0055 - 2007	0.0025 - 2.65	122 / 183	5.20E+02	sat	***Yes***	
Trichlorofluoromethane	0.038 - 0.046	-	3 / 3	2.00E+03	sat	No	
Xylenes (total)	0.029 - 4981	0.0025 - 75	24 / 59	4.20E+02	sat	***Yes***	

Notes:

All units are milligrams per kilogram (mg/kg).

All non-detects are presented as one-half the detection limits.

PRG – Preliminary Remediation Goal

nc – PRG based on noncarcinogenic (systemic) effects
ca – PRG based on carcinogenic effects
sat – PRG based on the soil saturation concentration
max – PRG based on a ceiling limit
SVOC – Semi-volatile organic compound
VOC – Volatile organic compound
surr – No toxicity information is available for this chemical. The value presented is a surrogate value selected based on structural similarities. The surrogates used are defined below.
a – Naphthalene used as a surrogate for 1-methylnaphthalene and 2-methylnaphthalene.
b – Acenaphthene used as a surrogate for acenaphthylene.
c – Pyrene used as a surrogate for benzo(g,h,i)perylene.
d – No risk-based screening values are available for Hopane (T19) and Retene.
e – m-Cresol used as a surrogate for m & p-cresol.
f – Anthracene used as a surrogate for phenanthrene.
g – 1,2,4-Trimethyl benzene used as a surrogate for 1,2,3-trimethylbenzene.
h – Total xylenes used as a surrogate for m-, o-, &p-xylenes.
i – Isopropylbenzene used as a surrogate for p-isopropyltoluene.

Table 15
Selection of Chemicals of Potential Concern
Recreational Receptors Exposed to Chequamegon Bay Sediment

Analyte	Detection Range (mg/kg)	Nondetect Range (mg/kg)	Detection Frequency	Region 9 Residential Soil PRG (mg/kg)	Basis	Exceeds?	Notes
Inorganics							
Aluminum	980 – 1800	–	2 / 2	7.61E+03	nc	No	
Ammonia (as N)	97 – 97	–	1 / 1	No Value			
Antimony	2.3 – 2.3	2 – 2	1 / 2	3.13E+00	nc	No	
Arsenic	3.1 – 10	–	2 / 2	1.59E+00			
Barium	65 – 110	–	2 / 2	5.37E+02	nc	No	
Beryllium	0.15 – 0.37	–	2 / 2	1.54E+01	nc	No	
Cadmium	0.48 – 0.61	–	2 / 2	3.70E+00	nc	No	
Calcium	4400 – 14000	–	2 / 2	Essential Nutrient			
Chromium	5.4 – 5.9	–	2 / 2	2.11E+02	ca	No	
Chromium (III)	5.4 – 5.9	–	2 / 2	2.11E+02	ca	No	
Cobalt	1.9 – 2.5	–	2 / 2	9.03E+02	ca	No	
Copper	33 – 44	–	2 / 2	3.13E+02	nc	No	
Cyanide	0.61 – 0.61	0.65 – 0.65	1 / 2	1.22E+02	nc	No	
Iron	9000 – 27000	–	2 / 2	2.35E+03	nc	**Yes**	
Lead	57 – 200	–	2 / 2	4.00E+02			
Magnesium	960 – 1300	–	2 / 2	Essential Nutrient			
Manganese	110 – 300	–	2 / 2	1.76E+02	nc	**Yes**	
Mercury	0.087 – 0.088	–	2 / 2	2.35E+00	nc	No	
Nickel	5.7 – 6.4	–	2 / 2	1.56E+02	nc	No	
Potassium	200 – 210	–	2 / 2	Essential Nutrient			
Selenium	1.3 – 2	–	2 / 2	3.91E+01	nc	No	
Sodium	210 – 580	–	2 / 2	Essential Nutrient			
Vanadium	9 – 9.7	–	2 / 2	7.82E+00	nc	**Yes**	
Zinc	110 – 140	–	2 / 2	2.35E+03	nc	No	
SVOCs							
1-Methylnaphthalene	0.084 – 19		3 / 3	5.59E+00	surr	**Yes**	a
2-Methylnaphthalene	0.059 – 2380	0.55 – 10	7 / 13	5.59E+00	surr	**Yes**	a
Acenaphthene	0.12 – 833	0.55 – 10	8 / 14	3.68E+02	nc	**Yes**	
Acenaphthylene	0.076 – 75	0.55 – 15.5	4 / 13	3.68E+02	surr	No	b
Anthracene	0.13 – 360	0.55 – 10.5	7 / 14	2.19E+03	nc	No	
Benzo(a)anthracene	0.86 – 150	0.55 – 10.5	8 / 14	6.21E-01	ca	**Yes**	
Benzo(a)pyrene	0.88 – 100	0.55 – 15.5	7 / 14	6.21E-02	ca	**Yes**	
Benzo(b)fluoranthene	0.99 – 50	0.55 – 15.5	5 / 14	6.21E-01	ca	**Yes**	
Benzo(e)pyrene	0.68 – 10	–	3 / 3	No Value	-		
Benzo(g,h,i)perylene	0.79 – 6.2	0.55 – 21	4 / 14	2.32E+02	surr	No	c
Benzo(k)fluoranthene	0.8 – 71	0.55 – 15.5	5 / 14	6.21E+00	ca	**Yes**	
Chrysene	0.96 – 130	0.55 – 10.5	8 / 14	6.21E+01	ca	**Yes**	
Dibenzo(a,h)anthracene	0.27 – 1.9	0.55 – 21	3 / 13	6.21E-02	ca	**Yes**	
Dibenzofuran	0.016 – 1.8	–	3 / 3	1.45E+01	nc	No	
Fluoranthene	1.6 – 330	0.55 – 3.15	4 / 14	2.29E+02	nc	**Yes**	
Fluorene	0.047 – 500	0.55 – 10.5	6 / 13	2.75E+02	nc	**Yes**	
Indeno(1,2,3-cd)pyrene	0.6 – 5.3	0.55 – 21	4 / 13	6.21E-01	ca	**Yes**	
Naphthalene	0.094 – 2710	0.0295 – 0.0295	13 / 14	5.59E+00	nc	**Yes**	
p-Cresol	0.15 – 0.15	0.0165 – 0.0175	1 / 3	3.06E+01	nc	No	
Phenanthrene	0.44 – 917	0.01 – 3.15	9 / 14	2.19E+03	surr	No	d
Pyrene	1.6 – 410	0.55 – 3.15	9 / 14	2.32E+02	nc	**Yes**	

Table 15
Selection of Chemicals of Potential Concern
Recreational Receptors Exposed to Chequamegon Bay Sediment

Analyte	Detection Range (mg/kg)	Nondetect Range (mg/kg)	Detection Frequency	Region 9 Residential Soil PRG (mg/kg)	Basis	Exceeds?	Notes
VOCs							
1,2,4-Trimethylbenzene	0.11 – 1.43	0.005 – 25	8 / 13	5.16E+00	nc	No	
1,3,5-Trimethylbenzene	0.055 – 0.27	0.005 – 25	4 / 13	2.13E+00	nc	No	
Benzene	0.098 – 0.54	0.005 – 25	3 / 13	6.43E-01	ca	No	
Ethylbenzene	0.018 – 31.1	0.0025 – 25	3 / 13	3.95E+02	sat	No	
Isopropylbenzene	0.07 – 0.07	0.0295 – 25	1 / 10	5.72E+01	nc	No	
Methylene chloride	0.15 – 36.8	0.0295 – 25	5 / 10	9.11E+00	ca	**Yes**	
p-Isopropyltoluene	0.11 – 13.9	0.0295 – 25	7 / 10	5.72E+01	surr	No	e
Toluene	0.016 – 2.2	0.005 – 25	8 / 14	5.20E+02	sat	No	
Xylenes (total)	0.069 – 31.1	0.0025 – 37.5	5 / 14	2.71E+01	nc	**Yes**	

Notes:

All units are milligrams per kilogram (mg/kg).

All non-detects are presented as one-half the detection limits.

PRG – Preliminary Remediation Goal

nc – PRG based on noncarcinogenic (systemic) effects
ca – PRG based on carcinogenic effects
sat – PRG based on the soil saturation concentration
max – PRG based on a ceiling limit
SVOC – Semi-volatile organic compound
VOC – Volatile organic compound
surr – No toxicity information is available for this chemical. The value presented is a surrogate value selected based on structural similarities. The surrogates used are defined below.
a – Naphthalene used as a surrogate for 1-methylnaphthalene and 2-methylnaphthalene.
b – Acenaphthene used as a surrogate for acenaphthylene.
c – Pyrene used as a surrogate for benzo(g,h,i)perylene.
d – Anthracene used as a surrogate for phenanthrene.
e – Isopropylbenzene used as a surrogate for p-isopropyltoluene.

Table 16
Selection of Chemicals of Potential Concern
Recreational Receptors Exposed to Chequamegon Bay Surface Water

Analyte	Detection Range (mg/kg)	Nondetect Range (mg/kg)	Detection Frequency	AWQC[a] Human Health (µg/L)	Basis	Exceeds AWQC?	Notes
High Energy Event							
SVOCs							
1-Methylnaphthalene	1.1 – 1.1	0.55 – 0.55	2 / 10	**6.2**	surr	No	a, b
2-Methylnaphthalene	1.1 – 1.2	0.55 – 0.55	2 / 10	**6.2**	surr	No	a, b
Acenaphthene	1.1 – 1.1	0.55 – 0.55	1 / 10	990	nc	No	
Naphthalene	1.1 – 1.8	0.55 – 0.55	5 / 19	**6.2**	nc	No	a
VOCs							
1,2,4-Trimethylbenzene	0.17 – 0.17	0.08 – 0.08	1 / 8	**12**	nc	No	a
1,3,5-Trimethylbenzene	0.23 – 0.23	0.075 – 0.075	1 / 8	**12**	nc	No	a
Benzene	0.15 – 0.74	0.07 – 0.07	5 / 8	51	ca	No	
Ethylbenzene	0.19 – 0.48	0.085 – 0.085	6 / 8	2,100	nc	No	
m & p-Xylenes	0.37 – 0.44	0.175 – 0.175	2 / 8	206	nc	No	
Toluene	0.14 – 0.31	0.125 – 0.125	2 / 8	15000	nc	No	
Low Energy Event							
SVOCs							
Anthracene	0.1 – 0.1	0.49 – 0.49	1 / 13	40000	nc	No	
Benzo(a)anthracene	0.29 – 0.29	0.46 – 0.46	1 / 13	0.018	ca	Yes	
Benzo(a)pyrene	0.33 – 0.33	0.5 – 0.5	1 / 13	0.018	ca	Yes	
Benzo(b)fluoranthene	0.17 – 0.17	0.46 – 0.46	1 / 13	0.018	ca	Yes	
Benzo(g,h,i)perylene	0.22 – 0.22	0.5 – 0.5	1 / 13	4000	surr	No	b
Benzo(k)fluoranthene	0.095 – 0.095	0.6 – 0.6	1 / 13	0.018	ca	Yes	
Chrysene	0.27 – 0.27	0.5 – 0.5	1 / 13	0.018	ca	Yes	
Dibenzo(a,h)anthracene	0.17 – 0.17	0.495 – 0.495	1 / 13	0.018	ca	Yes	
Fluoranthene	0.46 – 0.46	0.485 – 0.485	1 / 13	140	nc	No	
Indeno(1,2,3-cd)pyrene	0.42 – 0.42	0.47 – 0.47	1 / 13	0.018	ca	Yes	
Phenanthrene	0.66 – 0.66	0.49 – 0.49	1 / 13	40000	surr	No	b
Pyrene	0.7 – 0.7	0.47 – 0.47	1 / 13	4000	nc	No	
VOCs							
Benzene	0.26 – 0.47	0.07 – 0.07	4 / 13	51	ca	No	
Ethylbenzene	0.32 – 0.32	0.085 – 0.085	1 / 13	2,100	nc	No	
Toluene	0.25– 0.355	0.125 – 0.125	6 / 13	15,000	nc	No	
Xylenes (total)	1.54 – 1.54	No data	1 / 5	**210**	nc	No	a

Notes:
All units are micrograms per liter (µg/L).
All non-detects are presented as one-half the detection limits.

nc – AWQC based on noncarcinogenic (systemic) effects
ca – AWQC based on carcinogenic effects
SVOC – Semi-volatile organic compound
VOC – Volatile organic compound

a – For those chemicals lacking an AWQC, the Region 9 tap water PRG was used as the screening value. PRG values are presented in bold.
b – Naphthalene used as the surrogate for 1-methylnaphthalene and 2-methylnaphthalene.
Anthracene used as a surrogate for phenanthrene.
Pyrene used as a surrogate for benzo(g,h,i)perylene.

Table 17
Selection of Chemicals of Potential Concern
Residential Receptors Exposed to Chemicals in Soil Gas

Analyte	Detection Range (ug/m³)	Maximum	Detection Frequency	Target Shallow Gas[a] Concentration (ug/m³)	Basis	Exceeds Target Concentration?	Notes
1,2,4-Trimethylbenzene	2.01 - 2.01	2.01	1 / 7	6.00E+01	nc	No	
Benzene	1.69 - 1.69	1.69	1 / 7	3.10E+01	ca	No	
Chloromethane	1.3 - 1.3	1.3	1 / 7	2.40E+02	ca	No	
Dichlorodifluoromethane	2.57 - 16.82	16.82	6 / 7	2.00E+03	nc	No	
Ethylbenzene	2.17 - 2.17	2.17	1 / 7	2.20E+02	ca	No	
m & p-Xylenes	1.65 - 5.2	5.2	2 / 7	7.00E+04	nc	No	b
o-Xylene	1.73 - 1.73	1.73	1 / 7	7.00E+04	nc	No	
Toluene	1.85 - 7.15	7.15	3 / 7	4.00E+03	nc	No	
Trichlorofluoromethane	1.4 - 9.52	9.52	5 / 7	7.00E+03	nc	No	

Notes:

a – Target Shallow Gas Concentration Corresponding to Target Indoor Air Concentration Where the Soil Gas to Indoor Air Attenuation Factor Equals 0.1.

b – The concentration for m-xylene was used for the comparison.

nc – noncarcinogenic

ca – carcinogenic

Table 18
Selection of Chemicals of Potential Concern
Subsistence Fisher Exposed to Chemical in Finfish Tissue

Chemical	Frequency of Detection	Range of Detected Concentrations (mg/kg)	Range of Detected Limits (ug/kg)	Region 3 Fish Ingestion RBC (mg/kg)	Basis	Exceeds RBC?
Shorthead Redhorse & Walleye						
Naphthalene	16 / 16	0.0029 – 0.33	2 – 2	2.70	nc	No
2-Methylnaphthalene	16 / 16	0 – 0.23	2 – 2	0.54	nc	No
1-Methylnaphthalene	16 / 16	0.0 – 0.62	2 – 2	0.54	surr	***Yes***
Biphenyl	13 / 16	0.0 – 0.049	2 – 2	6.76	nc	No
2,6 Dimethylnaphthalene	15 / 16	0.0 – 0.23	2 – 2	0.54	surr	No
Acenaphthylene	7 / 16	0.0 – 0.019	2 – 2	8.11	surr	No
Acenaphthene	15 / 16	0.0 – 0.58	2 – 2	8.11	nc	No
2,3,5 Trimethylnaphthalene	14 / 16	0.0 – 0.051	2 – 2	0.54	surr	No
Fluorene	14 / 16	0.0 – 0.17	2 – 2	5.41	nc	No
Phenanthrene	15 / 16	0.0 – 0.24	2 – 2	40.56	nc	No
Anthracene	14 / 16	0 – 0.043	2 – 2	40.56	nc	No
1-Methylphenanthrene	4 / 16	0.0 – 0.0043	2 – 2	NV	–	–
Fluoranthene	14 / 16	0.0 – 0.017	2 – 2	5.41	nc	No
Pyrene	14 / 16	0.0 – 0.0097	2 – 2	4.06	nc	No
Smelt						
Naphthalene	9 / 9	0.014 – 0.026	2 – 2	2.70	nc	No
2-Methylnaphthalene	9 / 9	0.012 – 0.024	2 – 2	0.54	nc	No
1-Methylnaphthalene	9 / 9	0.012 – 0.032	2 – 2	0.54	surr	No
Biphenyl	5 / 9	0.0021 – 0.0032	2 – 2	6.76	nc	No
2,6 Dimethylnaphthalene	9 / 9	0.004 – 0.011	2 – 2	0.54	surr	No
Acenaphthylene	0 / 9	0 – 0	2 – 2	8.11	surr	No
Acenaphthene	9 / 9	0.0097 – 0.028	2 – 2	8.11	nc	No
2,3,5 Trimethylnaphthalene	3 / 9	0.002 – 0.003	2 – 2	0.54	surr	No
Fluorene	9 / 9	0.004 – 0.01	2 – 2	5.41	nc	No
Phenanthrene	8 / 9	0.0079 – 0.018	2 – 2	40.56	nc	No
Anthracene	8 / 9	0.0021 – 0.0051	2 – 2	40.56	nc	No
1-Methylphenanthrene	2 / 9	0.002 – 0.003	2 – 2	NV	–	–
Fluoranthene	8 / 9	0.0028 – 0.03	2 – 2	5.41	nc	No
Pyrene	8 / 9	0.0032 – 0.027	2 – 2	4.06	nc	No
Benzo(a)anthracene	3 / 9	0.0021 – 0.017	2 – 2	0.0043	ca	***Yes***
Chrysene	2 / 9	0.0026 – 0.017	2 – 2	0.43	ca	No
Benzo(b)fluoranthene	2 / 9	0.0034 – 0.019	2 – 2	0.0043	ca	***Yes***
Benzo(k)fluoranthene	2 / 9	0.0027 – 0.017	2 – 2	0.04	ca	No
Benzo(e)pyrene	1 / 9	0.0098 – 0.0098	2 – 2	0.00043	ca	***Yes***
Benzo(a)pyrene	2 / 9	0.0024 – 0.015	2 – 2	0.00043	ca	***Yes***
Perylene	1 / 9	0.0042 – 0.0042	2 – 2	NV	–	No
Indeno(1,2,3-cd)pyrene	1 / 9	0.0076 – 0.0076	2 – 2	0.0043	ca	***Yes***
Dibenzo(a,h)anthracene	1 / 9	0.0026 – 0.0026	2 – 2	0.00043	ca	***Yes***
Benzo(g,h,i)perylene	1 / 9	0.0075 – 0.0075	2 – 2	4.06	nc	No

Notes:

All units are milligrams per kilogram (mg/kg).

RBC – Region 3 Risk-based Concentration (USEPA, October 2005).

nc – Region 3 RBC based on noncarcinogenic effects.

surr – An RBC is not available for this chemical. A surrogate chemical was selected for this chemical based on structural similarities.

ca – Region 3 RBC is based on carcinogenic effects.

Table 19
Selection of Chemicals of Potential Concern
Industrial Worker Receptors Exposed to Volatile Organic Chemicals in Indoor Air

Analyte	Detection Range (µg/m3)	Detection Frequency	Max	Region 9 Ambient Air PRG (µg/m3)	Basis	Notes	Exceeds?
Indoor Air							
1,1,1-Trichloroethane	12.51 - 12.51	1 / 2	12.51	2.30E+03	nc		No
1,2,4-Trimethylbenzene	3.97 - 38.76	2 / 2	38.76	6.21E+00	nc		***Yes***
1,3,5-Trimethylbenzene	9.32 - 9.32	1 / 2	9.32	6.21E+00	nc		***Yes***
1,4-Dichlorobenzene	41.47 - 961.57	2 / 2	961.57	3.06E-01	ca		***Yes***
Benzene	7.98 - 38.32	2 / 2	38.32	2.49E-01	ca		***Yes***
Carbon tetrachloride	10.7 - 10.7	1 / 2	10.70	1.28E-01	ca		***Yes***
Chloromethane	2.68 - 4.13	2 / 2	4.13	9.49E+01	nc		No
Dichlorodifluoromethane	5.44 - 20.28	2 / 2	20.28	2.09E+02	nc		No
Ethylbenzene	11.27 - 26.87	2 / 2	26.87	1.06E+03	nc		No
m & p-Xylenes	39 - 73.67	2 / 2	73.67	1.06E+02	nc	a	No
Methylene chloride	4.86 - 4.86	1 / 2	4.86	4.09E+00	ca		***Yes***
o-Xylene	7.8 - 18.63	2 / 2	18.63	1.06E+02	nc	a	No
Styrene	5.53 - 5.53	1 / 2	5.53	1.06E+03	nc		No
Toluene	101.66 - 109.19	2 / 2	109.19	4.02E+02	nc		No
Trichloroethylene	3.16 - 3.16	1 / 2	3.16	1.68E-02	ca		***Yes***
Trichlorofluoromethane	2.63 - 8.4	2 / 2	8.40	7.30E+02	nc		No
Ambient Air							
Chloromethane	1.78 - 8.05	2 / 3	8.05	9.49E+01	ca		No
Dichlorodifluoromethane	3.26 - 14.84	3 / 3	14.84	2.09E+02	ca		No
Toluene	1.43 - 1.43	1 / 3	1.43	4.02E+02	nc		No
Trichlorofluoromethane	1.57 - 1.9	2 / 3	1.90	7.30E+02	nc		No

Notes:
All units are microgram per cubic meter (µg/m^3).

PRG – Preliminary Remediation Goal
nc – PRG based on noncarcinogenic (systemic) effects
ca – PRG based on carcinogenic effects

a – Total xylenes used as a surrogate for m-, o-, & p-xylenes.

Table 20
Residential Risk Summary (0-10 feet)
Reasonable Maximum Exposure

	Total Carcinogenic Risk								Total Noncarcinogenic Risk						
Equation unitless	CR unitless	=	Ingestion unitless	+	Dermal Contact unitless	+	Inhalation unitless	Target Organs	HQ unitless	=	Ingestion unitless	+	Dermal Contact unitless	+	Inhalation unitless
Inorganics															
Arsenic	1.31E-05	=	1.19E-05	+	1.18E-06	+	1.72E-09	*Skin, Circulatory*	2.35E-01	=	2.16E-01	+	1.91E-02	+	NA
Lead	NA	=	NA	+	NA	+	NA		NA	=	NA	+	NA	+	NA
Thallium	NA	=	NA	+	NA	+	NA	*Systemic*	1.31E-02	=	1.31E-02	+	NA	+	NA
SVOCs															
1-Methylnaphthalene	NA	=	NA	+	NA	+	NA	*Respiratory*	3.74E-02	=	2.74E-02	+	9.98E-03	+	NA
2-Methylnaphthalene	NA	=	NA	+	NA	+	NA	*Respiratory*	5.89E-01	=	4.60E-01	+	1.29E-01	+	NA
Acenaphthene	NA	=	NA	+	NA	+	NA	*Systemic*	1.06E-03	=	7.76E-04	+	2.82E-04	+	NA
Benzo(a)anthracene	3.96E-05	=	2.81E-05	+	1.15E-05	+	NA		NA	=	NA	+	NA	+	NA
Benzo(a)pyrene	3.14E-04	=	2.23E-04	+	9.14E-05	+	1.36E-09		NA	=	NA	+	NA	+	NA
Benzo(b)fluoranthene	3.43E-05	=	2.43E-05	+	9.97E-06	+	NA		NA	=	NA	+	NA	+	NA
Benzo(k)fluoranthene	1.63E-06	=	1.16E-06	+	4.74E-07	+	NA		NA	=	NA	+	NA	+	NA
Chrysene	3.77E-07	=	2.67E-07	+	1.10E-07	+	NA		NA	=	NA	+	NA	+	NA
Dibenzo(a,h)anthracene	5.02E-05	=	3.56E-05	+	1.46E-05	+	NA		NA	=	NA	+	NA	+	NA
Dibenzofuran	NA	=	NA	+	NA	+	NA	*Systemic*	8.66E-02	=	6.35E-02	+	2.31E-02	+	NA
Fluoranthene	NA	=	NA	+	NA	+	NA	*Systemic, Circulatory*	2.77E-03	=	2.03E-03	+	7.40E-04	+	NA
Fluorene	NA	=	NA	+	NA	+	NA	*Circulatory*	2.00E-03	=	1.47E-03	+	5.34E-04	+	NA
Indeno(1,2,3-cd)pyrene	8.61E-06	=	6.11E-06	+	2.50E-06	+	NA		NA	=	NA	+	NA	+	NA
Naphthalene	NA	=	NA	+	NA	+	NA	*Respiratory, Systemic*	1.10E+01	=	1.19E-01	+	4.33E-02	+	1.08E+01
Phenanthrene	NA	=	NA	+	NA	+	NA		NA	=	NA	+	NA	+	NA
Pyrene	NA	=	NA	+	NA	+	NA	*Systemic*	4.98E-03	=	3.65E-03	+	1.33E-03	+	NA
VOCs															
1,2,4-Trichlorobenzene	NA	=	NA	+	NA	+	NA	*Systemic, Adrenal*	2.96E-01	=	3.79E-02	+	NA	+	2.58E-01
1,2,4-Trimethylbenzene	NA	=	NA	+	NA	+	NA	*Systemic, CNS*	9.29E-01	=	1.55E-03	+	NA	+	9.27E-01
1,3,5-Trimethylbenzene	NA	=	NA	+	NA	+	NA	*Systemic, CNS*	2.93E-01	=	4.02E-04	+	NA	+	2.92E-01
Benzene	1.94E-05	=	1.23E-06	+	NA	+	1.82E-05	*Circulatory*	7.92E-01	=	4.57E-02	+	NA	+	7.46E-01
Xylenes (total)	NA	=	NA	+	NA	+	NA	*Systemic, CNS*	5.90E-01	=	1.87E-03	+	NA	+	5.88E-01
Soil CR	***5E-04***	=	3E-04	+	1E-04	+	2E-05	**Soil HI**	***15***	=	1.0	+	0.2	+	14

Notes:

NA – Not Applicable
CR – Cancer Risk
HQ – Hazard Quotient
HI – Hazard Index
CNS – Central Nervous System

Target Organ HI	
Respiratory	1E+01
Systemic	1E+01
Circulatory	1E+00
Adrenal	3E-01
CNS	2E+00
Skin	2E-01

Table 21
Risk Summary - Recreational Adult Exposed to Surface Soil
Reasonable Maximum Exposure

	Total Carcinogenic Risk							Total Noncarcinogenic Hazard						
Equation	CR	=	Ingestion	+	Dermal	+	Inhalation	HQ	=	Ingestion	+	Dermal	+	Inhalation
unitless	*unitless*		*unitless*		*unitless*		*unitless*	*unitless*		*unitless*		*unitless*		*unitless*
Aluminum	NA	=	NA	+	NA	+	NA	5.06E-04	=	4.85E-04	+	NA	+	2.06E-05
Arsenic	2.49E-07	=	1.79E-07	+	6.94E-08	+	1.09E-10	1.29E-03	=	9.30E-04	+	3.60E-04	+	NA
Cadmium	1.13E-11	=	NA	+	NA	+	1.13E-11	1.03E-04	=	6.89E-05	+	3.37E-05	+	1.61E-08
Iron	NA	=	NA	+	NA	+	NA	4.32E-03	=	4.32E-03	+	NA	+	NA
Lead	NA	=	NA	+	NA	+	NA	NA	=	NA	+	NA	+	NA
Manganese	NA	=	NA	+	NA	+	NA	2.01E-04	=	1.26E-04	+	NA	+	7.52E-05
Thallium	NA	=	NA	+	NA	+	NA	6.60E-05	=	6.60E-05	+	NA	+	NA
Vanadium	NA	=	NA	+	NA	+	NA	2.66E-04	=	2.66E-04	+	NA	+	NA
Zinc	NA	=	NA	+	NA	+	NA	7.82E-05	=	7.82E-05	+	NA	+	NA
Benzo(a)anthracene	1.49E-07	=	5.74E-08	+	9.13E-08	+	NA	NA	=	NA	+	NA	+	NA
Benzo(a)pyrene	2.93E-06	=	1.13E-06	+	1.80E-06	+	2.90E-11	NA	=	NA	+	NA	+	NA
Benzo(b)fluoranthene	2.83E-07	=	1.09E-07	+	1.74E-07	+	NA	NA	=	NA	+	NA	+	NA
Benzo(k)fluoranthene	7.95E-09	=	3.07E-09	+	4.88E-09	+	NA	NA	=	NA	+	NA	+	NA
Indeno(1,2,3-cd)pyrene	1.95E-07	=	7.52E-08	+	1.20E-07	+	NA	NA	=	NA	+	NA	+	NA
Soil CR	**4E-06**	=	2E-06	+	2E-06	+	1E-10	**Soil HI** **0.006**	=	0.01	+	0.0004	+	8E-05

Notes:

NA – Not Applicable
CR – Cancer Risk
HI – Hazard Index

Table 22
Risk Summary - Recreational Adolescent Exposed to Surface Soil
Reasonable Maximum Exposure

	Total Carcinogenic Risk							Total Noncarcinogenic Hazard						
Equation unitless	CR unitless	=	Ingestion unitless	+	Dermal unitless	+	Inhalation unitless	HQ unitless	=	Ingestion unitless	+	Dermal unitless	+	Inhalation unitless
Aluminum	NA	=	NA	+	NA	+	NA	7.53E-04	=	7.23E-04	+	NA	+	3.07E-05
Arsenic	1.16E-07	=	1.07E-07	+	9.54E-09	+	6.52E-11	1.92E-03	=	1.39E-03	+	5.36E-04	+	NA
Cadmium	6.74E-12	=	NA	+	NA	+	6.74E-12	1.53E-04	=	1.03E-04	+	5.03E-05	+	2.40E-08
Iron	NA	=	NA	+	NA	+	NA	6.43E-03	=	6.43E-03	+	NA	+	NA
Lead	NA	=	NA	+	NA	+	NA	NA	=	NA	+	NA	+	NA
Manganese	NA	=	NA	+	NA	+	NA	3.00E-04	=	1.88E-04	+	NA	+	1.12E-04
Thallium	NA	=	NA	+	NA	+	NA	9.83E-05	=	9.83E-05	+	NA	+	NA
Vanadium	NA	=	NA	+	NA	+	NA	3.96E-04	=	3.96E-04	+	NA	+	NA
Zinc	NA	=	NA	+	NA	+	NA	1.17E-04	=	1.17E-04	+	NA	+	NA
Benzo(a)anthracene	4.67E-08	=	3.42E-08	+	1.26E-08	+	NA	NA	=	NA	+	NA	+	NA
Benzo(a)pyrene	9.20E-07	=	6.73E-07	+	2.47E-07	+	1.73E-11	NA	=	NA	+	NA	+	NA
Benzo(b)fluoranthene	8.89E-08	=	6.50E-08	+	2.39E-08	+	NA	NA	=	NA	+	NA	+	NA
Benzo(k)fluoranthene	2.50E-09	=	1.83E-09	+	6.71E-10	+	NA	NA	=	NA	+	NA	+	NA
Indeno(1,2,3-cd)pyrene	6.12E-08	=	4.48E-08	+	1.65E-08	+	NA	NA	=	NA	+	NA	+	NA
Soil CR	**1E-06**	=	9E-07	+	3E-07	+	9E-11	**Soil HI** **0.009**	=	0.009	+	0.001	+	1E-04

Notes:

NA – Not Applicable
CR – Cancer Risk
HI – Hazard Index

Table 23
Risk Summary - Recreational Child Exposed to Surface Soil
Reasonable Maximum Exposure

	Total Carcinogenic Risk							Total Noncarcinogenic Hazard						
Equation unitless	CR unitless	=	Ingestion unitless	+	Dermal unitless	+	Inhalation unitless	HQ unitless	=	Ingestion unitless	+	Dermal unitless	+	Inhalation unitless
Aluminum	NA	=	NA	+	NA	+	NA	1.82E-02	=	1.81E-02	+	NA	+	1.16E-04
Arsenic	1.46E-06	=	1.34E-06	+	1.18E-07	+	1.23E-10	3.78E-02	=	3.47E-02	+	3.07E-03	+	NA
Cadmium	1.27E-11	=	NA	+	NA	+	1.27E-11	2.86E-03	=	2.57E-03	+	2.88E-04	+	9.03E-08
Iron	NA	=	NA	+	NA	+	NA	1.61E-01	=	1.61E-01	+	NA	+	NA
Lead	NA	=	NA	+	NA	+	NA	NA	=	NA	+	NA	+	NA
Manganese	NA	=	NA	+	NA	+	NA	5.14E-03	=	4.71E-03	+	NA	+	4.21E-04
Thallium	NA	=	NA	+	NA	+	NA	2.46E-03	=	2.46E-03	+	NA	+	NA
Vanadium	NA	=	NA	+	NA	+	NA	9.92E-03	=	9.92E-03	+	NA	+	NA
Zinc	NA	=	NA	+	NA	+	NA	2.92E-03	=	2.92E-03	+	NA	+	NA
Benzo(a)anthracene	5.84E-07	=	4.28E-07	+	1.56E-07	+	NA	NA	=	NA	+	NA	+	NA
Benzo(a)pyrene	1.15E-05	=	8.44E-06	+	3.07E-06	+	3.25E-11	NA	=	NA	+	NA	+	NA
Benzo(b)fluoranthene	1.11E-06	=	8.15E-07	+	2.96E-07	+	NA	NA	=	NA	+	NA	+	NA
Benzo(k)fluoranthene	3.12E-08	=	2.29E-08	+	8.34E-09	+	NA	NA	=	NA	+	NA	+	NA
Indeno(1,2,3-cd)pyrene	7.66E-07	=	5.61E-07	+	2.04E-07	+	NA	NA	=	NA	+	NA	+	NA
Soil CR	**2E-05**	=	1E-05	+	4E-06	+	2E-10	**Soil HI** **0.22**	=	0.2	+	0.003	+	4E-04

Notes:

NA – Not Applicable
CR – Cancer Risk
HI – Hazard Index

Table 24
Adult Swimmer Risk Summary
Reasonable Maximum Exposure

Equation unitless	Total Carcinogenic Risk: CR unitless	=	Ingestion unitless	+	Dermal Contact unitless		Total Noncarcinogenic Risk: HQ unitless	=	Ingestion unitless	+	Dermal Contact unitless
Iron	NA	=	NA	+	NA		1.18E-05	=	1.18E-05	+	NA
Manganese	NA	=	NA	+	NA		8.45E-07	=	8.45E-07	+	NA
Vanadium	NA	=	NA	+	NA		1.28E-06	=	1.28E-06	+	NA
1-Methylnaphthalene	NA	=	NA	+	NA		NA	=	NA	+	NA
2-Methylnaphthalene	NA	=	NA	+	NA		3.64E-02	=	1.80E-05	+	3.64E-02
Acenaphthene	NA	=	NA	+	NA		1.07E-03	=	4.05E-07	+	1.07E-03
Benzo(a)anthracene	4.29E-06	=	2E-09	+	4.29E-06		NA	=	NA	+	NA
Benzo(a)pyrene	3.10E-05	=	1E-08	+	3.10E-05		NA	=	NA	+	NA
Benzo(b)fluoranthene	1.63E-06	=	6E-10	+	1.63E-06		NA	=	NA	+	NA
Benzo(k)fluoranthene	2.07E-07	=	8E-11	+	2.07E-07		NA	=	NA	+	NA
Chrysene	3.83E-08	=	1E-11	+	3.83E-08		NA	=	NA	+	NA
Dibenzo(a,h)anthracene	2.06E-06	=	8E-10	+	2.06E-06		NA	=	NA	+	NA
Fluoranthene	NA	=	NA	+	NA		7.49E-04	=	2.84E-07	+	7.49E-04
Fluorene	NA	=	NA	+	NA		9.75E-04	=	3.70E-07	+	9.75E-04
Indeno(1,2,3-cd)pyrene	5.74E-07	=	2E-10	+	5.74E-07		NA	=	NA	+	NA
Naphthalene	NA	=	NA	+	NA		9.92E-03	=	3.77E-06	+	9.92E-03
Pyrene	NA	=	NA	+	NA		3.86E-05	=	4.83E-07	+	3.81E-05
Methylene chloride	5.49E-12	=	5E-12	+	NA		2.85E-08	=	2.85E-08	+	NA
Xylenes (total)	NA	=	NA	+	NA		6.77E-09	=	6.77E-09	+	NA
Sediment CR	**4E-05**	=	2E-08	+	4E-05	**Sediment HI**	**0.05**	=	4E-05	+	0.05

Notes:

NA – Not Applicable
CR – Cancer Risk
HI – Hazard Index

Table 25
Adolescent Swimmer Risk Summary
Reasonable Maximum Exposure

	Total Carcinogenic Risk						Total Noncarcinogenic Risk				
Equation unitless	CR unitless	=	Ingestion unitless	+	Dermal Contact unitless		HQ unitless	=	Ingestion unitless	+	Dermal Contact unitless
Iron	NA	=	NA	+	NA		1.76E-05	=	1.76E-05	+	NA
Manganese	NA	=	NA	+	NA		1.26E-06	=	1.26E-06	+	NA
Vanadium	NA	=	NA	+	NA		1.90E-06	=	1.90E-06	+	NA
1-Methylnaphthalene	NA	=	NA	+	NA		NA	=	NA	+	NA
2-Methylnaphthalene	NA	=	NA	+	NA		3.87E-02	=	2.67E-05	+	4E-02
Acenaphthene	NA	=	NA	+	NA		1.13E-03	=	0.00E+00	+	1E-03
Benzo(a)anthracene	1.82E-06	=	1E-09	+	2E-06		NA	=	NA	+	NA
Benzo(a)pyrene	1.32E-05	=	7E-09	+	1E-05		NA	=	NA	+	NA
Benzo(b)fluoranthene	6.93E-07	=	4E-10	+	7E-07		NA	=	NA	+	NA
Benzo(k)fluoranthene	8.81E-08	=	5E-11	+	9E-08		NA	=	NA	+	NA
Chrysene	1.63E-08	=	9E-12	+	2E-08		NA	=	NA	+	NA
Dibenzo(a,h)anthracene	8.75E-07	=	5E-10	+	9E-07		NA	=	NA	+	NA
Fluoranthene	NA	=	NA	+	NA		7.96E-04	=	4.24E-07	+	8E-04
Fluorene	NA	=	NA	+	NA		1.04E-03	=	5.51E-07	+	1E-03
Indeno(1,2,3-cd)pyrene	2.44E-07	=	1E-10	+	2E-07		NA	=	NA	+	NA
Naphthalene	NA	=	NA	+	NA		1.05E-02	=	5.61E-06	+	1E-02
Pyrene	NA	=	NA	+	NA		4.12E-05	=	7.19E-07	+	4E-05
Methylene chloride	3.27E-12	=	3E-12	+	NA		4.24E-08	=	4.24E-08	+	NA
Xylenes (total)	NA	=	NA	+	NA		1.01E-08	=	1.01E-08	+	NA
Sediment CR	**2E-05**	=	9E-09	+	2E-05	**Sediment HI**	**0.05**	=	6E-05	+	0.05

Notes:

NA – Not Applicable
CR – Cancer Risk
HI – Hazard Index

Table 26
Adult Wader Risk Summary
Reasonable Maximum Exposure

Equation unitless	Total Carcinogenic Risk: Total Risk unitless	=	Ingestion unitless	+	Dermal Contact unitless		Total Noncarcinogenic Risk: Total Hazard unitless	=	Ingestion unitless	+	Dermal Contact unitless
Iron	NA	=	NA	+	NA		2.37E-05	=	2.37E-05	+	NA
Manganese	NA	=	NA	+	NA		8.45E-07	=	8.45E-07	+	NA
Vanadium	NA	=	NA	+	NA		1.28E-06	=	1.28E-06	+	NA
1-Methylnaphthalene	NA	=	NA	+	NA		NA	=	NA	+	NA
2-Methylnaphthalene	NA	=	NA	+	NA		3.64E-02	=	1.80E-05	+	3.64E-02
Acenaphthene	NA	=	NA	+	NA		1.07E-03	=	2.19E-09	+	1.07E-03
Benzo(a)anthracene	4.29E-06	=	3.26E-09	+	4.29E-06		NA	=	NA	+	NA
Benzo(a)pyrene	3.10E-05	=	2.35E-08	+	3.10E-05		NA	=	NA	+	NA
Benzo(b)fluoranthene	1.63E-06	=	1.24E-09	+	1.63E-06		NA	=	NA	+	NA
Benzo(k)fluoranthene	2.07E-07	=	1.57E-10	+	2.07E-07		NA	=	NA	+	NA
Chrysene	3.83E-08	=	2.91E-11	+	3.83E-08		NA	=	NA	+	NA
Dibenzo(a,h)anthracene	2.06E-06	=	1.56E-09	+	2.06E-06		NA	=	NA	+	NA
Dibenzofuran	NA	=	NA	+	NA		1.73E-04	=	6.58E-08	+	1.73E-04
Fluoranthene	NA	=	NA	+	NA		7.49E-04	=	3.29E-09	+	7.49E-04
Fluorene	NA	=	NA	+	NA		9.75E-04	=	3.29E-09	+	9.75E-04
Indeno(1,2,3-cd)pyrene	5.75E-07	=	4.36E-10	+	5.74E-07		NA	=	NA	+	NA
Naphthalene	NA	=	NA	+	NA		9.92E-03	=	3.77E-06	+	9.92E-03
Pyrene	NA	=	NA	+	NA		8.29E-07	=	4.83E-07	+	3.46E-07
Methylene chloride	5.49E-12	=	5.49E-12	+	NA		2.85E-08	=	2.85E-08	+	NA
Xylenes (total)	NA	=	NA	+	NA		6.77E-09	=	6.77E-09	+	NA
Sediment CR	**4E-05**	=	3E-08	+	4E-05	**Sediment HI**	**0.05**	=	5E-05	+	0.046

Notes:

NA – Not Applicable
CR – Cancer Risk
HI – Hazard Index

Table 27
Adolescent Wader Risk Summary
Reasonable Maximum Exposure

Equation unitless	Total Carcinogenic Risk: CR unitless	=	Ingestion unitless	+	Dermal Contact unitless		Total Noncarcinogenic Risk: HQ unitless	=	Ingestion unitless	+	Dermal Contact unitless
Iron	NA	=	NA	+	NA		4E-05	=	3.53E-05	+	NA
Manganese	NA	=	NA	+	NA		3E-06	=	2.52E-06	+	NA
Vanadium	NA	=	NA	+	NA		4E-06	=	3.80E-06	+	NA
1-Methylnaphthalene	NA	=	NA	+	NA		NA	=	NA	+	NA
2-Methylnaphthalene	NA	=	NA	+	NA		4E-02	=	5.35E-05	+	4E-02
Acenaphthene	NA	=	NA	+	NA		1E-03	=	0.00E+00	+	1E-03
Benzo(a)anthracene	2E-06	=	2E-09	+	2E-06		NA	=	NA	+	NA
Benzo(a)pyrene	1E-05	=	1E-08	+	1E-05		NA	=	NA	+	NA
Benzo(b)fluoranthene	7E-07	=	7E-10	+	7E-07		NA	=	NA	+	NA
Benzo(k)fluoranthene	9E-08	=	9E-11	+	9E-08		NA	=	NA	+	NA
Chrysene	2E-08	=	2E-11	+	2E-08		NA	=	NA	+	NA
Dibenzo(a,h)anthracene	9E-07	=	9E-10	+	9E-07		NA	=	NA	+	NA
Fluoranthene	NA	=	NA	+	NA		8E-04	=	8.47E-07	+	8E-04
Fluorene	NA	=	NA	+	NA		1E-03	=	1.10E-06	+	1E-03
Indeno(1,2,3-cd)pyrene	2E-07	=	3E-10	+	2E-07		NA	=	NA	+	NA
Naphthalene	NA	=	NA	+	NA		1E-02	=	1.12E-05	+	1E-02
Pyrene	NA	=	NA	+	NA		4E-05	=	7.19E-07	+	4E-05
Methylene chloride	3E-13	=	3E-13	+	NA		3E-09	=	3.26E-09	+	NA
o-Xylene	NA	=	NA	+	NA		1E-09	=	9.79E-10	+	NA
sec-Butyl benzene	NA	=	NA	+	NA		5E-09	=	4.90E-09	+	NA
Styrene	NA	=	NA	+	NA		1E-09	=	9.79E-10	+	NA
Xylenes (total)	NA	=	NA	+	NA		1E-08	=	1.01E-08	+	NA
Sediment CR	**2E-05**	=	2E-08	+	2E-05	**Sediment HI**	**0.05**	=	1E-04	+	0.052

Notes:

NA – Not Applicable
CR – Cancer Risk
HI – Hazard Index

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Table 28
Industrial Worker Risk Summary
Reasonable Maximum Exposure

	Carcinogenic Risk							Noncarcinogenic Hazard						
Equation	CR	=	Ingestion	+	Dermal	+	Inhalation	HI	=	Ingestion	+	Dermal	+	Inhalation
Units	unitless		unitless		unitless		unitless	unitless		unitless		unitless		unitless
Inorganics														
Arsenic	1E-06	=	9.58E-07	+	2.00E-07	+	3.03E-10	7.20E-03	=	5.96E-03	+	1.24E-03	+	NA
SVOCs														
Benzo(a)pyrene	2E-07	=	2.31E-07	+	NA	+	3.07E-12	NA	=	NA	+	NA	+	NA
Soil CR	**1E-06**	=	1E-06	+	2E-07	+	3E-10	**Soil HI** **0.007**	=	0.01	+	0.001	+	NA

Notes:

NA – Not Applicable
CR – Cancer Risk
HI – Hazard Index
SVOC – Semivolatile organic compound

Table 29
Industrial Worker Indoor Air Risk Summary
Reasonable Maximum Exposure

	Carcinogenic Risk unitless	Target Organs	Noncarcinogenic Hazard unitless
1,2,4-Trimethylbenzene	NA	*Systemic, CNS*	1.77E+00
1,3,5-Trimethylbenzene	NA	*CNS*	4.27E-01
1,4-Dichlorobenzene	NA	*Systemic*	3.29E-01
Benzene	2.92E-05	*Circulatory*	3.50E-01
Carbon tetrachloride	1.57E-05		NA
Methylene chloride	2.24E-07	*CNS*	4.44E-04
Trichloroethylene	3.53E-05	*Systemic, CNS*	2.17E-02
Air CR	**8E-05**	**Air HI**	**3**

Notes:

NA – Not Applicable
CR – Cancer Risk
HQ – Hazard Quotient
HI – Hazard Index

Target Organ HI	
Circulatory	4E-01
CNS	2E+00
Systemic	3E+00

Table 30
Maintenance Worker Risk Summary
Reasonable Maximum Exposure

	Carcinogenic Risk							Noncarcinogenic Hazard						
Equation	CR	=	Ingestion	+	Dermal	+	Inhalation	HQ	=	Ingestion	+	Dermal	+	Inhalation
Units	unitless		unitless		unitless		unitless	unitless		unitless		unitless		unitless
Arsenic	1.39E-07	=	1.15E-07	+	2.40E-08	+	6.98E-11	8.65E-04	=	7.16E-04	+	1.49E-04	+	NA
Lead	NA	=	NA	+	NA	+	NA	NA	=	NA	+	NA	+	NA
Benzo(a)anthracene	5.9E-08	=	3.17E-08	+	2.72E-08	+	NA	NA	=	NA	+	NA	+	NA
Benzo(a)pyrene	1.2E-06	=	6.37E-07	+	5.47E-07	+	1.63E-11	NA	=	NA	+	NA	+	NA
Benzo(b)fluoranthene	1.1E-07	=	5.89E-08	+	5.05E-08	+	NA	NA	=	NA	+	NA	+	NA
Indeno(1,2,3-cd)pyrene	7.5E-08	=	4.06E-08	+	3.48E-08	+	NA	NA	=	NA	+	NA	+	NA
Soil CR	**2E-06**	=	9E-07	+	7E-07	+	9E-11	**Soil HI** **0.0009**	=	0.0007	+	0.0001	+	NA

Notes:

NA – Not Applicable
CR – Cancer Risk
HQ – Hazard Quotient
HI – Hazard Index

Table 31
Construction Worker Risk Summary
Reasonable Maximum Exposure

	Carcinogenic Risk								Target Organs	Noncarcinogenic Hazard						
Equation	CR	=	Ingestion	+	Dermal	+	Inhalation			HQ	=	Ingestion	+	Dermal	+	Inhalation
Units	unitless		unitless		unitless		unitless			unitless		unitless		unitless		unitless
Inorganics																
Arsenic	3.38E-07	=	3.12E-07	+	1.73E-08	+	8.32E-09		*Skin, Circulatory*	5.13E-02	=	4.86E-02	+	2.69E-03	+	NA
Lead	NA	=	NA	+	NA	+	NA			NA	=	NA	+	NA	+	NA
Manganese	NA	=	NA	+	NA	+	NA		*CNS*	1.92E-01	=	7.11E-03	+	NA	+	1.85E-01
Vanadium	NA	=	NA	+	NA	+	NA		*Skin*	1.45E-02	=	1.45E-02	+	NA	+	NA
SVOCs																
1-Methylnaphthalene	NA	=	NA	+	NA	+	NA		*Respiratory*	8.13E-02	=	6.62E-02	+	1.51E-02	+	NA
2-Methylnaphthalene	NA	=	NA	+	NA	+	NA		*Respiratory*	1.51E+00	=	1.29E+00	+	2.26E-01	+	NA
Acenaphthene	NA	=	NA	+	NA	+	NA		*Systemic*	2.55E-03	=	2.08E-03	+	4.74E-04	+	NA
Acenaphthylene	NA	=	NA	+	NA	+	NA			NA	=	NA	+	NA	+	NA
Benzo(a)anthracene	7.05E-06	=	5.74E-06	+	1.31E-06	+	NA			NA	=	NA	+	NA	+	NA
Benzo(a)pyrene	5.07E-05	=	4.13E-05	+	9.41E-06	+	4.62E-08			NA	=	NA	+	NA	+	NA
Benzo(b)fluoranthene	3.08E-06	=	2.51E-06	+	5.72E-07	+	NA			NA	=	NA	+	NA	+	NA
Benzo(k)fluoranthene	3.82E-07	=	3.11E-07	+	7.09E-08	+	NA			NA	=	NA	+	NA	+	NA
Chrysene	6.31E-08	=	5.14E-08	+	1.17E-08	+	NA			NA	=	NA	+	NA	+	NA
Dibenzo(a,h)anthracene	7.59E-06	=	6.18E-06	+	1.41E-06	+	NA			NA	=	NA	+	NA	+	NA
Dibenzofuran	NA	=	NA	+	NA	+	NA		*Systemic*	1.96E-01	=	1.60E-01	+	3.64E-02	+	NA
Fluoranthene	NA	=	NA	+	NA	+	NA		*Systemic, Circulatory*	2.80E-03	=	2.28E-03	+	5.20E-04	+	NA
Fluorene	NA	=	NA	+	NA	+	NA		*Circulatory*	2.85E-03	=	2.32E-03	+	5.29E-04	+	NA
Indeno(1,2,3-cd)pyrene	2.78E-06	=	2.26E-06	+	5.16E-07	+	NA			NA	=	NA	+	NA	+	NA
Naphthalene	NA	=	NA	+	NA	+	NA		*Respiratory, Systemic*	3.07E+01	=	1.89E-01	+	4.30E-02	+	3.05E+01
Pyrene	NA	=	NA	+	NA	+	NA		*Systemic*	5.23E-03	=	4.26E-03	+	9.71E-04	+	NA
VOCs																
1,2,3-Trimethylbenzene	NA	=	NA	+	NA	+	NA			NA	=	NA	+	NA	+	NA
1,2,4-Trichlorobenzene	NA	=	NA	+	NA	+	NA		*Systemic, Adrenal*	1.13E+00	=	8.54E-02	+	NA	+	1.04E+00
1,2,4-Trimethylbenzene	NA	=	NA	+	NA	+	NA		*Systemic, CNS*	2.90E-01	=	2.72E-04	+	NA	+	2.90E-01
1,3,5-Trimethylbenzene	NA	=	NA	+	NA	+	NA		*Systemic, CNS*	1.65E-01	=	1.27E-04	+	NA	+	1.65E-01
Benzene	1.22E-06	=	3.83E-08	+	NA	+	1.19E-06		*Circulatory*	3.67E-01	=	1.22E-02	+	NA	+	3.55E-01
Ethylbenzene	NA	=	NA	+	NA	+	NA		*Systemic, Teratology*	2.90E-02	=	1.48E-03	+	NA	+	2.76E-02
n-Butyl benzene	NA	=	NA	+	NA	+	NA		*Systemic*	4.78E-03	=	4.78E-03	+	NA	+	NA
p-Isopropyltoluene	NA	=	NA	+	NA	+	NA			NA	=	NA	+	NA	+	NA
sec-Butyl benzene	NA	=	NA	+	NA	+	NA		*Systemic*	4.05E-03	=	4.05E-03	+	NA	+	NA
Toluene	NA	=	NA	+	NA	+	NA		*Systemic, CNS*	2.41E-02	=	5.43E-05	+	NA	+	2.40E-02
Xylenes (total)	NA	=	NA	+	NA	+	NA		*Systemic, CNS*	1.19E+00	=	2.13E-03	+	NA	+	1.19E+00
Soil CR	**7E-05**	=	6E-05	+	1E-05	+	1E-06		**Soil HI**	**36**	=	2	+	0.3	+	34

Notes:

NA – Not Applicable
CR – Cancer Risk
HQ – Hazard Quotient
HI – Hazard Index
CNS – Central Nervous System

Target Organ HI	
Respiratory	3E+01
Circulatory	4E-01
Systemic	3E+01
Adrenal	1E+00
CNS	2E+00
Teratology	3E-02
Skin	1E-02

Table 32
Subsistence Fisher Risk Summary
Reasonable Maximum Exposure

	Carcinogenic Risk unitless		Noncarcinogenic Hazard unitless
Shorthead Redhorse & Walleye			
1-Methylnaphthalene	NA		0.01
Smelt			
Benzo(a)anthracene	5.90E-05		NA
Benzo(a)pyrene	5.21E-05		NA
Benzo(b)fluoranthene	6.60E-06		NA
Benzo(e)pyrene	NA		NA
Dibenzo(a,h)anthracene	9.03E-06		NA
Indeno(1,2,3-cd)pyrene	2.64E-06		NA
Fish CR	**1E-04**	**Fish HI**	0.01

Notes:

NA – Not Applicable
CR – Cancer Risk
HI – Hazard Index

Table 33
Residential Risk Summary (0-1 feet)
Reasonable Maximum Exposure

Chemical	Total Carcinogenic Risk: Total Risk	=	Ingestion		Dermal Contact	+	Inhalation	Total Noncarcinogenic Risk: Total Hazard	=	Ingestion	+	Dermal Contact	+	Inhalation
Inorganics														
Arsenic	9.47E-06	=	8.61E-06	+	8.58E-07	+	1.25E-09	1.70E-01	=	1.56E-01	+	1.38E-02	+	NA
Lead	NA	=	NA	+	NA	+	NA	NA	=	NA	+	NA	+	NA
SVOCs														
Benzo(a)pyrene	2.93E-06	=	2.08E-06	+	8.53E-07	+	1.27E-11	NA	=	NA	+	NA	+	NA
Soil CR	**1E-05**	=	1E-05	+	2E-06	+	1E-09	**Soil HI** **0.2**	=	0.2	+	0.01	+	NA

Notes:

NA – Not Applicable
CR – Cancer Risk
HQ – Hazard Quotient
HI – Hazard Index

Table 34
Residential Risk Summary (0-3 feet)
Reasonable Maximum Exposure

	Total Carcinogenic Risk								Total Noncarcinogenic Risk						
Equation unitless	Total Risk unitless	=	Ingestion unitless	+	Dermal Contact unitless	+	Inhalation unitless		Total Hazard unitless	=	Ingestion unitless	+	Dermal Contact unitless	+	Inhalation unitless
Inorganics															
Arsenic	9.65E-06	=	8.78E-06	+	8.74E-07	+	1.27E-09		1.73E-01	=	1.59E-01	+	1.41E-02	+	NA
Lead	NA	=	NA	+	NA	+	NA		NA	=	NA	+	NA	+	NA
SVOCs															
1-Methylnaphthalene	NA	=	NA	+	NA	+	NA		4.14E-03	=	3.03E-03	+	1.10E-03	+	NA
Benzo(a)anthracene	2.19E-05	=	1.55E-05	+	6.36E-06	+	NA		NA	=	NA	+	NA	+	NA
Benzo(a)pyrene	2.60E-04	=	1.84E-04	+	7.55E-05	+	1.12E-09		NA	=	NA	+	NA	+	NA
Benzo(b)fluoranthene	2.17E-05	=	1.54E-05	+	6.31E-06	+	NA		NA	=	NA	+	NA	+	NA
Benzo(k)fluoranthene	6.81E-07	=	4.83E-07	+	1.98E-07	+	NA		NA	=	NA	+	NA	+	NA
Dibenzo(a,h)anthracene	1.23E-05	=	8.70E-06	+	3.57E-06	+	NA		NA	=	NA	+	NA	+	NA
Fluoranthene	NA	=	NA	+	NA	+	NA		1.15E-03	=	8.46E-04	+	3.08E-04	+	NA
Indeno(1,2,3-cd)pyrene	5.47E-06	=	3.88E-06	+	1.59E-06	+	NA		NA	=	NA	+	NA	+	NA
Naphthalene	NA	=	NA	+	NA	+	NA		3.80E-01	=	4.13E-03	+	1.50E-03	+	3.75E-01
Pyrene	NA	=	NA	+	NA	+	NA		2.73E-03	=	2.00E-03	+	7.29E-04	+	NA
VOCs															
1,2,4-Trimethylbenzene	NA	=	NA	+	NA	+	NA		2.01E-01	=	3.35E-04	+	NA	+	2.00E-01
1,3,5-Trimethylbenzene	NA	=	NA	+	NA	+	NA		8.92E-03	=	1.23E-05	+	NA	+	8.91E-03
Benzene	2.62E-06	=	1.66E-07	+	NA	+	2.45E-06		1.07E-01	=	6.17E-03	+	NA	+	1.01E-01
Soil CR	***3E-04***	=	2E-04	+	9E-05	+	2E-06	**Soil HI**	***0.9***	=	0.2	+	0.02	+	0.7

Notes:

NA – Not Applicable
CR – Cancer Risk
HQ – Hazard Quotient
HI – Hazard Index

Table 35
Residential Risk Summary (0-10 feet)
Central Tendency Evaluation

Equation	Total Carcinogenic Risk								Total Noncarcinogenic Risk						
	CR		Ingestion		Dermal Contact		Inhalation		HQ		Ingestion		Dermal Contact		Inhalation
Units	unitless	=	unitless	+	unitless	+	unitless	Target Organs	unitless	=	unitless	+	unitless	+	unitless
Inorganics															
Arsenic	4.24E-06	=	4.10E-06	+	1.45E-07	+	NA	*Skin, Circulatory*	9.48E-02	=	9.16E-02	+	3.24E-03	+	NA
Lead	NA	=	NA	+	NA	+	NA		NA	=	NA	+	NA	+	NA
Thallium	NA	=	NA	+	NA	+	NA	*Systemic*	6.26E-02	=	6.26E-02	+	NA	+	NA
SVOCs															
1-Methylnaphthalene	NA	=	NA	+	NA	+	NA	*Respiratory*	7.88E-03	=	6.88E-03	+	1.00E-03	+	NA
2-Methylnaphthalene	NA	=	NA	+	NA	+	NA	*Respiratory*	1.43E-01	=	1.29E-01	+	1.44E-02	+	NA
Acenaphthene	NA	=	NA	+	NA	+	NA	*Systemic*	2.66E-03	=	2.32E-03	+	3.38E-04	+	NA
Benzo(a)anthracene	7.78E-06	=	6.79E-06	+	9.91E-07	+	NA		NA	=	NA	+	NA	+	NA
Benzo(a)pyrene	6.53E-05	=	5.70E-05	+	8.31E-06	+	3.61E-10		NA	=	NA	+	NA	+	NA
Benzo(b)fluoranthene	6.93E-06	=	6.05E-06	+	8.83E-07	+	NA		NA	=	NA	+	NA	+	NA
Benzo(k)fluoranthene	3.38E-07	=	2.95E-07	+	4.31E-08	+	NA		NA	=	NA	+	NA	+	NA
Chrysene	7.46E-08	=	6.51E-08	+	9.50E-09	+	NA		NA	=	NA	+	NA	+	NA
Dibenzo(a,h)anthracene	1.08E-05	=	9.42E-06	+	1.37E-06	+	NA		NA	=	NA	+	NA	+	NA
Dibenzofuran	NA	=	NA	+	NA	+	NA	*Systemic*	1.71E-02	=	1.49E-02	+	2.17E-03	+	NA
Fluoranthene	NA	=	NA	+	NA	+	NA	*Systemic, Circulatory*	6.73E-03	=	5.87E-03	+	8.55E-04	+	NA
Fluorene	NA	=	NA	+	NA	+	NA	*Circulatory*	4.64E-03	=	4.05E-03	+	5.90E-04	+	NA
Indeno(1,2,3-cd)pyrene	1.83E-06	=	1.59E-06	+	2.33E-07	+	NA		NA	=	NA	+	NA	+	NA
Naphthalene	NA	=	NA	+	NA	+	NA	*Respiratory, Systemic*	2.76E+00	=	1.55E-02	+	2.26E-03	+	2.74E+00
Phenanthrene	NA	=	NA	+	NA	+	NA		NA	=	NA	+	NA	+	NA
Pyrene	NA	=	NA	+	NA	+	NA	*Systemic*	1.20E-03	=	1.05E-03	+	1.53E-04	+	NA
VOCs															
1,2,4-Trichlorobenzene	NA	=	NA	+	NA	+	NA	*Systemic, Adrenal*	3.52E-01	=	9.87E-03	+	NA	+	3.42E-01
1,2,4-Trimethylbenzene	NA	=	NA	+	NA	+	NA	*Systemic, CNS*	2.64E-01	=	4.82E-04	+	NA	+	2.64E-01
1,3,5-Trimethylbenzene	NA	=	NA	+	NA	+	NA	*Systemic, CNS*	5.60E-01	=	8.80E-04	+	NA	+	5.59E-01
Benzene	2.94E-07	=	2.94E-07	+	NA	+	1.01E-15	*Circulatory*	2.69E-01	=	1.34E-02	+	NA	+	2.56E-01
Xylenes (total)	NA	=	NA	+	NA	+	NA	*Systemic, CNS*	1.36E-01	=	1.86E-03	+	NA	+	1.34E-01
ILCR	***1E-04***	=	9E-05	+	1E-05	+	4E-10	**Soil HI**	5	=	0.4	+	0.03	+	4

Notes:

NA – Not Applicable
CR – Cancer Risk
HQ – Hazard Quotient
HI – Hazard Index
CNS – Central Nervous System

Target Organ HI	
Respiratory	3E+00
Systemic	4E+00
Circulatory	4E-01
Adrenal	4E-01
CNS	1E+00
Skin	9E-02

Table 36
Residential Risk Summary (0-3 feet)
Central Tendency Evaluation

	Total Carcinogenic Risk								Total Noncarcinogenic Risk						
Equation	CR	=	Ingestion	+	Dermal Contact	+	Inhalation		HQ	=	Ingestion	+	Dermal Contact	+	Inhalation
Units	unitless		unitless		unitless		unitless		unitless		unitless		unitless		unitless
Inorganics															
Arsenic	2.93E-06	=	2.83E-06	+	1.00E-07	+	4.27E-10		7.45E-02	=	6.33E-02	+	1.12E-02	+	NA
Lead	NA	=	NA	+	NA	+	NA		NA	=	NA	+	NA	+	NA
SVOCs															
1-Methylnaphthalene	NA	=	NA	+	NA	+	NA		1.14E-03	=	6.58E-04	+	4.79E-04	+	NA
Benzo(a)anthracene	3.07E-06	=	2.68E-06	+	3.91E-07	+	NA		NA	=	NA	+	NA	+	NA
Benzo(a)pyrene	3.71E-05	=	3.24E-05	+	4.73E-06	+	2.05E-10		NA	=	NA	+	NA	+	NA
Benzo(b)fluoranthene	3.26E-06	=	2.84E-06	+	4.15E-07	+	NA		NA	=	NA	+	NA	+	NA
Benzo(k)fluoranthene	1.05E-07	=	9.19E-08	+	1.34E-08	+	NA		NA	=	NA	+	NA	+	NA
Dibenzo(a,h)anthracene	2.10E-06	=	1.83E-06	+	2.67E-07	+	NA		NA	=	NA	+	NA	+	NA
Fluoranthene	NA	=	NA	+	NA	+	NA		3.09E-04	=	1.79E-04	+	1.30E-04	+	NA
Indeno(1,2,3-cd)pyrene	8.22E-07	=	7.17E-07	+	1.05E-07	+	NA		NA	=	NA	+	NA	+	NA
Naphthalene	NA	=	NA	+	NA	+	NA		1.19E-01	=	9.00E-04	+	6.55E-04	+	1.18E-01
Pyrene	NA	=	NA	+	NA	+	NA		7.03E-04	=	4.07E-04	+	2.96E-04	+	NA
VOCs															
1,2,4-Trimethylbenzene	NA	=	NA	+	NA	+	NA		6.76E-02	=	7.84E-05	+	NA	+	6.75E-02
1,3,5-Trimethylbenzene	NA	=	NA	+	NA	+	NA		3.12E-03	=	2.98E-06	+	NA	+	3.12E-03
Benzene	5.10E-07	=	3.12E-08	+	NA	+	4.78E-07		3.49E-02	=	1.43E-03	+	NA	+	3.35E-02
Soil CR	***5E-05***	=	4E-05	+	6E-06	+	5E-07	**Soil HI**	***0.3***	=	0.07	+	0.01	+	0.2

Notes:

NA – Not Applicable
CR – Cancer Risk
HQ – Hazard Quotient
HI – Hazard Index

Table 36
Residential Risk Summary (0-1 feet)
Reasonable Maximum Exposure

	Total Carcinogenic Risk								Total Noncarcinogenic Risk							
Chemical	**Total Risk**	**=**	**Ingestion**		**Dermal Contact**	**+**	**Inhalation**		**Total Hazard**	**=**	**Ingestion**	**+**	**Dermal Contact**	**+**	**Inhalation**
Inorganics															
Arsenic	3.62E-06	=	3.50E-06	+	1.24E-07	+	5.27E-10		8.09E-02	=	7.82E-02	+	2.76E-03	+	NA
Lead	NA	=	NA	+	NA	+	NA		NA	=	NA	+	NA	+	NA
SVOCs															
Benzo(a)pyrene	9.69E-07	=	8.46E-07	+	1.23E-07	+	5.35E-12		NA	=	NA	+	NA	+	NA
Soil CR	**5E-06**	=	4E-06	+	2E-07	+	5E-10	**Soil HI**	**0.1**	=	0.1	+	0.00	+	NA

Notes:

NA – Not Applicable
CR – Cancer Risk
HQ – Hazard Quotient
HI – Hazard Index

Table 38
Construction Worker Risk Summary
Reasonable Maximum Exposure

	Carcinogenic Risk								Target Organs	Noncarcinogenic Hazard						
Equation	CR	=	Ingestion	+	Dermal	+	Inhalation			HQ	=	Ingestion	+	Dermal	+	Inhalation
Units	unitless		unitless		unitless		unitless			unitless		unitless		unitless		unitless
Inorganics																
Arsenic	9.42E-08	=	8.29E-08	+	5.05E-09	+	6.31E-09		*Skin, Circulatory*	1.37E-02	=	1.29E-02	+	7.86E-04	+	NA
Lead	NA	=	NA	+	NA	+	NA			NA	=	NA	+	NA	+	NA
Manganese	NA	=	NA	+	NA	+	NA		*CNS*	1.42E-01	=	1.89E-03	+	NA	+	1.40E-01
Vanadium	NA	=	NA	+	NA	+	NA		*Skin*	3.86E-03	=	3.86E-03	+	NA	+	NA
SVOCs																
1-Methylnaphthalene	NA	=	NA	+	NA	+	NA		*Respiratory*	2.20E-02	=	1.76E-02	+	4.41E-03	+	NA
2-Methylnaphthalene	NA	=	NA	+	NA	+	NA		*Respiratory*	4.08E-01	=	3.42E-01	+	6.60E-02	+	NA
Acenaphthene	NA	=	NA	+	NA	+	NA		*Systemic*	6.90E-04	=	5.52E-04	+	1.38E-04	+	NA
Acenaphthylene	NA	=	NA	+	NA	+	NA			NA	=	NA	+	NA	+	NA
Benzo(a)anthracene	1.91E-06	=	1.52E-06	+	3.82E-07	+	NA			NA	=	NA	+	NA	+	NA
Benzo(a)pyrene	1.37E-05	=	1.10E-05	+	2.75E-06	+	3.51E-08			NA	=	NA	+	NA	+	NA
Benzo(b)fluoranthene	8.33E-07	=	6.66E-07	+	1.67E-07	+	NA			NA	=	NA	+	NA	+	NA
Benzo(k)fluoranthene	1.03E-07	=	8.25E-08	+	2.07E-08	+	NA			NA	=	NA	+	NA	+	NA
Chrysene	1.71E-08	=	1.36E-08	+	3.42E-09	+	NA			NA	=	NA	+	NA	+	NA
Dibenzo(a,h)anthracene	2.05E-06	=	1.64E-06	+	4.11E-07	+	NA			NA	=	NA	+	NA	+	NA
Dibenzofuran	NA	=	NA	+	NA	+	NA		*Systemic*	5.30E-02	=	4.24E-02	+	1.06E-02	+	NA
Fluoranthene	NA	=	NA	+	NA	+	NA		*Systemic, Circulatory*	7.57E-04	=	6.05E-04	+	1.52E-04	+	NA
Fluorene	NA	=	NA	+	NA	+	NA		*Circulatory*	7.71E-04	=	6.16E-04	+	1.55E-04	+	NA
Indeno(1,2,3-cd)pyrene	7.51E-07	=	6.01E-07	+	1.51E-07	+	NA			NA	=	NA	+	NA	+	NA
Naphthalene	NA	=	NA	+	NA	+	NA		*Respiratory, Systemic*	2.32E+01	=	5.00E-02	+	1.26E-02	+	2.32E+01
Pyrene	NA	=	NA	+	NA	+	NA		*Systemic*	1.41E-03	=	1.13E-03	+	2.83E-04	+	NA
VOCs																
1,2,3-Trimethylbenzene	NA	=	NA	+	NA	+	NA			NA	=	NA	+	NA	+	NA
1,2,4-Trichlorobenzene	NA	=	NA	+	NA	+	NA		*Systemic, Adrenal*	8.13E-01	=	2.27E-02	+	NA	+	7.90E-01
1,2,4-Trimethylbenzene	NA	=	NA	+	NA	+	NA		*Systemic, CNS*	2.20E-01	=	7.22E-05	+	NA	+	2.20E-01
1,3,5-Trimethylbenzene	NA	=	NA	+	NA	+	NA		*Systemic, CNS*	1.25E-01	=	3.37E-05	+	NA	+	1.25E-01
Benzene	9.10E-07	=	1.02E-08	+	NA	+	9.00E-07		*Circulatory*	2.73E-01	=	3.24E-03	+	NA	+	2.69E-01
Ethylbenzene	NA	=	NA	+	NA	+	NA		*Systemic, Teratology*	2.13E-02	=	3.93E-04	+	NA	+	2.09E-02
n-Butyl benzene	NA	=	NA	+	NA	+	NA		*Systemic*	1.27E-03	=	1.27E-03	+	NA	+	NA
p-Isopropyltoluene	NA	=	NA	+	NA	+	NA			NA	=	NA	+	NA	+	NA
sec-Butyl benzene	NA	=	NA	+	NA	+	NA		*Systemic*	1.08E-03	=	1.08E-03	+	NA	+	NA
Toluene	NA	=	NA	+	NA	+	NA		*Systemic, CNS*	1.83E-02	=	1.44E-05	+	NA	+	1.83E-02
Xylenes (total)	NA	=	NA	+	NA	+	NA		*Systemic, CNS*	9.05E-01	=	5.64E-04	+	NA	+	9.05E-01
Soil CR	**2E-05**	=	2E-05	+	4E-06	+	9E-07		**Soil HI**	**26**	=	1	+	0.1	+	26

Notes:

NA – Not Applicable
CR – Cancer Risk
HQ – Hazard Quotient
HI – Hazard Index
CNS – Central Nervous System

Target Organ HI	
Respiratory	2E+01
Circulatory	3E-01
Systemic	2E+01
Adrenal	8E-01
CNS	1E+00
Teratology	2E-02
Skin	4E-03

Table 39
Industrial Worker Indoor Air Risk Summary
Central Tendency Evaluation

	Carcinogenic Risk unitless		Noncarcinogenic Hazard unitless
1,2,4-Trimethylbenzene	NA		8.55E-01
1,3,5-Trimethylbenzene	NA		2.06E-01
1,4-Dichlorobenzene	NA		1.58E-01
Benzene	6.76E-06		1.69E-01
Carbon tetrachloride	3.63E-06		NA
Methylene chloride	5.18E-08		2.14E-04
Trichloroethylene	8.17E-06		1.05E-02
Air CR	**2E-05**	**Air HI**	**1**

Notes:

NA – Not Applicable
CR – Cancer Risk
HQ – Hazard Quotient
HI – Hazard Index

Table 40
Subsistence Fisher Risk Summary
Central Tendency Exposure

	Risk	Hazard
Shorthead Redhorse & Walleye		
1-Methylnaphthalene	NA	3.40E-04
Smelt		
Benzo(a)anthracene	1.64E-07	NA
Benzo(a)pyrene	1.92E-06	NA
Benzo(b)fluoranthene	2.48E-07	NA
Benzo(e)pyrene	NA	NA
Dibenzo(a,h)anthracene	5.75E-07	NA
Indeno(1,2,3-cd)pyrene	1.68E-07	NA
Total CR	3E-06	***HI*** 3E-04

Notes:

NA – Not Applicable
CR – Cancer Risk
HI – Hazard Index

Table 41
Residential Risk Summary (0-10 feet)
Excluding VOC Concentrations Exceeding Csat Values
Reasonable Maximum Exposure

	Total Carcinogenic Risk									Total Noncarcinogenic Risk						
Chemical	**Total Risk**	=	**Ingestion**		**Dermal Contact**	+	**Inhalation**	**Target Organs**	**Total Hazard**	=	**Ingestion**	+	**Dermal Contact**	+	**Inhalation**
Inorganics															
Arsenic	1.31E-05	=	1.19E-05	+	1.18E-06	+	1.72E-09	*Skin, Circulatory*	2.35E-01	=	2.16E-01	+	1.91E-02	+	NA
Lead	NA	=	NA	+	NA	+	NA		NA	=	NA	+	NA	+	NA
Thallium	NA	=	NA	+	NA	+	NA	*Systemic*	1.31E-02	=	1.31E-02	+	NA	+	NA
SVOCs															
1-Methylnaphthalene	NA	=	NA	+	NA	+	NA	*Respiratory*	3.74E-02	=	2.74E-02	+	9.98E-03	+	NA
2-Methylnaphthalene	NA	=	NA	+	NA	+	NA	*Respiratory*	5.89E-01	=	4.60E-01	+	1.29E-01	+	NA
Acenaphthene	NA	=	NA	+	NA	+	NA	*Systemic*	1.06E-03	=	7.76E-04	+	2.82E-04	+	NA
Benzo(a)anthracene	3.96E-05	=	2.81E-05	+	1.15E-05	+	NA		NA	=	NA	+	NA	+	NA
Benzo(a)pyrene	3.14E-04	=	2.23E-04	+	9.14E-05	+	1.36E-09		NA	=	NA	+	NA	+	NA
Benzo(b)fluoranthene	3.43E-05	=	2.43E-05	+	9.97E-06	+	NA		NA	=	NA	+	NA	+	NA
Benzo(k)fluoranthene	1.63E-06	=	1.16E-06	+	4.74E-07	+	NA		NA	=	NA	+	NA	+	NA
Chrysene	3.77E-07	=	2.67E-07	+	1.10E-07	+	NA		NA	=	NA	+	NA	+	NA
Dibenzo(a,h)anthracene	5.02E-05	=	3.56E-05	+	1.46E-05	+	NA		NA	=	NA	+	NA	+	NA
Dibenzofuran	NA	=	NA	+	NA	+	NA	*Systemic*	8.66E-02	=	6.35E-02	+	2.31E-02	+	NA
Fluoranthene	NA	=	NA	+	NA	+	NA	*Systemic, Circulatory*	2.77E-03	=	2.03E-03	+	7.40E-04	+	NA
Fluorene	NA	=	NA	+	NA	+	NA	*Circulatory*	2.00E-03	=	1.47E-03	+	5.34E-04	+	NA
Indeno(1,2,3-cd)pyrene	8.61E-06	=	6.11E-06	+	2.50E-06	+	NA		NA	=	NA	+	NA	+	NA
Naphthalene	NA	=	NA	+	NA	+	NA	*Systemic, Respiratory*	1.10E+01	=	1.19E-01	+	4.33E-02	+	1.08E+01
Phenanthrene	NA	=	NA	+	NA	+	NA		NA	=	NA	+	NA	+	NA
Pyrene	NA	=	NA	+	NA	+	NA	*Kidney*	4.98E-03	=	3.65E-03	+	1.33E-03	+	NA
VOCs															
1,2,4-Trichlorobenzene	NA	=	NA	+	NA	+	NA	*Systemic, Adrenal*	2.97E-01	=	3.79E-02	+	NA	+	2.59E-01
1,2,4-Trimethylbenzene	NA	=	NA	+	NA	+	NA	*Systemic, CNS*	9.26E-01	=	1.55E-03	+	NA	+	9.24E-01
1,3,5-Trimethylbenzene	NA	=	NA	+	NA	+	NA	*Systemic, CNS*	4.12E-02	=	5.66E-05	+	NA	+	4.12E-02
Benzene	1.92E-05	=	1.22E-06	+	NA	+	1.80E-05	*Circulatory*	7.83E-01	=	4.52E-02	+	NA	+	7.38E-01
Xylenes (total)	NA	=	NA	+	NA	+	NA	*Systemic, CNS*	1.47E-01	=	4.67E-04	+	NA	+	1.47E-01
Soil CR	***5E-04***	=	3E-04	+	1E-04	+	2E-05	**Soil HI**	***14***	=	1.0	+	0.2	+	13

Notes:

NA – Not Applicable
CR – Cancer Risk
HQ – Hazard Quotient
HI – Hazard Index

Target Organ HI	
Respiratory	1E+01
Systemic	1E+01
Circulatory	1E+00
Adrenal	3E-01
CNS	1E+00
Skin	2E-01

Table 42
Construction Worker Risk Summary
Excluding VOC Concentrations Exceeding Csat Values
Reasonable Maximum Exposure

	Carcinogenic Risk								Noncarcinogenic Hazard						
Equation	CR	=	Ingestion	+	Dermal	+	Inhalation	Target Organs	HQ	=	Ingestion	+	Dermal	+	Inhalation
Units	unitless		unitless		unitless		unitless		unitless		unitless		unitless		unitless
Inorganics															
								Skin							
Arsenic	3.67E-07	=	3.39E-07	+	1.88E-08	+	9.02E-09	*Circulatory*	5.56E-02	=	5.27E-02	+	2.92E-03	+	NA
Lead	NA	=	NA	+	NA	+	NA		NA	=	NA	+	NA	+	NA
SVOCs															
1-Methylnaphthalene	NA	=	NA	+	NA	+	NA	*Respiratory*	7.44E-02	=	6.06E-02	+	1.38E-02	+	NA
2-Methylnaphthalene	NA	=	NA	+	NA	+	NA	*Respiratory*	1.34E+00	=	1.14E+00	+	2.00E-01	+	NA
Acenaphthene	NA	=	NA	+	NA	+	NA	*Systemic*	2.91E-03	=	2.37E-03	+	5.41E-04	+	NA
Benzo(a)anthracene	8.16E-06	=	6.64E-06	+	1.52E-06	+	NA		NA	=	NA	+	NA	+	NA
Benzo(a)pyrene	7.15E-05	=	5.82E-05	+	1.33E-05	+	6.52E-08		NA	=	NA	+	NA	+	NA
Benzo(b)fluoranthene	4.22E-06	=	3.43E-06	+	7.83E-07	+	NA		NA	=	NA	+	NA	+	NA
Benzo(k)fluoranthene	4.91E-07	=	4.00E-07	+	9.11E-08	+	NA		NA	=	NA	+	NA	+	NA
Chyrsene	7.50E-08	=	6.11E-08	+	1.39E-08	+	NA		NA	=	NA	+	NA	+	NA
Dibenzo(a,h)anthracene	1.08E-05	=	8.82E-06	+	2.01E-06	+	NA		NA	=	NA	+	NA	+	NA
Dibenzofuran	NA	=	NA	+	NA	+	NA	*Systemic*	1.77E-01	=	1.44E-01	+	3.29E-02	+	NA
Fluoranthene	NA	=	NA	+	NA	+	NA	*Systemic, Circulatory*	3.26E-03	=	2.66E-03	+	6.06E-04	+	NA
Fluorene	NA	=	NA	+	NA	+	NA	*Circulatory*	2.80E-03	=	2.28E-03	+	5.20E-04	+	NA
Indeno(1,2,3-cd)pyrene	3.86E-06	=	3.15E-06	+	7.18E-07	+	NA		NA	=	NA	+	NA	+	NA
Naphthalene	NA	=	NA	+	NA	+	NA	*Respiratory, Systemic*	3.09E+01	=	1.89E-01	+	4.32E-02	+	3.06E+01
Pyrene	NA	=	NA	+	NA	+	NA	*Systemic*	6.41E-03	=	5.22E-03	+	1.19E-03	+	NA
VOCs															
1,2,4-Trichlorobenzene	NA	=	NA	+	NA	+	NA	*Systemic, Adrenal*	7.18E-02	=	5.45E-03	+	NA	+	6.64E-02
1,2,4-Trimethylbenzene	NA	=	NA	+	NA	+	NA	*Systemic, CNS*	2.45E-01	=	2.30E-04	+	NA	+	2.45E-01
1,3,5-Trimethylbenzene	NA	=	NA	+	NA	+	NA	*Systemic, CNS*	1.20E-02	=	9.28E-06	+	NA	+	1.20E-02
Benzene	1.15E-06	=	3.61E-08	+	NA	+	1.12E-06	*Circulatory*	3.46E-01	=	1.15E-02	+	NA	+	3.34E-01
Soil CR	***1E-04***	=	8E-05	+	2E-05	+	1E-06	**Soil HI**	**33**	=	2	+	0.3	+	31

Notes:

NA – Not Applicable
CR – Cancer Risk
HQ – Hazard Quotient
HI – Hazard Index

Target Organ HI	
Respiratory	3E+01
Systemic	3E+01
Circulatory	4E-01
Adrenal	7E-02
CNS	3E-01
Skin	6E-02

Table 43
Residential Risk Summary (0-10 feet)
Excluding VOC Concentrations Exceeding Csat Values
Central Tendency Evaluation

Chemical	Total Carcinogenic Risk							Target Organs	Total Noncarcinogenic Risk						
	Total Risk	=	Ingestion		Dermal Contact	+	Inhalation		Total Hazard	=	Ingestion		Dermal Contact	+	Inhalation
Inorganics															
Arsenic	4E-06	=	4E-06	+	1E-07	+	NA	*Skin* *Circulatory*	9E-02	=	9.16E-02	+	3.24E-03	+	3.40E-07
Lead	NA	=	NA	+	NA	+	NA		NA	=	NA	+	NA	+	NA
Thallium	NA	=	NA	+	NA	+	NA	*Systemic*	6E-02	=	6.26E-02	+	NA	+	NA
SVOCs															
1-Methylnaphthalene	NA	=	NA	+	NA	+	NA	*Respiratory*	8E-03	=	6.88E-03	+	1.00E-03	+	NA
2-Methylnaphthalene	NA	=	NA	+	NA	+	NA	*Respiratory*	1E-01	=	1.29E-01	+	1.44E-02	+	NA
Acenaphthene	NA	=	NA	+	NA	+	NA	*Systemic*	3E-03	=	2.32E-03	+	3.38E-04	+	NA
Benzo(a)anthracene	8E-06	=	7E-06	+	1E-06	+	NA		NA	=	NA	+	NA	+	NA
Benzo(a)pyrene	7E-05	=	6E-05	+	8E-06	+	4E-10		NA	=	NA	+	NA	+	NA
Benzo(b)fluoranthene	7E-06	=	6E-06	+	9E-07	+	NA		NA	=	NA	+	NA	+	NA
Benzo(k)fluoranthene	3E-07	=	3E-07	+	4E-08	+	NA		NA	=	NA	+	NA	+	NA
Chrysene	7E-08	=	7E-08	+	9E-09	+	NA		NA	=	NA	+	NA	+	NA
Dibenzo(a,h)anthracene	1E-05	=	9E-06	+	1E-06	+	NA		NA	=	NA	+	NA	+	NA
Dibenzofuran	NA	=	NA	+	NA	+	NA	*Systemic*	2E-02	=	1.49E-02	+	2.17E-03	+	NA
Fluoranthene	NA	=	NA	+	NA	+	NA	*Systemic, Circulatory*	7E-03	=	5.87E-03	+	8.55E-04	+	NA
Fluorene	NA	=	NA	+	NA	+	NA	*Circulatory*	5E-03	=	4.05E-03	+	5.90E-04	+	NA
Indeno(1,2,3-cd)pyrene	2E-06	=	2E-06	+	2E-07	+	NA		NA	=	NA	+	NA	+	NA
Naphthalene	NA	=	NA	+	NA	+	NA	*Respiratory, Systemic*	3E+00	=	1.55E-02	+	2.26E-03	+	2.74E+00
Phenanthrene	NA	=	NA	+	NA	+	NA		NA	=	NA	+	NA	+	NA
Pyrene	NA	=	NA	+	NA	+	NA	*Systemic*	1E-03	=	1.05E-03	+	1.53E-04	+	NA
VOCs															
1,2,4-Trichlorobenzene	NA	=	NA	+	NA	+	NA	*Systemic, Adrenal*	4E-01	=	9.87E-03	+	NA	+	3.42E-01
1,2,4-Trimethylbenzene	NA	=	NA	+	NA	+	NA	*Systemic, CNS*	3E-01	=	4.82E-04	+	NA	+	2.64E-01
1,3,5-Trimethylbenzene	NA	=	NA	+	NA	+	NA	*Systemic, CNS*	1E-01	=	1.87E-04	+	NA	+	1.19E-01
Benzene	3E-07	=	3E-07	+	NA	+	1E-15	*Circulatory*	3E-01	=	1.34E-02	+	NA	+	2.56E-01
Xylenes (total)	NA	=	NA	+	NA	+	NA	*Systemic, CNS*	3E-02	=	4.70E-04	+	NA	+	3.38E-02
Soil CR	***1E-04***	=	9E-05	+	1E-05	+	4E-10	**Soil HI**	**4**	=	0.4	+	0.03	+	4

Notes:

NA – Not Applicable
CR – Cancer Risk
HQ – Hazard Quotient
HI – Hazard Index

Target Organ HI	
Respiratory	3E+00
Systemic	4E+00
Circulatory	4E-01
Adrenal	4E-01
CNS	4E-01
Skin	9E-02

Table 44
Construction Worker Risk Summary
Excluding VOC Concentrations Exceeding Csat Values
Central Tendency Evaluation

	Carcinogenic Risk								Noncarcinogenic Hazard							
Equation	CR	=	Ingestion	+	Dermal	+	Inhalation	Target Organs	HQ	=	Ingestion	+	Dermal	+	Inhalation
Units	unitless		unitless		unitless		unitless		unitless		unitless		unitless		unitless
Inorganics															
Arsenic	8.82E-08	=	7.76E-08	+	4.73E-09	+	5.91E-09	*Skin Circulatory*	1.28E-02	=	1.21E-02	+	7.35E-04	+	NA
Lead	NA	=	NA	+	NA	+	NA		NA	=	NA	+	NA	+	NA
SVOCs															
1-Methylnaphthalene	NA	=	NA	+	NA	+	NA	*Respiratory*	1.38E-02	=	1.10E-02	+	2.76E-03	+	NA
2-Methylnaphthalene	NA	=	NA	+	NA	+	NA	*Respiratory*	2.47E-01	=	2.07E-01	+	3.99E-02	+	NA
Acenaphthene	NA	=	NA	+	NA	+	NA	*Systemic*	5.67E-04	=	4.53E-04	+	1.14E-04	+	NA
Benzo(a)anthracene	1.64E-06	=	1.31E-06	+	3.28E-07	+	NA		NA	=	NA	+	NA	+	NA
Benzo(a)pyrene	1.42E-05	=	1.13E-05	+	2.84E-06	+	3.63E-08		NA	=	NA	+	NA	+	NA
Benzo(b)fluoranthene	8.59E-07	=	6.86E-07	+	1.72E-07	+	NA		NA	=	NA	+	NA	+	NA
Benzo(k)fluoranthene	9.75E-08	=	7.79E-08	+	1.96E-08	+	NA		NA	=	NA	+	NA	+	NA
Chrysene	1.51E-08	=	1.20E-08	+	3.02E-09	+	NA		NA	=	NA	+	NA	+	NA
Dibenzo(a,h)anthracene	2.17E-06	=	1.74E-06	+	4.36E-07	+	NA		NA	=	NA	+	NA	+	NA
Dibenzofuran	NA	=	NA	+	NA	+	NA	*Systemic*	3.38E-02	=	2.70E-02	+	6.78E-03	+	NA
Fluoranthene	NA	=	NA	+	NA	+	NA	*Systemic, Circulatory*	6.57E-04	=	5.26E-04	+	1.32E-04	+	NA
Fluorene	NA	=	NA	+	NA	+	NA	*Circulatory*	5.54E-04	=	4.43E-04	+	1.11E-04	+	NA
Indeno(1,2,3-cd)pyrene	7.65E-07	=	6.12E-07	+	1.53E-07	+	NA		NA	=	NA	+	NA	+	NA
Naphthalene	NA	=	NA	+	NA	+	NA	*Respiratory, Systemic*	8.08E+00	=	3.43E-02	+	8.61E-03	+	8.04E+00
Pyrene	NA	=	NA	+	NA	+	NA	*Systemic*	1.29E-03	=	1.03E-03	+	2.58E-04	+	NA
VOCs															
1,2,4-Trichlorobenzene	NA	=	NA	+	NA	+	NA	*Systemic, Adrenal*	1.67E-02	=	7.88E-04	+	NA	+	1.59E-02
1,2,4-Trimethylbenzene	NA	=	NA	+	NA	+	NA	*Systemic, CNS*	5.91E-02	=	3.83E-05	+	NA	+	5.90E-02
1,3,5-Trimethylbenzene	NA	=	NA	+	NA	+	NA	*Systemic, CNS*	3.13E-03	=	1.67E-06	+	NA	+	3.13E-03
Benzene	2.44E-07	=	5.33E-09	+	NA	+	2.39E-07	*Circulatory*	7.31E-02	=	1.70E-03	+	NA	+	7.15E-02
Soil CR	**2E-05**	=	2E-05	+	4E-06	+	3E-07	**Soil HI**	9	=	0.3	+	0.06	+	8

Notes:

NA – Not Applicable
CR – Cancer Risk
HQ – Hazard Quotient
HI – Hazard Index

Target Organ HI	
Respiratory	8E+00
Systemic	6E-04
Circulatory	9E-02
Adrenal	2E-02
CNS	6E-02
Skin	1E-02

Table 45
Residential Risk Summary (0-3 feet)
Excluding VOCs Exceeding Csat Values
Reasonable Maximum Exposure

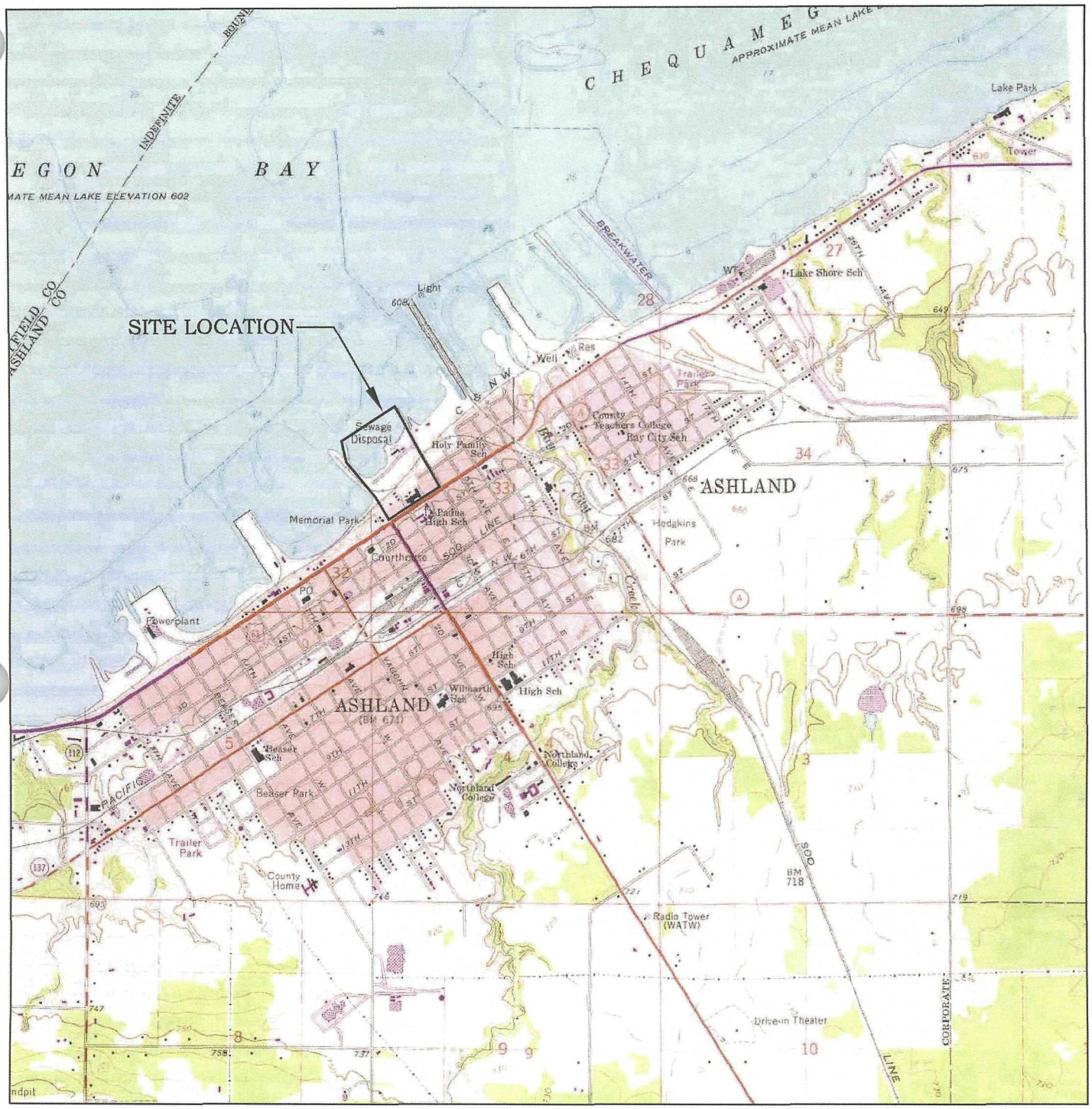
	Total Carcinogenic Risk								Total Noncarcinogenic Risk						
Chemical	**Total Risk**	**=**	**Ingestion**		**Dermal Contact**	**+**	**Inhalation**		**Total Hazard**	**=**	**Ingestion**	**+**	**Dermal Contact**	**+**	**Inhalation**
Inorganics															
Arsenic	9.65E-06	=	8.78E-06	+	8.74E-07	+	1.27E-09		1.73E-01	=	1.59E-01	+	1.41E-02	+	NA
Lead	NA	=	NA	+	NA	+	NA		NA	=	NA	+	NA	+	NA
SVOCs															
1-Methylnaphthalene	NA	=	NA	+	NA	+	NA		4.14E-03	=	3.03E-03	+	1.10E-03	+	NA
Benzo(a)anthracene	2.19E-05	=	1.55E-05	+	6.36E-06	+	NA		NA	=	NA	+	NA	+	NA
Benzo(a)pyrene	2.60E-04	=	1.84E-04	+	7.55E-05	+	1.12E-09		NA	=	NA	+	NA	+	NA
Benzo(b)fluoranthene	2.17E-05	=	1.54E-05	+	6.31E-06	+	NA		NA	=	NA	+	NA	+	NA
Benzo(k)fluoranthene	6.81E-07	=	4.83E-07	+	1.98E-07	+	NA		NA	=	NA	+	NA	+	NA
Dibenzo(a,h)anthracene	1.23E-05	=	8.70E-06	+	3.57E-06	+	NA		NA	=	NA	+	NA	+	NA
Fluoranthene	NA	=	NA	+	NA	+	NA		1.15E-03	=	8.46E-04	+	3.08E-04	+	NA
Indeno(1,2,3-cd)pyrene	5.47E-06	=	3.88E-06	+	1.59E-06	+	NA		NA	=	NA	+	NA	+	NA
Naphthalene	NA	=	NA	+	NA	+	NA		3.80E-01	=	4.13E-03	+	1.50E-03	+	3.75E-01
Pyrene	NA	=	NA	+	NA	+	NA		2.73E-03	=	2.00E-03	+	7.29E-04	+	NA
VOCs															
1,2,4-Trimethylbenzene	NA	=	NA	+	NA	+	NA		2.02E-01	=	3.38E-04	+	NA	+	2.02E-01
1,3,5-Trimethylbenzene	NA	=	NA	+	NA	+	NA		9.04E-03	=	1.24E-05	+	NA	+	9.02E-03
Benzene	2.64E-06	=	1.67E-07	+	NA	+	2.47E-06		1.08E-01	=	6.22E-03	+	NA	+	1.02E-01
Soil CR	3E-04	=	2E-04	+	9E-05	+	2E-06	**Soil HI**	0.9	=	0.2	+	0.02	+	0.7

Notes:

NA – Not Applicable
CR – Cancer Risk
HQ – Hazard Quotient
HI – Hazard Index

FIGURES

FIGURES



BASE MAP SOURCE: USGS 7.5 MINUTE TOPOGRAPHIC QUADRANGLE, ASHLAND, WISCONSIN, DATED 1964, PHOTOREVISED 1975.

QUADRANGLE LOCATION

NORTH

0 1320 2640 5280

SCALE IN FEET

PROJECT: ASHLAND/NSP LAKEFRONT SITE ASHLAND, WISCONSIN		
TITLE: FIGURE 1 SITE LOCATION MAP		
DRAWN BY: DDZ	SCALE: 1" = 2640'	PROJ. NO. 25688375
CHECKED BY: PJS	DATE: 31.JULY.2007	SHEET: 1 OF 7
APPROVED BY: DPT		

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10200 INNOVATION DRIVE, SUITE 500
MILWAUKEE, WISCONSIN 53226
414-831-4100

PROJECT: ASHLAND/NSP LAKEFRONT SITE ASHLAND, WISCONSIN			
TITLE: FIGURE 2 SITE FEATURES			
DRAWN BY: DDZ	SCALE: AS SHOWN	PROJECT: 25688375	
CHECKED BY: PLH	DATE: 31.JULY.2007	SHEET: 2 OF 7	
APPROVED BY: DPT			

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414-831-4100

PROJECT: ASHLAND/NSP LAKEFRONT SITE ASHLAND, WISCONSIN			
TITLE: FIGURE 3 FILLED RAVINE DETAIL			
DRAWN BY: DDZ	SCALE: AS SHOWN	PROJECT: 25688375	
CHECKED BY: PLH	DATE: 31.JULY.2007	SHEET: 3 OF 7	
APPROVED BY: DPT			

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MILWAUKEE, WISCONSIN 53226
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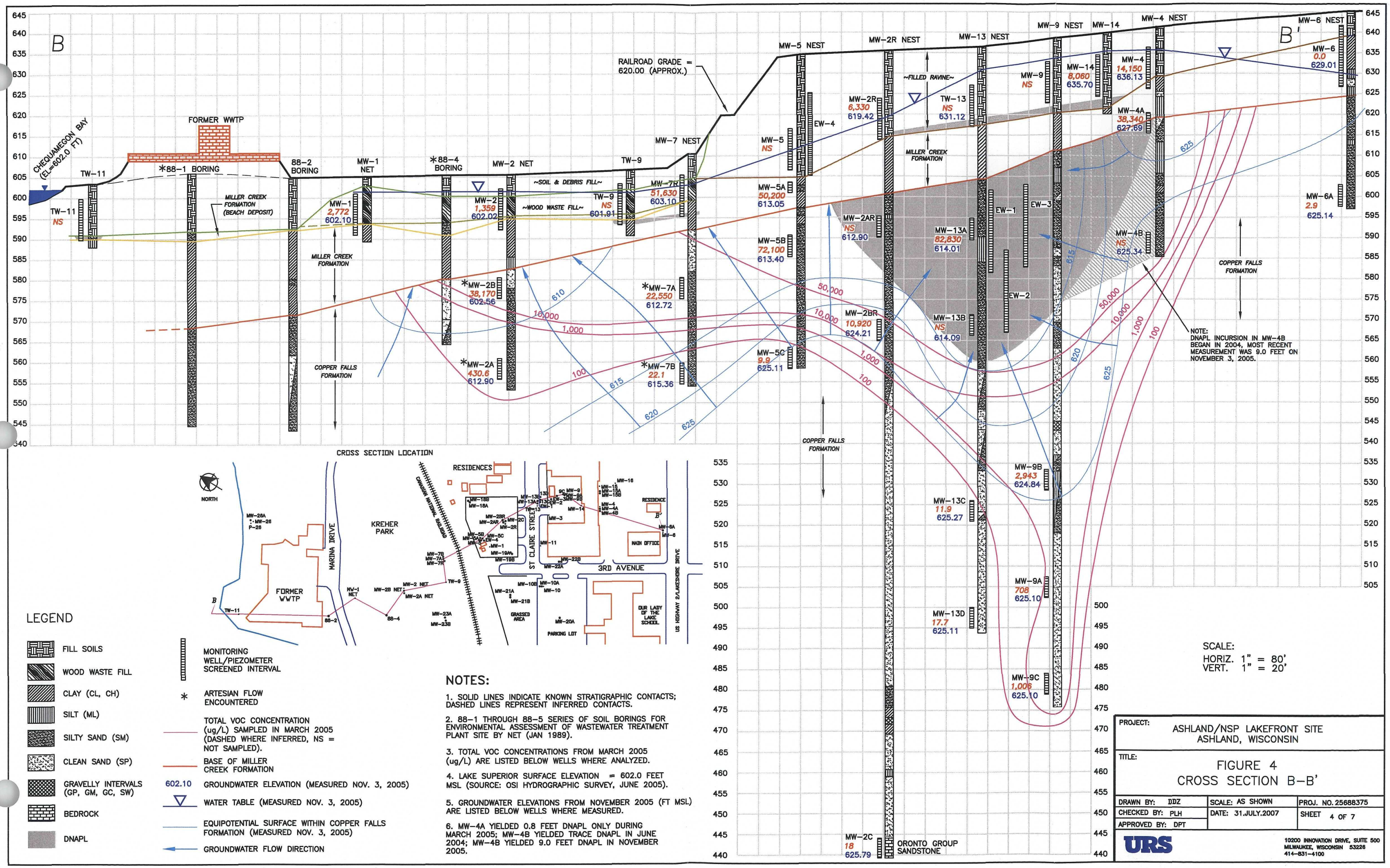
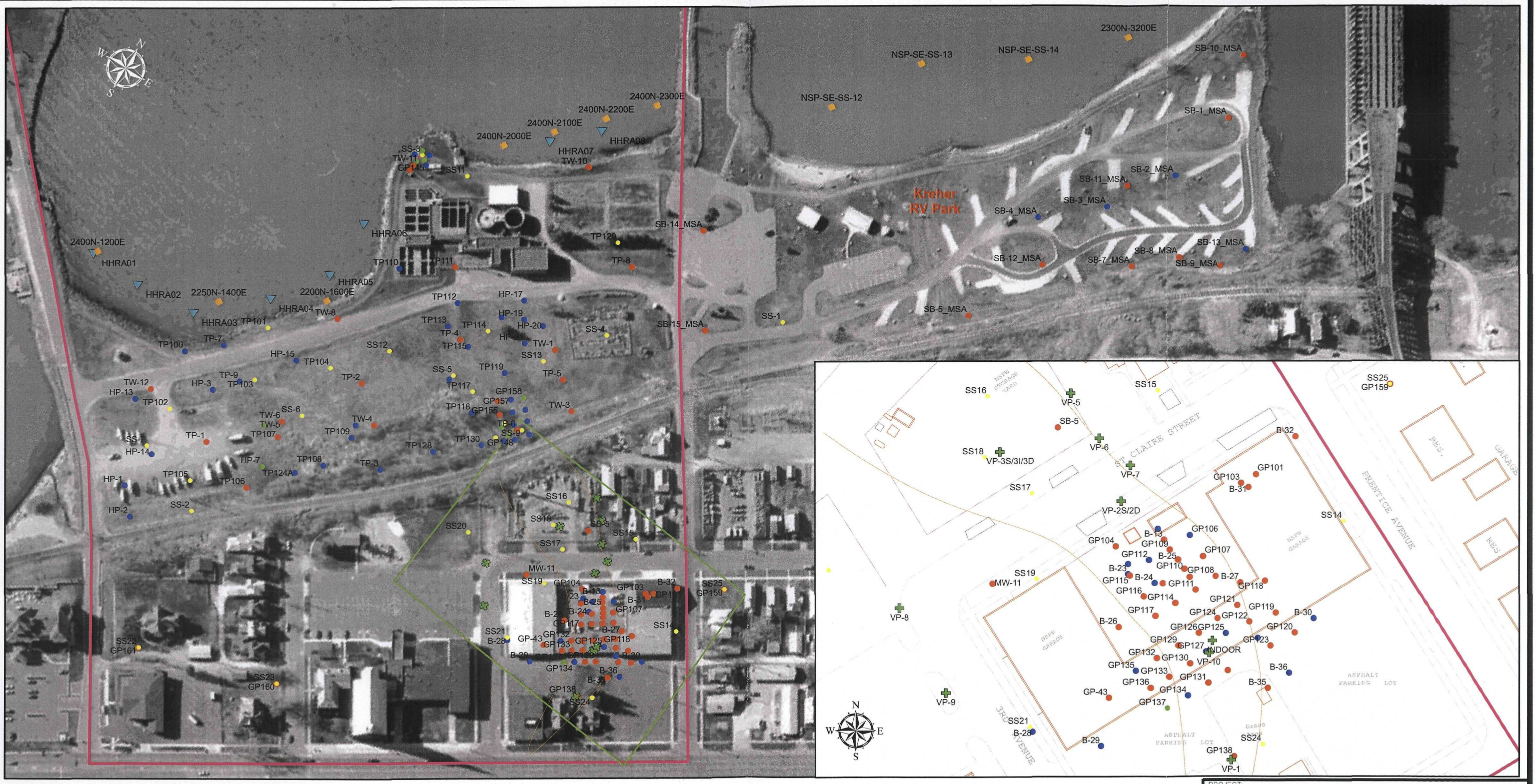

B
B'
CHEQUAMEGON BAY (EL=602.0 FT)
FORMER WWTP
TW-11
TW-11 NS
*88-1 BORING
MILLER CREEK FORMATION (BEACH DEPOSIT)
88-2 BORING
MW-1 NET
MW-1 2,772 602.10
*88-4 BORING
MW-2 NET
MW-2 1,359 602.02
~SOIL & DEBRIS FILL~
~WOOD WASTE FILL~
TW-9
TW-9 NS 601.91
MW-7 NEST
MW-7R 51,630 603.10
RAILROAD GRADE = 620.00 (APPROX.)
MILLER CREEK FORMATION
COPPER FALLS FORMATION
*MW-2B 38,170 602.56
*MW-2A 430.6 612.90
*MW-7A 22,550 612.72
*MW-7B 22.1 615.36
MW-5 NEST
MW-5 NS
MW-5A 50,200 613.05
MW-5B 72,100 613.40
MW-5C 9.9 625.11
EW-4
MW-2R NEST
MW-2R 6,330 619.42
MW-2AR NS 612.90
MW-2BR 10,920 624.21
~FILLED RAVINE~
TW-13 NS 631.12
MW-13 NEST
MW-13A 82,830 614.01
MW-13B NS 614.09
EW-1
EW-2
EW-3
MW-9 NEST
MW-9 NS
MW-9B 2,943 624.84
MW-9A 708 625.10
MW-9C 1,006 625.10
MW-14
MW-14 8,060 635.70
MW-4 NEST
MW-4 14,150 636.13
MW-4A 38,340 627.69
MW-4B NS 625.34
MW-6 NEST
MW-6 0.0 629.01
MW-6A 2.9 625.14
MW-13C 11.9 625.27
MW-13D 17.7 625.11
MW-2C 18 625.79
ORONTO GROUP SANDSTONE
NOTE: DNAPL INCURSION IN MW-4B BEGAN IN 2004, MOST RECENT MEASUREMENT WAS 9.0 FEET ON NOVEMBER 3, 2005.
CROSS SECTION LOCATION
NORTH
KREHER PARK
MARINA DRIVE
FORMER WWTP
CANADIAN NATIONAL RAILROAD
RESIDENCES
ST CLAIRE STREET
3RD AVENUE
MAIN OFFICE
RESIDENCE
GRASSED AREA
PARKING LOT
OUR LADY OF THE LAKE SCHOOL
US HIGHWAY 2/LAKESHORE DRIVE
LEGEND
FILL SOILS
WOOD WASTE FILL
CLAY (CL, CH)
SILT (ML)
SILTY SAND (SM)
CLEAN SAND (SP)
GRAVELLY INTERVALS (GP, GM, GC, SW)
BEDROCK
DNAPL
MONITORING WELL/PIEZOMETER SCREENED INTERVAL
ARTESIAN FLOW ENCOUNTERED
TOTAL VOC CONCENTRATION (ug/L) SAMPLED IN MARCH 2005 (DASHED WHERE INFERRED, NS = NOT SAMPLED).
BASE OF MILLER CREEK FORMATION
602.10 GROUNDWATER ELEVATION (MEASURED NOV. 3, 2005)
WATER TABLE (MEASURED NOV. 3, 2005)
EQUIPOTENTIAL SURFACE WITHIN COPPER FALLS FORMATION (MEASURED NOV. 3, 2005)
GROUNDWATER FLOW DIRECTION
NOTES:
1. SOLID LINES INDICATE KNOWN STRATIGRAPHIC CONTACTS; DASHED LINES REPRESENT INFERRED CONTACTS.
2. 88-1 THROUGH 88-5 SERIES OF SOIL BORINGS FOR ENVIRONMENTAL ASSESSMENT OF WASTEWATER TREATMENT PLANT SITE BY NET (JAN 1989).
3. TOTAL VOC CONCENTRATIONS FROM MARCH 2005 (ug/L) ARE LISTED BELOW WELLS WHERE ANALYZED.
4. LAKE SUPERIOR SURFACE ELEVATION = 602.0 FEET MSL (SOURCE: OSI HYDROGRAPHIC SURVEY, JUNE 2005).
5. GROUNDWATER ELEVATIONS FROM NOVEMBER 2005 (FT MSL) ARE LISTED BELOW WELLS WHERE MEASURED.
6. MW-4A YIELDED 0.8 FEET DNAPL ONLY DURING MARCH 2005; MW-4B YIELDED TRACE DNAPL IN JUNE 2004; MW-4B YIELDED 9.0 FEET DNAPL IN NOVEMBER 2005.
SCALE:
HORIZ. 1" = 80'
VERT. 1" = 20'
PROJECT: ASHLAND/NSP LAKEFRONT SITE ASHLAND, WISCONSIN
TITLE: FIGURE 4 CROSS SECTION B-B'
DRAWN BY: DDZ
CHECKED BY: PLH
APPROVED BY: DPT
SCALE: AS SHOWN
DATE: 31.JULY.2007
PROJ. NO. 25688375
SHEET 4 OF 7
URS
10200 INNOVATION DRIVE, SUITE 500 MILWAUKEE, WISCONSIN 53226 414-831-4100



Legend

- Sediment Locations
- Vapor probe locations
- Surface water locations
- NPL Site Boundary
- Former MGP inset map

Soil Samples - by depth

End depth - feet

- 0 - 1.0
- 1.1 - 2.0
- 2.1 - 5.0
- 5.1 - 10

0 75 150 300 450 600 Feet

Former MGP area detail

Note:
Site layout has been rotated from true north for display purposes only; Inset of former MGP area is shown in true north.

PROJECT: ASHLAND/NSP LAKEFRONT SITE
ASHLAND, WISCONSIN

TITLE: Figure 5
HHRA Sample Locations

DRAWN BY:	DDZ	SCALE:	AS SHOWN	PROJECT:	25688375
CHECKED BY:	PLH	DATE:	31.JULY.2007	SHEET:	5 of 7
APPROVED BY:	DPT				

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10200 INNOVATION DRIVE, SUITE 500
MILWAUKEE, WISCONSIN 53226
414-831-4100

Legend

- Fish Sample Locations
- NPL Site Boundary

0 500 1,000 2,000 3,000 4,000 Feet

PROJECT: ASHLAND/NSP LAKEFRONT SITE
ASHLAND, WISCONSIN

TITLE: Figure 6
Fish Sampling Locations

DRAWN BY:	DDZ	SCALE:	AS SHOWN	PROJECT:	25688375
CHECKED BY:	PLH	DATE:	31.JULY.2007	SHEET:	6 of 7
APPROVED BY:	DPT				

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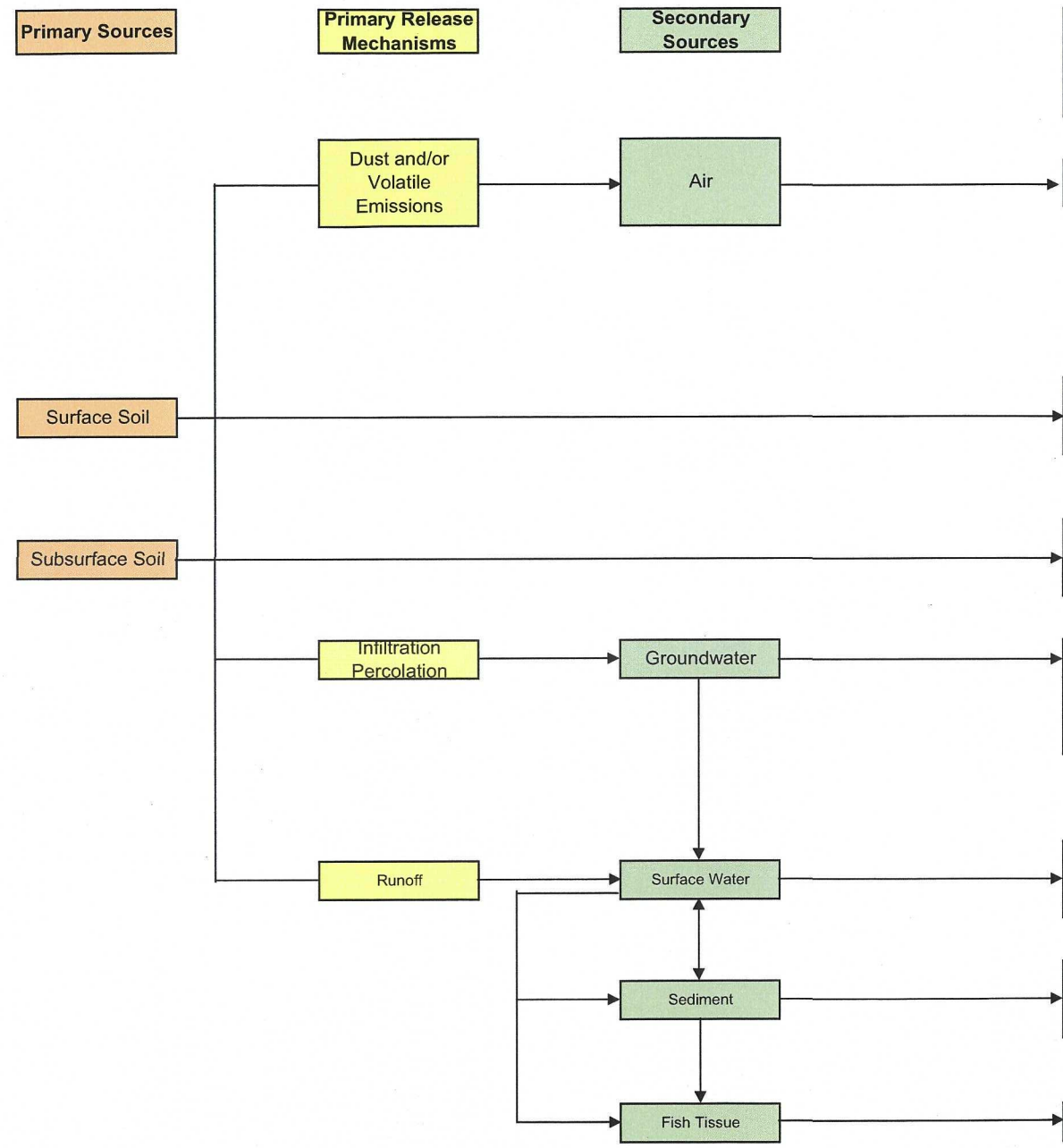
10200 INNOVATION DRIVE, SUITE 500
MILWAUKEE, WISCONSIN 53226
414-831-4100



Exposure Route	Human Receptors					
	Industrial Worker (a)	Maintenance Worker (b)	Construction Worker (b)	Recreational (c)	Fisher (d)	Residents (e)
Inhalation	●	●	●	●	○	●
Ingestion	●	●	●	●	○	●
Dermal	●	●	●	●	○	●
Ingestion	●	●	●	●	○	●
Dermal	●	●	●	●	○	●
Inhalation	○	○	●	●	○	○
Ingestion	○	○	○	●	○	○
Dermal	○	○	●	●	○	○
Ingestion	○	○	○	●	○	○
Dermal	○	○	○	●	○	○
Ingestion	○	○	○	●	○	○
Dermal	○	○	○	●	○	○
Ingestion	○	○	○	○	●	○

● Pathway potentially complete, further evaluation completed.

○ Pathway considered to be incomplete, no further evaluation recommended.

(a) Operational activities occur in limited areas.
(b) Maintenance activities include grass mowing or utility repair. Potential construction exposures may include workers who encounter impacted materials in the filled ravine or at Kreher Park.
(c) Representative of young children (0-6 years of age), older children and adolescents (7-18 years of age) and adults.
(d) Representative of adult subsistence fishers.
(e) Representative of children and adult residents in houses located near the filled ravine.

PROJECT: ASHLAND/NSP LAKEFRONT SITE ASHLAND, WISCONSIN		
TITLE: HUMAN HEALTH RISK ASSESSMENT FIGURE 7 CONCEPTUAL SITE MODEL		
DRAWN BY: JAH	SCALE:	PROJ. NO.:
CHECKED BY:	DATE: 09/06/07	SHEET
APPROVED BY: PLH		
URS		

ATTACHMENT A

EXPOSURE PARAMETERS, TOXICITY VALUES, AND CHEMICAL-SPECIFIC VALUES

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Attachment A

Table 1
Exposure Parameters – Residential Adult and Child

Exposure Pathway	Parameter	Description	Resident			
			CTE	Source	RME	Source
Incidental Soil Ingestion	SIRc	Soil Ingestion Rate, child (mg/day)	100	(a)	200	(a)
	SIRa	Soil Ingestion Rate, adult (mg/day)	50	(a)	100	(a)
	SIRadj	Age-adjusted Soil Ingestion Rate (mg-yr/kg-day)	46.43	Calculated	114.28	Equation 1
	EF	Exposure Frequency (days/year)	350	(b)	350	(b)
	EDc	Exposure Duration, child (years)	6	(b)	6	(b)
	EDa	Exposure Duration, adult (years)	9	(f)	24	(b)
	FI	Fraction Ingested (unitless)	1	(c)	1	(c)
	CF	Conversion Factor (kg/mg)	1E-06	–	1E-06	–
	BWc	Body Weight, child (kg)	15	(b)	15	(b)
	BWa	Body Weight, adult (kg)	70	(d)	70	(d)
	ATc	Averaging Time for Carcinogens (days)	25,550	(b)	25,550	(b)
	ATn	Averaging Time for Noncarcinogens (days)	2,190	(e)	2,190	(e)
Dermal Contact with Soil	SAc	Skin Surface Area Exposed, child (cm^2)	2,800	(f)	2,800	(f)
	SAa	Skin Surface Area Exposed, adult (cm^2)	5,700	(f)	5,700	(f)
	AFc	Soil-to-skin adherence factor, child (mg/cm^2-event)	0.04	(f)	0.2	(f)
	AFa	Soil-to-skin adherence factor, adult (mg/cm^2-event)	0.01	(f)	0.1	(f)
	CF	Conversion Factor (kg/mg)	1E-06	–	1E-06	–
	EDc	Exposure Duration, Child (years)	6	(b)	6	(b)
	EDa	Exposure Duration, adult (years)	9	(f)	24	(b)
	SCRadj	Age-adjusted Soil Contact Rate (mg-yr/k-event)	52.13	Calculated	360.28	Equation 2
	EF	Exposure Frequency (day/year)	350	(b)	350	(b)
	EV	Event Frequency (events/day)	1	(f)	1	(f)
	BWc	Body Weight, child (kg)	15	(b)	15	(b)
	BWa	Body Weight, adult (kg)	70	(d)	70	(d)
	ATc	Averaging Time for Carcinogens (days)	25,550	(b)	25,550	(b)
	ATn	Averaging Time for Noncarcinogens (days)	2,190	(e)	2,190	(e)
Inhalation of Soil-derived Chemicals	InhRc	Inhalation Rate, child (m^3/day)	7.2	(i)	10	(g)
	InhRa	Inhalation Rate, adult (m^3/day)	13.25	(j)	20	(b)
	SInhRadj	Age-adjusted Inhalation Rate (m^3-yr/kg-day)	4.58	Calculated	10.86	Equation 3
	EF	Exposure Frequency (day/year)	350	(b)	350	(b)
	EDc	Exposure Duration, child (years)	6	(b)	6	(b)
	EDa	Exposure Duration, adult (years)	9	(f)	24	(b)
	BWc	Body Weight, child (kg)	15	(b)	15	(b)
	BWa	Body Weight, adult (kg)	70	(d)	70	(d)
	ATc	Averaging Time for Carcinogens (days)	25,550	(b)	25,550	(b)
	ATn	Averaging Time for Noncarcinogens (days)	2,190	(e)	2,190	(e)
	PEF	Particulate Emission Factor (m^3/kg)	Calculated	See Table 3e	Calculated	See Table 3e
	VF	Volatilization Factor (m^3/kg)	Calculated	(h)	Calculated	(h)

Attachment A

Table 1
Exposure Parameters – Residential Adult and Child

Notes:
(a) US EPA, 1997. Table 1-2 of *Exposure Factors Handbook*, Volume I based on the mean or average value.
(b) US EPA, 1989. Exhibit 6-14 of RAGS, *Volume I, Human Health Evaluation Manual (Part A).*
(c) US EPA, 1989. *RAGS, Volume I, Human Health Evaluation Manual (Part A).* Default value that assumes all soil ingested is from the contaminated source.
(d) 70 kg body weight and 70 year lifetime are used to be consistent with the development of cancer slope factors.
(e) Averaging Time for noncarcinogens is calculated using the equation ED × 365 days.
(f) US EPA, 2004. RAGS, Volume 1, Human Health Evaluation Manual (Part E) Exhibit 3-2. Default event frequency.
(g) US EPA, 1997. Table 5-23 of *Exposure Factors Handbook,* Volume I.
(h) US EPA, 1996. Exhibit 1-1of *Soil Screening Guidance: User's Guide.*
(i) US EPA, 1997. Table 5-23. InhRc for the child resident was derived using time-weighted average InhRs of the infant and children long-term exposures for the <1 year, 1-2 year, and 3-5 year groups.
(j) US EPA, 1997. Table 5-23. InhRa for the adult resident was derived by averaging the male and female long-term exposure inhalation rates.

RME

The age-adjusted soil ingestion rate were calculated using Equation 1.

Equation 1: $$SIRadj=\left(\frac{IRa\times EDa}{BWa}\right)+\left(\frac{IRc\times EDc}{BWc}\right)=\left(\frac{100mg/day\times 24yr}{70kg}\right)+\left(\frac{200mg/day\times 6yr}{15kg}\right)=114.28\,{}^{mg-yr}/_{kg-day}$$

The age-adjusted soil contact rates were calculated using Equation 2.

Equation 2: $$SCRadj=\left(\frac{SAa\times EDa\times AFa}{BWa}\right)+\left(\frac{SAc\times EDc\times AFc}{BWc}\right)=\left(\frac{5700cm^2\times 24yr\times 0.07mg/cm^2-event}{70kg}\right)+\left(\frac{2800cm^2\times 6yr\times 0.2mg/cm^2-event}{15kg}\right)=360.28\,{}^{mg-yr}/_{kg-event}$$

The age-adjusted inhalation rates were calculated using Equation 3.

Equation 3: $$SInhRadj=\left(\frac{InhRa\times EDa}{BWa}\right)+\left(\frac{InhRc\times EDc}{BWc}\right)=\left(\frac{20\,m^3/day\times 24\,yr}{70kg}\right)+\left(\frac{10\,m^3/day\times 6yr}{15kg}\right)=6.857+4\,{}^{m^3-yr}/_{kg-day}=10.857\,{}^{m^3-yr}/_{kg-day}$$

CTE

The age-adjusted soil ingestion rate were calculated using Equation 1.

Equation 1: $$SIRadj=\left(\frac{IRa\times EDa}{BWa}\right)+\left(\frac{IRc\times EDc}{BWc}\right)=\left(\frac{50mg/day\times 9yr}{70kg}\right)+\left(\frac{100mg/day\times 6yr}{15kg}\right)=46.43\,{}^{mg-yr}/_{kg-day}$$

The age-adjusted soil contact rates were calculated using Equation 2.

Equation 2: $$SCRadj=\left(\frac{SAa\times EDa\times AFa}{BWa}\right)+\left(\frac{SAc\times EDc\times AFc}{BWc}\right)=\left(\frac{5700cm^2\times 9yr\times 0.01mg/cm^2-event}{70kg}\right)+\left(\frac{2800cm^2\times 6yr\times 0.04mg/cm^2-event}{15kg}\right)=52.13\,{}^{mg-yr}/_{kg-event}$$

The age-adjusted inhalation rates were calculated using Equation 3.

Equation 3: $$SInhRadj=\left(\frac{InhRa\times EDa}{BWa}\right)+\left(\frac{InhRc\times EDc}{BWc}\right)=\left(\frac{13.25\,m^3/day\times 9yr}{70kg}\right)+\left(\frac{7.2\,m^3/day\times 6yr}{15kg}\right)=4.58\,{}^{m^3-yr}/_{kg-day}$$

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Table 2
Exposure Parameters – Recreational Adult

Exposure Pathway	Parameter	Description	Exposure Parameters			
			CTE	Source	RME	Source
Incidental Soil Ingestion	IR	Soil Ingestion Rate (mg/day)	50	(a)	50	(a)
	EF	Exposure Frequency (days/year)	40	(b)	52	(c)
	ED	Exposure Duration (years)	9	(d)	30	(d)
	FI	Fraction Ingested (unitless)	0.5	(e)	0.5	(e)
	BW	Body Weight (kg)	70	(f)	70	(f)
	ATnc	Averaging Time for Noncarcinogenic Effects (days)	3,285	(g)	10,950	(g)
	ATc	Averaging Time for Carcinogenic Effects (days)	25,550	(h,i)	25,550	(h,i)
	CF	Unit Conversion Factor	1E-06	–	1E-06	–
Dermal Contact with Soil	SA	Skin Surface Area Exposed (cm^2)	4,373	(j)	4,373	(j)
	SSAF	Soil-to-Skin Adherence Factor (mg/cm^2)	0.01	(k)	0.07	(k)
	EF	Exposure Frequency (days/year)	40	(b)	52	(c)
	ED	Exposure Duration (years)	9	(d)	30	(d)
	EV	Event Frequency (event/day)	1	(l)	1	(l)
	BW	Body Weight (kg)	70	(f)	70	(f)
	ATnc	Averaging Time for Noncarcinogenic Effects (days)	3,285	(g)	10,950	(g)
	ATc	Averaging Time for Carcinogenic Effects (days)	25,550	(h,i)	25,550	(h,i)
	CF	Unit Conversion Factor	1E-06	–	1E-06	–
Inhalation of Soil-derived Chemicals	IR	Inhalation Rate (m^3/hour)	0.52	(m)	1	(n)
	ET	Exposure Time (hour/day)	2	(o)	2	(o)
	EF	Exposure Frequency (days/year)	40	(b)	52	(c)
	ED	Exposure Duration (years)	9	(d)	30	(d)
	PEF	Particulate Emission Factor (m^3/kg)	Calculated	See Attachment G	Calculated	See Attachment G
	VF	Volatilization Factor (m^3/kg)	Calculated	See Table 6c	Calculated	See Table 6c
	BW	Body Weight (kg)	70	(f)	70	(f)
	ATnc	Averaging Time for Noncarcinogenic Effects (days)	3,285	(g)	10,950	(g)
	ATc	Averaging Time for Carcinogenic Effects (days)	25,550	(h,i)	25,550	(h,i)
	CF	Unit Conversion Factor	1E-06	–	1E-06	–

Notes:
CTE - Central Tendency Evaluation
RME - Reasonable Maximum Exposure.

(a) US EPA, 1997. Exposure Factors Handbook, Table 1-2. The mean soil ingestion rate for children over 6 years of age.
(b) Conservative assumption for CTE (2 days/week during June, July, and August and 1 day/week during April, May, September, and October).
(c) Conservative assumption for RME (3 days/week during June, July, and August and 1 day/week during April, May, September, and October).
(d) Recreational adolescent is assumed to range in age from 7 to 18. Therefore, total exposure duration is 12 years.
(e) Site-specific value based on conclusions reached for the 1998 baseline human health risk assessment completed by SEH. The existing data for the site indicated that approximately half of the surface soil data have chemical detections.
(f) US EPA, 1997. Exposure Factors Handbook, Table 7-3. Body weight is the average of males and females aged 7 to 18.
(g) US EPA, 1989. RAGS, Volume I, Human Health Evaluation Manual (Part A). Averaging Time for noncarcinogens is equal to ED (year) × 365 days/year (US EPA, 1989).
(h) US EPA, 1989. RAGS, Volume I, Human Health Evaluation Manual (Part A).
(i) 70 year lifetime is used to be consistent with the development of cancer slope factors.
(j) US EPA, 1997. Exposure Factors Handbook. Average surface area of head, hands, forearms, and lower legs of males and females aged 7-18.
(k) US EPA, 2004. RAGS, Part E, Exhibit 3-5. Recommended soil-to-skin adherence factor for older children and adults, greater than 6 years of age.
(l) USEPA, 2004. RAGS, Part E, Exhibit 3-5. Default event frequency.
(m) US EPA, 1997. Exposure Factors Handbook, Table 5-23. Average inhalation rate of males and females aged 7 to 18.
(n) US EPA, 1997. Exposure Factors Handbook, Table 5-23. Inhalation rate for short-term exposure, light activity (adults and children).
(o) Each recreational event is assumed to last for 2 hours, based on the estimated time spent on outdoor activities (US EPA, 1997).
(p) USEPA. 1996. Soil Screening Guidance User's Guide. Equation 5. Default particulate emission factor.

Attachment A

Table 3
Exposure Parameters – Recreational Adolescent

Exposure Pathway	Parameter	Description	Exposure Parameters			
			CTE	Source	RME	Source
Incidental Soil Ingestion	IR	Soil Ingestion Rate (mg/day)	50	(a)	50	(a)
	EF	Exposure Frequency (days/year)	40	(b)	52	(c)
	ED	Exposure Duration (years)	12	(d)	12	(d)
	FI	Fraction Ingested (unitless)	0.5	(e)	0.5	(e)
	BW	Body Weight (kg)	47	(f)	47	(f)
	ATnc	Averaging Time for Noncarcinogenic Effects (days)	4,380	(g)	4,380	(g)
	ATc	Averaging Time for Carcinogenic Effects (days)	25,550	(h,i)	25,550	(h,i)
	CF	Unit Conversion Factor	1E-06	–	1E-06	–
Dermal Contact with Soil	SA	Skin Surface Area Exposed (cm^2)	4,373	(j)	4,373	(j)
	SSAF	Soil-to-Skin Adherence Factor (mg/cm^2)	0.01	(k)	0.07	(k)
	EF	Exposure Frequency (days/year)	40	(b)	52	(c)
	ED	Exposure Duration (years)	12	(d)	12	(d)
	EV	Event Frequency (event/day)	1	(l)	1	(l)
	BW	Body Weight (kg)	47	(f)	47	(f)
	ATnc	Averaging Time for Noncarcinogenic Effects (days)	4,380	(g)	4,380	(g)
	ATc	Averaging Time for Carcinogenic Effects (days)	25,550	(h,i)	25,550	(h,i)
	CF	Unit Conversion Factor	1E-06	–	1E-06	–
Inhalation of Soil-derived Chemicals	IR	Inhalation Rate (m^3/hour)	0.52	(m)	1	(n)
	ET	Exposure Time (hour/day)	2	(o)	2	(o)
	EF	Exposure Frequency (days/year)	40	(b)	52	(b)
	ED	Exposure Duration (years)	12	(d)	12	(d)
	PEF	Particulate Emission Factor (m^3/kg)	Calculated	See Attachment G	Calculated	See Attachment G
	VF	Volatilization Factor (m^3/kg)	Calculated	See Table 9c	Calculated	See Table 9c
	BW	Body Weight (kg)	47	(f)	47	(f)
	ATnc	Averaging Time for Noncarcinogenic Effects (days)	4,380	(g)	4,380	(g)
	ATc	Averaging Time for Carcinogenic Effects (days)	25,550	(h,i)	25,550	(h,i)
	CF	Unit Conversion Factor	1E-06	–	1E-06	–

Notes:
CTE - Central Tendency Evaluation
RME - Reasonable Maximum Exposure.

(a) US EPA, 1997. Exposure Factors Handbook, Table 1-2. The mean soil ingestion rate for children over 6 years of age.
(b) Conservative assumption for CTE (2 days/week during June, July, and August and 1 day/week during April, May, September, and October).
(c) Conservative assumption for RME (3 days/week during June, July, and August and 1 day/week during April, May, September, and October).
(d) Recreational adolescent is assumed to range in age from 7 to 18. Therefore, total exposure duration is 12 years.
(e) Site-specific value based on conclusions reached for the 1998 baseline human health risk assessment completed by SEH. The existing data for the site indicated that approximately half of the surface soil data have chemical detections.
(f) US EPA, 1997. Exposure Factors Handbook, Table 7-3. Body weight is the average of males and females aged 7 to 18.
(g) US EPA, 1989. RAGS, Volume I, Human Health Evaluation Manual (Part A). Averaging Time for noncarcinogens is equal to ED (year) × 365 days/year (US EPA, 1989).
(h) US EPA, 1989. RAGS, Volume I, Human Health Evaluation Manual (Part A).
(i) 70 year lifetime is used to be consistent with the development of cancer slope factors.
(j) US EPA, 1997. Exposure Factors Handbook. Average surface area of head, hands, forearms, and lower legs of males and females aged 7-18.
(k) US EPA, 2004. RAGS, Part E, Exhibit 3-5. Recommended soil-to-skin adherence factor for older children and adults, greater than 6 years of age.
(l) USEPA, 2004. RAGSE, Part E, Exhibit 3-5. Default event frequency.
(m) US EPA, 1997. Exposure Factors Handbook, Table 5-23. Average inhalation rate of males and females aged 7 to 18.
(n) US EPA, 1997. Exposure Factors Handbook, Table 5-23. Inhalation rate for short-term exposure, light activity (adults and children).
(o) Each recreational event is assumed to last for 2 hours, based on the estimated time spent on outdoor activities (US EPA, 1997).
(p) USEPA. 1996. Soil Screening Guidance User's Guide. Equation 5. Default particulate emission factor.

Attachment A

Table 4
Exposure Parameters – Recreational Child

Exposure Pathway	Parameter	Description	Exposure Parameters			
			CTE	Source	RME	Source
Incidental Soil Ingestion	IR	Soil Ingestion Rate (mg/day)	100	(a)	400	(b)
	EF	Exposure Frequency (days/year)	40	(c)	52	(c)
	ED	Exposure Duration (years)	6	(d)	6	(d)
	FI	Fraction Ingested (unitless)	0.5	(e)	0.5	(e)
	BW	Body Weight (kg)	15	(f)	15	(f)
	ATnc	Averaging Time for Noncarcinogenic Effects (days)	2,190	(g)	2,190	(g)
	ATc	Averaging Time for Carcinogenic Effects (days)	25,550	(h,i)	25,550	(h,i)
	CF	Unit Conversion Factor (kg/mg)	1E-06	–	1E-06	–
Dermal Contact with Soil	SA	Skin Surface Area Exposed (cm^2)	2,800	(j)	2,800	(j)
	SSAF	Soil-to-Skin Adherence Factor (mg/cm^2)	0.2	(k)	0.2	(k)
	EF	Exposure Frequency (days/year)	40	(c)	52	(c)
	ED	Exposure Duration (years)	6	(d)	6	(d)
	EV	Event Frequency (events/day)	1	(l)	1	(L)
	BW	Body Weight (kg)	15	(f)	15	(f)
	ATnc	Averaging Time for Noncarcinogenic Effects (days)	2,190	(g)	2,190	(g)
	ATc	Averaging Time for Carcinogenic Effects (days)	25,550	(h,i)	25,550	(h,i)
	CF	Unit Conversion Factor (kg/mg)	1E-06	–	1E-06	–
Inhalation of Soil-derived Chemicals	IR	Inhalation Rate (m^3/hour)	1	(m)	1.2	(n)
	ET	Exposure Time (hour/day)	2	(o)	2	(o)
	EF	Exposure Frequency (days/year)	40	(c)	52	(c)
	ED	Exposure Duration (years)	6	(d)	6	(d)
	PEF	Particulate Emission Factor (m^3/kg)	Calculated	See Attachment G	Calculated	See Attachment G
	VF	Volatilization Factor (m^3/kg)	Calculated	See Table 12c	Calculated	See Table 12c
	BW	Body Weight (kg)	15	(f)	15	(f)
	ATnc	Averaging Time for Noncarcinogenic Effects (days)	2,190	(g)	2,190	(g)
	ATc	Averaging Time for Carcinogenic Effects (days)	25,550	(h,i)	25,550	(h,i)
	CF	Unit Conversion Factor	1E-06	–	1E-06	–

Notes:
CTE - Central Tendency Evaluation
RME - Reasonable Maximum Exposure.

(a) US EPA, 1997. Exposure Factors Handbook, Table 4-23. The mean soil ingestion rate for children under 6 years of age over a short duration.
(b) US EPA, 1997. Exposure Factors Handbook, Table 4-23. The upperbound soil ingestion rate for children under 6 years of age over a short duration.
(c) Conservative assumption for CTE (2 days/week during June, July, and August and 1 day/week during April, May, September, and October) and RME (3 days/week during June, July, and August and 1 day/week during April, May, September, and October).
(d) Recreational children are assumed to range in age from 0 to 6. Therefore, total exposure duration is 6 years.
(e) Site-specific value based on conclusions reached for the 1998 baseline human health risk assessment completed by SEH. The existing data for the site indicated that approximately half of the surface soil data have chemical detections.
(f) US EPA, 1997. Exposure Factors Handbook, Table 7-3. Body weight is the average weight for children.
(g) Averaging Time for noncarcinogens is equal to ED (year) × 365 days/year (US EPA, 1989).
(h) US EPA, 1989. RAGS, Volume I, Human Health Evaluation Manual (Part A) .
(i) 70 year lifetime is used to be consistent with the development of cancer slope factors.
(j) US EPA, 2004. RAGS, Part E, Exhibit 3-5. Recommended default skin surface area for children.
(k) US EPA, 2004. RAGS, Part E, Exhibit 3-5. Recommended soil-to-skin adherence factor for older children.
(l) USEPA, 2004. RAGSE, Part E, Exhibit 3-5. Default event frequency.
(m) US EPA, 1997. Exposure Factors Handbook, Table 5-23. Mean inhalation rate for children at light activity level.
(n) US EPA, 1997. Exposure Factors Handbook, Table 5-23. Mean inhalation rate for children at moderate activity level.
(o) Each recreational event is assumed to last for 2 hours, based on the estimated time spent on outdoor activities (US EPA, 1997).
(p) USEPA. 1996. Soil Screening Guidance User's Guide. Equation 5. Default particulate emission factor.

Attachment A

Table 5
Exposure Parameters – Adult Swimmer and Wader

Exposure Pathway	Parameter	Description	Exposure Parameters			
			CTE	Source	RME	Source
Incidental Surface Water Ingestion	$WIR_{swimming}$	Surface Water Ingestion Rate (L/hour)	0.01	(a)	0.05	(b)
	WIR_{wading}	Surface Water Ingestion Rate (L/hour)	0.01	(a)	0.01	(a)
	EF	Exposure Frequency (events/year)	6	(c)	12	(d)
	ED	Exposure Duration (years)	9	(e)	30	(e)
	ET	Exposure Time (hours/event)	1 (swimming) 2 (wading)	(f)	1 (swimming) 2 (wading)	(f)
	BW	Body Weight (kg)	70	(g)	70	(g)
	ATn	Averaging Time for Noncarcinogenic Effects (days)	3,285	(l)	10,950	(l)
	ATc	Averaging Time Carcinogens (days)	25,550	(h,i, j)	25,550	(h,i, j)
Dermal Contact with Surface Water	$SA_{swimming}$	Skin Surface Area Exposed (cm²)	18,000	(k)	18,000	(k)
	SA_{wading}	Skin Surface Area Exposed (cm²)	9,000	(k)	9,000	(k)
	EF	Exposure Frequency (days/year)	6	(c)	12	(d)
	ED	Exposure Duration (years)	9	(e)	30	(e)
	EV	Event Frequency (events/day)	1		1	
	ET	Exposure Time (hours/event)	1 (swimming) 2 (wading)	(f)	1 (swimming) 2 (wading)	(f)
	BW	Body Weight (kg)	70	(g)	70	(g)
	ATn	Averaging Time for Noncarcinogenic Effects (days)	3,285	(l)	10,950	(l)
	ATc	Averaging Time Carcinogens (days)	25,550	(h,i, j)	25,550	(h,i, j)
Incidental Sediment Ingestion	SIR	Sediment Ingestion Rate (mg/hour)	5.60E-02	(m)	2.80E-01	(m)
	CF	Conversion Factor (kg/mg)	1E-06	--	1E-06	--
	EF	Exposure Frequency (days/year)	6	(c)	12	(d)
	ED	Exposure Duration (years)	9	(e)	30	(e)
	FI	Fraction Ingested from Contaminated Source (unitless)	1		1	
	BW	Body Weight (kg)	70	(g)	70	(g)
	ATn	Averaging Time for Noncarcinogenic Effects (days)	3,285	(l)	10,950	(l)
	ATc	Averaging Time Carcinogens (days)	25,550	(h,i, j)	25,550	(h,i, j)
Dermal Contact with Sediment	SA	Skin Surface Area Exposed (cm²)	4,499	(n)	5,672	(n)
	SSAF	Soil-to-Skin Adherence Factor (mg/cm²)	0.5	(o)	1	(o)
	CF	Conversion Factor (kg/mg)	1E-06	--	1E-06	--
	EF	Exposure Frequency (days/year)	6	(c)	12	(d)
	ED	Exposure Duration (years)	9	(e)	30	(e)
	EV	Event Frequency (events/day)	1		1	
	BW	Body Weight (kg)	70	(g)	70	(g)
	ATn	Averaging Time for Noncarcinogenic Effects (days)	3,285	(l)	10,950	(l)
	ATc	Averaging Time Carcinogens (days)	25,550	(h,i, j)	25,550	(h,i, j)

Notes:
CTE - Central Tendency Evaluation
RME - Reasonable Maximum Exposure.

(a) US EPA, 1989. Risk Assessment Guidance for Superfund, Volume I (RAGS, Part A). Value is one-fifth of that assumed to occur during a swimming event.
(b) US EPA, 1989. RAGS Part A. Value for a swimming event.
(c) One event per month for the 6 warmest months of the year.
(d) Two events per month for the 6 warmest months of the year.
(e) The default values for a residential adult, assuming the individual resides in the same residence for 9 (average) or 30 years (upperbound).
(f) US EPA, 1997. Exposure Factors Handbook, Table 1-2. Recommended values for exposure frequency of 1event per month and exposure time of 1 hours per event for swimming. The exposure time of 2 hours per event for wading is based on the estimated time spent on outdoor activities.
(g) US EPA, 1997. Exposure Factors Handbook. Body weight is the average of adult males and females
(h) 70 year lifetime is used to be consistent with the development of cancer slope factors.
(i) US EPA, 1991. Standard Default Exposure Factors. Value for adult.
(j) US EPA, 1989. RAGS, Part A.
(k) Value represents recommended skin surface area (100% for swimmers and 50% for waders).
(l) US EPA, 1989. RAGS, Part A. Averaging Time for noncarcinogens is equal to ED (year) × 365 days/year.
(m) Calculated using the equation: Sediment Ingestion Rate (mg/hour) = Surface water ingestion rate (ml/hour) × total solids (mg/ml). The average total suspended solids is 0.0056 mg/mL.
(n) The RME skin surface area represents the sum of the surface area for forearms, hands, lower legs and feet. The CTE skin surface area represents the sum of the skin surface area for hands, lower legs and feet.
(o) US EPA, 1997. Exposure Factors Handbook, (Table ??). Using loadings for the hands, arms, and legs for the irrigation worker, a weighted soil-to-skin adherence factor of 2.0 mg/cm² was calculated. One-half this value (1.0 mg/cm²) used as RME assuming some

Attachment A

Table 6
Exposure Parameters – Adolescent Swimmer and Wader

Exposure Pathway	Parameter	Description	Exposure Parameters			
			CTE	Source	RME	Source
Incidental Surface Water Ingestion	$WIR_{swimming}$	Surface Water Ingestion Rate (L/hour)	0.01	(a)	0.05	(b)
	WIR_{wading}	Surface Water Ingestion Rate (L/hour)	0.01	(a)	0.01	(a)
	EF	Exposure Frequency (events/year)	6	(c)	12	(d)
	ED	Exposure Duration (years)	12	(e)	12	(e)
	ET	Exposure Time (hours/event)	1 (swimming) 2 (wading)	(f)	1 (swimming) 2 (wading)	(f)
	BW	Body Weight (kg)	47	(g)	47	(g)
	ATnc	Averaging Time for Non-Carcinogenic Effects (days)	4,380	(l)	4,380	(l)
	ATc	Averaging Time Carcinogens (days)	25,550	(h,i, j)	25,550	(h,i, j)
Dermal Contact with Surface Water	$SA_{swimming}$	Skin Surface Area Exposed (cm^2)	13,533	(k)	13,533	(k)
	SA_{wading}	Skin Surface Area Exposed (cm^2)	6,767	(k)	6,767	(k)
	EF	Exposure Frequency (days/year)	6	(c)	12	(d)
	ED	Exposure Duration (years)	12	(e)	12	(e)
	EV	Event Frequency (events/day)	1		1	
	ET	Exposure Time (hours/event)	1 (swimming) 2 (wading)	(f)	1 (swimming) 2 (wading)	(f)
	BW	Body Weight (kg)	47	(g)	47	(g)
	ATnc	Averaging Time for Non-Carcinogenic Effects (days)	4,380	(l)	4,380	(l)
	ATc	Averaging Time Carcinogens (days)	25,550	(h,i, j)	25,550	(h,i, j)
Incidental Sediment Ingestion	SIR	Sediment Ingestion Rate (mg/day)	5.60E-02	(m)	2.80E-04	(m)
	CF	Conversion Factor (kg/mg)	1E-06	–	1E-06	--
	EF	Exposure Frequency (days/year)	6	(c)	12	(d)
	ED	Exposure Duration (years)	12	(e)	12	(e)
	FI	Fraction Ingested from Contaminated Source (unitless)	1		1	
	BW	Body Weight (kg)	47	(g)	47	(g)
	ATnc	Averaging Time for Non-carcinogenic Effects (days)	4,380	(l)	4,380	(l)
	ATc	Averaging Time Carcinogens (days)	25,550	(h,i, j)	25,550	(h,i, j)
Dermal Contact with Sediment	SA	Skin Surface Area Exposed (cm^2)	3,259	(n)	4,046	(n)
	SSAF	Soil-to-Skin Adherence Factor (mg/cm^2-event)	0.5	(o)	1	(o)
	CF	Conversion Factor (kg/mg)	1E-06	--	1E-06	--
	EF	Exposure Frequency (days/year)	6	(c)	12	(d)
	ED	Exposure Duration (years)	12	(e)	12	(e)
	EV	Event Frequency (events/day)	1		1	
	BW	Body Weight (kg)	47	(d)	47	(d)
	ATnc	Averaging Time for Non-carcinogenic Effects (days)	4,380	(e)	4,380	(e)
	ATc	Averaging Time Carcinogens (days)	25,550	(h,i, j)	25,550	(h,i, j)

Notes:
CTE - Central Tendency Evaluation
RME - Reasonable Maximum Exposure.

(a) US EPA, 1989. Risk Assessment Guidance for Superfund, Volume I (Part A) . Value is one-fifth of that assumed to occur during a swimming event.
(b) US EPA, 1989. RAGS Part A. Value for a swimming event.
(c) One event per month for the 6 warmest months of the year.
(d) Two events per month for the 6 warmest months of the year.
(e) Recreational adolescent is assumed to range in age from 7 to 18. Therefore, total exposure duration is 12 years.
(f) US EPA. 1997. Exposure Factors Handbook, Table 1-2. Recommended exposure frequency of 1 event/month and exposure time of 1 hour per event for swimming. The exposure time of 2 hours per event for wading is based on the estimated time spent on outdoor activities .
(g) US EPA, 1997. Exposure Factors Handbook . Body weight is the average of males and females aged 7 to 18.
(h) 70 year lifetime is used to be consistent with the development of cancer slope factors.
(i) US EPA, 1991. Standard Default Exposure Factors. Value for adult.
(j) US EPA, 1989. RAGS Part A.
(k) Value represents average total body surface area of males and females aged 7 to 18. Assumes 100% of skin surface exposed while swimming. The wader's skin surface area is 50% of the swimming value.
(l) US EPA, 1989. RAGS Part A. Averaging Time for noncarcinogens is equal to ED (year) × 365 days/year .
(m) Calculated using the following equation: Sediment Ingestion Rate (mg/day) = Surface water ingestion rate (ml/day) × total solids (mg/ml).
(n) The RME skin surface area represents the sum of the surface area for forearms, hands, lower legs and feet. The CTE skin surface area represents the sum of the skin surface area for hands, lower legs and feet.
(o) US EPA, 1997. Exposure Factors Handbook. Using loadings for the hands, arms, and legs for the irrigation worker reported in, a weighted soil-to-skin adherence factor of 2.0 mg/cm^2 was calculated. One-half this value (1.0 mg/cm^2) used as RME assuming some washoff; 0.5 mg/cm^2 used for CTE.

Attachment A

Table 7
Exposure Parameters – Industrial Worker

Exposure Pathway	Parameter	Description	Exposure Parameters			
			CTE	Source	RME	Source
Incidental Soil Ingestion	IR	Soil Ingestion Rate (mg/day)	50	(a)	50	(a)
	EF	Exposure Frequency (days/year)	219	(c,e)	250	(c,e)
	ED	Exposure Duration (years)	6.6	(d,e)	25	(d,e)
	FI	Fraction Ingested (unitless)	1	(e)	1	(e)
	BW	Body Weight (kg)	70	(b,g,h)	70	(b,g,h)
	ATnc	Averaging Time for Noncarcinogenic Effects (days)	2,409	(f)	9,125	(f)
	ATc	Averaging Time for Carcinogenic Effects (days)	25,550	(b,g,h)	25,550	(b,g,h)
	CF	Unit Conversion Factor (kg/mg)	1E-06	–	1E-06	–
Dermal Contact with Soil	SA	Skin Surface Area Exposed (cm²)	3,300	(j)	3,300	(j)
	SSAF	Soil-to-Skin Adherence Factor (mg/cm²)	0.02	(k)	0.1	(k)
	CF	Conversion Factor (kg/mg)	1E-06	--	1E-06	--
	EF	Exposure Frequency (days/year)	219	(c,e)	250	(c,e)
	ED	Exposure Duration (years)	6.6	(d,e)	25	(d,e)
	EV	Event Frequency (events/day)	1	(i)	1	(i)
	BW	Body Weight (kg)	70	(b,g,h)	70	(b,g,h)
	ATnc	Averaging Time for Noncarcinogenic Effects (days)	2,409	(f)	9,125	(f)
	ATc	Averaging Time for Carcinogen Effects (days)	25,550	(b,g,h)	25,550	(b,g,h)
	CF	Unit Conversion Factor (kg/mg)	1E-06	–	1E-06	–
Inhalation of Soil-derived Chemicals	IR	Inhalation Rate (m³/hour)	0.55	(m)	1	(n)
	ET	Exposure Time (hour/day)	8	(o)	8	(o)
	EF	Exposure Frequency (days/year)	219	(c,e)	250	(c,e)
	ED	Exposure Duration (years)	6.6	(d,e)	25	(d,e)
	BW	Body Weight (kg)	70	(b,g,h)	70	(b,g,h)
	PEF	Particulate Emission Factor (m³/kg)	Calculated	See Attachment G	Calculated	See Attachment G
	VF	Volatilization Factor (m³/kg)	Calculated		Calculated	
	ATnc	Averaging Time for Noncarcinogenic Effects (days)	2,409	(f)	9,125	(f)
	ATc	Averaging Time for Carcinogen Effects (days)	25,550	(b,g,h)	25,550	(b,g,h)
	CF	Unit Conversion Factor (kg/mg)	1E-06	–	1E-06	–

Notes:
CTE - Central Tendency Evaluation
RME - Reasonable Maximum Exposure.

(a) US EPA, 1997. Table 1-2 of Exposure Factors Handbook , Volume I based on mean or average value.
(b) US EPA, 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites.
(c) The value of 250 days/year is recommended as the upperbound EF value in the Exposure Factors Handbook . The value of 219 days/year was used as the average EF value in USEPA's Adult Lead Model (US EPA, 2003).
(d) Values represent the 95th (for RME) and 50th percentile (for CTE) of occupational tenure, as provided in the Exposure Factors Handbook.
(e) Site-/Area-specific values to be used, if available.

(f) US EPA, 1989. RAGS, Volume I, Human Health Evaluation Manual (Part A). Averaging Time for noncarcinogens is equal to ED (year) × 365 days/year.
(g) US EPA, 1989. RAGS, Volume I, Human Health Evaluation Manual (Part A).
(h) 70 kg body weight and 70 year lifetime are used to be consistent with the development of cancer slope factors.
(i) US EPA, 2004. RAGS Part E, Exhibit 3-5.
(j) Suggested value represents the total skin surface area for head and hands, assuming that a worker wears long sleeve shirts, long pants and shoes. *This value may be adjusted based on the following body part-specific surface area (mean, in cm²), as provided in Table 6-4 of the* Exposure Factors Handbook (US EPA, 1997).

Change note for (j) to Exhibit 3-5 USEPA 2004.

	Men	Women
Head	1,180	1,100
Hands	840	746
Forearms	1,140	1,360
Lower Legs	2,070	1,940
Feet	1,120	975

(k) USEPA, 2004. RAGS Part E, Exhibit 3-3. *Recommended default values for a groundskeeper.*
(l) USEPA, 2004. RAGSE, Part E, Exhibit 3-5. Default event frequency.
(m) US EPA, 1997. Exposure Factors Handbook, Table 5-23. Based on the calculated average of recommended mean values for male and female.
(n) US EPA, 1997. Exposure Factors Handbook, Table 5-23. Value for short-term exposures for adults at light activity level.
(o) US EPA, 1997. Exposure Factors Handbook, Table 15-68. Represents the 50th percentile value for time spent at work (males and females, all ages).
(p) USEPA. 1996. Soil Screening Guidance User's Guide. Equation 5. Default particulate emission factor.

Attachment A

Table 8
Exposure Parameters – Maintenance/Utility Worker

Exposure Pathway	Parameter	Description	Exposure Parameters			
			CTE	Source	RME	Source
Incidental Soil Ingestion	IR	Soil Ingestion Rate (mg/day)	50	(a)	100	(b)
	EF	Exposure Frequency (days/year)	22	(c)	22	(c)
	ED	Exposure Duration (years)	6.6	(d)	25	(d)
	FI	Fraction Ingested (unitless)	0.5	(e)	0.5	(e)
	BW	Body Weight (kg)	70	(f,g,h)	70	(f,g,h)
	ATnc	Averaging Time for Noncarcinogenic Effects (days)	2,409	(i)	9,125	(i)
	ATc	Averaging Time for Carcinogenic Effects (days)	25,550	(f,g,h)	25,550	(f,g,h)
Dermal Contact with Soil	SA	Skin Surface Area Exposed (cm^2)	3,300	(j)	3,300	(j)
	SSAF	Soil to Skin Adherence Factor (mg/cm^2)	0.02	(k)	0.1(Maintenance) 0.2 (Utility)	(k)
	CF	Conversion Factor (kg/mg)	1E-06	--	1E-06	--
	EF	Exposure Frequency (days/year)	22	(c)	22	(c)
	ED	Exposure Duration (years)	6.6	(d)	25	(d)
	EV	Event Frequency (events/day)	1		1	
	BW	Body Weight (kg)	70	(f,g,h)	70	(f,g,h)
	ATnc	Averaging Time for Noncarcinogenic Effects (days)	2,409	(i)	9,125	(i)
	ATc	Averaging Time for Carcinogenic Effects (days)	25,550	(f,g,h)	25,550	(f,g,h)
Inhalation of Soil Derived Chemicals or Groundwater VOCs	IR	Inhalation Rate (m^3/hour)	1.3	(l)	1.5	(m)
	ET	Exposure Time (hour/day)	8	(n)	8	(n)
	EF	Exposure Frequency (days/year)	22	(c)	22	(c)
	ED	Exposure Duration (years)	6.6	(d)	25	(d)
	BW	Body Weight (kg)	70	(f,g,h)	70	(f,g,h)
	PEF	Particulate Emission Factor (m^3/kg)	Calculated	See Attachment G	Calculated	See Attachment G
	VF	Volatilization Factor (m^3/kg)	Calculated		Calculated	
	ATnc	Averaging Time for Noncarcinogenic Effects (days)	2,409	(i)	9,125	(i)
	ATc	Averaging Time for Carcinogenic Effects (days)	25,550	(f,g,h)	25,550	(f,g,h)

Notes:
CTE - Central Tendency Exposure
RME - Reasonable Maximum Exposure.

(a) US EPA, 1997. Table 1-2 of Exposure Factors Handbook, Volume I based on mean or average value for adults.
(b) US EPA, 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites, Exhibit 1-2.
(c) Assumes that maintenance workers is present mowing the grass 1 day per week between May 1 and October 1. Assumption based on Chequamegon Bay being icebound between December and April and the average maximum temperatures between May and October.
(d) USEPA, 1997. Table 1-2 of Exposure Factors Handbook, Volume I based on the occupational tenure for adults 16 years and older.
(e) Based on value provided in the SEH Baseline Human Health Risk Assessment (SEH, 1998) which assumes that FI is based on the distribution of contaminants.
(f) US EPA, 1991. Human Health Evaluation Manual, Supplemental Guidance, Standard Default Exposure Factors.
(g) US EPA, 1989. RAGS, Volume I, Human Health Evaluation Manual (Part A).
(h) 70 kg body weight and 70 year lifetime are used to be consistent with the development of cancer slope factors.
(i) Averaging Time for noncarcinogens is equal to ED (year) × 365 days/year (US EPA, 1989).
(j) Suggested value represents the total skin surface area for head and hands, assuming that a worker wears long sleeve shirts, long pants and shoes. This value may be adjusted based on the following body part-specific surface area (mean, in cm^2), as provided in Table 6-4 of the Exposure Factors Handbook (US EPA, 1997). The skin surface area of 3,300 cm^2 is recommended by USEPA for industrial workers (Exhibit 3-5, RAGS, Part E, USEPA, 2004).

	Men	Women
Head	1,180	1,100
Hands	840	746
Forearms	1,140	1,360
Lower Legs	2,070	1,940
Feet	1,120	975

(k) USEPA's recommended default values for groundskeepers (Exhibit 3-3 in RAGS Part E, USEPA, 2004) were adopted for maintenance workers and the recommended default for a high-end soil contact activity (Exhibit 3-5 in RAGS, Part E, 2004) were adopted for utility workers.
(l) US EPA, 1997. Exposure Factors Handbook, Volume 1, Table 5-23. Short-term exposures for outdoor workers, moderate activity.
(m) US EPA, 1997. Exposure Factors Handbook, Volume 1, Table 5-23. Hourly average for short-term exposures for outdoor workers.
(n) US EPA, 1997. Table 15-68 of Exposure Factors Handbook, Volume 3, 50th percentile value for time spent at work (males and females of all ages).

Attachment A

Table 9
Exposure Parameters – Construction Worker

Exposure Pathway	Parameter	Description	Exposure Parameters			
			CTE	Source	RME	Source
Incidental Soil Ingestion	IR	Soil Ingestion Rate (mg/day)	100	(a)	330	(b)
	EF	Exposure Frequency (days/year)	219	(c)	250	(d)
	ED	Exposure Duration (years)	1	(e)	1	(e)
	FI	Fraction Ingested (unitless)	1	(f)	1	(f)
	BW	Body Weight (kg)	70	(g,h,i)	70	(g,h,i)
	ATnc	Averaging Time for Noncarcinogenic Effects (days)	365	(j)	365	(j)
	ATc	Averaging Time for Carcinogenic Effects (days)	25,550	(g,h,i)	25,550	(g,h,i)
Dermal Contact with Soil	SA	Skin Surface Area Exposed (cm^2)	1,930	(k)	1,930	(k)
	SSAF	Soil-to-Skin Adherence Factor (mg/cm^2)	0.1	(l)	0.3	(l)
	CF	Conversion Factor (kg/mg)	1E-06	–	1E-06	–
	EF	Exposure Frequency (days/year)	219	(c)	250	(d)
	ED	Exposure Duration (years)	1	(e)	1	(e)
	EV	Event Frequency (events/day)	1	(m)	1	(m)
	BW	Body Weight (kg)	70	(g,h,i)	70	(g,h,i)
	ATnc	Averaging Time for Noncarcinogenic Effects (days)	365	(j)	365	(j)
	ATc	Averaging Time for Carcinogenic Effects (days)	25,550	(g,h,i)	25,550	(g,h,i)
Inhalation of Soil Derived Chemicals or Groundwater VOCs	IR	Inhalation Rate (m^3/hour)	1.3	(n)	1.5	(o)
	ET	Exposure Time (hour/day)	8	(p)	8	(p)
	EF	Exposure Frequency (days/year)	219	(c)	250	(d)
	ED	Exposure Duration (years)	1	(e)	1	(e)
	BW	Body Weight (kg)	70	(g,h,i)	70	(g,h,i)
	PEF	Particulate Emission Factor (m^3/kg)	Calculated	Attachment G	Calculated	Attachment G
	VF	Volatilization Factor (m^3/kg)	Calculated	See Table 21c	Calculated	See Table 21c
	ATnc	Averaging Time for Noncarcinogenic Effects (days)	365	(j)	365	(j)
	ATc	Averaging Time for Carcinogenic Effects (days)	25,550	(g,h,i)	25,550	(g,h,i)

Notes:
CTE - Central Tendency Exposure
RME - Reasonable Maximum Exposure.

(a) US EPA, 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites, Exhibit 1-2. Value for outdoor workers.
(b) US EPA, 2002, Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites, Exhibit 1-2. Recommended ingestion rate for construction workers.
(c) In the absence of Site-specific information regarding future construction activities, the default industrial worker exposure frequency was used.
(d) In the absence of Site-specific information regarding future construction activities, the default industrial worker exposure frequency was used.
(e) Construction activities are assumed to occur over a 1 year period.
(f) Based on information provided in the HHRA completed by SEH (SEH, 1998) an FI of 1 was selected as the fraction ingested based on the percentage of chemical detections in subsurface soil.
(g) US EPA, 1991. Human Health Evaluation Manual, Supplemental Guidance, Standard Default Exposure Factors.
(h) US EPA, 1989. RAGS, Volume I, Human Health Evaluation Manual (Part A).
(i) 70 kg body weight and 70 year lifetime are used to be consistent with the development of cancer slope factors.
(j) US EPA, 1989. RAGS, Part A. Averaging Time for noncarcinogens is equal to ED (year) × 365 days/year.
(k) Suggested value represents the total skin surface area for head and hands, assuming that a worker wears long sleeve shirts, long pants and shoes. This value may be adjusted based on the following body part-specific surface area (mean, in cm^2), as provided in Table 6-4 of the Exposure Factors Handbook (US EPA, 1997). The skin surface area of 3,300 cm^2 is recommended by USEPA for industrial workers (Exhibit 3-5, RAGS, Part E, USEPA, 2004).

	Men	Women
Head	1,180	1,100
Hands	840	746
Forearms	1,140	1,360
Lower Legs	2,070	1,940
Feet	1,120	975

(l) USEPA, 2004. RAGS Part E, Exhibit 3-3. Recommended default values for a construction worker.
(m) USEPA, 2004. RAGS Part E, Exhibit 3-5. Recommended RME and CTE parameters for industrial scenario.
(n) US EPA, 1997. Exposure Factors Handbook, Volume 1, Table 5-23. Short-term exposures for outdoor workers, moderate activity.
(o) US EPA, 1997. Exposure Factors Handbook, Volume 1, Table 5-23. Hourly average for short-term exposures for outdoor workers.
(p) US EPA, 1997. Exposure Factors Handbook, Table 15-68. Represents the 50th percentile value for time spent at work (males and females, all ages).
(q) Chemical-specific PEF calculated using methodology. Calculated PEFs for construction worker are presented in Table 21e.

Attachment A

Table 10
Exposure Parameters – Adult Fisher Exposed to Biota

Exposure Pathway	Parameter	Description	Exposure Parameters			
			CTE	Source	RME	Source
Ingestion of Fish	FIR	Fish Ingestion Rate (kg/event)	0.0172	(a)	0.081	(a)
	FI	Fraction Ingested (unitless)	1	(b)	1	(b)
	EF	Exposure Frequency (events/year)	350	(c)	350	(c)
	ED	Exposure Duration (years)	9	(d)	30	(d)
	BW	Body Weight (kg)	70	(e,f)	70	(e,f)
	ATn	Averaging Time for Noncarcinogenic Effects (days)	3,285	(g)	10,950	(g)
	ATc	Averaging Time for Carcinogenic Effects (days)	25,550	(h)	25,550	(h)

Notes:

CTE - Central Tendency Evaluation

RME - Reasonable Maximum Exposure.

(a) US EPA, 1997. Exposure Factors Handbook . The RME value is US EPA's recommended mean intake values for subsistence fisher and the CTE value is the recommended mean intake for sport anglers. The RME fish ingestion rate is based on the median intake value recommended by USEPA for the Native American subsistence populations using data collected from 94 Native American Communities in Alaska. However, this ingestion rate is expoected to be protective of any non-Native American subsistence fisher populations near the Site.

(b) A conservative value of 1 (assuming all of the fish ingested is from the impacted area) is used due to the lack of site-/area-specific information.

(c) US EPA's current guidance indicates that the fish ingestion rates are expressed in terms of daily ingestion over a long period.

(d) US EPA, 1997. Exposure Factors Handbook, Table 1-2. Recommended average (9 years) and 95th percentile (30 years) for time residing in a household.

(e) US EPA, 1989. RAGS, Volume I, Human Health Evaluation Manual (Part A).

(f) 70 kg body weight and 70 year lifetime are used to be consistent with the development of cancer slope factors.

(g) US EPA, 1989. RAGS, Part A. Averaging Time for noncarcinogens is equal to ED (year) × 365 days/year.

(h) US EPA, 1989. RAGS, Part A. Averaging time for carcinogenic effects is equal to 70 year (average life expectancy) × 365 days/year.

Attachment A

Table 11a
Carcinogenic Toxicity Values

CAS Number	Chemical	SFi	SFi Source	Inhalation WOE	SFo	SFo Source	Oral WOE
	Inorganics						
7429905	Aluminum	NV	NCEA	c	NV	NCEA	c
7440360	Antimony	NV	-	NA	NV	IRIS	NA
7440382	Arsenic	1.51E+01	IRIS	A	1.50E+00	IRIS	A
7440393	Barium	NV	IRIS	D	NV	IRIS	D
7440417	Beryllium	8.40E+00	IRIS	B1	NV	IRIS	NA
7440439	Cadmium	6.30E+00	IRIS	NA	NV	IRIS	NA
7440473	Chromium, Total	4.20E+01	IRIS	A	NV	IRIS	D
16065831	Chromium (III)	NV	IRIS	D	NV	IRIS	D
18540299	Chromium (VI)	4.20E+01	IRIS	A	NV	IRIS	D
7440484	Cobalt	9.80E-07	PPRTV	B1	NV	NCEA	NA
7440508	Copper	NV	IRIS	D	NV	IRIS	D
57125	Cyanides	NV	IRIS	D	NV	IRIS	D
7439896	Iron	NV	-	NA	NV		NA
7439921	Lead	a	-	NA	a	IRIS	B2
7439965	Manganese	NV	IRIS	D	NV	IRIS	D
7439976	Mercury	NV	IRIS	D	NV	IRIS	D
7440020	Nickel	NV	IRIS	NA	NV	IRIS	NA
7782492	Selenium	NV	IRIS	D	NV	IRIS	D
7440224	Silver	NV	IRIS	D	NV	IRIS	D
7440280	Thallium	NV	IRIS	D	NV	IRIS	D
7440622	Vanadium	NV	IRIS	NA	NV	IRIS	NA
7440666	Zinc	NV	IRIS	D	NV	IRIS	D
	SVOCs						
90120	1-Methylnaphthalene	NV	-	-	NV	-	-
832699	1-Methylphenanthrene	NV	-	-	NV	-	-
2245387	2,3,5-Trimethylnaphthalene	NV	-	-	NV	-	-
581420	2,6-Dimethylnaphthalene	NV	-	-	NV	-	-
91576	2-Methylnaphthalene	NV	IRIS	NA	NV	IRIS	NA
83329	Acenaphthene	NV	IRIS	NA	NV	IRIS	NA
208968	Acenaphthylene	NV	IRIS	D	NV	IRIS	D
120127	Anthracene	NV	IRIS	D	NV	IRIS	D
56553	Benzo(a)anthracene	NV	IRIS	NA	7.30E-01	R9NCEA	B2
50328	Benzo(a)pyrene	3.08E+00	R4NCEA	B2	7.30E+00	IRIS	B2
205992	Benzo(b)fluoranthene	NV	IRIS	NA	7.30E-01	R9NCEA	B2
192972	Benzo(e)pyrene	NV	-	-	NV	-	-
191242	Benzo(g,h,i)perylene	NV	IRIS	NA	NV	IRIS	D
207089	Benzo(k)fluoranthene	NV	IRIS	NA	7.30E-02	R9NCEA	B2
92524	Biphenyl	NV	IRIS	D	NV	IRIS	D
218019	Chrysene	NV	IRIS	NA	7.30E-03	R9NCEA	B2
53703	Dibenzo(a,h)anthracene	NV	IRIS	NA	7.30E+00	R9NCEA	B2
132649	Dibenzofuran	NV	IRIS	D	NV	IRIS	D
206440	Fluoranthene	NV	IRIS	D	NV	IRIS	D
86737	Fluorene	NV	IRIS	D	NV	IRIS	D
193395	Indeno(1,2,3-cd)pyrene	NV	IRIS	NA	7.30E-01	R9NCEA	B2
NA	m & p-Cresols	NV	IRIS	C	NV	IRIS	C
91203	Naphthalene	NV	IRIS	C	NV	IRIS	C
95487	o-Cresol	NV	IRIS	C	NV	IRIS	C
106445	p-Cresol	NV	IRIS	C	NV	IRIS	C
198550	Perylene (Dibenz[de,kl]anthracene)	NV	-	-	NV	-	-
85018	Phenanthrene	NV	IRIS	D	NV	IRIS	D
108952	Phenol	NV	IRIS	D	NV	IRIS	D
129000	Pyrene	NV	IRIS	D	NV	IRIS	D
	VOCs						

Attachment A

Table 11a
Carcinogenic Toxicity Values

CAS Number	Chemical	SFi	SFi Source	Inhalation WOE	SFo	SFo Source	Oral WOE
71556	1,1,1-Trichloroethane	NV	-	D	NV	-	D
526738	1,2,3-Trimethylbenzene	NV	-	-	NV	-	-
120821	1,2,4-Trichlorobenzene	NV	IRIS	D	NV	IRIS	D
95636	1,2,4-Trimethylbenzene	NV	NCEA	c	NV	NCEA	c
108678	1,3,5-Trimethylbenzene	NV	NCEA	c	NV	NCEA	c
106467	1,4-Dichlorobenzene	NV	-	d	2.40E-02	HEAST	d
71432	Benzene	2.73E-02	IRIS	A	5.50E-02	IRIS	A
56235	Carbon tetrachloride	5.25E-02	IRIS	B2	1.30E-01	IRIS	B2
74873	Chloromethane	6.30E-03	HEAST		NV	IRIS	e
75718	Dichlorodifluoromethane	NV	IRIS	d	NV	IRIS	d
100414	Ethylbenzene	NV	IRIS	D	NV	IRIS	D
75092	Methylene chloride	1.65E-03	IRIS	B2	7.50E-03	IRIS	B2
NA	m & p-Xylenes	NV	IRIS	b	NV	IRIS	b
95476	o-Xylene	NV	IRIS	b	NV	IRIS	b
104518	n-Butyl benzene	NV	-	NA	NV	-	NA
135988	sec-Butyl benzene	NV	-	NA	NV	-	NA
100425	Styrene	NV	IRIS	D	NV	IRIS	D
108883	Toluene	NV	-	C	NV	-	C
79016	Trichloroethene	4.00E-01	NCEA	-	4.00E-01	NCEA	-
75694	Trichlorofluoromethane	NV	IRIS	-	NV	IRIS	-
1330207	Xylenes (total)	NV	IRIS	-	NV	IRIS	-

Attachment A

Table 11a
Carcinogenic Toxicity Values

Table values are current for 02/06.

SF_i – Inhalation Slope Factor (kg-day/mg)
SF_o – Oral Slope Factor (kg-day/mg)
WOE – Weight of Evidence
NV – No Value
NCEA – National Center for Environmental Assessment
NA – Not Applicable
IRIS – Integrated Risk Information System
PPRTV – Provisional Peer Reviewed Toxicity Values
SVOC – Semivolatile organic compound
R9NCEA – Value presented in the Region 9 Preliminary Remediation Goal Table (USEPA, 2004). Source of the value is the National Center for Environmental Assessment.
R4NCEA – Value presented in the Region 4 Human Health Risk Assessment Bulletins (USEPA, 1995). The source of the value is the National Center for Environmental Assessment.
VOC – Volatile organic compound

WOE Classification System
Group Description

A Human Carcinogen
D Not classifieable as to human carcinogenicity
B1 Probable human carcinogen based on limited human data.
B2 Probable human carcinogen based on sufficient evidence in animals and inadequate or no evidence in humans
C Possible human carcinogen

a – Quantifying lead's cancer risk involves many uncertainties, some of which may be unique to lead. USEPAs Carcinogen Assessment Group does not recommend a numerical estimate of toxicity.
b – This substance has not been assessed under the 1986 Cancer guidelines. Under the Draft Guidelines for Carcinogens Risk Assessment (USEPA, 1999), data are inadequate for an assessment of the carcinogenic potential of xylenes.
c – Under the Guidelines for Carcinogen Risk Assessement (U.S. EPA, 2005), there is inadequate information to assess the carcinogenic potential of toluene because studies of humans chronically exposed to toluene are inconclusive.
d – This substance/agent has not undergone a complete evaluation and determination under US EPA's IRIS program for evidence of human carcinogenic potential.
e – Not applicable. Chloromethane exists primarily as a gas. No adequate oral exposure studies exist from which an oral RfD may be derived.

Attachment A

Table 11b
Noncarcinogenic Toxicity Values

CAS Numbe	Chemical	RfDi_C	Chronic Source	RfDi_S	Subchronic Source	Inhalation Target Organ	RfDo_C	Chronic Source	RfDo_S	Subchronic Source	Oral Target Organ
	Inorganics										
7429905	Aluminum	1.43E-03	PPRTV	NV		nervous system	1.00E+00	PPRTV	NV		LOAEL
7440360	Antimony	NV		1.10E-04	PPRTV	lung	4.00E-04	IRIS	2.00E-04	PPRTV	blood
7440382	Arsenic	NV		NV		NA	3.00E-04	IRIS	3.00E-04	HEAST	vascular system/skin
7440393	Barium	1.43E-04	HEAST	1.43E-03	HEAST	fetus	2.00E-01	IRIS	7.00E-02	HEAST	kidney
7440417	Beryllium	5.71E-06	IRIS	NV		immune system/lung	2.00E-03	IRIS	5.00E-03	HEAST	GI tract
7440439	Cadmium	NV		2.60E-04	PPRTV	kidney	1.00E-03	IRIS	NV		kidney
7440439	Cadmium (diet)					-	1.00E-03	IRIS	-	-	-
7440439	Cadmium (water)					-	5.00E-04	IRIS	-	-	-
16065831	Chromium (III)	NV	IRIS	NV	-	NA	1.50E+00	IRIS	1.50E+00	HEAST	NOAEL
18540299	Chromium (VI)	2.86E-05	IRIS	NV		lung	3.00E-03	IRIS	2.00E-02	HEAST	NOAEL
7440484	Cobalt	5.71E-06	PPRTV	NV		respiratory tract	2.00E-02	PPRTV	NV	-	blood
7440508	Copper	NV		NV			3.00E-02	NCEA	4.00E-02	HEAST	GI tract
57125	Cyanides	NV		NV		NA	2.00E-02	IRIS	2.00E-02	HEAST	thyroid/decreased weight
7439896	Iron	NV		NV		NA	3.00E-01	R9NCEA	NV	-	blood/liver/GI tract
7439921	Lead	NV		NV		NA	NV	IRIS	NV	-	NA
7439965	Manganese (diet)	1.43E-05	IRIS	NV	-	LOAEL	1.40E-01	IRIS	-	-	CNS
7439965	Manganese (water)	1.43E-05	IRIS	NV	-	LOAEL	4.60E-02	IRIS	-	-	CNS
7439976	Mercury	8.57E-05	IRIS	8.57E-05		nervous system	3.00E-04	IRIS	3.00E-03	HEAST	Autoimmune effects
7440020	Nickel	NV	IRIS	NV		lung	2.00E-02	IRIS	2.00E-02	HEAST	kidney/liver/spleen
7782492	Selenium	NV	IRIS	NV		NA	5.00E-03	IRIS	5.00E-03	HEAST	liver/nails/CNS
7440224	Silver	NV	IRIS	NV		NA	5.00E-03	IRIS	5.00E-03	HEAST	skin
7440280	Thallium	NV	IRIS	NV		NA	8.00E-05	IRIS	8.00E-04	HEAST	nervous system/skin
7440622	Vanadium	NV	IRIS	NV		NA	7.00E-03	HEAST	7.00E-03	HEAST	blood
7440666	Zinc	NV	IRIS	NV		NA	3.00E-01	IRIS	3.00E-01	HEAST	blood/kidney/pancreas
	SVOCs										
90120	1-Methylnaphthalene	NV		NV		NA	7.00E-02	ATSDR	NV	-	LOAEL
832699	1-Methylphenanthrene	NV		NV		NA	NV	-	NV	-	NA
2245387	2,3,5-Trimethylnaphthalene	NV		NV		NA	NV	-	NV	-	NA
581420	2,6-Dimethylnaphthalene	NV		NV		NA	NV	-	NV	-	NA
91576	2-Methylnaphthalene	NV		NV		NA	4.00E-03	IRIS	NV	-	kidney/thymus
83329	Acenaphthene	NV		NV		NA	6.00E-02	IRIS	6.00E-01	HEAST	liver
208968	Acenaphthylene	NV		NV		NA	NV		NV		liver
120127	Anthracene	NV		NV		NA	3.00E-01	IRIS	3.00E+00	HEAST	NOAEL
56553	Benzo(a)anthracene	NV		NV		NA	NV	IRIS	NV	-	NA
50328	Benzo(a)pyrene	NV		NV		NA	NV	IRIS	NV	-	NA
205992	Benzo(b)fluoranthene	NV		NV		NA	NV	IRIS	NV	-	NA
192972	Benzo(e)pyrene	NV		NV		NA	NV	IRIS	NV	-	NA
191242	Benzo(g,h,i)perylene	NV		NV		NA	NV	IRIS	NV	-	kidney
207089	Benzo(k)fluoranthene	NV		NV		NA	NV	IRIS	NV	-	NA
92524	Biphenyl	NV		NV		NA	5.00E-02	IRIS	5.00E-02	HEAST	kidney
218019	Chrysene	NV		NV		NA	NV	IRIS	NV	-	NA
53703	Dibenzo(a,h)anthracene	NV		NV		NA	NV	IRIS	NV	-	NA

Attachment A

Table 11b
Noncarcinogenic Toxicity Values

CAS Numbe	Chemical	RfDi_C	Chronic Source	RfDi_S	Subchronic Source	Inhalation Target Organ	RfDo_C	Chronic Source	RfDo_S	Subchronic Source	Oral Target Organ
132649	Dibenzofuran	NV	IRIS	NV	-	NA	2.00E-03	R9NCEA	NV	-	NA
206440	Fluoranthene	NV	IRIS	NV	-	NA	4.00E-02	IRIS	4.00E-01	HEAST	kidney/liver/blood
86737	Fluorene	NV	IRIS	NV	-	NA	4.00E-02	IRIS	4.00E-01	HEAST	blood
193395	*Indeno(1,2,3-cd)pyrene*	NV	IRIS	NV	-	NA	NV	IRIS	NV	-	NA
NA	m & p-Cresols	NV	IRIS	NV	-	NA	NV	-	NV	-	NA
108-39-4	m-Cresol	NV	IRIS	NV	-	NA	5.00E-02	IRIS	5.00E-01	HEAST	Weight/neurotoxicity
91203	Naphthalene	8.57E-04	IRIS	NV	-	respiratory tract	2.00E-02	IRIS	NV		blood
95487	o-Cresol	NV	IRIS	NV	-	NA	5.00E-02	IRIS	5.00E-01	HEAST	weight/neurotoxicity
106445	p-Cresol	NV	IRIS	NV	-	NA	5.00E-03	HEAST	5.00E-03	HEAST	CNS/respiratory tract/whole body
198550	Perylene (Dibenz[de,kl]anthracene)	NV	-	NV	-	NA	NV	-	NV	-	NA
85018	Phenanthrene	NV	-	NV	-	NA	NV	-	NV	-	
108952	Phenol	NV	IRIS	NV	-	NA	3.00E-01	IRIS	6.00E-01	HEAST	maternal weight decrease
129000	Pyrene	NV	IRIS	NV	-	NA	3.00E-02	IRIS	3.00E-01	HEAST	kidney
	VOCs										
71556	1,1,1-Trichloroethane	6.29E-01	PPRTV	NV	-	-	2.00E-01	PPRTV	NV	-	-
526738	1,2,3-Trimethylbenzene	NV	-	NV	-	NA	NV	-	NV	-	NA
120821	1,2,4-Trichlorobenzene	1.00E-03	PPRTV	NV	-	liver	1.00E-02	IRIS	1.00E-02	HEAST	adrenal glands
95636	1,2,4-Trimethylbenzene	1.71E-03	PPRTV	NV	-	CNS/blood/lung	5.00E-02	PPRTV	NV	-	whole body/blood/liver/kidney
108678	1,3,5-Trimethylbenzene	1.71E-03	PPRTV	1.70E-02	PPRTV	CNS/blood/lung	5.00E-02	PPRTV	5.00E-01	PPRTV	whole body/blood/liver/kidney
106467	1,4-Dichlorobenzene	2.29E-01	IRIS	NV	-	increased liver weight	NV	IRIS	NV	-	-
71432	Benzene	8.57E-03	IRIS	NV	-	bone marrow	4.00E-03	IRIS	NV	-	bone marrow
56235	Carbon tetrachloride	NV	-	NV	-	-	7.00E-04	IRIS	NV	-	liver lesions
74873	Chloromethane	2.57E-02	IRIS	NV	-	Cerebellar lesions	NV	IRIS	NV	-	-
75718	Dichlorodifluoromethane	5.71E-02	HEAST	NV	-	NOAEL	2.00E-01	IRIS	NV	-	reduced body weight
100414	Ethylbenzene	2.86E-01	PPRTV	2.90E-01	PPRTV	NA	1.00E-01	IRIS	1.10E-01	PPRTV	liver/kidney
75092	Methylene chloride	8.57E-01	HEAST	NV	-	toxicity	6.00E-02	IRIS	NV	-	liver toxicity
NA	m & p-Xylenes	NV	-	NV	-	NA	NV	-	NV	-	-
95476	*o-Xylene*	NV	-	NV	-	NA	2.00E+00	HEAST	NV	-	CNS
104518	n-Butyl benzene	NV	-	NV	-	NA	4.00E-02	NCEA	-	-	NOAEL
135988	sec-Butyl benzene	NV	-	NV	-	NA	4.00E-02	NCEA	-	-	NOAEL
100425	Styrene	2.86E-01	IRIS	8.60E-01	HEAST	CNS	2.00E-01	IRIS	NV	-	blood/liver/GI tract
108883	Toluene	1.43E+00	IRIS	2.60E-01	PPRTV	lung/nervous system	8.00E-02	IRIS	2.00E+00	HEAST	kidney/liver
79016	Trichloroethene	1.14E-02	PPRTV	NV	-	-	3.00E-04	PPRTV	NV	-	-
1330207	*Xylenes (total)*	2.86E-02	IRIS	NV	-	nervous system	2.00E-01	IRIS	3.57E-01		CNS/developmental
108383	m-Xylene	NV	IRIS	NV	-	NA	2.00E+00	HEAST	6.67E-02	NCEA	CNS
75694	Trichlorofluoromethane	2.00E-01	HEAST	NV	-	increased mortality	3.00E-01	IRIS	NV	-	survival and histopathology
106423	p-Xylene	NV	IRIS	NV	-	NA	NV	-	2.67E-01	NCEA	CNS

Attachment A

Table 11b
Noncarcinogenic Toxicity Values

Table values are current for 02/06.

RfDi_C – Inhalation Reference Dose (Chronic) (mg/kg-day)
RfDi_S – Inhalation Reference Dose (Subchronic)(mg/kg-day)
RfDo_C – Inhalation Reference Dose (Oral)(mg/kg-day)
RfDo_S – Inhalation Reference Dose (Subchronic)(mg/kg-day)
PPRTV – Provisional Peer Reviewed Toxicity Value
NV – No Value
LOAEL – Lowest-observed-adverse-effect-Level
IRIS – Integrated Risk Information System
NA – Not Applicable
HEAST – Health Effects Assessment Summary Table (HEAST), 1997
GI – Gastrointestinal (USEPA, 1997)
NCEA – National Center for Environmental Assessment
NCEA – Value presented in the Region 9 Preliminary Remediation Goal Table (USEPA, 2004). Source of the value is the National Center for Environmental Assessment.
CNS – Central Nervous System
SVOC – Semivolatile Organic Compound
ATSDR – Agency for Toxic Substances Disease Registry
NOAEL – No-observed-adverse-effect Level
VOC – Volatile Organic Compound

Attachment A
TABLE 12
IEUBK LEAD WORKSHEET
Site Name: NSP Ashland Lakefront
Receptor: Residential Child (Age 0 - 84 Months) Exposure to Soil

1. Lead Screening Questions

Medium	Lead Concentration Used in Model Run		Basis for Lead Concentration Used For Model Run	Lead Screening Concentration		Basis for Lead Screening Level
	Value	Units		Value	Units	
Soil	Site-specific	mg/kg	Average Detected Value	400	mg/kg	Recommended Soil Screening Level
Water	4	ug/L	Model Default Value	15	ug/L	Recommended Drinking Water Action Level

2. Lead Model Questions

Question	Response for Residential Lead Model
What lead model (version and date) was used?	IEUBKwin v1.0 build 263; December 2005
Where are the input values located in the risk assessment report?	Output that identifies the input values used in the IEUBK model are provided in **Attachments D** (surface and subsurface soil) and **F** (surface soil only and soil [0-3 feet bgs]).
What range of media concentrations were used for the model?	The average concentration was used for soil. Data are located in **Attachment B1, Table 1** (surface and subsurface soil) and **Attachment F Tables 1** and **2** (surface soil only and soil [0-3 feet bgs]). Model default values were used for all other media.
What statistics were used to represent the exposure concentration terms and where are the data on concentrations in the risk assessment that support use of these statistics?	Average concentration was used for soil. Data are located in **Table 4-8A** and **4-8B** of the RI Report.
Was soil sample taken from top 2 cm? If not, why?	As outlined in the approved RI/FS Workplan (URS, 2005), residential receptors are assumed to be exposed to exposed soil at depths between 0 and 10 feet. However, surface soil (0-1 foot) and surface and subsurface soil (0-3 feet bgs) was also evaluated. **Section 3.1.4.1** of the HHRA discusses the rationale for this approach.
Was soil sample sieved? What size screen was used? If not sieved, provide rationale.	Samples were prepared in accordance with the Field Sampling Plan approved by the USEPA (URS, 2005).
What was the point of exposure/location?	Samples were collected from the upper bluff area near the former ravine.
Where are the output values located in the risk assessment report?	Output that identifies the input values used in the IEUBK model are provided in **Attachments D** (surface and subsurface soil) and **F** (surface soil only and soil [0-3 feet bgs]).
Was the model run using default values only?	No. Site-specific soil concentration values used. All other model values are default.
Was the default soil bioavailability used?	Yes. Default is 30%
Was the default soil ingestion rate used?	Yes Default values for 7 age groups are 85, 135, 135, 100, 090, and 85 mg/day.
If non-default values were used, where is the rationale for the values located in the risk assessment report?	Not applicable to this evaluation.

Attachment A

Table 13
Exposure Variables Used in the Calculation of Blood Lead Concentrations (PbBs) for Non-residential Receptors (Construction and Maintenance Workers)

Exposure Variable	PbB Equation[1] 1*	2**	Description of Exposure Variable	Units	Values for Non-Residential Exposure Scenario[2] Using Equation 1 GSDi = Hom	Using Equation 1 GSDi = Het	Using Equation 2 GSDi = Hom	Using Equation 2 GSDi = Het
PbS	X	X	Soil lead concentration	ug/g or ppm	Site-specific	Site-specific	Site-specific	Site-specific
$R_{fetal/maternal}$	X	X	Fetal/maternal PbB ratio	--	0.9	0.9	0.9	0.9
BKSF	X	X	Biokinetic Slope Factor	ug/dL per ug/day	0.4	0.4	0.4	0.4
GSD_i	X	X	Geometric standard deviation PbB	--	2.1	2.3	2.1	2.3
PbB_0	X	X	Baseline PbB	ug/dL	1.5	1.7	1.5	1.7
IR_S	X		Soil ingestion rate (including soil-derived indoor dust)	g/day	0.050	0.050	--	--
IR_{S+D}		X	Total ingestion rate of outdoor soil and indoor dust	g/day	--	--	0.050	0.050
W_S		X	Weighting factor; fraction of IR_{S+D} ingested as outdoor soil	--	--	--	1.0	1.0
K_{SD}		X	Mass fraction of soil in dust	--	--	--	0.7	0.7
$AF_{S,D}$	X	X	Absorption fraction (same for soil and dust)	--	0.12	0.12	0.12	0.12
$EF_{S,D}$	X	X	Exposure frequency (same for soil and dust)	days/yr	219	219	219	219
$AT_{S,D}$	X	X	Averaging time (same for soil and dust)	days/yr	365	365	365	365
PbB_t			**Target PbB level of concern (e.g., 10 ug/dL)**	**ug/dL**	**10.0**	**10.0**	**10.0**	**10.0**

[1] Equation 1 does not apportion exposure between soil and dust ingestion (excludes W_S, K_{SD}).
When $IR_S = IR_{S+D}$ and $W_S = 1.0$, the equations yield the same $PbB_{fetal,0.95}$.

[2] Except for the soil lead concentration (PbS), all exposure variable values are the default values presented in the Adult Lead Model (USEPA, 2003).

***Equation 1, based on Eq. 1, 2 in USEPA (1996).**

PbB_{adult} =	$(PbS*BKSF*IR_{S+D}*AF_{S,D}*EF_S/AT_{S,D}) + PbB_0$
$PbB_{fetal,0.95}$ =	$PbB_{adult} * (GSD_i^{1.645} * R)$

****Equation 2, alternate approach based on Eq. 1, 2, and A-19 in USEPA (1996).**

PbB_{adult} =	$PbS*BKSF*([(IR_{S+D})*AF_S*EF_S*W_S]+[K_{SD}*(IR_{S+D})*(1-W_S)*AF_D*EF_D])/365+PbB_0$
$PbB_{fetal,0.95}$ =	$PbB_{adult} * (GSD_i^{1.645} * R)$

Source: U.S. EPA Technical Review Workgroup for Lead, Adult Lead Committee
Version data 05/19/03

Attachment A

Table 14
Chemical - Specific Values

Chemical	Solubility	Solubility Source	GIABS	GIABS Source	ABSd	ABSd Source	DI	DI Source	Dw	Dw Source	VOCs	HLC	HLC Source	Koc	Koc Source	Kd	Kd Source
Inorganics																	
Aluminum	NV	ORNL	NV	USEPA 2004	NA	USEPA 2004	NV	NV	NV	NV	No	NV	NV	NV		8.58E-02	ORNL
Antimony	NV	ORNL	1.50E-01	USEPA 2004	NA	USEPA 2004	NV	NV	NV	NV	No	NV	NV	NV		8.58E-02	ORNL
Arsenic	NV	ORNL	9.50E-01	USEPA 2004	3.00E-02	USEPA 2004	NV	NV	NV	NV	No	NV	NV	NV		8.58E-02	ORNL
Barium	NV	ORNL	7.00E-02	USEPA 2004	NA	USEPA 2004	NV	NV	NV	NV	No	NV	NV	NV		8.58E-02	ORNL
Beryllium	NV	ORNL	7.00E-03	USEPA 2004	NA	USEPA 2004	NV	NV	NV	NV	No	NV	NV	NV		8.58E-02	ORNL
Cadmium	NV	ORNL	2.50E-02	USEPA 2004	1.00E-03	USEPA 2004	NV	NV	NV	NV	No	NV	NV	NV		8.58E-02	ORNL
Chromium, Total	NV		2.50E-02	USEPA 2004	NA	USEPA 2004	NV	NV	NV	NV	No	NV	NV	NV			
Chromium (III)	NV	ORNL	1.30E-02	USEPA 2004	NA	USEPA 2004	NV	NV	NV	NV	No	NV	NV	NV		1.80E+06	ORNL
Chromium (VI)	1.69E+06	ORNL	2.50E-02	USEPA 2004	NA	USEPA 2004	NV	NV	NV	NV	No	NV	NV	NV		1.90E+01	ORNL
Cobalt	NV	ORNL	NV	USEPA 2004	NA	USEPA 2004	NV	NV	NV	NV	No	NV	NV	NV		8.58E-02	ORNL
Copper	NV	ORNL	NV	USEPA 2004	NA	USEPA 2004	NV	NV	NV	NV	No	NV	NV	NV		8.58E-02	ORNL
Cyanides	*NV*		*1.00E+00*	*USEPA 2004*	*NA*	*USEPA 2004*	*NV*	*NV*	*NV*	*NV*	*No*	*NV*	*NV*	*NV*		*1.63E-02*	*ORNL*
Iron	NV	ORNL	NV	USEPA 2004	NA	USEPA 2004	NV	NV	NV	NV	No	NV	NV	NV		8.58E-02	ORNL
Lead	NV	ORNL	NV	USEPA 2004	NA	USEPA 2004	NV	NV	NV	NV	No	NV	NV	NV		8.58E-02	ORNL
Manganese	NV	ORNL	4.00E-02	USEPA 2004	NA	USEPA 2004	NV	NV	NV	NV	No	NV	NV	NV		8.58E-02	ORNL
Mercury	6.00E-02	ORNL	7.00E-02	USEPA 2004	NA	USEPA 2004	NV	NV	NV	NV	No	NV	NV	NV		8.58E-02	ORNL
Nickel	NV	ORNL	4.00E-02	USEPA 2004	NA	USEPA 2004	NV	NV	NV	NV	No	NV	NV	NV		8.58E-02	ORNL
Selenium	NV	ORNL	1.00E+00	USEPA 2004	NA	USEPA 2004	NV	NV	NV	NV	No	NV	NV	NV		8.58E-02	ORNL
Silver	NV	ORNL	4.00E-02	USEPA 2004	NA	USEPA 2004	NV	NV	NV	NV	No	NV	NV	NV		8.58E-02	ORNL
Thallium	NV	ORNL	1.00E+00	USEPA 2004	NA	USEPA 2004	NV	NV	NV	NV	No	NV	NV	NV		8.58E-02	ORNL
Vanadium	NV	ORNL	2.60E-02	USEPA 2004	NA	USEPA 2004	NV	NV	NV	NV	No	NV	NV	NV		8.58E-02	ORNL
Zinc	NV	ORNL	1.00E+00	USEPA 2004	NA	USEPA 2004	NV	NV	NV	NV	No	NV	NV	NV		8.58E-02	ORNL
SVOCs																	
1-Methylnaphthalene	2.50E+01	ORNL	1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	6.31E-02	TCEQ	7.13E-06	TCEQ	Yes	1.64E-02	TCEQ	1.84E+03	ORNL	3.75E+01	ORNL calculated
1-Methylphenanthrene	NV		1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	NV	NV	NV	NV	NV	NV	NV	NV		NV	
2,3,5-Trimethylnaphthalene	NV		1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	NV	NV	NV	NV	NV	NV	NV	NV		NV	
2,6-Dimethylnaphthalene	NV		1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	NV	NV	NV	NV	NV	NV	NV	NV		NV	
2-Methylnaphthalene	2.46E+01	ORNL	1.00E+00	USEPA 2004	1.00E-01	USEPA 2004	4.80E-02	ORNL	7.84E-06	ORNL	Yes	2.12E-02	ORNL	2.98E+03	ORNL	1.79E+01	ORNL
Acenaphthene	3.90E+00	ORNL	1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	4.21E-02	ORNL	7.69E-06	ORNL	Yes	7.44E-03	ORNL	6.12E+03	ORNL	3.67E+01	ORNL
Acenaphthylene	1.61E+01	ORNL	1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	4.39E-02	ORNL	7.53E-06	ORNL	Yes	5.11E-03	ORNL	6.12E+03	ORNL	3.67E+01	ORNL
Anthracene	4.34E-02	ORNL	1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	3.24E-02	ORNL	7.74E-06	ORNL	Yes	2.27E-03	ORNL	2.04E+04	ORNL	1.22E+02	ORNL
Benzo(a)anthracene	9.40E-03	ORNL	1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	5.10E-02	ORNL	9.00E-06	ORNL	No	4.91E-04	ORNL	2.31E+05	ORNL	1.39E+03	ORNL
Benzo(a)pyrene	1.62E-03	ORNL	1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	4.30E-02	ORNL	9.00E-06	ORNL	No	4.63E-05	USEPA 2002	7.87E+05	ORNL	4.72E+03	ORNL
Benzo(b)fluoranthene	1.50E-03	ORNL	1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	2.26E-02	ORNL	5.56E-06	ORNL	No	2.69E-05	ORNL	8.03E+05	ORNL	4.82E+03	ORNL
Benzo(e)pyrene	8.60E-04	TCEQ	1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	4.05E-02	TCEQ	5.49E-06	TCEQ	No	2.38E-05	TCEQ	1.58E+06	TCEQ	9.51E+03	TCEQ calculated
Benzo(g,h,i)perylene	2.60E-04	ORNL	1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	4.90E-02	TCEQ	5.65E-05	TCEQ	No	1.35E-05	ORNL	2.68E+06	ORNL	1.61E+04	ORNL
Benzo(k)fluoranthene	8.00E-04	ORNL	1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	2.26E-02	ORNL	5.56E-06	ORNL	No	2.39E-05	ORNL	7.87E+05	ORNL	4.72E+03	ORNL
Biphenyl	6.94E+00	ORNL	1.00E+00	USEPA 2004	1.00E-01	USEPA 2004	4.04E-02	ORNL	8.15E-06	ORNL	Yes	1.26E-02	ORNL	6.25E+03	ORNL	3.75E+01	ORNL calculated
Chrysene	2.00E-03	ORNL	1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	2.48E-02	ORNL	6.21E-06	ORNL	No	2.14E-04	ORNL	2.36E+05	ORNL	1.42E+03	ORNL
Dibenzo(a,h)anthracene	1.03E-03	ORNL	1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	2.02E-02	ORNL	5.18E-06	ORNL	No	5.03E-06	ORNL	2.62E+06	ORNL	1.57E+04	ORNL
Dibenzofuran	3.10E+00	ORNL	1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	5.51E-02	TCEQ	7.04E-06	TCEQ	Yes	8.71E-03	ORNL	1.13E+04	ORNL	6.78E+01	ORNL
Fluoranthene	2.60E-01	ORNL	1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	3.02E-02	ORNL	6.35E-06	ORNL	No	3.62E-04	ORNL	7.09E+04	ORNL	4.25E+02	ORNL
Fluorene	1.89E+00	ORNL	1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	3.63E-02	ORNL	7.88E-06	ORNL	Yes	3.93E-03	ORNL	1.13E+04	ORNL	6.78E+01	ORNL
Indeno(1,2,3-cd)pyrene	1.90E-04	ORNL	1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	1.90E-02	ORNL	5.66E-06	ORNL	No	1.42E-05	ORNL	2.68E+06	ORNL	1.61E+04	ORNL
m & p-Cresols	22700/21500	ORNL	1.00E+00	USEPA 2004	1.00E-01	USEPA 2004	7.40E-02	ORNL	1.00E-05	ORNL	No	4.09E-05	ORNL	4.34E+02	ORNL	2.60E+00	ORNL
Naphthalene	3.10E+01	ORNL	1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	5.90E-02	ORNL	7.50E-06	ORNL	Yes	1.80E-02	ORNL	1.84E+03	ORNL	1.10E+01	ORNL
o-Cresol	*2.59E+04*	*ORNL*	*1.00E+00*	*USEPA 2004*	*1.00E-01*	*USEPA 2004*	*7.40E-02*	*ORNL*	*8.30E-06*	*ORNL*	*No*	*4.91E-05*	*ORNL*	*4.43E+02*	*ORNL*	*2.66E+00*	*ORNL*
p-Cresol	2.15E+04	ORNL	1.00E+00	USEPA 2004	1.00E-01	USEPA 2004	7.40E-02	ORNL	1.00E-05	ORNL	No	4.09E-05	ORNL	4.34E+02	ORNL	2.60E+00	ORNL
Perylene (Dibenz[de,kl]anthracene)	NV		1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	NV		NV		NV	3.50E-13	TCEQ	3.89E+06	TCEQ	2.33E+04	TCEQ calculated
Phenanthrene	1.15E+00	ORNL	1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	3.33E-02	TCEQ	7.47E-06	TCEQ	Yes	1.73E-03	ORNL	2.08E+04	ORNL	1.25E+02	ORNL
Phenol	8.28E+04	ORNL	1.00E+00	USEPA 2004	1.00E-01	USEPA 2004	8.20E-02	ORNL	9.10E-06	ORNL	No	1.36E-05	ORNL	2.68E+02	ORNL	1.61E+00	ORNL
Pyrene	1.35E-01	ORNL	1.00E+00	USEPA 2004	1.30E-01	USEPA 2004	2.72E-02	ORNL	7.24E-06	ORNL	Yes	4.87E-04	ORNL	6.94E+04	ORNL	4.16E+02	ORNL
VOCs																	
1,1,1-Trichloroethane	1.29E+03	ORNL	1.00E+00	USEPA 2004	NA	USEPA 2004	7.80E-02	ORNL	8.80E-06	ORNL	Yes	7.03E-01	ORNL	4.86E+01	ORNL	2.92E-01	calculated
1,2,3-Trimethylbenzene	7.52E+01	TCEQ	1.00E+00	USEPA 2004	NA	USEPA 2004	6.77E-02	TCEQ	7.41E-06	TCEQ	NV	1.33E-01	TCEQ	5.89E+02	TCEQ	3.53E+00	TCEQ calculated
1,2,4-Trichlorobenzene	4.90E+01	ORNL	NV	USEPA 2004	NV	USEPA 2004	3.00E-02	USEPA 2001	8.23E-06	USEPA 2001	Yes	1.42E-03	USEPA 2001	1.78E+03	USEPA 2001	1.07E+01	calculated
1,2,4-Trimethylbenzene	5.70E+01	ORNL	1.00E+00	USEPA 2004	NA	USEPA 2004	6.44E-02	ORNL	7.92E-06	ORNL	Yes	2.52E-01	ORNL	7.18E+02	ORNL	4.31E+00	ORNL calculated
1,3,5-Trimethylbenzene	4.82E+01	ORNL	1.00E+00	USEPA 2004	NA	USEPA 2004	6.02E-02	ORNL	8.67E-06	ORNL	Yes	3.59E-01	ORNL	7.03E+02	ORNL	4.22E+00	ORNL calculated
1,4-Dichlorobenzene	8.13E+01	ORNL	1.00E+00	USEPA 2004	NA	USEPA 2005	6.90E-02	ORNL	7.90E-06	ORNL	Yes	9.85E-02	ORNL	4.34E+02	ORNL	2.60E+00	calculated
Benzene	1.79E+03	ORNL	1.00E+00	USEPA 2004	NA	USEPA 2006	8.80E-02	ORNL	9.80E-06	ORNL	Yes	2.27E-01	ORNL	1.66E+02	ORNL	9.93E-01	ORNL

Attachment A

Table 14
Chemical - Specific Values

Chemical	Solubility	Solubility Source	GIABS	GIABS Source	ABSd	ABSd Source	Di	Di Source	Dw	Dw Source	VOCs	HLC	HLC Source	Koc	Koc Source	Kd	Kd Source
Carbon tetrachloride	7.93E+02	ORNL	1.00E+00	USEPA 2004	NA	USEPA 2007	7.80E-02	ORNL	8.80E-06	ORNL	Yes	1.13E+00	ORNL	4.86E+01	ORNL	2.92E-01	calculated
Chloromethane	5.32E+03	ORNL	1.00E+00	USEPA 2004	NA	USEPA 2008	1.26E-01	ORNL	6.50E-06	ORNL	Yes	3.61E-01	ORNL	1.43E+01	ORNL	8.58E-02	calculated
Dichlorodifluoromethane	2.80E+02	ORNL	1.00E+00	USEPA 2004	NA	USEPA 2009	5.20E-02	ORNL	1.05E-05	ORNL	Yes	1.40E+01	ORNL	4.86E+01	ORNL	2.92E-01	calculated
Ethylbenzene	1.69E+02	ORNL	1.00E+00	USEPA 2004	NA	USEPA 2010	7.50E-02	ORNL	7.80E-06	ORNL	Yes	3.22E-01	ORNL	5.18E+02	ORNL	3.11E+00	ORNL
Methylene chloride	1.30E+04	ORNL	1.00E+00	USEPA 2004	NA	USEPA 2011	1.01E-01	ORNL	1.17E-05	ORNL	Yes	1.33E-01	ORNL	2.37E+01	ORNL	1.42E-01	calculated
m & p-Xylenes	161/162	ORNL	1.00E+00	USEPA 2004	NA	USEPA 2012	7.14E-02	ORNL	9.34E-06	ORNL	Yes	2.71E-01	ORNL	4.43E+02	ORNL	2.66E+00	ORNL calculated
n-Butyl benzene	1.08E+01	TCEQ	1.00E+00	USEPA 2004	NA	USEPA 2013	5.70E-02	TCEQ	6.74E-06	TCEQ	Yes	5.57E-01	TCEQ	2.51E+03	HSDB	1.51E+01	calculated
o-Xylene	1.06E+02	ORNL	1.00E+00	USEPA 2004	NA	USEPA 2014	8.70E-02	ORNL	1.00E-05	ORNL	Yes	2.12E-01	ORNL	4.43E+02	ORNL	2.66E+00	ORNL calculated
sec-Butyl benzene	1.81E+01	TCEQ	1.00E+00	USEPA 2004	NA	USEPA 2015	5.76E-02	TCEQ	6.75E-06	TCEQ	Yes	5.07E-01	TCEQ	2.09E+03	TCEQ	1.25E+01	TCEQ calculated
Styrene	3.10E+02	ORNL	1.00E+00	USEPA 2004	NA	USEPA 2016	7.10E-02	ORNL	8.00E-06	ORNL	Yes	1.12E-01	ORNL	5.18E+02	ORNL	3.11E+00	ORNL
Toluene	5.26E+02	ORNL	1.00E+00	USEPA 2004	NA	USEPA 2017	8.70E-02	ORNL	8.60E-06	ORNL	Yes	2.71E-01	ORNL	2.68E+02	ORNL	1.61E+00	ORNL
Trichloroethene	1.28E+03	ORNL	1.00E+00	USEPA 2004	NA	USEPA 2018	7.90E-02	ORNL	9.10E-06	ORNL	Yes	4.03E-01	ORNL	6.77E+01	ORNL	4.06E-01	calculated
Trichlorofluoromethane	1.10E+03	ORNL	1.00E+00	USEPA 2004	NA	USEPA 2019	8.70E-02	ORNL	9.70E-06	ORNL	Yes	3.97E+00	ORNL	4.86E+01	ORNL	2.92E-01	calculated
Xylenes (total)	1.06E+02	ORNL	1.00E+00	USEPA 2004	NA	USEPA 2020	7.14E-02	ORNL	9.34E-06	ORNL	Yes	2.71E-01	ORNL	4.43E+02	ORNL	2.66E+00	ORNL

Volatile organic compounds (VOCs) are defined as those chemicals having a Henry's Law constant greater than 10^{-5} (atm-m^3/mol) and a molecular weight less than 200 g/mole).

Except for Solubility, table values are current as of 02/06. Values for solubility were downloaded from ORNL and TCEQ 01/07.

GIABS – Gastrointestinal Absorption
ABSd – Dermal Absorption Factor
Di – Diffusivity in Air (cm^2/s)
Dw – Diffusivity in Water (cm^2/s)
HLC – Dimensionless Henry's Law Constant
Koc – Organic Carbon Partition Coefficient (cm^3/g)
Kd – Soil - Water Partition Coefficient (cm^3/g). The Kd was calculated assuming the fraction organic carbon (foc) was 0.006 g/g. The equation used. Kd = Koc × foc.
NV – No Value
USEPA – United States Environmental Protection Agency
NA – Not Applicable
ORNL – Oak Ridge National Laboratory Risk Assessment Information System
SVOC – Semivolatile organic compound
TCEQ – Texas Commission on Environmental Quality
VOC – Volatile organic compound

APPENDIX B

Attachment B1

Exposure Point Concentration Summary

Attachment B1

Table 1
Exposure Point Concentration
Residential Receptor Exposed to Surface and Subsurface Soil (0-10 feet)

Analyte	Detection Range (mg/kg)	Frequency of Detection	Maximum Concentration (mg/kg)	Average Concentration (mg/kg)	95% UCL (mg/kg)	Distribution	Statistic used for 95% UCL	Rationale	Selected EPC (mg/kg)
Inorganics									
Arsenic	0.87 - 23.4	87 / 103	23.4	4.3	5.07	lognormal	Standard Boostrap		5.07
Lead	6 - 4000	82 / 103	4000	90.5	-	-	-	Per EPA Guidance	90.48
Thallium	1.5 - 1.8	2 / 86	1.8	0.8	0.82	normal	Student's-t UCL		0.82
SVOCs									
1-Methylnaphthalene	0.051 - 3800	36 / 86	3800	75.3	150	non-parametric	Standard Boostrap		150
2-Methylnaphthalene	0.0076 - 3300	47 / 102	3300	80.5	144	non-parametric	Standard Boostrap		144
Acenaphthene	0.00059 - 670	33 / 101	670	21.8	36	non-parametric	Standard Boostrap		36.42
Benzo(a)anthracene	0.044 - 520	50 / 102	520	14.6	24.6	non-parametric	Standard Boostrap		24.59
Benzo(a)pyrene	0.06 - 340	50 / 104	340	12.3	20	non-parametric	Standard Boostrap		19.51
Benzo(b)fluoranthene	0.07 - 410	50 / 102	410	13	21.3	non-parametric	Standard Boostrap		21.28
Benzo(k)fluoranthene	0.045 - 170	35 / 102	170	6.4	10.1	non-parametric	Standard Boostrap		10.12
Chrysene	0.059 - 470	47 / 102	470	14	23.4	non-parametric	Standard Boostrap		23.38
Dibenzo(a,h)anthracene	0.12 - 3.8	6 / 102	3.8	2	3.1	non-parametric	Standard Boostrap		3.12
Dibenzofuran	0.079 - 280	14 / 86	280	4.7	9.9	non-parametric	Standard Boostrap		9.93
Fluoranthene	0.047 - 1400	57 / 103	1400	36.7	64	non-parametric	Standard Boostrap		63.62
Fluorene	0.0015 - 1200	34 / 101	1200	25.4	46	non-parametric	Standard Boostrap		45.88
Indeno(1,2,3-cd)pyrene	0.037 - 100	32 / 102	100	3.4	5.3	non-parametric	Standard Boostrap		5.34
Naphthalene	0.00024 - 2900	62 / 123	2900	121.2	186	non-parametric	Standard Boostrap		186
Phenanthrene	0.0054 - 3700	60 / 104	3700	79.4	144	non-parametric	Standard Boostrap		144
Pyrene	0.048 - 2000	64 / 104	2000	49.2	86	non-parametric	Standard Boostrap		85.60
VOCs									
1,2,4-Trichlorobenzene	0.0097 - 120	7 / 16	120	15.4	30	non-parametric	Standard Boostrap		29.62
1,2,4-Trimethylbenzene	0.02 - 100	43 / 87	100	3.8	6.1	non-parametric	Standard Boostrap		6.06
1,3,5-Trimethylbenzene	0.0079 - 560	37 / 103	560	6.9	15.7	non-parametric	Standard Boostrap		15.74
Benzene	0.016 - 230	49 / 104	230	8.4	14.3	non-parametric	Standard Boostrap		14.29
Xylenes (total)	0.046 - 320	7 / 18	320	23.3	52	non-parametric	Standard Boostrap		52.19

All units are milligrams per kilogram (mg/kg).

EPC– Exposure Point Concentration
mg/kg – milligrams per kilogram
SVOC– Semi-volatile organic compound
UCL– Upper Confidence Limit
VOC– Volatile organic compound

Attachment B1

Table 2
Exposure Point Concentration
Recreational Receptor Exposed to Surface Soil

Analyte	Detection Range (mg/kg)	Frequency of Detection	Maximum Concentration (mg/kg)	Average Concentration (mg/kg)	95% UCL (mg/kg)	Distribution	Statistic used for 95% UCL	Rationale	Selected EPC (mg/kg)
Inorganics									
Aluminum	870 - 14000	24 / 24	14000	8561	9535	normal	Standard Bootstrap		9,534.67
Arsenic	1.3 - 13.9	30 / 33	13.9	4.43	5.48	non-parametric	Standard Bootstrap	-	5.48
Cadmium	0.145 - 12	18 / 33	12	0.77	1.35	non-parametric	Standard Bootstrap	-	1.35
Iron	4100 - 94000	33 / 33	94000	21375	25468	non-parametric	Standard Bootstrap		25,467.52
Lead	5.2 - 950	32 / 33	950	107.94	-	-	-	Per EPA Guidance	107.94
Manganese	100 - 670	24 / 24	670	311.25	347.47	non-parametric	Standard Bootstrap		347.47
Thallium	2.4 - 2.4	1 / 24	2.4	0.93	1.04	non-parametric	Standard Bootstrap	-	1.04
Vanadium	8.9 - 63	24 / 24	63	33.37	36.57	non-parametric	Standard Bootstrap		36.57
Zinc	4.3 - 4100	33 / 33	4100	261.23	461.31	lognormal	Standard Bootstrap		461.31
SVOCs									
Benzo(a)anthracene	0.24 - 25	15 / 27	25	2.10	3.60	lognormal	Standard Bootstrap	-	3.60
Benzo(a)pyrene	0.29 - 68	16 / 33	68	3.64	7.10	non-parametric	Standard Bootstrap	-	7.10
Benzo(b)fluoranthene	0.33 - 53	16 / 27	53	3.67	6.85	lognormal	Standard Bootstrap	-	6.85
Benzo(k)fluoranthene	0.19 - 15	5 / 24	15	0.93	1.93	non-parametric	Standard Bootstrap	-	1.93
Indeno(1,2,3-cd)pyrene	0.155 - 33	9 / 27	33	2.54	4.72	non-parametric	Standard Bootstrap	-	4.72

All units are milligrams per kilogram (mg/kg).

EPC– Exposure Point Concentration
mg/kg – milligrams per kilogram
Sd– Standard Deviation
SVOC– Semi-volatile organic compound
UCL– Upper Confidence Limit
VOC– Volatile organic compound

Attachment B1

Table 3
Exposure Point Concentration
Recreational Receptor Exposed to Sediment

Analyte	Detection Range (mg/kg)	Maximum Concentration (mg/kg)	Average Concentration (mg/kg)	95% UCL (mg/kg)	Distribution	Statistic used for 95% UCL	Rationale	Selected EPC (mg/kg)
Inorganics								
Iron	9000 – 27000	27000	18000	NA	NA	Maximum Concentration		27,000
Manganese	110 – 300	300	205	NA	NA	Maximum Concentration		300
Vanadium	9 – 9.7	9.7	9.35	NA	NA	Maximum Concentration		9.7
SVOCs								
1-Methylnaphthalene	0.084 – 19	19	11.36	NA	NA	Maximum Concentration		19
2-Methylnaphthalene	0.059 – 2380	2380	248.293	546.04	gamma	Standard Bootstrap		546
Acenaphthene	0.12 – 833	833	89.39428571	184.8	gamma	Standard Bootstrap		184.8
Benzo(a)anthracene	0.86 – 150	150	22.92928571	39.56	gamma	Standard Bootstrap		39.56
Benzo(a)pyrene	0.88 – 100	100	17.43071429	28.60	gamma	Standard Bootstrap		28.6
Benzo(b)fluoranthene	0.99 – 50	50	9.874285714	15.05	gamma	Standard Bootstrap		15.05
Benzo(k)fluoranthene	0.8 – 71	71	11.46785714	19.12	gamma	Standard Bootstrap		19.12
Chrysene	0.96 – 130	130	21.04357143	35.31	gamma	Standard Bootstrap		35.31
Dibenzo(a,h)anthracene	0.27 – 1.9	1.9	6.516923077	9.60	gamma	Standard Bootstrap		1.9
Fluoranthene	1.6 – 330	330	48.52857143	86.5	gamma	Standard Bootstrap		86.5
Fluorene	0.047 – 500	500	52.12284615	112.59	lognormal	Standard Bootstrap		113
Indeno(1,2,3-cd)pyrene	0.6 – 5.3	5.3	6.839285714	9.56	gamma	Standard Bootstrap		5.30
Naphthalene	0.094 – 2710	2710	266.3885357	572.82	lognormal	Standard Bootstrap		573
Pyrene	1.6 – 410	410	61.95357143	110.09	gamma	Standard Bootstrap		110
VOCs								
Methylene chloride	0.15 – 36.8	36.8	6.494	12.99	non-parametric	Standard Bootstrap		12.99
Xylenes (total)	0.069 – 31.1	31.1	5.008785714	10.29	non-parametric	Standard Bootstrap		10.29

Notes:

All units are milligrams per kilogram (mg/kg).

EPC – Exposure point concentration
mg/kg – milligrams per kilogram
SVOC – Semivolatile organic compound
VOC – Volatile organic compound
UCL – Upper confidence Limit

Attachment B1

Table 4
Exposure Point Concentration
Industrial Worker Receptor Exposed to Surface Soil

Analyte	Detection Range (mg/kg)	Maximum Concentration (mg/kg)	Average Concentration (mg/kg)	95% UCL (mg/kg)	Distribution	Statistic used for 95% UCL	Rationale	Selected EPC (mg/kg)
Inorganics								
Arsenic	1.4 - 8.5	8.5	2.75	3.65	Gamma	Standard Bootstrap		3.65
SVOCs								
Benzo(a)pyrene	0.034 - 0.39	0.39	0.14	0.18	Gamma	Standard Bootstrap		0.18

All units are milligrams per kilogram (mg/kg).
EPC– Exposure Point Concentration
mg/kg – milligrams per kilogram
SVOC– Semi-volatile organic compound
UCL– Upper Confidence Limit
VOC– Volatile organic compound

Attachment B1

Table 5
Exposure Point Concentration
Maintenance Worker Receptor Exposed to Surface Soil

Analyte	Detection Range (mg/kg)	Frequency of Detection	Maximum Concentration (mg/kg)	Average Concentration (mg/kg)	95% UCL (mg/kg)	Distribution	Statistic used for 95% UCL	Rationale	Selected EPC (mg/kg)
Inorganics									
Arsenic	1.3 - 13.9	38 / 42	13.9	4.11	4.99	lognormal	Standard Bootstrap		4.99
Lead	5.2 - 950	40 / 42	950	95.07		-	-	per EPA Guidance	95.07
SVOCs									
Benzo(a)anthracene	0.16 - 25	18 / 36	25	1.61	2.83	lognormal	Standard Bootstrap		2.83
Benzo(a)pyrene	0.17 - 68	19 / 42	68	2.89	5.68	non-parametric	Standard Bootstrap		5.68
Benzo(b)fluoranthene	0.23 - 53	20 / 36	53	2.81	5.25	lognormal	Standard Bootstrap		5.25
Indeno(1,2,3-cd)pyrene	0.05 - 33	10 / 36	33	1.92	3.62	non-parametric	Standard Bootstrap		3.62

All units are milligrams per kilogram (mg/kg).

EPC– Exposure Point Concentration
mg/kg – milligrams per kilogram
SVOC– Semi-volatile organic compound
UCL– Upper Confidence Limit
VOC– Volatile organic compound

Attachment B1

Table 6
Exposure Point Concentration
Construction Worker Receptor Exposed to Surface and Subsurface Soil

Analyte	Detection Range (mg/kg)	Frequency of Detection	Maximum Concentration (mg/kg)	Average Concentration (mg/kg)	95% UCL (mg/kg)	Distribution	Statistic used for 95% UCL	Rationale	Selected EPC (mg/kg)
Inorganics									
Arsenic	0.83 - 23.4	126 / 165	23.4	3.94	4.51	non-parametric	Standard Bootstrap		4.51
Lead	3.32 - 4000	151 / 182	4000	95.78	-	-	-	Per EPA Guidance	95.78
Manganese	12 - 2000	124 / 124	2000	275.27	308.25	non-parametric	Standard Bootstrap		308.25
Vanadium	8.3 - 110	124 / 124	110	29.52	31.48				31.48
SVOCs									
1-Methylnaphthalene	0.04 - 21000	63 / 150	21000	942.19	1,435.41	non-parametric	Standard Bootstrap		1,435
2-Methylnaphthalene	0.0076 - 30000	85 / 178	30000	1,044.71	1,596.29	non-parametric	Standard Bootstrap		1,596
Acenaphthene	0.00059 - 8200	76 / 180	8200	263.44	386.26	non-parametric	Standard Bootstrap		386.26
Acenaphthylene	0.045 - 9700	89 / 180	9700	255.20	412.39	non-parametric	Standard Bootstrap		412.39
Benzo(a)anthracene	0.0173 - 2400	116 / 193	2400	121.92	170.53	non-parametric	Standard Bootstrap		170.53
Benzo(a)pyrene	0.0219 - 1800	118 / 209	1800	88.21	122.56	non-parametric	Standard Bootstrap		122.56
Benzo(b)fluoranthene	0.0149 - 1200	118 / 192	1200	54.24	74.53	non-parametric	Standard Bootstrap		74.53
Benzo(k)fluoranthene	0.00669 - 1500	87 / 185	1500	64.99	92.29	non-parametric	Standard Bootstrap		92.29
Chrysene	0.0118 - 2300	108 / 188	2300	109.65	152.62	non-parametric	Standard Bootstrap		152.62
Dibenzo(a,h)anthracene	0.12 - 290	37 / 172	290	12.97	18.34	non-parametric	Standard Bootstrap		18.34
Dibenzofuran	0.042 - 1400	41 / 150	1400	66.95	98.90	non-parametric	Standard Bootstrap		98.90
Fluoranthene	0.047 - 5400	126 / 196	5400	202.71	282.42	non-parametric	Standard Bootstrap		282.42
Fluorene	0.0015 - 4600	80 / 183	4600	203.80	287.57	non-parametric	Standard Bootstrap		287.57
Indeno(1,2,3-cd)pyrene	0.0113 - 1200	89 / 189	1200	47.14	67.20	non-parametric	Standard Bootstrap		67.20
Naphthalene	0.00024 - 37000	130 / 246	37000	769.61	1,167.64	non-parametric	Standard Bootstrap		1,168
Pyrene	0.042 - 7600	137 / 209	7600	285.55	395.41	non-parametric	Standard Bootstrap		395.41
VOCs									
1,2,3-Trimethylbenzene	0.037 - 37	23 / 124	37	9.68	19.60	non-parametric	Standard Bootstrap		19.60
1,2,4-Trichlorobenzene	0.0097 - 2994	21 / 31	2994	109.92	264.44	non-parametric	Standard Bootstrap		264.44
1,2,4-Trimethylbenzene	0.019 - 100	47 / 125	100	2.63	4.21	non-parametric	Standard Bootstrap		4.21
1,3,5-Trimethylbenzene	0.0079 - 742	47 / 152	742	9.68	19.68	non-parametric	Standard Bootstrap		19.68
Benzene	0.005 - 645	68 / 183	645	8.39	15.10	non-parametric	Standard Bootstrap		15.10
Ethylbenzene	0.014 - 2973	54 / 167	2973	21.94	50.43	non-parametric	Standard Bootstrap		50.43
n-Butyl benzene	0.0094 - 648	18 / 31	648	25.97	59.25	non-parametric	Standard Bootstrap		59.25
p-Isopropyltoluene	0.019 - 213	20 / 32	213	11.20	23.01	non-parametric	Standard Bootstrap		23.01
sec-Butyl benzene	0.03 - 2688	10 / 143	2688	19.94	50.17	non-parametric	Standard Bootstrap		50.17
Toluene	0.0055 - 2007	122 / 183	2007	15.43	33.63	non-parametric	Standard Bootstrap		33.63
Xylenes (total)	0.029 - 4981	24 / 59	4981	96.17	234.96	non-parametric	Standard Bootstrap		234.96

All units are milligrams per kilogram (mg/kg).

EPC– Exposure Point Concentration
mg/kg – milligrams per kilogram
SVOC– Semi-volatile organic compound
UCL– Upper Confidence Limit
VOC– Volatile organic compound

Attachment B1

Table 7
Exposure Point Concentration
Subsistence Fisher Receptor Exposed to Chemicals in Fin Fish

Analyte	Detection Range (mg/kg)	Maximum Concentration (mg/kg)	Average Concentration (mg/kg)	95% UCL (mg/kg)	Distribution	Statistic used for 95% UCL	Rationale	Selected EPC (mg/kg)
Shorthead Redhorse & Walleye								
1-Methylnaphthalene	0.0031 – 0.62	0.62	109	204	non-parametric	Approximate Gamma		0.62
Smelt								
Benzo(a)anthracene	0.0021 – 0.017	0.17	NC	NC	–	–		0.17
Benzo(b)fluoranthene	0.0034 – 0.019	0.019	NC	NC	–	–		0.019
Benzo(e)pyrene	0.0098 – 0.0098	0.0098	NC	NC	–	–		0.0098
Benzo(a)pyrene	0.0024 – 0.015	0.015	NC	NC	–	–		0.015
Indeno(1,2,3-cd)pyrene	0.0076 – 0.0076	0.0076	NC	NC	–	–		0.0076
Dibenzo(a,h)anthracene	0.0026 – 0.0026	0.0026	NC	NC	–	–		0.0026

Notes:

All units are milligrams per kilogram (mg/kg).
EPC– Exposure Point Concentration
mg/kg – milligrams per kilogram
UCL– Upper Confidence Limit

Attachment B2

ProUCL Output Tables

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
1,2,3-Trimethylbenzene

Raw Statistics	
Number of Valid Samples	152
Number of Unique Samples	52
Minimum	0.0079
Maximum	742
Mean	9.67654868
Median	0.008
Standard Deviation	75.1458279
Variance	5646.89546
Coefficient of Variation	7.76576757
Skewness	8.84528234

Gamma Statistics	
k hat	0.14069604
k star (bias corrected)	0.14230511
Theta hat	68.7762702
Theta star	67.9986046
nu hat	42.7715954
nu star	43.2607525
Approx.Chi Square Value (.05)	29.1765675
Adjusted Level of Significance	0.04842105
Adjusted Chi Square Value	29.0660968

Log-transformed Statistics	
Minimum of log data	-4.84089252
Maximum of log data	6.60934924
Mean of log data	-3.24357976
Standard Deviation of log data	2.57699203
Variance of log data	6.6408879

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.47663899
Lilliefors 5% Critical Value	0.07186409
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	19.7640341

Gamma Distribution Test	
A-D Test Statistic	34.8544031
A-D 5% Critical Value	1.00203191
K-S Test Statistic	0.33588309
K-S 5% Critical Value	0.08683465
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	14.3476362
Adjusted Gamma UCL	14.4021669

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.35570782
Lilliefors 5% Critical Value	0.07186409
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	2.44318935
95% Chebyshev (MVUE) UCL	2.65370955
97.5% Chebyshev (MVUE) UCL	3.37458318
99% Chebyshev (MVUE) UCL	4.79059983

95% Non-parametric UCLs	
CLT UCL	19.7021479
Adj-CLT UCL (Adjusted for skewness)	24.3746919
Mod-t UCL (Adjusted for skewness)	20.4928566
Jackknife UCL	19.7640341
Standard Bootstrap UCL	19.6016945
Bootstrap-t UCL	187.581516
Hall's Bootstrap UCL	127.764647
Percentile Bootstrap UCL	20.2203546
BCA Bootstrap UCL	26.7031349
95% Chebyshev (Mean, Sd) UCL	36.2446109
97.5% Chebyshev (Mean, Sd) UCL	47.7406327
99% Chebyshev (Mean, Sd) UCL	70.3223414

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
1,2,4-Trichlorobenzene

Raw Statistics	
Number of Valid Samples	31
Number of Unique Samples	24
Minimum	0.0097
Maximum	2994
Mean	109.919345
Median	0.159
Standard Deviation	536.569737
Variance	287907.082
Coefficient of Variation	4.88148593
Skewness	5.52541904

Gamma Statistics	
k hat	0.14136119
k star (bias corrected)	0.14918646
Theta hat	777.577929
Theta star	736.79172
nu hat	8.76439408
nu star	9.24956025
Approx.Chi Square Value (.05)	3.47761021
Adjusted Level of Significance	0.0413
Adjusted Chi Square Value	3.28273402

Log-transformed Statistics	
Minimum of log data	-4.63562939
Maximum of log data	8.00436557
Mean of log data	-0.78390612
Standard Deviation of log data	3.11503665
Variance of log data	9.70345331

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.21595119
Shapiro-Wilk 5% Critical Value	0.929
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	273.48566

Gamma Distribution Test	
A-D Test Statistic	5.30983979
A-D 5% Critical Value	0.92818353
K-S Test Statistic	0.35170066
K-S 5% Critical Value	0.17685798
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	292.357551
Adjusted Gamma UCL	309.713062

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.82955596
Shapiro-Wilk 5% Critical Value	0.929
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	1391.08227
95% Chebyshev (MVUE) UCL	129.233323
97.5% Chebyshev (MVUE) UCL	172.249923
99% Chebyshev (MVUE) UCL	256.747704

95% Non-parametric UCLs	
CLT UCL	268.435155
Adj-CLT UCL (Adjusted for skewness)	370.625559
Mod-t UCL (Adjusted for skewness)	289.425298
Jackknife UCL	273.48566
Standard Bootstrap UCL	264.438416
Bootstrap-t UCL	2594.15149
Hall's Bootstrap UCL	2089.93722
Percentile Bootstrap UCL	298.976
BCA Bootstrap UCL	483.832936
95% Chebyshev (Mean, Sd) UCL	529.989791
97.5% Chebyshev (Mean, Sd) UCL	711.754609
99% Chebyshev (Mean, Sd) UCL	1068.7964

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
1,2,4-Trimethylbenzene

Raw Statistics	
Number of Valid Samples	125
Number of Unique Samples	42
Minimum	0.0075
Maximum	100
Mean	2.629408
Median	0.0075
Standard Deviation	10.7368124
Variance	115.27914
Coefficient of Variation	4.08335732
Skewness	7.12503476

Gamma Statistics	
k hat	0.17698369
k star (bias corrected)	0.17806942
Theta hat	14.8567812
Theta star	14.7661965
nu hat	44.2459232
nu star	44.5173543
Approx.Chi Square Value (.05)	30.2113949
Adjusted Level of Significance	0.04808
Adjusted Chi Square Value	30.0741965

Log-transformed Statistics	
Minimum of log data	-4.89285226
Maximum of log data	4.60517019
Mean of log data	-3.27039013
Standard Deviation of log data	2.64386699
Variance of log data	6.99003264

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.40527026
Lilliefors 5% Critical Value	0.07924625
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	4.22089968

Gamma Distribution Test	
A-D Test Statistic	24.5872873
A-D 5% Critical Value	0.94214115
K-S Test Statistic	0.36050829
K-S 5% Critical Value	0.09289881
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	3.87450789
Adjusted Gamma UCL	3.89218337

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.34628338
Lilliefors 5% Critical Value	0.07924625
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	3.26124009
95% Chebyshev (MVUE) UCL	3.2170805
97.5% Chebyshev (MVUE) UCL	4.12728736
99% Chebyshev (MVUE) UCL	5.91521249

95% Non-parametric UCLs	
CLT UCL	4.20900978
Adj-CLT UCL (Adjusted for skewness)	4.86294203
Mod-t UCL (Adjusted for skewness)	4.32289989
Jackknife UCL	4.22089968
Standard Bootstrap UCL	4.212669
Bootstrap-t UCL	6.36348835
Hall's Bootstrap UCL	11.0422899
Percentile Bootstrap UCL	4.382248
BCA Bootstrap UCL	5.204116
95% Chebyshev (Mean, Sd) UCL	6.81538809
97.5% Chebyshev (Mean, Sd) UCL	8.62666502
99% Chebyshev (Mean, Sd) UCL	12.1845678

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
1,3,5-Trimethylbenzene

Raw Statistics	
Number of Valid Samples	152
Number of Unique Samples	52
Minimum	0.0079
Maximum	742
Mean	9.67654868
Median	0.008
Standard Deviation	75.1458279
Variance	5646.89546
Coefficient of Variation	7.76576757
Skewness	8.84528234

Gamma Statistics	
k hat	0.14069604
k star (bias corrected)	0.14230511
Theta hat	68.7762702
Theta star	67.9986046
nu hat	42.7715954
nu star	43.2607525
Approx.Chi Square Value (.05)	29.1765675
Adjusted Level of Significance	0.04842105
Adjusted Chi Square Value	29.0660968

Log-transformed Statistics	
Minimum of log data	-4.84089252
Maximum of log data	6.60934924
Mean of log data	-3.24357976
Standard Deviation of log data	2.57699203
Variance of log data	6.6408879

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.47663899
Lilliefors 5% Critical Value	0.07186409
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	19.7640341

Gamma Distribution Test	
A-D Test Statistic	34.8544031
A-D 5% Critical Value	1.00203191
K-S Test Statistic	0.33588309
K-S 5% Critical Value	0.08683465
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	14.3476362
Adjusted Gamma UCL	14.4021669

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.35570782
Lilliefors 5% Critical Value	0.07186409
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	2.44318935
95% Chebyshev (MVUE) UCL	2.65370955
97.5% Chebyshev (MVUE) UCL	3.37458318
99% Chebyshev (MVUE) UCL	4.79059983

95% Non-parametric UCLs	
CLT UCL	19.7021479
Adj-CLT UCL (Adjusted for skewness)	24.3746919
Mod-t UCL (Adjusted for skewness)	20.4928566
Jackknife UCL	19.7640341
Standard Bootstrap UCL	19.6799243
Bootstrap-t UCL	188.000811
Hall's Bootstrap UCL	125.60266
Percentile Bootstrap UCL	20.6603599
BCA Bootstrap UCL	26.0620428
95% Chebyshev (Mean, Sd) UCL	36.2446109
97.5% Chebyshev (Mean, Sd) UCL	47.7406327
99% Chebyshev (Mean, Sd) UCL	70.3223414

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
1-Methylnaphthalene

Raw Statistics	
Number of Valid Samples	150
Number of Unique Samples	64
Minimum	0.016
Maximum	21000
Mean	942.191487
Median	0.1225
Standard Deviation	3535.54124
Variance	12500051.9
Coefficient of Variation	3.7524657
Skewness	4.54435523

Gamma Statistics	
k hat	0.11143506
k star (bias corrected)	0.1136508
Theta hat	8455.07231
Theta star	8290.23168
nu hat	33.4305179
nu star	34.0952409
Approx.Chi Square Value (.05)	21.7383432
Adjusted Level of Significance	0.0484
Adjusted Chi Square Value	21.6427214

Log-transformed Statistics	
Minimum of log data	-4.13516656
Maximum of log data	9.95227772
Mean of log data	-0.33880031
Standard Deviation of log data	4.27466805
Variance of log data	18.272787

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.45315516
Lilliefors 5% Critical Value	0.0723416
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	1419.99144

Gamma Distribution Test	
A-D Test Statistic	21.4525893
A-D 5% Critical Value	1.04432726
K-S Test Statistic	0.29486479
K-S 5% Critical Value	0.08868243
Data do not follow gamma distribution at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	1477.76881
Adjusted Gamma UCL	1484.29789

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.20995738
Lilliefors 5% Critical Value	0.0723416
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	55475.2689
95% Chebyshev (MVUE) UCL	16642.4212
97.5% Chebyshev (MVUE) UCL	22166.7848
99% Chebyshev (MVUE) UCL	33018.3274

95% Non-parametric UCLs	
CLT UCL	1417.02081
Adj-CLT UCL (Adjusted for skewness)	1531.47122
Mod-t UCL (Adjusted for skewness)	1437.84339
Jackknife UCL	1419.99144
Standard Bootstrap UCL	1435.41394
Bootstrap-t UCL	1699.64993
Hall's Bootstrap UCL	1473.12112
Percentile Bootstrap UCL	1474.42427
BCA Bootstrap UCL	1531.86316
95% Chebyshev (Mean, Sd) UCL	2200.49984
97.5% Chebyshev (Mean, Sd) UCL	2744.97086
99% Chebyshev (Mean, Sd) UCL	3814.47877

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
2-Methylnaphthalene

Raw Statistics	
Number of Valid Samples	178
Number of Unique Samples	91
Minimum	0.0076
Maximum	30000
Mean	1044.70618
Median	0.18
Standard Deviation	4294.05217
Variance	18438884.1
Coefficient of Variation	4.11029654
Skewness	5.18593958

Gamma Statistics	
k hat	0.11174535
k star (bias corrected)	0.11360732
Theta hat	9348.99052
Theta star	9195.76501
nu hat	39.781343
nu star	40.4442042
Approx.Chi Square Value (.05)	26.8692334
Adjusted Level of Significance	0.04865169
Adjusted Chi Square Value	26.7791131

Log-transformed Statistics	
Minimum of log data	-4.87960703
Maximum of log data	10.3089527
Mean of log data	-0.21294248
Standard Deviation of log data	4.15906551
Variance of log data	17.2978259

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.44860322
Lilliefors 5% Critical Value	0.06640851
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	1576.89227

Gamma Distribution Test	
A-D Test Statistic	25.3963173
A-D 5% Critical Value	1.06460897
K-S Test Statistic	0.28762518
K-S 5% Critical Value	0.0808715
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	1572.51639
Adjusted Gamma UCL	1577.80841

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.1697604
Lilliefors 5% Critical Value	0.06640851
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	28535.2627
95% Chebyshev (MVUE) UCL	12365.3808
97.5% Chebyshev (MVUE) UCL	16401.4815
99% Chebyshev (MVUE) UCL	24329.6197

95% Non-parametric UCLs	
CLT UCL	1574.10695
Adj-CLT UCL (Adjusted for skewness)	1707.78349
Mod-t UCL (Adjusted for skewness)	1597.7431
Jackknife UCL	1576.89227
Standard Bootstrap UCL	1596.29456
Bootstrap-t UCL	1940.7321
Hall's Bootstrap UCL	1650.04257
Percentile Bootstrap UCL	1622.77981
BCA Bootstrap UCL	1762.04257
95% Chebyshev (Mean, Sd) UCL	2447.63008
97.5% Chebyshev (Mean, Sd) UCL	3054.67638
99% Chebyshev (Mean, Sd) UCL	4247.10128

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
Acenaphthene

Raw Statistics	
Number of Valid Samples	180
Number of Unique Samples	84
Minimum	0.00059
Maximum	8200
Mean	263.443911
Median	0.16
Standard Deviation	995.967976
Variance	991952.209
Coefficient of Variation	3.78056936
Skewness	5.59623273

Gamma Statistics	
k hat	0.12863573
k star (bias corrected)	0.1301955
Theta hat	2047.98395
Theta star	2023.44859
nu hat	46.3088627
nu star	46.8703817
Approx.Chi Square Value (.05)	32.1574792
Adjusted Level of Significance	0.04866667
Adjusted Chi Square Value	32.0594073

Log-transformed Statistics	
Minimum of log data	-7.43538802
Maximum of log data	9.01188943
Mean of log data	-0.53273609
Standard Deviation of log data	3.84697354
Variance of log data	14.7992054

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.43687555
Lilliefors 5% Critical Value	0.06603854
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	386.18495

Gamma Distribution Test	
A-D Test Statistic	23.3767863
A-D 5% Critical Value	1.03695049
K-S Test Statistic	0.26270408
K-S 5% Critical Value	0.07953016
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	383.97651
Adjusted Gamma UCL	385.151121

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.17290212
Lilliefors 5% Critical Value	0.06603854
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	4572.95121
95% Chebyshev (MVUE) UCL	2661.81928
97.5% Chebyshev (MVUE) UCL	3509.63358
99% Chebyshev (MVUE) UCL	5175.00059

95% Non-parametric UCLs	
CLT UCL	385.549735
Adj-CLT UCL (Adjusted for skewness)	418.636099
Mod-t UCL (Adjusted for skewness)	391.345754
Jackknife UCL	386.18495
Standard Bootstrap UCL	386.256922
Bootstrap-t UCL	440.309992
Hall's Bootstrap UCL	443.175263
Percentile Bootstrap UCL	390.369069
BCA Bootstrap UCL	423.501039
95% Chebyshev (Mean, Sd) UCL	587.027078
97.5% Chebyshev (Mean, Sd) UCL	727.041774
99% Chebyshev (Mean, Sd) UCL	1002.07353

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
Acenaphthylene

Raw Statistics	
Number of Valid Samples	180
Number of Unique Samples	96
Minimum	0.00011
Maximum	9700
Mean	255.196609
Median	0.225
Standard Deviation	1250.15177
Variance	1562879.46
Coefficient of Variation	4.89877894
Skewness	6.20109716

Gamma Statistics	
k hat	0.12947221
k star (bias corrected)	0.13101804
Theta hat	1971.05319
Theta star	1947.79746
nu hat	46.6099949
nu star	47.166495
Approx.Chi Square Value (.05)	32.4031186
Adjusted Level of Significance	0.04866667
Adjusted Chi Square Value	32.3046488

Log-transformed Statistics	
Minimum of log data	-9.11503019
Maximum of log data	9.17988116
Mean of log data	-0.51964412
Standard Deviation of log data	3.61375292
Variance of log data	13.0592102

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.45218759
Lilliefors 5% Critical Value	0.06603854
Data not normal at 5% significance level	
95% UCL (Assuming Normal Distribution)	
Student's-t UCL	409.262735

Gamma Distribution Test	
A-D Test Statistic	24.1403648
A-D 5% Critical Value	1.03550737
K-S Test Statistic	0.26940067
K-S 5% Critical Value	0.07949131
Data do not follow gamma distribution	
at 5% significance level	
95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	371.468245
Adjusted Gamma UCL	372.60054

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.15086579
Lilliefors 5% Critical Value	0.06603854
Data not lognormal at 5% significance level	
95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	1632.37802
95% Chebyshev (MVUE) UCL	1134.36911
97.5% Chebyshev (MVUE) UCL	1487.8588
99% Chebyshev (MVUE) UCL	2182.22086

95% Non-parametric UCLs	
CLT UCL	408.465405
Adj-CLT UCL (Adjusted for skewness)	454.484618
Mod-t UCL (Adjusted for skewness)	416.440802
Jackknife UCL	409.262735
Standard Bootstrap UCL	412.38793
Bootstrap-t UCL	523.900336
Hall's Bootstrap UCL	401.795514
Percentile Bootstrap UCL	413.673473
BCA Bootstrap UCL	469.029353
95% Chebyshev (Mean, Sd) UCL	661.36235
97.5% Chebyshev (Mean, Sd) UCL	837.110591
99% Chebyshev (Mean, Sd) UCL	1182.33398

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
Arsenic

Raw Statistics	
Number of Valid Samples	165
Number of Unique Samples	93
Minimum	0.325
Maximum	23.4
Mean	3.93739394
Median	2.3
Standard Deviation	4.54754338
Variance	20.6801508
Coefficient of Variation	1.15496276
Skewness	2.10911869

Gamma Statistics	
k hat	1.00230574
k star (bias corrected)	0.98812241
Theta hat	3.9283362
Theta star	3.98472285
nu hat	330.760896
nu star	326.080394
Approx.Chi Square Value (.05)	285.234063
Adjusted Level of Significance	0.04854545
Adjusted Chi Square Value	284.900879

Log-transformed Statistics	
Minimum of log data	-1.1239301
Maximum of log data	3.15273602
Mean of log data	0.79478673
Standard Deviation of log data	1.11077973
Variance of log data	1.23383162

RECOMMENDATION

Data are Non-parametric (0.05)

Use 97.5% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.23452201
Lilliefors 5% Critical Value	0.06897501
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	4.52302254

Gamma Distribution Test	
A-D Test Statistic	2.97415127
A-D 5% Critical Value	0.78353451
K-S Test Statistic	0.11154395
K-S 5% Critical Value	0.0748109
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	4.50123999
Adjusted Gamma UCL	4.50650406

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.12192368
Lilliefors 5% Critical Value	0.06897501
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	4.98938566
95% Chebyshev (MVUE) UCL	6.0016874
97.5% Chebyshev (MVUE) UCL	6.83386773
99% Chebyshev (MVUE) UCL	8.46852484

95% Non-parametric UCLs	
CLT UCL	4.51971452
Adj-CLT UCL (Adjusted for skewness)	4.58182635
Mod-t UCL (Adjusted for skewness)	4.53271073
Jackknife UCL	4.52302254
Standard Bootstrap UCL	4.51169902
Bootstrap-t UCL	4.60126322
Hall's Bootstrap UCL	4.56887097
Percentile Bootstrap UCL	4.55424242
BCA Bootstrap UCL	4.59278788
95% Chebyshev (Mean, Sd) UCL	5.4805565
97.5% Chebyshev (Mean, Sd) UCL	6.14828418
99% Chebyshev (Mean, Sd) UCL	7.45990589

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
Benzene

Raw Statistics	
Number of Valid Samples	183
Number of Unique Samples	68
Minimum	0.0025
Maximum	645
Mean	8.38851093
Median	0.0075
Standard Deviation	54.6173724
Variance	2983.05736
Coefficient of Variation	6.5109735
Skewness	9.65014649

Gamma Statistics	
k hat	0.14367517
k star (bias corrected)	0.14496282
Theta hat	58.3852528
Theta star	57.8666359
nu hat	52.5851111
nu star	53.0563934
Approx.Chi Square Value (.05)	37.3203754
Adjusted Level of Significance	0.04868852
Adjusted Chi Square Value	37.2159921

Log-transformed Statistics	
Minimum of log data	-5.99146455
Maximum of log data	6.46925032
Mean of log data	-3.25595207
Standard Deviation of log data	2.86869751
Variance of log data	8.22942542

Normal Distribution Test	
Lilliefors Test Statisitic	0.4496538
Lilliefors 5% Critical Value	0.065495
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	15.0634716

Gamma Distribution Test	
A-D Test Statistic	34.0392321
A-D 5% Critical Value	1.01244273
K-S Test Statistic	0.3134769
K-S 5% Critical Value	0.07798835
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	11.9255
Adjusted Gamma UCL	11.9589486

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.30056661
Lilliefors 5% Critical Value	0.065495
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	5.76587612
95% Chebyshev (MVUE) UCL	6.01685136
97.5% Chebyshev (MVUE) UCL	7.70413289
99% Chebyshev (MVUE) UCL	11.0184707

95% Non-parametric UCLs	
CLT UCL	15.0294962
Adj-CLT UCL (Adjusted for skewness)	18.1069676
Mod-t UCL (Adjusted for skewness)	15.5434949
Jackknife UCL	15.0634716
Standard Bootstrap UCL	15.1023888
Bootstrap-t UCL	26.6577423
Hall's Bootstrap UCL	32.255411
Percentile Bootstrap UCL	15.9342869
BCA Bootstrap UCL	19.03947
95% Chebyshev (Mean, Sd) UCL	25.9872705
97.5% Chebyshev (Mean, Sd) UCL	33.6022678
99% Chebyshev (Mean, Sd) UCL	48.5604555

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
Benzo(a)anthracene

Raw Statistics	
Number of Valid Samples	193
Number of Unique Samples	121
Minimum	0.00011
Maximum	2400
Mean	121.919896
Median	0.42
Standard Deviation	415.630698
Variance	172748.877
Coefficient of Variation	3.40904733
Skewness	4.11232135

Gamma Statistics	
k hat	0.15144071
k star (bias corrected)	0.15254094
Theta hat	805.066837
Theta star	799.260139
nu hat	58.4561155
nu star	58.8808046
Approx.Chi Square Value (.05)	42.2351676
Adjusted Level of Significance	0.04875648
Adjusted Chi Square Value	42.1295514

Log-transformed Statistics	
Minimum of log data	-9.11503019
Maximum of log data	7.78322402
Mean of log data	-0.26470863
Standard Deviation of log data	3.48474983
Variance of log data	12.1434814

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.43394701
Lilliefors 5% Critical Value	0.06377568
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	171.368804

Gamma Distribution Test	
A-D Test Statistic	20.8661628
A-D 5% Critical Value	1.00301596
K-S Test Statistic	0.25749245
K-S 5% Critical Value	0.07480809
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	169.970714
Adjusted Gamma UCL	170.39682

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.13195249
Lilliefors 5% Critical Value	0.06377568
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	1155.61765
95% Chebyshev (MVUE) UCL	919.289831
97.5% Chebyshev (MVUE) UCL	1199.88977
99% Chebyshev (MVUE) UCL	1751.07403

95% Non-parametric UCLs	
CLT UCL	171.13022
Adj-CLT UCL (Adjusted for skewness)	180.592979
Mod-t UCL (Adjusted for skewness)	172.844803
Jackknife UCL	171.368804
Standard Bootstrap UCL	170.53162
Bootstrap-t UCL	185.662597
Hall's Bootstrap UCL	179.797155
Percentile Bootstrap UCL	172.301019
BCA Bootstrap UCL	181.384084
95% Chebyshev (Mean, Sd) UCL	252.328355
97.5% Chebyshev (Mean, Sd) UCL	308.7562
99% Chebyshev (Mean, Sd) UCL	419.597773

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
Benzo(a)pyrene

Raw Statistics	
Number of Valid Samples	209
Number of Unique Samples	112
Minimum	0.00011
Maximum	1800
Mean	88.2059637
Median	0.348
Standard Deviation	305.286197
Variance	93199.662
Coefficient of Variation	3.46106073
Skewness	4.27317732

Gamma Statistics	
k hat	0.15417027
k star (bias corrected)	0.15514709
Theta hat	572.133416
Theta star	568.531205
nu hat	64.4431732
nu star	64.8514849
Approx.Chi Square Value (.05)	47.3193087
Adjusted Level of Significance	0.04885167
Adjusted Chi Square Value	47.2158253

Log-transformed Statistics	
Minimum of log data	-9.11503019
Maximum of log data	7.49554194
Mean of log data	-0.48569231
Standard Deviation of log data	3.52066907
Variance of log data	12.3951107

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.43005282
Lilliefors 5% Critical Value	0.0612859
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	123.095871

Gamma Distribution Test	
A-D Test Statistic	20.433842
A-D 5% Critical Value	1.0040512
K-S Test Statistic	0.23174772
K-S 5% Critical Value	0.07157813
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	120.886967
Adjusted Gamma UCL	121.151916

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.14263604
Lilliefors 5% Critical Value	0.0612859
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	1017.65711
95% Chebyshev (MVUE) UCL	834.57188
97.5% Chebyshev (MVUE) UCL	1088.3468
99% Chebyshev (MVUE) UCL	1586.83848

95% Non-parametric UCLs	
CLT UCL	122.940482
Adj-CLT UCL (Adjusted for skewness)	129.609966
Mod-t UCL (Adjusted for skewness)	124.136176
Jackknife UCL	123.095871
Standard Bootstrap UCL	122.555722
Bootstrap-t UCL	132.848066
Hall's Bootstrap UCL	127.899024
Percentile Bootstrap UCL	122.217648
BCA Bootstrap UCL	128.36733
95% Chebyshev (Mean, Sd) UCL	180.253215
97.5% Chebyshev (Mean, Sd) UCL	220.082135
99% Chebyshev (Mean, Sd) UCL	298.318334

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
Benzo(b)fluoranthene

Raw Statistics	
Number of Valid Samples	192
Number of Unique Samples	110
Minimum	0.00011
Maximum	1200
Mean	54.2351475
Median	0.6205
Standard Deviation	171.160867
Variance	29296.0424
Coefficient of Variation	3.15590305
Skewness	4.22370363

Gamma Statistics	
k hat	0.17981062
k star (bias corrected)	0.1804733
Theta hat	301.623715
Theta star	300.516181
nu hat	69.0472784
nu star	69.301748
Approx.Chi Square Value (.05)	51.1352409
Adjusted Level of Significance	0.04875
Adjusted Chi Square Value	51.0178129

Log-transformed Statistics	
Minimum of log data	-9.11503019
Maximum of log data	7.09007684
Mean of log data	-0.16715969
Standard Deviation of log data	3.24164977
Variance of log data	10.5082933

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.40609121
Lilliefors 5% Critical Value	0.06394154
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	74.6521794

Gamma Distribution Test	
A-D Test Statistic	15.7958992
A-D 5% Critical Value	0.95087083
K-S Test Statistic	0.22378188
K-S 5% Critical Value	0.0737441
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	73.5029396
Adjusted Gamma UCL	73.6721217

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.11879534
Lilliefors 5% Critical Value	0.06394154
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	481.687951
95% Chebyshev (MVUE) UCL	436.888802
97.5% Chebyshev (MVUE) UCL	566.216319
99% Chebyshev (MVUE) UCL	820.255183

95% Non-parametric UCLs	
CLT UCL	74.5531552
Adj-CLT UCL (Adjusted for skewness)	78.5764059
Mod-t UCL (Adjusted for skewness)	75.2797252
Jackknife UCL	74.6521794
Standard Bootstrap UCL	74.5310096
Bootstrap-t UCL	82.1094279
Hall's Bootstrap UCL	79.0316385
Percentile Bootstrap UCL	77.3328401
BCA Bootstrap UCL	79.3057657
95% Chebyshev (Mean, Sd) UCL	108.078323
97.5% Chebyshev (Mean, Sd) UCL	131.376308
99% Chebyshev (Mean, Sd) UCL	177.140688

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
Benzo(k)fluoranthene

Raw Statistics	
Number of Valid Samples	185
Number of Unique Samples	93
Minimum	0.00011
Maximum	1500
Mean	64.9922384
Median	0.21
Standard Deviation	226.451732
Variance	51280.3868
Coefficient of Variation	3.48428885
Skewness	4.25052837

Gamma Statistics	
k hat	0.15723057
k star (bias corrected)	0.15828449
Theta hat	413.356234
Theta star	410.603951
nu hat	58.1753127
nu star	58.5652626
Approx.Chi Square Value (.05)	41.9677102
Adjusted Level of Significance	0.0487027
Adjusted Chi Square Value	41.857852

Log-transformed Statistics	
Minimum of log data	-9.11503019
Maximum of log data	7.31322039
Mean of log data	-0.68041687
Standard Deviation of log data	3.26442337
Variance of log data	10.6564599

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.42566546
Lilliefors 5% Critical Value	0.06514002
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	92.5160821

Gamma Distribution Test	
A-D Test Statistic	22.1022754
A-D 5% Critical Value	0.98946136
K-S Test Statistic	0.27039216
K-S 5% Critical Value	0.07679096
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	90.6956203
Adjusted Gamma UCL	90.9336559

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.14647881
Lilliefors 5% Critical Value	0.06514002
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	322.567312
95% Chebyshev (MVUE) UCL	283.358739
97.5% Chebyshev (MVUE) UCL	367.85343
99% Chebyshev (MVUE) UCL	533.826887

95% Non-parametric UCLs	
CLT UCL	92.3775091
Adj-CLT UCL (Adjusted for skewness)	97.9369005
Mod-t UCL (Adjusted for skewness)	93.3832348
Jackknife UCL	92.5160821
Standard Bootstrap UCL	92.2879491
Bootstrap-t UCL	100.536367
Hall's Bootstrap UCL	96.3150683
Percentile Bootstrap UCL	93.9186596
BCA Bootstrap UCL	98.3384297
95% Chebyshev (Mean, Sd) UCL	137.563819
97.5% Chebyshev (Mean, Sd) UCL	168.9656
99% Chebyshev (Mean, Sd) UCL	230.648318

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
Chrysene

Raw Statistics	
Number of Valid Samples	188
Number of Unique Samples	109
Minimum	0.00011
Maximum	2300
Mean	109.652457
Median	0.595
Standard Deviation	364.676577
Variance	132989.005
Coefficient of Variation	3.32574925
Skewness	4.09410951

Gamma Statistics	
k hat	0.15616564
k star (bias corrected)	0.15721974
Theta hat	702.154818
Theta star	697.447156
nu hat	58.7182809
nu star	59.1146204
Approx.Chi Square Value (.05)	42.4334364
Adjusted Level of Significance	0.0487234
Adjusted Chi Square Value	42.324716

Log-transformed Statistics	
Minimum of log data	-9.11503019
Maximum of log data	7.7406644
Mean of log data	-0.19536504
Standard Deviation of log data	3.46298776
Variance of log data	11.9922843

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.42630907
Lilliefors 5% Critical Value	0.06461819
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	153.618054

Gamma Distribution Test	
A-D Test Statistic	19.112101
A-D 5% Critical Value	0.99247461
K-S Test Statistic	0.25227547
K-S 5% Critical Value	0.07599437
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	152.758388
Adjusted Gamma UCL	153.150782

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.12422847
Lilliefors 5% Critical Value	0.06461819
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	1153.10601
95% Chebyshev (MVUE) UCL	913.315546
97.5% Chebyshev (MVUE) UCL	1192.10806
99% Chebyshev (MVUE) UCL	1739.74197

95% Non-parametric UCLs	
CLT UCL	153.400255
Adj-CLT UCL (Adjusted for skewness)	161.885999
Mod-t UCL (Adjusted for skewness)	154.941658
Jackknife UCL	153.618054
Standard Bootstrap UCL	152.618064
Bootstrap-t UCL	165.263632
Hall's Bootstrap UCL	160.247944
Percentile Bootstrap UCL	154.706914
BCA Bootstrap UCL	162.461615
95% Chebyshev (Mean, Sd) UCL	225.585102
97.5% Chebyshev (Mean, Sd) UCL	275.74925
99% Chebyshev (Mean, Sd) UCL	374.287006

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
Dibenz(a,h)anthracene

Raw Statistics	
Number of Valid Samples	172
Number of Unique Samples	64
Minimum	0.00011
Maximum	290
Mean	12.9722739
Median	0.17
Standard Deviation	41.7783232
Variance	1745.42829
Coefficient of Variation	3.22058596
Skewness	4.15897631

Gamma Statistics	
k hat	0.19095356
k star (bias corrected)	0.19149894
Theta hat	67.9341832
Theta star	67.7407085
nu hat	65.6880235
nu star	65.8756355
Approx.Chi Square Value (.05)	48.1955855
Adjusted Level of Significance	0.04860465
Adjusted Chi Square Value	48.068385

Log-transformed Statistics	
Minimum of log data	-9.11503019
Maximum of log data	5.66988092
Mean of log data	-1.31890218
Standard Deviation of log data	2.84619979
Variance of log data	8.10085325

Normal Distribution Test	
Lilliefors Test Statisitic	0.41828303
Lilliefors 5% Critical Value	0.06755687
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	18.2406063

Gamma Distribution Test	
A-D Test Statistic	19.4127003
A-D 5% Critical Value	0.92869343
K-S Test Statistic	0.2687715
K-S 5% Critical Value	0.07891651
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	17.7310178
Adjusted Gamma UCL	17.7779384

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.16066275
Lilliefors 5% Critical Value	0.06755687
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	38.221039
95% Chebyshev (MVUE) UCL	39.3242992
97.5% Chebyshev (MVUE) UCL	50.3991852
99% Chebyshev (MVUE) UCL	72.1536544

95% Non-parametric UCLs	
CLT UCL	18.2120658
Adj-CLT UCL (Adjusted for skewness)	19.2914832
Mod-t UCL (Adjusted for skewness)	18.4089736
Jackknife UCL	18.2406063
Standard Bootstrap UCL	18.3418382
Bootstrap-t UCL	19.8748651
Hall's Bootstrap UCL	19.1260046
Percentile Bootstrap UCL	18.4203721
BCA Bootstrap UCL	20.2300665
95% Chebyshev (Mean, Sd) UCL	26.8578398
97.5% Chebyshev (Mean, Sd) UCL	32.8661352
99% Chebyshev (Mean, Sd) UCL	44.6682681

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
Dibenzofuran

Raw Statistics	
Number of Valid Samples	150
Number of Unique Samples	51
Minimum	0.016
Maximum	1400
Mean	66.9486767
Median	0.08
Standard Deviation	238.655828
Variance	56956.604
Coefficient of Variation	3.56475795
Skewness	4.20783983

Gamma Statistics	
k hat	0.14051232
k star (bias corrected)	0.14214652
Theta hat	476.461257
Theta star	470.983585
nu hat	42.153696
nu star	42.6439554
Approx.Chi Square Value (.05)	28.6698173
Adjusted Level of Significance	0.0484
Adjusted Chi Square Value	28.5588985

Log-transformed Statistics	
Minimum of log data	-4.13516656
Maximum of log data	7.24422752
Mean of log data	-1.31759516
Standard Deviation of log data	3.2935922
Variance of log data	10.8477496

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg)

Normal Distribution Test	
Lilliefors Test Statisitic	0.45633689
Lilliefors 5% Critical Value	0.0723416
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	99.2010919

Gamma Distribution Test	
A-D Test Statistic	23.783707
A-D 5% Critical Value	1.00129582
K-S Test Statistic	0.31306131
K-S 5% Critical Value	0.08740448
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	99.5805574
Adjusted Gamma UCL	99.9673142

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.19614528
Lilliefors 5% Critical Value	0.0723416
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	221.634798
95% Chebyshev (MVUE) UCL	167.493517
97.5% Chebyshev (MVUE) UCL	218.721828
99% Chebyshev (MVUE) UCL	319.349924

95% Non-parametric UCLs	
CLT UCL	99.0005687
Adj-CLT UCL (Adjusted for skewness)	106.154099
Mod-t UCL (Adjusted for skewness)	100.316898
Jackknife UCL	99.2010919
Standard Bootstrap UCL	98.9047532
Bootstrap-t UCL	114.285822
Hall's Bootstrap UCL	104.601885
Percentile Bootstrap UCL	100.267757
BCA Bootstrap UCL	109.51262
95% Chebyshev (Mean, Sd) UCL	151.886908
97.5% Chebyshev (Mean, Sd) UCL	188.639749
99% Chebyshev (Mean, Sd) UCL	260.833588

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
Ethylbenzene

Raw Statistics	
Number of Valid Samples	167
Number of Unique Samples	55
Minimum	0.0025
Maximum	2973
Mean	21.9396317
Median	0.0075
Standard Deviation	231.147462
Variance	53429.149
Coefficient of Variation	10.5356127
Skewness	12.6968785

Gamma Statistics	
k hat	0.11865802
k star (bias corrected)	0.12051846
Theta hat	184.898003
Theta star	182.043746
nu hat	39.6317802
nu star	40.2531654
Approx.Chi Square Value (.05)	26.7133723
Adjusted Level of Significance	0.04856287
Adjusted Chi Square Value	26.6175523

Log-transformed Statistics	
Minimum of log data	-5.99146455
Maximum of log data	7.99732682
Mean of log data	-3.60510732
Standard Deviation of log data	2.83061583
Variance of log data	8.01238596

Normal Distribution Test	
Lilliefors Test Statisitic	0.47168162
Lilliefors 5% Critical Value	0.06856074
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	51.5257893

Gamma Distribution Test	
A-D Test Statistic	39.5701795
A-D 5% Critical Value	1.04526964
K-S Test Statistic	0.36199503
K-S 5% Critical Value	0.08362115
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	33.0598329
Adjusted Gamma UCL	33.1788443

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.33410445
Lilliefors 5% Critical Value	0.06856074
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	3.73943562
95% Chebyshev (MVUE) UCL	3.82861547
97.5% Chebyshev (MVUE) UCL	4.90806488
99% Chebyshev (MVUE) UCL	7.02843423

95% Non-parametric UCLs	
CLT UCL	51.3606805
Adj-CLT UCL (Adjusted for skewness)	70.1387109
Mod-t UCL (Adjusted for skewness)	54.4547825
Jackknife UCL	51.5257893
Standard Bootstrap UCL	50.4298214
Bootstrap-t UCL	361.45285
Hall's Bootstrap UCL	336.934093
Percentile Bootstrap UCL	56.7756737
BCA Bootstrap UCL	93.5737305
95% Chebyshev (Mean, Sd) UCL	99.9060697
97.5% Chebyshev (Mean, Sd) UCL	133.64221
99% Chebyshev (Mean, Sd) UCL	199.910324

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
Fluoranthene

Raw Statistics	
Number of Valid Samples	196
Number of Unique Samples	121
Minimum	0.00011
Maximum	5400
Mean	202.713436
Median	0.725
Standard Deviation	687.276276
Variance	472348.68
Coefficient of Variation	3.39038344
Skewness	4.53640045

Gamma Statistics	
k hat	0.14748927
k star (bias corrected)	0.14863314
Theta hat	1374.42836
Theta star	1363.85084
nu hat	57.815794
nu star	58.2641917
Approx.Chi Square Value (.05)	41.7126405
Adjusted Level of Significance	0.04877551
Adjusted Chi Square Value	41.6093373

Log-transformed Statistics	
Minimum of log data	-9.11503019
Maximum of log data	8.59415423
Mean of log data	0.08792872
Standard Deviation of log data	3.62364589
Variance of log data	13.1308096

Normal Distribution Test	
Lilliefors Test Statisitic	0.44104611
Lilliefors 5% Critical Value	0.06328571
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	283.846643

Gamma Distribution Test	
A-D Test Statistic	20.3460504
A-D 5% Critical Value	1.01159854
K-S Test Statistic	0.25736206
K-S 5% Critical Value	0.07415153
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	283.150008
Adjusted Gamma UCL	283.852983

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.12511585
Lilliefors 5% Critical Value	0.06328571
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	2925.45852
95% Chebyshev (MVUE) UCL	2157.56834
97.5% Chebyshev (MVUE) UCL	2825.41969
99% Chebyshev (MVUE) UCL	4137.28434

95% Non-parametric UCLs	
CLT UCL	283.461213
Adj-CLT UCL (Adjusted for skewness)	300.458009
Mod-t UCL (Adjusted for skewness)	286.4978
Jackknife UCL	283.846643
Standard Bootstrap UCL	282.420055
Bootstrap-t UCL	310.371006
Hall's Bootstrap UCL	297.854148
Percentile Bootstrap UCL	287.167674
BCA Bootstrap UCL	303.783166
95% Chebyshev (Mean, Sd) UCL	416.696852
97.5% Chebyshev (Mean, Sd) UCL	509.287648
99% Chebyshev (Mean, Sd) UCL	691.164336

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
Fluorene

Raw Statistics	
Number of Valid Samples	183
Number of Unique Samples	88
Minimum	0.0015
Maximum	4600
Mean	203.79649
Median	0.16
Standard Deviation	693.573554
Variance	481044.275
Coefficient of Variation	3.40326546
Skewness	4.08107242

Gamma Statistics	
k hat	0.13174473
k star (bias corrected)	0.13322797
Theta hat	1546.90432
Theta star	1529.68251
nu hat	48.2185708
nu star	48.7614358
Approx.Chi Square Value (.05)	33.7289017
Adjusted Level of Significance	0.04868852
Adjusted Chi Square Value	33.629975

Log-transformed Statistics	
Minimum of log data	-6.50229017
Maximum of log data	8.43381158
Mean of log data	-0.62559526
Standard Deviation of log data	3.77791545
Variance of log data	14.2726451

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.43833741
Lilliefors 5% Critical Value	0.065495
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	288.560293

Gamma Distribution Test	
A-D Test Statistic	23.65025
A-D 5% Critical Value	1.0333183
K-S Test Statistic	0.27868863
K-S 5% Critical Value	0.07854537
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	294.625943
Adjusted Gamma UCL	295.49262

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.15459429
Lilliefors 5% Critical Value	0.065495
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	2995.81482
95% Chebyshev (MVUE) UCL	1870.98078
97.5% Chebyshev (MVUE) UCL	2462.50515
99% Chebyshev (MVUE) UCL	3624.44025

95% Non-parametric UCLs	
CLT UCL	288.128847
Adj-CLT UCL (Adjusted for skewness)	304.655928
Mod-t UCL (Adjusted for skewness)	291.138184
Jackknife UCL	288.560293
Standard Bootstrap UCL	287.570828
Bootstrap-t UCL	309.931324
Hall's Bootstrap UCL	309.696833
Percentile Bootstrap UCL	291.676092
BCA Bootstrap UCL	300.963512
95% Chebyshev (Mean, Sd) UCL	427.279124
97.5% Chebyshev (Mean, Sd) UCL	523.980238
99% Chebyshev (Mean, Sd) UCL	713.930852

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
Indeno(1,2,3-cd)pyrene

Raw Statistics	
Number of Valid Samples	189
Number of Unique Samples	101
Minimum	0.00011
Maximum	1200
Mean	47.1356165
Median	0.22
Standard Deviation	161.217947
Variance	25991.2265
Coefficient of Variation	3.42029997
Skewness	4.4950519

Gamma Statistics	
k hat	0.16610052
k star (bias corrected)	0.16699134
Theta hat	283.777665
Theta star	282.263844
nu hat	62.7859948
nu star	63.1227251
Approx.Chi Square Value (.05)	45.842851
Adjusted Level of Significance	0.04873016
Adjusted Chi Square Value	45.7302103

Log-transformed Statistics	
Minimum of log data	-9.11503019
Maximum of log data	7.09007684
Mean of log data	-0.70513409
Standard Deviation of log data	3.28070713
Variance of log data	10.7630393

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.40850919
Lilliefors 5% Critical Value	0.06444702
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	66.5201355

Gamma Distribution Test	
A-D Test Statistic	19.040202
A-D 5% Critical Value	0.97498082
K-S Test Statistic	0.23423895
K-S 5% Critical Value	0.07524341
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	64.9027818
Adjusted Gamma UCL	65.0626476

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.13508641
Lilliefors 5% Critical Value	0.06444702
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	330.815706
95% Chebyshev (MVUE) UCL	291.586488
97.5% Chebyshev (MVUE) UCL	378.521439
99% Chebyshev (MVUE) UCL	549.288316

95% Non-parametric UCLs	
CLT UCL	66.4246178
Adj-CLT UCL (Adjusted for skewness)	70.5216248
Mod-t UCL (Adjusted for skewness)	67.1591858
Jackknife UCL	66.5201355
Standard Bootstrap UCL	67.2020699
Bootstrap-t UCL	73.9542256
Hall's Bootstrap UCL	71.227428
Percentile Bootstrap UCL	67.3733477
BCA Bootstrap UCL	73.7955805
95% Chebyshev (Mean, Sd) UCL	98.2519018
97.5% Chebyshev (Mean, Sd) UCL	120.369959
99% Chebyshev (Mean, Sd) UCL	163.8166

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
Lead

Raw Statistics	
Number of Valid Samples	182
Number of Unique Samples	138
Minimum	0.5
Maximum	4000
Mean	95.7825275
Median	18.7
Standard Deviation	329.224553
Variance	108388.807
Coefficient of Variation	3.43720887
Skewness	9.76569544

Gamma Statistics	
k hat	0.44740106
k star (bias corrected)	0.44368932
Theta hat	214.086503
Theta star	215.877471
nu hat	162.853984
nu star	161.502911
Approx.Chi Square Value (.05)	133.115256
Adjusted Level of Significance	0.04868132
Adjusted Chi Square Value	132.911268

Log-transformed Statistics	
Minimum of log data	-0.69314718
Maximum of log data	8.29404964
Mean of log data	3.11738763
Standard Deviation of log data	1.6332858
Variance of log data	2.66762251

RECOMMENDATION

Data are Non-parametric (0.05)

Use 97.5% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.38613197
Lilliefors 5% Critical Value	0.06567469
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	136.129619

Gamma Distribution Test	
A-D Test Statistic	8.16289231
A-D 5% Critical Value	0.83320447
K-S Test Statistic	0.14486685
K-S 5% Critical Value	0.07273946
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	116.208746
Adjusted Gamma UCL	116.3871

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.07147425
Lilliefors 5% Critical Value	0.06567469
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	119.878292
95% Chebyshev (MVUE) UCL	149.964892
97.5% Chebyshev (MVUE) UCL	178.376231
99% Chebyshev (MVUE) UCL	234.184805

95% Non-parametric UCLs	
CLT UCL	135.92312
Adj-CLT UCL (Adjusted for skewness)	154.798879
Mod-t UCL (Adjusted for skewness)	139.073856
Jackknife UCL	136.129619
Standard Bootstrap UCL	136.514235
Bootstrap-t UCL	194.176533
Hall's Bootstrap UCL	293.863702
Percentile Bootstrap UCL	139.724341
BCA Bootstrap UCL	162.847253
95% Chebyshev (Mean, Sd) UCL	202.155997
97.5% Chebyshev (Mean, Sd) UCL	248.183882
99% Chebyshev (Mean, Sd) UCL	338.59675

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
Manganese

Raw Statistics	
Number of Valid Samples	124
Number of Unique Samples	61
Minimum	12
Maximum	2000
Mean	275.266129
Median	275
Standard Deviation	222.885041
Variance	49677.7416
Coefficient of Variation	0.80970747
Skewness	4.29887388

Gamma Statistics	
k hat	2.08395827
k star (bias corrected)	2.03891626
Theta hat	132.08812
Theta star	135.006098
nu hat	516.82165
nu star	505.651234
Approx.Chi Square Value (.05)	454.494136
Adjusted Level of Significance	0.04806452
Adjusted Chi Square Value	453.929676

Log-transformed Statistics	
Minimum of log data	2.48490665
Maximum of log data	7.60090246
Mean of log data	5.35902381
Standard Deviation of log data	0.78268492
Variance of log data	0.61259569

RECOMMENDATION

Data are Non-parametric (0.05)

Use 95% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.19045467
Lilliefors 5% Critical Value	0.07956515
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	308.438816

Gamma Distribution Test	
A-D Test Statistic	3.31528991
A-D 5% Critical Value	0.76482602
K-S Test Statistic	0.14925804
K-S 5% Critical Value	0.08431677
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	306.249623
Adjusted Gamma UCL	306.630443

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.19037063
Lilliefors 5% Critical Value	0.07956515
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	332.570417
95% Chebyshev (MVUE) UCL	387.492832
97.5% Chebyshev (MVUE) UCL	430.648442
99% Chebyshev (MVUE) UCL	515.419281

95% Non-parametric UCLs	
CLT UCL	308.188973
Adj-CLT UCL (Adjusted for skewness)	316.445442
Mod-t UCL (Adjusted for skewness)	309.726658
Jackknife UCL	308.438816
Standard Bootstrap UCL	308.247205
Bootstrap-t UCL	322.894302
Hall's Bootstrap UCL	348.837293
Percentile Bootstrap UCL	311.943548
BCA Bootstrap UCL	315.040323
95% Chebyshev (Mean, Sd) UCL	362.512401
97.5% Chebyshev (Mean, Sd) UCL	400.263933
99% Chebyshev (Mean, Sd) UCL	474.419507

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
Naphthalene

Raw Statistics	
Number of Valid Samples	246
Number of Unique Samples	136
Minimum	0.00024
Maximum	37000
Mean	769.608058
Median	0.155
Standard Deviation	3805.35746
Variance	14480745.4
Coefficient of Variation	4.94453952
Skewness	6.87105268

Gamma Statistics	
k hat	0.11208139
k star (bias corrected)	0.11342457
Theta hat	6866.51059
Theta star	6785.19694
nu hat	55.1440444
nu star	55.8048894
Approx.Chi Square Value (.05)	39.6336747
Adjusted Level of Significance	0.04902439
Adjusted Chi Square Value	39.5537106

Log-transformed Statistics	
Minimum of log data	-8.33487164
Maximum of log data	10.5186732
Mean of log data	-0.49424959
Standard Deviation of log data	4.06307069
Variance of log data	16.5085434

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.44143172
Lilliefors 5% Critical Value	0.0564893
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	1170.19829

Gamma Distribution Test	
A-D Test Statistic	35.3000595
A-D 5% Critical Value	1.1114062
K-S Test Statistic	0.28051921
K-S 5% Critical Value	0.06908465
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	1083.62126
Adjusted Gamma UCL	1085.81197

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.1563625
Lilliefors 5% Critical Value	0.0564893
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	10022.2069
95% Chebyshev (MVUE) UCL	6559.80585
97.5% Chebyshev (MVUE) UCL	8635.70036
99% Chebyshev (MVUE) UCL	12713.3932

95% Non-parametric UCLs	
CLT UCL	1168.68365
Adj-CLT UCL (Adjusted for skewness)	1282.25376
Mod-t UCL (Adjusted for skewness)	1187.91293
Jackknife UCL	1170.19829
Standard Bootstrap UCL	1167.63687
Bootstrap-t UCL	1413.86677
Hall's Bootstrap UCL	1262.03708
Percentile Bootstrap UCL	1205.82854
BCA Bootstrap UCL	1332.33934
95% Chebyshev (Mean, Sd) UCL	1827.1673
97.5% Chebyshev (Mean, Sd) UCL	2284.77403
99% Chebyshev (Mean, Sd) UCL	3183.65384

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
n-Butyl benzene

Raw Statistics	
Number of Valid Samples	31
Number of Unique Samples	23
Minimum	0.0094
Maximum	648
Mean	25.9693839
Median	0.098
Standard Deviation	116.253618
Variance	13514.9038
Coefficient of Variation	4.47656436
Skewness	5.44926017

Gamma Statistics	
k hat	0.16848403
k star (bias corrected)	0.1736845
Theta hat	154.135578
Theta star	149.520443
nu hat	10.4460101
nu star	10.7684392
Approx.Chi Square Value (.05)	4.42665655
Adjusted Level of Significance	0.0413
Adjusted Chi Square Value	4.20191109

Log-transformed Statistics	
Minimum of log data	-4.66704559
Maximum of log data	6.4738907
Mean of log data	-1.22718182
Standard Deviation of log data	2.77208074
Variance of log data	7.68443165

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.2394792
Shapiro-Wilk 5% Critical Value	0.929
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	61.4077895

Gamma Distribution Test	
A-D Test Statistic	5.02130265
A-D 5% Critical Value	0.91422612
K-S Test Statistic	0.34410636
K-S 5% Critical Value	0.17585335
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	63.1740296
Adjusted Gamma UCL	66.5529864

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.83608292
Shapiro-Wilk 5% Critical Value	0.929
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	173.449517
95% Chebyshev (MVUE) UCL	34.4460055
97.5% Chebyshev (MVUE) UCL	45.6334862
99% Chebyshev (MVUE) UCL	67.6091258

95% Non-parametric UCLs	
CLT UCL	60.3135431
Adj-CLT UCL (Adjusted for skewness)	82.1490231
Mod-t UCL (Adjusted for skewness)	64.8136831
Jackknife UCL	61.4077895
Standard Bootstrap UCL	59.251052
Bootstrap-t UCL	317.183539
Hall's Bootstrap UCL	272.070197
Percentile Bootstrap UCL	66.8262194
BCA Bootstrap UCL	90.3260129
95% Chebyshev (Mean, Sd) UCL	116.982175
97.5% Chebyshev (Mean, Sd) UCL	156.363482
99% Chebyshev (Mean, Sd) UCL	233.720435

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
p-Isopropyltoluene

Raw Statistics	
Number of Valid Samples	32
Number of Unique Samples	24
Minimum	0.019
Maximum	213
Mean	11.2011875
Median	0.122
Standard Deviation	40.6403306
Variance	1651.63647
Coefficient of Variation	3.62821626
Skewness	4.49865229

Gamma Statistics	
k hat	0.19610868
k star (bias corrected)	0.19855683
Theta hat	57.1172448
Theta star	56.4130067
nu hat	12.5509556
nu star	12.7076368
Approx.Chi Square Value (.05)	5.69562093
Adjusted Level of Significance	0.0416
Adjusted Chi Square Value	5.44492563

Log-transformed Statistics	
Minimum of log data	-3.9633163
Maximum of log data	5.36129217
Mean of log data	-1.34809015
Standard Deviation of log data	2.58267332
Variance of log data	6.67020147

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.31593867
Shapiro-Wilk 5% Critical Value	0.93
Data not normal at 5% significance level	
95% UCL (Assuming Normal Distribution)	
Student's-t UCL	23.3822386

Gamma Distribution Test	
A-D Test Statistic	4.39039063
A-D 5% Critical Value	0.90042349
K-S Test Statistic	0.27714998
K-S 5% Critical Value	0.17232551
Data do not follow gamma distribution	
at 5% significance level	
95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	24.9912387
Adjusted Gamma UCL	26.1418855

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.85717332
Shapiro-Wilk 5% Critical Value	0.93
Data not lognormal at 5% significance level	
95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	64.5305452
95% Chebyshev (MVUE) UCL	19.2467011
97.5% Chebyshev (MVUE) UCL	25.3761451
99% Chebyshev (MVUE) UCL	37.416251

95% Non-parametric UCLs	
CLT UCL	23.0182491
Adj-CLT UCL (Adjusted for skewness)	29.1230302
Mod-t UCL (Adjusted for skewness)	24.3344611
Jackknife UCL	23.3822386
Standard Bootstrap UCL	23.0126909
Bootstrap-t UCL	93.0197419
Hall's Bootstrap UCL	115.523479
Percentile Bootstrap UCL	23.7263125
BCA Bootstrap UCL	31.8225313
95% Chebyshev (Mean, Sd) UCL	42.5166654
97.5% Chebyshev (Mean, Sd) UCL	56.0668976
99% Chebyshev (Mean, Sd) UCL	82.6837051

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
Pyrene

Raw Statistics	
Number of Valid Samples	209
Number of Unique Samples	134
Minimum	0.00011
Maximum	7600
Mean	285.546618
Median	0.64
Standard Deviation	978.34971
Variance	957168.156
Coefficient of Variation	3.42623463
Skewness	4.51515435

Gamma Statistics	
k hat	0.13924036
k star (bias corrected)	0.14043149
Theta hat	2050.74603
Theta star	2033.35179
nu hat	58.202471
nu star	58.7003621
Approx.Chi Square Value (.05)	42.0822063
Adjusted Level of Significance	0.04885167
Adjusted Chi Square Value	41.9849313

Log-transformed Statistics	
Minimum of log data	-9.11503019
Maximum of log data	8.93590353
Mean of log data	0.07523231
Standard Deviation of log data	3.84516047
Variance of log data	14.785259

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.44164601
Lilliefors 5% Critical Value	0.0612859
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	397.358195

Gamma Distribution Test	
A-D Test Statistic	20.9214418
A-D 5% Critical Value	1.03324891
K-S Test Statistic	0.24562708
K-S 5% Critical Value	0.07230406
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	398.308249
Adjusted Gamma UCL	399.231091

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.1243586
Lilliefors 5% Critical Value	0.0612859
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	7318.65821
95% Chebyshev (MVUE) UCL	4888.45281
97.5% Chebyshev (MVUE) UCL	6426.48353
99% Chebyshev (MVUE) UCL	9447.64705

95% Non-parametric UCLs	
CLT UCL	396.86022
Adj-CLT UCL (Adjusted for skewness)	419.444219
Mod-t UCL (Adjusted for skewness)	400.880843
Jackknife UCL	397.358195
Standard Bootstrap UCL	395.413419
Bootstrap-t UCL	434.538709
Hall's Bootstrap UCL	425.533214
Percentile Bootstrap UCL	405.81595
BCA Bootstrap UCL	419.62678
95% Chebyshev (Mean, Sd) UCL	580.530155
97.5% Chebyshev (Mean, Sd) UCL	708.169767
99% Chebyshev (Mean, Sd) UCL	958.893069

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
sec-Butyl benzene

Raw Statistics	
Number of Valid Samples	143
Number of Unique Samples	28
Minimum	0.0095
Maximum	2688
Mean	19.9383846
Median	0.0095
Standard Deviation	224.798911
Variance	50534.5503
Coefficient of Variation	11.2746802
Skewness	11.9402004

Gamma Statistics	
k hat	0.11972967
k star (bias corrected)	0.12187986
Theta hat	166.52835
Theta star	163.590472
nu hat	34.242686
nu star	34.8576413
Approx.Chi Square Value (.05)	22.3487353
Adjusted Level of Significance	0.04832168
Adjusted Chi Square Value	22.2468684

Log-transformed Statistics	
Minimum of log data	-4.65646348
Maximum of log data	7.8965527
Mean of log data	-3.63281602
Standard Deviation of log data	2.06939339
Variance of log data	4.28238901

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.49887328
Lilliefors 5% Critical Value	0.07409104
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	51.062427

Gamma Distribution Test	
A-D Test Statistic	44.7822614
A-D 5% Critical Value	1.0273239
K-S Test Statistic	0.43092596
K-S 5% Critical Value	0.09027184
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	31.098183
Adjusted Gamma UCL	31.2405794

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.36790184
Lilliefors 5% Critical Value	0.07409104
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	0.39889749
95% Chebyshev (MVUE) UCL	0.482131
97.5% Chebyshev (MVUE) UCL	0.59769227
99% Chebyshev (MVUE) UCL	0.82469001

95% Non-parametric UCLs	
CLT UCL	50.8593782
Adj-CLT UCL (Adjusted for skewness)	70.9156495
Mod-t UCL (Adjusted for skewness)	54.1908001
Jackknife UCL	51.062427
Standard Bootstrap UCL	50.1746029
Bootstrap-t UCL	1054.97199
Hall's Bootstrap UCL	1058.67451
Percentile Bootstrap UCL	57.627028
BCA Bootstrap UCL	77.0616259
95% Chebyshev (Mean, Sd) UCL	101.87971
97.5% Chebyshev (Mean, Sd) UCL	137.335787
99% Chebyshev (Mean, Sd) UCL	206.982384

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
Toluene

Raw Statistics	
Number of Valid Samples	183
Number of Unique Samples	92
Minimum	0.0025
Maximum	2007
Mean	15.426276
Median	0.033
Standard Deviation	151.067352
Variance	22821.3449
Coefficient of Variation	9.79285943
Skewness	12.7916753

Gamma Statistics	
k hat	0.13898202
k star (bias corrected)	0.14034662
Theta hat	110.994757
Theta star	109.915553
nu hat	50.8674207
nu star	51.3668619
Approx.Chi Square Value (.05)	35.9040221
Adjusted Level of Significance	0.04868852
Adjusted Chi Square Value	35.8017582

Log-transformed Statistics	
Minimum of log data	-5.99146455
Maximum of log data	7.60439635
Mean of log data	-2.85494543
Standard Deviation of log data	2.74347003
Variance of log data	7.52662779

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg)

Normal Distribution Test	
Lilliefors Test Statisitic	0.47938558
Lilliefors 5% Critical Value	0.065495
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	33.8886916

Gamma Distribution Test	
A-D Test Statistic	32.4016136
A-D 5% Critical Value	1.02065467
K-S Test Statistic	0.3054693
K-S 5% Critical Value	0.07820747
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	22.0699337
Adjusted Gamma UCL	22.1329741

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.13729935
Lilliefors 5% Critical Value	0.065495
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	5.64811812
95% Chebyshev (MVUE) UCL	6.15147004
97.5% Chebyshev (MVUE) UCL	7.83520275
99% Chebyshev (MVUE) UCL	11.1425696

95% Non-parametric UCLs	
CLT UCL	33.7947183
Adj-CLT UCL (Adjusted for skewness)	45.0777905
Mod-t UCL (Adjusted for skewness)	35.6486229
Jackknife UCL	33.8886916
Standard Bootstrap UCL	33.6278379
Bootstrap-t UCL	222.358169
Hall's Bootstrap UCL	127.90108
Percentile Bootstrap UCL	36.8700601
BCA Bootstrap UCL	51.4264618
95% Chebyshev (Mean, Sd) UCL	64.1030594
97.5% Chebyshev (Mean, Sd) UCL	85.1655425
99% Chebyshev (Mean, Sd) UCL	126.538712

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
Xylenes (Total)

Raw Statistics	
Number of Valid Samples	59
Number of Unique Samples	29
Minimum	0.0025
Maximum	4981
Mean	96.1730678
Median	0.075
Standard Deviation	648.825979
Variance	420975.151
Coefficient of Variation	6.74644153
Skewness	7.6134503

Gamma Statistics	
k hat	0.10909296
k star (bias corrected)	0.1148453
Theta hat	881.569874
Theta star	837.414078
nu hat	12.8729694
nu star	13.551745
Approx.Chi Square Value (.05)	6.2645243
Adjusted Level of Significance	0.0459322
Adjusted Chi Square Value	6.1401462

Log-transformed Statistics	
Minimum of log data	-5.99146455
Maximum of log data	8.51338595
Mean of log data	-2.7955884
Standard Deviation of log data	3.54000431
Variance of log data	12.5316305

RECOMMENDATION
Data are Non-parametric (0.05)

Use Hall's Bootstrap UCL

All units are milligrams per kilogram (mg/kg).
an erratic, unreasonably large UCL value,
use 99% Chebyshev (Mean, Sd) UCL

Normal Distribution Test	
Lilliefors Test Statisitic	0.46381472
Lilliefors 5% Critical Value	0.11534738
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	237.369019

Gamma Distribution Test	
A-D Test Statistic	11.1499052
A-D 5% Critical Value	0.97661822
K-S Test Statistic	0.35550441
K-S 5% Critical Value	0.13167329
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	208.046585
Adjusted Gamma UCL	212.260888

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.22346069
Lilliefors 5% Critical Value	0.11534738
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	440.558923
95% Chebyshev (MVUE) UCL	75.2571496
97.5% Chebyshev (MVUE) UCL	100.29471
99% Chebyshev (MVUE) UCL	149.476148

95% Non-parametric UCLs	
CLT UCL	235.113768
Adj-CLT UCL (Adjusted for skewness)	324.575674
Mod-t UCL (Adjusted for skewness)	251.323269
Jackknife UCL	237.369019
Standard Bootstrap UCL	234.964611
Bootstrap-t UCL	2425.57324
Hall's Bootstrap UCL	2028.62301
Percentile Bootstrap UCL	264.952119
BCA Bootstrap UCL	368.527398
95% Chebyshev (Mean, Sd) UCL	464.369033
97.5% Chebyshev (Mean, Sd) UCL	623.687721
99% Chebyshev (Mean, Sd) UCL	936.638434

Attachment B2
ProUCL Summary Sheet
Construction Worker Receptor Exposed to Surface and Subsurface Soil
Vanadium

Raw Statistics	
Number of Valid Samples	124
Number of Unique Samples	42
Minimum	8.3
Maximum	110
Mean	29.5177419
Median	30
Standard Deviation	13.5125178
Variance	182.588138
Coefficient of Variation	0.45777614
Skewness	1.86551284

Gamma Statistics	
k hat	5.2098755
k star (bias corrected)	5.08920647
Theta hat	5.66572885
Theta star	5.80006767
nu hat	1292.04913
nu star	1262.12321
Approx.Chi Square Value (.05)	1180.61803
Adjusted Level of Significance	0.04806452
Adjusted Chi Square Value	1179.70194

Log-transformed Statistics	
Minimum of log data	2.11625552
Maximum of log data	4.70048037
Mean of log data	3.28596086
Standard Deviation of log data	0.45806699
Variance of log data	0.20982537

RECOMMENDATION

Data are Non-parametric (0.05)

Use Student's-t UCL
or Modified-t UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.10439864
Lilliefors 5% Critical Value	0.07956515
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	31.5288528

Gamma Distribution Test	
A-D Test Statistic	1.96877523
A-D 5% Critical Value	0.75462473
K-S Test Statistic	0.13921587
K-S 5% Critical Value	0.08338984
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	31.5555296
Adjusted Gamma UCL	31.5800337

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.16724043
Lilliefors 5% Critical Value	0.07956515
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	31.9811911
95% Chebyshev (MVUE) UCL	35.2362398
97.5% Chebyshev (MVUE) UCL	37.6472647
99% Chebyshev (MVUE) UCL	42.3832563

95% Non-parametric UCLs	
CLT UCL	31.5137059
Adj-CLT UCL (Adjusted for skewness)	31.7309226
Mod-t UCL (Adjusted for skewness)	31.5627342
Jackknife UCL	31.5288528
Standard Bootstrap UCL	31.4836972
Bootstrap-t UCL	31.7769482
Hall's Bootstrap UCL	32.0587304
Percentile Bootstrap UCL	31.6024194
BCA Bootstrap UCL	31.8782258
95% Chebyshev (Mean, Sd) UCL	34.8070911
97.5% Chebyshev (Mean, Sd) UCL	37.0957967
99% Chebyshev (Mean, Sd) UCL	41.5915157

Attachment B2
ProUCL Summary Sheet
Industrial Worker Receptor Exposed to Surface Soil
Benzo(a)pyrene

Raw Statistics	
Number of Valid Samples	12
Number of Unique Samples	7
Minimum	17
Maximum	390
Mean	138
Median	140
Standard Deviation	96.4327368
Variance	9299.27273
Coefficient of Variation	0.69878795
Skewness	1.55662458

Gamma Statistics	
k hat	2.10749407
k star (bias corrected)	1.63617611
Theta hat	65.4806114
Theta star	84.3429992
nu hat	50.5798576
nu star	39.2682266
Approx.Chi Square Value (.05)	25.9111531
Adjusted Level of Significance	0.02896
Adjusted Chi Square Value	24.265208

Log-transformed Statistics	
Minimum of log data	2.83321334
Maximum of log data	5.96614674
Mean of log data	4.67162781
Standard Deviation of log data	0.82915983
Variance of log data	0.68750602

RECOMMENDATION

Data follow gamma distribution (0.05)

Use Approximate Gamma UCL

All units are micrograms per kilogram (µg/kg).

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.83306158
Shapiro-Wilk 5% Critical Value	0.859
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	187.993358

Gamma Distribution Test	
A-D Test Statistic	0.53718836
A-D 5% Critical Value	0.74125019
K-S Test Statistic	0.2107995
K-S 5% Critical Value	0.24842746
Data follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	209.138329
Adjusted Gamma UCL	223.324493

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.890894
Shapiro-Wilk 5% Critical Value	0.859
Data are lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	289.982612
95% Chebyshev (MVUE) UCL	305.322535
97.5% Chebyshev (MVUE) UCL	374.520655
99% Chebyshev (MVUE) UCL	510.446962

95% Non-parametric UCLs	
CLT UCL	183.788997
Adj-CLT UCL (Adjusted for skewness)	197.155184
Mod-t UCL (Adjusted for skewness)	190.078213
Jackknife UCL	187.993358
Standard Bootstrap UCL	180.942897
Bootstrap-t UCL	207.051522
Hall's Bootstrap UCL	416.747465
Percentile Bootstrap UCL	186
BCA Bootstrap UCL	201.416667
95% Chebyshev (Mean, Sd) UCL	259.341866
97.5% Chebyshev (Mean, Sd) UCL	311.846589
99% Chebyshev (Mean, Sd) UCL	414.981949

Attachment B2
ProUCL Summary Sheet
Industrial Worker Receptor Exposed to Surface Soil
Arsenic

Raw Statistics	
Number of Valid Samples	12
Number of Unique Samples	12
Minimum	0.55
Maximum	8.5
Mean	2.75416667
Median	2.35
Standard Deviation	2.00742467
Variance	4.02975379
Coefficient of Variation	0.72886826
Skewness	2.35130527

Gamma Statistics	
k hat	2.74511977
k star (bias corrected)	2.11439538
Theta hat	1.00329563
Theta star	1.30257883
nu hat	65.8828744
nu star	50.7454891
Approx.Chi Square Value (.05)	35.3842552
Adjusted Level of Significance	0.02896
Adjusted Chi Square Value	33.4351191

Log-transformed Statistics	
Minimum of log data	-0.597837
Maximum of log data	2.14006616
Mean of log data	0.82005354
Standard Deviation of log data	0.65199177
Variance of log data	0.42509327

RECOMMENDATION

Data follow gamma distribution (0.05)

Use Approximate Gamma UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.75161292
Shapiro-Wilk 5% Critical Value	0.859
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	3.79487031

Gamma Distribution Test	
A-D Test Statistic	0.43342932
A-D 5% Critical Value	0.73964975
K-S Test Statistic	0.17047124
K-S 5% Critical Value	0.24770056
Data follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	3.9498227
Adjusted Gamma UCL	4.18008185

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.94544352
Shapiro-Wilk 5% Critical Value	0.859
Data are lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	4.45192516
95% Chebyshev (MVUE) UCL	5.09281551
97.5% Chebyshev (MVUE) UCL	6.10600154
99% Chebyshev (MVUE) UCL	8.09620931

95% Non-parametric UCLs	
CLT UCL	3.70734879
Adj-CLT UCL (Adjusted for skewness)	4.12763723
Mod-t UCL (Adjusted for skewness)	3.86042681
Jackknife UCL	3.79487031
Standard Bootstrap UCL	3.65370668
Bootstrap-t UCL	4.76959993
Hall's Bootstrap UCL	8.03577283
Percentile Bootstrap UCL	3.725
BCA Bootstrap UCL	4.2375
95% Chebyshev (Mean, Sd) UCL	5.28012064
97.5% Chebyshev (Mean, Sd) UCL	6.37310295
99% Chebyshev (Mean, Sd) UCL	8.52005504

Attachment B2
ProUCL Summary Sheet
Maintenance Worker Receptor Exposed to Surface Soil
Arsenic

Raw Statistics	
Number of Valid Samples	42
Number of Unique Samples	34
Minimum	0.47
Maximum	13.9
Mean	4.10714286
Median	2.55
Standard Deviation	3.5722421
Variance	12.7609136
Coefficient of Variation	0.86976329
Skewness	1.49170809

Gamma Statistics	
k hat	1.6073175
k star (bias corrected)	1.50838212
Theta hat	2.55527788
Theta star	2.72287957
nu hat	135.01467
nu star	126.704098
Approx.Chi Square Value (.05)	101.700395
Adjusted Level of Significance	0.04428571
Adjusted Chi Square Value	100.9038

Log-transformed Statistics	
Minimum of log data	-0.75502258
Maximum of log data	2.63188884
Mean of log data	1.07047124
Standard Deviation of log data	0.85886854
Variance of log data	0.73765516

RECOMMENDATION

Data are Non-parametric (0.05)

Use 95% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.77053161
Shapiro-Wilk 5% Critical Value	0.942
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	5.03476004

Gamma Distribution Test	
A-D Test Statistic	0.95994652
A-D 5% Critical Value	0.76512454
K-S Test Statistic	0.15202999
K-S 5% Critical Value	0.13871458
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	5.11691062
Adjusted Gamma UCL	5.15730661

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.91503674
Shapiro-Wilk 5% Critical Value	0.942
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	5.67356932
95% Chebyshev (MVUE) UCL	6.88975632
97.5% Chebyshev (MVUE) UCL	8.06723033
99% Chebyshev (MVUE) UCL	10.38015

95% Non-parametric UCLs	
CLT UCL	5.01380085
Adj-CLT UCL (Adjusted for skewness)	5.14936844
Mod-t UCL (Adjusted for skewness)	5.05590584
Jackknife UCL	5.03476004
Standard Bootstrap UCL	4.98846744
Bootstrap-t UCL	5.24431422
Hall's Bootstrap UCL	5.18632753
Percentile Bootstrap UCL	5.00857143
BCA Bootstrap UCL	5.16690476
95% Chebyshev (Mean, Sd) UCL	6.50980685
97.5% Chebyshev (Mean, Sd) UCL	7.54944147
99% Chebyshev (Mean, Sd) UCL	9.59160239

Attachment B2
ProUCL Summary Sheet
Maintenance Worker Receptor Exposed to Surface Soil
Benzo(a)anthracene

Raw Statistics	
Number of Valid Samples	36
Number of Unique Samples	21
Minimum	0.017
Maximum	25
Mean	1.60561111
Median	0.17
Standard Deviation	4.46974569
Variance	19.9786265
Coefficient of Variation	2.78382833
Skewness	4.50934347

Gamma Statistics	
k hat	0.36430273
k star (bias corrected)	0.35246269
Theta hat	4.40735404
Theta star	4.55540732
nu hat	26.2297966
nu star	25.3773136
Approx.Chi Square Value (.05)	14.8989667
Adjusted Level of Significance	0.0428
Adjusted Chi Square Value	14.5334767

Log-transformed Statistics	
Minimum of log data	-4.07454194
Maximum of log data	3.21887583
Mean of log data	-1.36032663
Standard Deviation of log data	1.81972994
Variance of log data	3.31141706

RECOMMENDATION

Data are lognormal (0.05)

Use 95% Chebyshev (MVUE) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.39642772
Shapiro-Wilk 5% Critical Value	0.935
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	2.86427073

Gamma Distribution Test	
A-D Test Statistic	2.97492418
A-D 5% Critical Value	0.84245938
K-S Test Statistic	0.21859202
K-S 5% Critical Value	0.15801936
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	2.73482701
Adjusted Gamma UCL	2.80360285

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.93738876
Shapiro-Wilk 5% Critical Value	0.935
Data are lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	3.9163537
95% Chebyshev (MVUE) UCL	3.36499778
97.5% Chebyshev (MVUE) UCL	4.2998359
99% Chebyshev (MVUE) UCL	6.13614438

95% Non-parametric UCLs	
CLT UCL	2.83095735
Adj-CLT UCL (Adjusted for skewness)	3.42919541
Mod-t UCL (Adjusted for skewness)	2.95758378
Jackknife UCL	2.86427073
Standard Bootstrap UCL	2.82595511
Bootstrap-t UCL	5.25239763
Hall's Bootstrap UCL	6.52586728
Percentile Bootstrap UCL	2.92277778
BCA Bootstrap UCL	3.79655556
95% Chebyshev (Mean, Sd) UCL	4.85280607
97.5% Chebyshev (Mean, Sd) UCL	6.25786993
99% Chebyshev (Mean, Sd) UCL	9.01784579

Attachment B2
ProUCL Summary Sheet
Maintenance Worker Receptor Exposed to Surface Soil
Benzo(a)pyrene

Raw Statistics	
Number of Valid Samples	42
Number of Unique Samples	23
Minimum	0.01
Maximum	68
Mean	2.89278571
Median	0.17
Standard Deviation	10.8086594
Variance	116.827118
Coefficient of Variation	3.73641897
Skewness	5.66045733

Gamma Statistics	
k hat	0.26212026
k star (bias corrected)	0.2592704
Theta hat	11.0361011
Theta star	11.1574082
nu hat	22.018102
nu star	21.7787137
Approx.Chi Square Value (.05)	12.1709485
Adjusted Level of Significance	0.04428571
Adjusted Chi Square Value	11.9143887

Log-transformed Statistics	
Minimum of log data	-4.60517019
Maximum of log data	4.21950771
Mean of log data	-1.62690921
Standard Deviation of log data	2.24493982
Variance of log data	5.03975481

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.29526538
Shapiro-Wilk 5% Critical Value	0.942
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	5.69950987

Gamma Distribution Test	
A-D Test Statistic	3.78098835
A-D 5% Critical Value	0.87622318
K-S Test Statistic	0.26037847
K-S 5% Critical Value	0.14926512
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	5.17635517
Adjusted Gamma UCL	5.28782079

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.89514728
Shapiro-Wilk 5% Critical Value	0.942
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	9.97090606
95% Chebyshev (MVUE) UCL	6.52689134
97.5% Chebyshev (MVUE) UCL	8.47145759
99% Chebyshev (MVUE) UCL	12.2911815

95% Non-parametric UCLs	
CLT UCL	5.63609292
Adj-CLT UCL (Adjusted for skewness)	7.1926121
Mod-t UCL (Adjusted for skewness)	5.94229541
Jackknife UCL	5.69950987
Standard Bootstrap UCL	5.67904145
Bootstrap-t UCL	16.1732535
Hall's Bootstrap UCL	14.3339075
Percentile Bootstrap UCL	5.97152381
BCA Bootstrap UCL	8.16554762
95% Chebyshev (Mean, Sd) UCL	10.1626112
97.5% Chebyshev (Mean, Sd) UCL	13.3082705
99% Chebyshev (Mean, Sd) UCL	19.4873091

Attachment B2
ProUCL Summary Sheet
Maintenance Worker Receptor Exposed to Surface Soil
Benzo(b)fluoranthene

Raw Statistics	
Number of Valid Samples	36
Number of Unique Samples	24
Minimum	0.019
Maximum	53
Mean	2.80745833
Median	0.345
Standard Deviation	9.000622
Variance	81.0111964
Coefficient of Variation	3.20596815
Skewness	5.27159029

Gamma Statistics	
k hat	0.33434801
k star (bias corrected)	0.32500419
Theta hat	8.39681492
Theta star	8.63822192
nu hat	24.0730565
nu star	23.4003018
Approx.Chi Square Value (.05)	13.3918367
Adjusted Level of Significance	0.0428
Adjusted Chi Square Value	13.0471759

Log-transformed Statistics	
Minimum of log data	-3.9633163
Maximum of log data	3.97029191
Mean of log data	-0.99396588
Standard Deviation of log data	1.95806671
Variance of log data	3.83402523

RECOMMENDATION

Data are lognormal (0.05)

Use 95% Chebyshev (MVUE) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.33430201
Shapiro-Wilk 5% Critical Value	0.935
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	5.34199168

Gamma Distribution Test	
A-D Test Statistic	2.58816958
A-D 5% Critical Value	0.84909135
K-S Test Statistic	0.22114645
K-S 5% Critical Value	0.15863643
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	4.90562825
Adjusted Gamma UCL	5.03521782

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.95346855
Shapiro-Wilk 5% Critical Value	0.935
Data are lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	8.50842574
95% Chebyshev (MVUE) UCL	6.50797514
97.5% Chebyshev (MVUE) UCL	8.37422761
99% Chebyshev (MVUE) UCL	12.0401192

95% Non-parametric UCLs	
CLT UCL	5.27490929
Adj-CLT UCL (Adjusted for skewness)	6.68319926
Mod-t UCL (Adjusted for skewness)	5.56165645
Jackknife UCL	5.34199168
Standard Bootstrap UCL	5.24566763
Bootstrap-t UCL	13.2246763
Hall's Bootstrap UCL	13.3659004
Percentile Bootstrap UCL	5.62336111
BCA Bootstrap UCL	7.427875
95% Chebyshev (Mean, Sd) UCL	9.34625862
97.5% Chebyshev (Mean, Sd) UCL	12.1756027
99% Chebyshev (Mean, Sd) UCL	17.7333014

Attachment B2
ProUCL Summary Sheet
Maintenance Worker Receptor Exposed to Surface Soil
Indeno(1,2,3-cd)pyrene

Raw Statistics	
Number of Valid Samples	36
Number of Unique Samples	14
Minimum	0.0155
Maximum	33
Mean	1.92413889
Median	0.08
Standard Deviation	6.15659619
Variance	37.9036766
Coefficient of Variation	3.19966309
Skewness	4.30925707

Gamma Statistics	
k hat	0.28445516
k star (bias corrected)	0.27926909
Theta hat	6.76429585
Theta star	6.88991007
nu hat	20.4807719
nu star	20.1073742
Approx.Chi Square Value (.05)	10.9288352
Adjusted Level of Significance	0.0428
Adjusted Chi Square Value	10.6208681

Log-transformed Statistics	
Minimum of log data	-4.16691526
Maximum of log data	3.49650756
Mean of log data	-1.79080333
Standard Deviation of log data	1.89079208
Variance of log data	3.57509468

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.35807613
Shapiro-Wilk 5% Critical Value	0.935
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	3.65780777

Gamma Distribution Test	
A-D Test Statistic	5.63896403
A-D 5% Critical Value	0.86345583
K-S Test Statistic	0.37804878
K-S 5% Critical Value	0.15985325
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	3.54011934
Adjusted Gamma UCL	3.64277011

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.83571046
Shapiro-Wilk 5% Critical Value	0.935
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	3.13085694
95% Chebyshev (MVUE) UCL	2.54010813
97.5% Chebyshev (MVUE) UCL	3.25779903
99% Chebyshev (MVUE) UCL	4.66756382

95% Non-parametric UCLs	
CLT UCL	3.61192215
Adj-CLT UCL (Adjusted for skewness)	4.39936852
Mod-t UCL (Adjusted for skewness)	3.7806335
Jackknife UCL	3.65780777
Standard Bootstrap UCL	3.61529801
Bootstrap-t UCL	10.628598
Hall's Bootstrap UCL	9.78148043
Percentile Bootstrap UCL	3.70533333
BCA Bootstrap UCL	4.74215278
95% Chebyshev (Mean, Sd) UCL	6.39680233
97.5% Chebyshev (Mean, Sd) UCL	8.33212737
99% Chebyshev (Mean, Sd) UCL	12.1336987

Attachment B2
ProUCL Summary Sheet
Recreational Receptor Exposed to Surface Soil
Aluminum

Raw Statistics	
Number of Valid Samples	24
Number of Unique Samples	20
Minimum	870
Maximum	14000
Mean	8561.25
Median	8100
Standard Deviation	2924.53536
Variance	8552907.07
Coefficient of Variation	0.34160144
Skewness	-0.32525241

Gamma Statistics	
k hat	5.4484837
k star (bias corrected)	4.79520102
Theta hat	1571.30873
Theta star	1785.37875
nu hat	261.527218
nu star	230.169649
Approx.Chi Square Value (.05)	196.044619
Adjusted Level of Significance	0.0392
Adjusted Chi Square Value	193.832737

Log-transformed Statistics	
Minimum of log data	6.76849321
Maximum of log data	9.54681261
Mean of log data	8.96043498
Standard Deviation of log data	0.54300005
Variance of log data	0.29484905

RECOMMENDATION

Data are normal (0.05)

Use Student's-t UCL

All units are milligrams per kilogram (mg/kg)

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.95950189
Shapiro-Wilk 5% Critical Value	0.916
Data are normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	9584.37676

Gamma Distribution Test	
A-D Test Statistic	1.10943124
A-D 5% Critical Value	0.74600496
K-S Test Statistic	0.21196523
K-S 5% Critical Value	0.17825977
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	10051.4868
Adjusted Gamma UCL	10166.1873

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.69847261
Shapiro-Wilk 5% Critical Value	0.916
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	11331.0264
95% Chebyshev (MVUE) UCL	13497.5309
97.5% Chebyshev (MVUE) UCL	15459.3582
99% Chebyshev (MVUE) UCL	19312.988

95% Non-parametric UCLs	
CLT UCL	9543.17544
Adj-CLT UCL (Adjusted for skewness)	9500.82611
Mod-t UCL (Adjusted for skewness)	9577.77112
Jackknife UCL	9584.37676
Standard Bootstrap UCL	9534.67079
Bootstrap-t UCL	9554.96656
Hall's Bootstrap UCL	9567.16849
Percentile Bootstrap UCL	9516.66667
BCA Bootstrap UCL	9475
95% Chebyshev (Mean, Sd) UCL	11163.3744
97.5% Chebyshev (Mean, Sd) UCL	12289.3157
99% Chebyshev (Mean, Sd) UCL	14501.0094

Attachment B2
ProUCL Summary Sheet
Recreational Receptor Exposed to Surface Soil
Arsenic

Raw Statistics	
Number of Valid Samples	33
Number of Unique Samples	29
Minimum	0.47
Maximum	13.9
Mean	4.42878788
Median	2.6
Standard Deviation	3.81276537
Variance	14.5371797
Coefficient of Variation	0.86090494
Skewness	1.32877513

Gamma Statistics	
k hat	1.55446583
k star (bias corrected)	1.43335277
Theta hat	2.84907381
Theta star	3.0898101
nu hat	102.594745
nu star	94.6012831
Approx.Chi Square Value (.05)	73.1647321
Adjusted Level of Significance	0.0419
Adjusted Chi Square Value	72.1942735

Log-transformed Statistics	
Minimum of log data	-0.75502258
Maximum of log data	2.63188884
Mean of log data	1.1332061
Standard Deviation of log data	0.8885076
Variance of log data	0.78944576

RECOMMENDATION

Data follow gamma distribution (0.05)

Use Approximate Gamma UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.81868781
Shapiro-Wilk 5% Critical Value	0.931
Data not normal at 5% significance level	
95% UCL (Assuming Normal Distribution)	
Student's-t UCL	5.55305091

Gamma Distribution Test	
A-D Test Statistic	0.67975454
A-D 5% Critical Value	0.7645076
K-S Test Statistic	0.14909195
K-S 5% Critical Value	0.15594525
Data follow gamma distribution	
at 5% significance level	
95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	5.72637942
Adjusted Gamma UCL	5.80335525

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.95725308
Shapiro-Wilk 5% Critical Value	0.931
Data are lognormal at 5% significance level	
95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	6.60042431
95% Chebyshev (MVUE) UCL	7.98209047
97.5% Chebyshev (MVUE) UCL	9.47374486
99% Chebyshev (MVUE) UCL	12.4038111

95% Non-parametric UCLs	
CLT UCL	5.52050562
Adj-CLT UCL (Adjusted for skewness)	5.68454875
Mod-t UCL (Adjusted for skewness)	5.57863833
Jackknife UCL	5.55305091
Standard Bootstrap UCL	5.48442395
Bootstrap-t UCL	5.78012589
Hall's Bootstrap UCL	5.67673146
Percentile Bootstrap UCL	5.46151515
BCA Bootstrap UCL	5.65272727
95% Chebyshev (Mean, Sd) UCL	7.32186433
97.5% Chebyshev (Mean, Sd) UCL	8.57370082
99% Chebyshev (Mean, Sd) UCL	11.0326912

Attachment B2
ProUCL Summary Sheet
Recreational Receptor Exposed to Surface Soil
Benzo(a)anthracene

Raw Statistics	
Number of Valid Samples	27
Number of Unique Samples	17
Minimum	0.017
Maximum	25
Mean	2.09522222
Median	0.28
Standard Deviation	5.08876308
Variance	25.8955096
Coefficient of Variation	2.42874623
Skewness	3.89221491

Gamma Statistics	
k hat	0.37217936
k star (bias corrected)	0.35551746
Theta hat	5.62960344
Theta star	5.89344397
nu hat	20.0976856
nu star	19.1979428
Approx.Chi Square Value (.05)	10.2606174
Adjusted Level of Significance	0.0401
Adjusted Chi Square Value	9.8431013

Log-transformed Statistics	
Minimum of log data	-4.07454194
Maximum of log data	3.21887583
Mean of log data	-1.0491376
Standard Deviation of log data	1.94650442
Variance of log data	3.78887947

RECOMMENDATION

Data are lognormal (0.05)

Use 95% Chebyshev (MVUE) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.45280693
Shapiro-Wilk 5% Critical Value	0.923
Data not normal at 5% significance level	
95% UCL (Assuming Normal Distribution)	
Student's-t UCL	3.76558977

Gamma Distribution Test	
A-D Test Statistic	1.64613043
A-D 5% Critical Value	0.83658423
K-S Test Statistic	0.21367677
K-S 5% Critical Value	0.18096409
Data do not follow gamma distribution	
at 5% significance level	
95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	3.92022767
Adjusted Gamma UCL	4.08651248

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.94847344
Shapiro-Wilk 5% Critical Value	0.923
Data are lognormal at 5% significance level	
95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	10.1563494
95% Chebyshev (MVUE) UCL	6.15076437
97.5% Chebyshev (MVUE) UCL	7.96474466
99% Chebyshev (MVUE) UCL	11.5279577

95% Non-parametric UCLs	
CLT UCL	3.70608151
Adj-CLT UCL (Adjusted for skewness)	4.48991839
Mod-t UCL (Adjusted for skewness)	3.88785248
Jackknife UCL	3.76558977
Standard Bootstrap UCL	3.60415813
Bootstrap-t UCL	6.51645284
Hall's Bootstrap UCL	8.61330043
Percentile Bootstrap UCL	3.87018519
BCA Bootstrap UCL	4.72777778
95% Chebyshev (Mean, Sd) UCL	6.36403541
97.5% Chebyshev (Mean, Sd) UCL	8.21115429
99% Chebyshev (Mean, Sd) UCL	11.8394617

Attachment B2
ProUCL Summary Sheet
Recreational Receptor Exposed to Surface Soil
Benzo(a)pyrene

Raw Statistics	
Number of Valid Samples	33
Number of Unique Samples	20
Minimum	0.01
Maximum	68
Mean	3.64260606
Median	0.29
Standard Deviation	12.1233632
Variance	146.975936
Coefficient of Variation	3.32821145
Skewness	5.01640977

Gamma Statistics	
k hat	0.25754952
k star (bias corrected)	0.25433794
Theta hat	14.1433232
Theta star	14.3219136
nu hat	16.9982681
nu star	16.7863043
Approx.Chi Square Value (.05)	8.51843657
Adjusted Level of Significance	0.0419
Adjusted Chi Square Value	8.21502742

Log-transformed Statistics	
Minimum of log data	-4.60517019
Maximum of log data	4.21950771
Mean of log data	-1.45195777
Standard Deviation of log data	2.46596726
Variance of log data	6.0809945

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.3332132
Shapiro-Wilk 5% Critical Value	0.931
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	7.21739963

Gamma Distribution Test	
A-D Test Statistic	2.32789362
A-D 5% Critical Value	0.87380247
K-S Test Statistic	0.24268085
K-S 5% Critical Value	0.16769945
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	7.17806529
Adjusted Gamma UCL	7.44317587

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.93041926
Shapiro-Wilk 5% Critical Value	0.931
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	34.799499
95% Chebyshev (MVUE) UCL	13.1333877
97.5% Chebyshev (MVUE) UCL	17.2533468
99% Chebyshev (MVUE) UCL	25.3462088

95% Non-parametric UCLs	
CLT UCL	7.1139161
Adj-CLT UCL (Adjusted for skewness)	9.08308331
Mod-t UCL (Adjusted for skewness)	7.52454992
Jackknife UCL	7.21739963
Standard Bootstrap UCL	7.09872532
Bootstrap-t UCL	20.4572615
Hall's Bootstrap UCL	18.6100448
Percentile Bootstrap UCL	7.71793939
BCA Bootstrap UCL	9.87324242
95% Chebyshev (Mean, Sd) UCL	12.8416554
97.5% Chebyshev (Mean, Sd) UCL	16.8220914
99% Chebyshev (Mean, Sd) UCL	24.6408871

Attachment B2
ProUCL Summary Sheet
Recreational Receptor Exposed to Surface Soil
Benzo(b)fluoranthene

Raw Statistics	
Number of Valid Samples	27
Number of Unique Samples	19
Minimum	0.019
Maximum	53
Mean	3.67007407
Median	0.53
Standard Deviation	10.2932435
Variance	105.950861
Coefficient of Variation	2.80464188
Skewness	4.57330934

Gamma Statistics	
k hat	0.34290885
k star (bias corrected)	0.32949922
Theta hat	10.7027687
Theta star	11.1383391
nu hat	18.5170777
nu star	17.792958
Approx.Chi Square Value (.05)	9.24004302
Adjusted Level of Significance	0.0401
Adjusted Chi Square Value	8.84637645

Log-transformed Statistics	
Minimum of log data	-3.9633163
Maximum of log data	3.97029191
Mean of log data	-0.66731843
Standard Deviation of log data	2.0793848
Variance of log data	4.32384117

RECOMMENDATION

Data are lognormal (0.05)

Use 97.5% Chebyshev (MVUE) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.38040092
Shapiro-Wilk 5% Critical Value	0.923
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	7.04879293

Gamma Distribution Test	
A-D Test Statistic	1.43816028
A-D 5% Critical Value	0.84326962
K-S Test Statistic	0.18894958
K-S 5% Critical Value	0.18166804
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	7.06722616
Adjusted Gamma UCL	7.38171998

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.95970235
Shapiro-Wilk 5% Critical Value	0.923
Data are lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	23.4427705
95% Chebyshev (MVUE) UCL	11.9302013
97.5% Chebyshev (MVUE) UCL	15.5317084
99% Chebyshev (MVUE) UCL	22.6061716

95% Non-parametric UCLs	
CLT UCL	6.92842322
Adj-CLT UCL (Adjusted for skewness)	8.79136582
Mod-t UCL (Adjusted for skewness)	7.33937433
Jackknife UCL	7.04879293
Standard Bootstrap UCL	6.85287743
Bootstrap-t UCL	16.2657925
Hall's Bootstrap UCL	17.7783235
Percentile Bootstrap UCL	7.25322222
BCA Bootstrap UCL	9.39237037
95% Chebyshev (Mean, Sd) UCL	12.3047723
97.5% Chebyshev (Mean, Sd) UCL	16.0410131
99% Chebyshev (Mean, Sd) UCL	23.3801347

Attachment B2
ProUCL Summary Sheet
Recreational Receptor Exposed to Surface Soil
Benzo(k)fluoranthene

Raw Statistics	
Number of Valid Samples	24
Number of Unique Samples	7
Minimum	0.019
Maximum	15
Mean	0.93216667
Median	0.095
Standard Deviation	3.03708546
Variance	9.22388806
Coefficient of Variation	3.25809275
Skewness	4.69951045

Gamma Statistics	
k hat	0.3940652
k star (bias corrected)	0.37258482
Theta hat	2.36551382
Theta star	2.50189113
nu hat	18.9151294
nu star	17.8840716
Approx.Chi Square Value (.05)	9.30576788
Adjusted Level of Significance	0.0392
Adjusted Chi Square Value	8.8714774

Log-transformed Statistics	
Minimum of log data	-3.9633163
Maximum of log data	2.7080502
Mean of log data	-1.74414321
Standard Deviation of log data	1.54161345
Variance of log data	2.37657203

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.30706603
Shapiro-Wilk 5% Critical Value	0.916
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	1.99466824

Gamma Distribution Test	
A-D Test Statistic	3.25144767
A-D 5% Critical Value	0.83010707
K-S Test Statistic	0.34217917
K-S 5% Critical Value	0.19080485
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	1.79146263
Adjusted Gamma UCL	1.87916112

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.86165431
Shapiro-Wilk 5% Critical Value	0.916
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	1.65948549
95% Chebyshev (MVUE) UCL	1.40227954
97.5% Chebyshev (MVUE) UCL	1.78364338
99% Chebyshev (MVUE) UCL	2.5327588

95% Non-parametric UCLs	
CLT UCL	1.95188129
Adj-CLT UCL (Adjusted for skewness)	2.58732752
Mod-t UCL (Adjusted for skewness)	2.09378501
Jackknife UCL	1.99466824
Standard Bootstrap UCL	1.92693545
Bootstrap-t UCL	8.41978481
Hall's Bootstrap UCL	5.84847087
Percentile Bootstrap UCL	2.10854167
BCA Bootstrap UCL	2.79370833
95% Chebyshev (Mean, Sd) UCL	3.63443326
97.5% Chebyshev (Mean, Sd) UCL	4.80370617
99% Chebyshev (Mean, Sd) UCL	7.10051638

Attachment B2
ProUCL Summary Sheet
Recreational Receptor Exposed to Surface Soil
Cadmium

Raw Statistics	
Number of Valid Samples	33
Number of Unique Samples	26
Minimum	0.095
Maximum	12
Mean	0.77342424
Median	0.161
Standard Deviation	2.07682953
Variance	4.31322088
Coefficient of Variation	2.68523976
Skewness	5.24783075

Gamma Statistics	
k hat	0.62538118
k star (bias corrected)	0.58873037
Theta hat	1.23672452
Theta star	1.31371555
nu hat	41.2751581
nu star	38.8562044
Approx.Chi Square Value (.05)	25.576253
Adjusted Level of Significance	0.0419
Adjusted Chi Square Value	25.020649

Log-transformed Statistics	
Minimum of log data	-2.35387839
Maximum of log data	2.48490665
Mean of log data	-1.23894657
Standard Deviation of log data	1.13093186
Variance of log data	1.27900688

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.33484378
Shapiro-Wilk 5% Critical Value	0.931
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	1.3858151

Gamma Distribution Test	
A-D Test Statistic	3.75860873
A-D 5% Critical Value	0.7993285
K-S Test Statistic	0.24025745
K-S 5% Critical Value	0.16059687
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	1.17500912
Adjusted Gamma UCL	1.20110116

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.8226638
Shapiro-Wilk 5% Critical Value	0.931
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	0.91758775
95% Chebyshev (MVUE) UCL	1.07565621
97.5% Chebyshev (MVUE) UCL	1.31055831
99% Chebyshev (MVUE) UCL	1.77197799

95% Non-parametric UCLs	
CLT UCL	1.36808754
Adj-CLT UCL (Adjusted for skewness)	1.72098387
Mod-t UCL (Adjusted for skewness)	1.4408598
Jackknife UCL	1.3858151
Standard Bootstrap UCL	1.3542689
Bootstrap-t UCL	3.24633921
Hall's Bootstrap UCL	3.38175151
Percentile Bootstrap UCL	1.46087879
BCA Bootstrap UCL	1.92706061
95% Chebyshev (Mean, Sd) UCL	2.3492953
97.5% Chebyshev (Mean, Sd) UCL	3.03117597
99% Chebyshev (Mean, Sd) UCL	4.3705985

Attachment B2
ProUCL Summary Sheet
Recreational Receptor Exposed to Surface Soil
Indeno(1,2,3-cd)pyrene

Raw Statistics	
Number of Valid Samples	27
Number of Unique Samples	12
Minimum	0.0155
Maximum	33
Mean	2.54042593
Median	0.155
Standard Deviation	7.03176398
Variance	49.4457047
Coefficient of Variation	2.76794687
Skewness	3.69333244

Gamma Statistics	
k hat	0.29128472
k star (bias corrected)	0.28361111
Theta hat	8.7214528
Theta star	8.95742745
nu hat	15.7293748
nu star	15.3149998
Approx.Chi Square Value (.05)	7.47996088
Adjusted Level of Significance	0.0401
Adjusted Chi Square Value	7.13066909

Log-transformed Statistics	
Minimum of log data	-4.16691526
Maximum of log data	3.49650756
Mean of log data	-1.44640333
Standard Deviation of log data	2.03367937
Variance of log data	4.1358518

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.41598363
Shapiro-Wilk 5% Critical Value	0.923
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	4.84857629

Gamma Distribution Test	
A-D Test Statistic	3.52353269
A-D 5% Critical Value	0.85678445
K-S Test Statistic	0.3405195
K-S 5% Critical Value	0.18301636
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	5.20144735
Adjusted Gamma UCL	5.4562373

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.85868734
Shapiro-Wilk 5% Critical Value	0.923
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	9.16882225
95% Chebyshev (MVUE) UCL	4.96547217
97.5% Chebyshev (MVUE) UCL	6.45304382
99% Chebyshev (MVUE) UCL	9.37509027

95% Non-parametric UCLs	
CLT UCL	4.76634648
Adj-CLT UCL (Adjusted for skewness)	5.79412453
Mod-t UCL (Adjusted for skewness)	5.00888889
Jackknife UCL	4.84857629
Standard Bootstrap UCL	4.72309956
Bootstrap-t UCL	10.3684621
Hall's Bootstrap UCL	11.9471028
Percentile Bootstrap UCL	4.90542593
BCA Bootstrap UCL	6.12911111
95% Chebyshev (Mean, Sd) UCL	8.43916524
97.5% Chebyshev (Mean, Sd) UCL	10.9915545
99% Chebyshev (Mean, Sd) UCL	16.0052288

Attachment B2
ProUCL Summary Sheet
Recreational Receptor Exposed to Surface Soil
Iron

Raw Statistics	
Number of Valid Samples	33
Number of Unique Samples	24
Minimum	4100
Maximum	94000
Mean	21374.8485
Median	20000
Standard Deviation	14696.0348
Variance	215973438
Coefficient of Variation	0.68753866
Skewness	3.97276766

Gamma Statistics	
k hat	3.8973024
k star (bias corrected)	3.5632042
Theta hat	5484.52398
Theta star	5998.77169
nu hat	257.221958
nu star	235.171477
Approx.Chi Square Value (.05)	200.664589
Adjusted Level of Significance	0.0419
Adjusted Chi Square Value	199.02666

Log-transformed Statistics	
Minimum of log data	8.31874225
Maximum of log data	11.4510501
Mean of log data	9.83622496
Standard Deviation of log data	0.49993536
Variance of log data	0.24993537

RECOMMENDATION

Data are Non-parametric (0.05)

Use Student's-t UCL
or Modified-t UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.60186644
Shapiro-Wilk 5% Critical Value	0.931
Data not normal at 5% significance level	
95% UCL (Assuming Normal Distribution)	
Student's-t UCL	25708.2409

Gamma Distribution Test	
A-D Test Statistic	1.46531804
A-D 5% Critical Value	0.75156444
K-S Test Statistic	0.17350965
K-S 5% Critical Value	0.15394671
Data do not follow gamma distribution	
at 5% significance level	
95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	25050.5319
Adjusted Gamma UCL	25256.6902

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.91041081
Shapiro-Wilk 5% Critical Value	0.931
Data not lognormal at 5% significance level	
95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	25112.4612
95% Chebyshev (MVUE) UCL	29465.9993
97.5% Chebyshev (MVUE) UCL	33086.4363
99% Chebyshev (MVUE) UCL	40198.0838

95% Non-parametric UCLs	
CLT UCL	25582.7974
Adj-CLT UCL (Adjusted for skewness)	27473.224
Mod-t UCL (Adjusted for skewness)	26003.1093
Jackknife UCL	25708.2409
Standard Bootstrap UCL	25467.5156
Bootstrap-t UCL	30135.4534
Hall's Bootstrap UCL	45102.3248
Percentile Bootstrap UCL	25757.5758
BCA Bootstrap UCL	27926.3636
95% Chebyshev (Mean, Sd) UCL	32526.0073
97.5% Chebyshev (Mean, Sd) UCL	37351.1227
99% Chebyshev (Mean, Sd) UCL	46829.1274

Attachment B2
ProUCL Summary Sheet
Recreational Receptor Exposed to Surface Soil
Lead

Raw Statistics	
Number of Valid Samples	33
Number of Unique Samples	33
Minimum	2.25
Maximum	950
Mean	107.941515
Median	53
Standard Deviation	177.763263
Variance	31599.7776
Coefficient of Variation	1.64684795
Skewness	3.7456398

Gamma Statistics	
k hat	0.72446449
k star (bias corrected)	0.6788061
Theta hat	148.994902
Theta star	159.016714
nu hat	47.814656
nu star	44.8012024
Approx.Chi Square Value (.05)	30.4455865
Adjusted Level of Significance	0.0419
Adjusted Chi Square Value	29.8354527

Log-transformed Statistics	
Minimum of log data	0.81093022
Maximum of log data	6.85646199
Mean of log data	3.85135745
Standard Deviation of log data	1.35007257
Variance of log data	1.82269594

RECOMMENDATION

Data follow gamma distribution (0.05)

Use Approximate Gamma UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.56317843
Shapiro-Wilk 5% Critical Value	0.931
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	160.358238

Gamma Distribution Test	
A-D Test Statistic	0.58335925
A-D 5% Critical Value	0.79010187
K-S Test Statistic	0.10201974
K-S 5% Critical Value	0.15952281
Data follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	158.83779
Adjusted Gamma UCL	162.086016

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.99214918
Shapiro-Wilk 5% Critical Value	0.931
Data are lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	231.258435
95% Chebyshev (MVUE) UCL	253.129107
97.5% Chebyshev (MVUE) UCL	314.437343
99% Chebyshev (MVUE) UCL	434.865503

95% Non-parametric UCLs	
CLT UCL	158.840873
Adj-CLT UCL (Adjusted for skewness)	180.400165
Mod-t UCL (Adjusted for skewness)	163.721052
Jackknife UCL	160.358238
Standard Bootstrap UCL	158.05742
Bootstrap-t UCL	220.911601
Hall's Bootstrap UCL	387.399202
Percentile Bootstrap UCL	167.471212
BCA Bootstrap UCL	189.957576
95% Chebyshev (Mean, Sd) UCL	242.825952
97.5% Chebyshev (Mean, Sd) UCL	301.190556
99% Chebyshev (Mean, Sd) UCL	415.836518

Attachment B2
ProUCL Summary Sheet
Recreational Receptor Exposed to Surface Soil
Manganese

Raw Statistics	
Number of Valid Samples	24
Number of Unique Samples	14
Minimum	100
Maximum	670
Mean	311.25
Median	280
Standard Deviation	110.34777
Variance	12176.6304
Coefficient of Variation	0.35453099
Skewness	1.5227098

Gamma Statistics	
k hat	8.94740227
k star (bias corrected)	7.85675477
Theta hat	34.7866331
Theta star	39.6155931
nu hat	429.475309
nu star	377.124229
Approx.Chi Square Value (.05)	333.106879
Adjusted Level of Significance	0.0392
Adjusted Chi Square Value	330.203011

Log-transformed Statistics	
Minimum of log data	4.60517019
Maximum of log data	6.50727771
Mean of log data	5.68367466
Standard Deviation of log data	0.35166004
Variance of log data	0.12366479

RECOMMENDATION

Data are Non-parametric (0.05)

Use Student's-t UCL
or Modified-t UCL

All units are milligrams per kilogram (mg/kg)

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.8531566
Shapiro-Wilk 5% Critical Value	0.916
Data not normal at 5% significance level	
95% UCL (Assuming Normal Distribution)	
Student's-t UCL	349.85434

Gamma Distribution Test	
A-D Test Statistic	1.02039843
A-D 5% Critical Value	0.74471736
K-S Test Statistic	0.21448992
K-S 5% Critical Value	0.17786789
Data do not follow gamma distribution	
at 5% significance level	
95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	352.379142
Adjusted Gamma UCL	355.478032

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.89309073
Shapiro-Wilk 5% Critical Value	0.916
Data not lognormal at 5% significance level	
95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	358.368612
95% Chebyshev (MVUE) UCL	411.547247
97.5% Chebyshev (MVUE) UCL	454.650053
99% Chebyshev (MVUE) UCL	539.317171

95% Non-parametric UCLs	
CLT UCL	348.299743
Adj-CLT UCL (Adjusted for skewness)	355.780575
Mod-t UCL (Adjusted for skewness)	351.021199
Jackknife UCL	349.85434
Standard Bootstrap UCL	347.472148
Bootstrap-t UCL	361.553263
Hall's Bootstrap UCL	387.109507
Percentile Bootstrap UCL	347.083333
BCA Bootstrap UCL	352.5
95% Chebyshev (Mean, Sd) UCL	409.432648
97.5% Chebyshev (Mean, Sd) UCL	451.916359
99% Chebyshev (Mean, Sd) UCL	535.367381

Attachment B2
ProUCL Summary Sheet
Recreational Receptor Exposed to Surface Soil
Thallium

Raw Statistics	
Number of Valid Samples	24
Number of Unique Samples	10
Minimum	0.7
Maximum	2.4
Mean	0.92916667
Median	0.85
Standard Deviation	0.34132627
Variance	0.11650362
Coefficient of Variation	0.36734666
Skewness	3.78752599

Gamma Statistics	
k hat	12.8683303
k star (bias corrected)	11.2875668
Theta hat	0.07220569
Theta star	0.08231771
nu hat	617.679852
nu star	541.803204
Approx.Chi Square Value (.05)	488.807264
Adjusted Level of Significance	0.0392
Adjusted Chi Square Value	485.275047

Log-transformed Statistics	
Minimum of log data	-0.35667494
Maximum of log data	0.87546874
Mean of log data	-0.11282517
Standard Deviation of log data	0.2555466
Variance of log data	0.06530406

RECOMMENDATION

Data are Non-parametric (0.05)

Use Student's-t UCL
or Modified-t UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.53789419
Shapiro-Wilk 5% Critical Value	0.916
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	1.0485771

Gamma Distribution Test	
A-D Test Statistic	2.53655674
A-D 5% Critical Value	0.74374199
K-S Test Statistic	0.29983324
K-S 5% Critical Value	0.17768403
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	1.02990588
Adjusted Gamma UCL	1.03740236

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.70877219
Shapiro-Wilk 5% Critical Value	0.916
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	1.01550811
95% Chebyshev (MVUE) UCL	1.13354688
97.5% Chebyshev (MVUE) UCL	1.22522803
99% Chebyshev (MVUE) UCL	1.40531789

95% Non-parametric UCLs	
CLT UCL	1.04376844
Adj-CLT UCL (Adjusted for skewness)	1.10132496
Mod-t UCL (Adjusted for skewness)	1.05755476
Jackknife UCL	1.0485771
Standard Bootstrap UCL	1.03729337
Bootstrap-t UCL	1.22086876
Hall's Bootstrap UCL	1.48359301
Percentile Bootstrap UCL	1.05833333
BCA Bootstrap UCL	1.11875
95% Chebyshev (Mean, Sd) UCL	1.23286394
97.5% Chebyshev (Mean, Sd) UCL	1.364274
99% Chebyshev (Mean, Sd) UCL	1.6224036

Attachment B2
ProUCL Summary Sheet
Recreational Receptor Exposed to Surface Soil
Vanadium

Raw Statistics	
Number of Valid Samples	24
Number of Unique Samples	15
Minimum	8.9
Maximum	63
Mean	33.3708333
Median	32
Standard Deviation	9.63803512
Variance	92.891721
Coefficient of Variation	0.28881614
Skewness	0.61959778

Gamma Statistics	
k hat	10.6166195
k star (bias corrected)	9.31731988
Theta hat	3.14326356
Theta star	3.58159146
nu hat	509.597738
nu star	447.231354
Approx.Chi Square Value (.05)	399.190851
Adjusted Level of Significance	0.0392
Adjusted Chi Square Value	396.005453

Log-transformed Statistics	
Minimum of log data	2.18605128
Maximum of log data	4.14313473
Mean of log data	3.4598476
Standard Deviation of log data	0.34461778
Variance of log data	0.11876141

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.88712908
Shapiro-Wilk 5% Critical Value	0.916
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	36.7426276

Gamma Distribution Test	
A-D Test Statistic	1.28779728
A-D 5% Critical Value	0.74420359
K-S Test Statistic	0.2394646
K-S 5% Critical Value	0.17773582
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	37.3868363
Adjusted Gamma UCL	37.6875694

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.78997524
Shapiro-Wilk 5% Critical Value	0.916
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	38.5597528
95% Chebyshev (MVUE) UCL	44.1995423
97.5% Chebyshev (MVUE) UCL	48.7554205
99% Chebyshev (MVUE) UCL	57.7045608

95% Non-parametric UCLs	
CLT UCL	36.6068455
Adj-CLT UCL (Adjusted for skewness)	36.8727144
Mod-t UCL (Adjusted for skewness)	36.7840978
Jackknife UCL	36.7426276
Standard Bootstrap UCL	36.5669628
Bootstrap-t UCL	37.0658361
Hall's Bootstrap UCL	38.2318154
Percentile Bootstrap UCL	36.5833333
BCA Bootstrap UCL	36.5375
95% Chebyshev (Mean, Sd) UCL	41.9463379
97.5% Chebyshev (Mean, Sd) UCL	45.6569656
99% Chebyshev (Mean, Sd) UCL	52.9457752

RECOMMENDATION

Data are Non-parametric (0.05)

Use Student's-t UCL
or Modified-t UCL

All units are milligrams per kilogram (mg/kg).

Attachment B2
ProUCL Summary Sheet
Recreational Receptor Exposed to Surface Soil
Zinc

Raw Statistics	
Number of Valid Samples	33
Number of Unique Samples	30
Minimum	4.3
Maximum	4100
Mean	261.230303
Median	100
Standard Deviation	704.490701
Variance	496307.148
Coefficient of Variation	2.69681845
Skewness	5.37577046

Gamma Statistics	
k hat	0.65091329
k star (bias corrected)	0.61194138
Theta hat	401.328881
Theta star	426.887792
nu hat	42.9602773
nu star	40.3881309
Approx.Chi Square Value (.05)	26.8234766
Adjusted Level of Significance	0.0419
Adjusted Chi Square Value	26.2534483

Log-transformed Statistics	
Minimum of log data	1.45861502
Maximum of log data	8.31874225
Mean of log data	4.62734321
Standard Deviation of log data	1.19168133
Variance of log data	1.4201044

RECOMMENDATION

Data are lognormal (0.05)

Use H-UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.31860175
Shapiro-Wilk 5% Critical Value	0.931
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	468.962166

Gamma Distribution Test	
A-D Test Statistic	2.56205529
A-D 5% Critical Value	0.79695095
K-S Test Statistic	0.22932698
K-S 5% Critical Value	0.1603201
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	393.334683
Adjusted Gamma UCL	401.874967

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.95026781
Shapiro-Wilk 5% Critical Value	0.931
Data are lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	363.074089
95% Chebyshev (MVUE) UCL	419.259066
97.5% Chebyshev (MVUE) UCL	513.761141
99% Chebyshev (MVUE) UCL	699.392169

95% Non-parametric UCLs	
CLT UCL	462.948721
Adj-CLT UCL (Adjusted for skewness)	585.574691
Mod-t UCL (Adjusted for skewness)	488.08934
Jackknife UCL	468.962166
Standard Bootstrap UCL	461.313181
Bootstrap-t UCL	1310.55827
Hall's Bootstrap UCL	1177.6346
Percentile Bootstrap UCL	495.169697
BCA Bootstrap UCL	631.378788
95% Chebyshev (Mean, Sd) UCL	795.788626
97.5% Chebyshev (Mean, Sd) UCL	1027.09244
99% Chebyshev (Mean, Sd) UCL	1481.44399

Attachment B2
ProUCL Summary Sheet
Recreational Receptors Exposed to Sediment
2-Methylnaphthalene

Raw Statistics	
Number of Valid Samples	13
Number of Unique Samples	13
Minimum	0.059
Maximum	2380
Mean	248.293
Median	3.15
Standard Deviation	665.926455
Variance	443458.043
Coefficient of Variation	2.68201864
Skewness	3.20779524

Gamma Statistics	
k hat	0.21781556
k star (bias corrected)	0.21883249
Theta hat	1139.92313
Theta star	1134.62587
nu hat	5.66320468
nu star	5.68964463
Approx.Chi Square Value (.05)	1.48290584
Adjusted Level of Significance	0.03009
Adjusted Chi Square Value	1.20023754

Log-transformed Statistics	
Minimum of log data	-2.83021784
Maximum of log data	7.77485577
Mean of log data	2.18098357
Standard Deviation of log data	2.9118647
Variance of log data	8.47895601

RECOMMENDATION

Assuming gamma distribution (0.05)

Use Adjusted Gamma UCL

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.43942992
Shapiro-Wilk 5% Critical Value	0.866
Data not normal at 5% significance level	
95% UCL (Assuming Normal Distribution)	
Student's-t UCL	577.472171

Gamma Distribution Test	
A-D Test Statistic	1.18094457
A-D 5% Critical Value	0.86132595
K-S Test Statistic	0.24497179
K-S 5% Critical Value	0.26040813
Data follow approximate gamma distibution	
at 5% significance level	
95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	952.655858
Adjusted Gamma UCL	1177.01612

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.95953436
Shapiro-Wilk 5% Critical Value	0.866
Data are lognormal at 5% significance level	
95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	185137.133
95% Chebyshev (MVUE) UCL	1011.26233
97.5% Chebyshev (MVUE) UCL	1353.04027
99% Chebyshev (MVUE) UCL	2024.39685

95% Non-parametric UCLs	
CLT UCL	552.088859
Adj-CLT UCL (Adjusted for skewness)	727.666807
Mod-t UCL (Adjusted for skewness)	604.858782
Jackknife UCL	577.472171
Standard Bootstrap UCL	546.044902
Bootstrap-t UCL	6163.21076
Hall's Bootstrap UCL	5001.72746
Percentile Bootstrap UCL	566.485308
BCA Bootstrap UCL	799.069923
95% Chebyshev (Mean, Sd) UCL	1053.35883
97.5% Chebyshev (Mean, Sd) UCL	1401.71145
99% Chebyshev (Mean, Sd) UCL	2085.98274

Attachment B2
ProUCL Summary Sheet
Recreational Receptors Exposed to Sediment
Acenaphthene

Raw Statistics	
Number of Valid Samples	14
Number of Unique Samples	14
Minimum	0.12
Maximum	833
Mean	89.3942857
Median	6.575
Standard Deviation	222.81005
Variance	49644.3183
Coefficient of Variation	2.49244175
Skewness	3.30910397

Gamma Statistics	
k hat	0.28118495
k star (bias corrected)	0.26855008
Theta hat	317.919885
Theta star	332.877528
nu hat	7.8731785
nu star	7.51940216
Approx.Chi Square Value (.05)	2.45932738
Adjusted Level of Significance	0.03122
Adjusted Chi Square Value	2.09276878

Log-transformed Statistics	
Minimum of log data	-2.12026354
Maximum of log data	6.72503364
Mean of log data	2.0146745
Standard Deviation of log data	2.51798054
Variance of log data	6.34022602

RECOMMENDATION

Data follow gamma distribution (0.05)

Use Adjusted Gamma UCL

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.46202091
Shapiro-Wilk 5% Critical Value	0.874
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	194.850685

Gamma Distribution Test	
A-D Test Statistic	0.8117492
A-D 5% Critical Value	0.83983072
K-S Test Statistic	0.23252803
K-S 5% Critical Value	0.24877086
Data follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	273.323344
Adjusted Gamma UCL	321.197255

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.98029614
Shapiro-Wilk 5% Critical Value	0.874
Data are lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	10000.9866
95% Chebyshev (MVUE) UCL	397.981658
97.5% Chebyshev (MVUE) UCL	529.200489
99% Chebyshev (MVUE) UCL	786.954475

95% Non-parametric UCLs	
CLT UCL	187.342837
Adj-CLT UCL (Adjusted for skewness)	243.615512
Mod-t UCL (Adjusted for skewness)	203.628085
Jackknife UCL	194.850685
Standard Bootstrap UCL	184.799576
Bootstrap-t UCL	781.247882
Hall's Bootstrap UCL	644.444653
Percentile Bootstrap UCL	202.792857
BCA Bootstrap UCL	261.287143
95% Chebyshev (Mean, Sd) UCL	348.960139
97.5% Chebyshev (Mean, Sd) UCL	461.274491
99% Chebyshev (Mean, Sd) UCL	681.894286

Attachment B2
ProUCL Summary Sheet
Recreational Receptors Exposed to Sediment
Benzo(a)anthrancene

Raw Statistics	
Number of Valid Samples	14
Number of Unique Samples	14
Minimum	0.55
Maximum	150
Mean	22.9292857
Median	8.6
Standard Deviation	39.1112881
Variance	1529.69285
Coefficient of Variation	1.70573513
Skewness	2.9992148

Gamma Statistics	
k hat	0.56155702
k star (bias corrected)	0.48884242
Theta hat	40.8316249
Theta star	46.9052697
nu hat	15.7235967
nu star	13.6875878
Approx.Chi Square Value (.05)	6.35690914
Adjusted Level of Significance	0.03122
Adjusted Chi Square Value	5.70624296

Log-transformed Statistics	
Minimum of log data	-0.597837
Maximum of log data	5.01063529
Mean of log data	2.02120998
Standard Deviation of log data	1.64712809
Variance of log data	2.71303094

RECOMMENDATION

Data follow gamma distribution (0.05)

Use Approximate Gamma UCL

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.59330513
Shapiro-Wilk 5% Critical Value	0.874
Data not normal at 5% significance level	
95% UCL (Assuming Normal Distribution)	
Student's-t UCL	41.440729

Gamma Distribution Test	
A-D Test Statistic	0.41024889
A-D 5% Critical Value	0.78769293
K-S Test Statistic	0.16918069
K-S 5% Critical Value	0.24063524
Data follow gamma distribution	
at 5% significance level	
95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	49.3709451
Adjusted Gamma UCL	55.0005695

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.96981991
Shapiro-Wilk 5% Critical Value	0.874
Data are lognormal at 5% significance level	
95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	181.482189
95% Chebyshev (MVUE) UCL	77.1964121
97.5% Chebyshev (MVUE) UCL	100.070859
99% Chebyshev (MVUE) UCL	145.003282

95% Non-parametric UCLs	
CLT UCL	40.1228279
Adj-CLT UCL (Adjusted for skewness)	49.0756935
Mod-t UCL (Adjusted for skewness)	42.8371951
Jackknife UCL	41.440729
Standard Bootstrap UCL	39.5615285
Bootstrap-t UCL	70.606479
Hall's Bootstrap UCL	102.621669
Percentile Bootstrap UCL	41.7257143
BCA Bootstrap UCL	51.0607143
95% Chebyshev (Mean, Sd) UCL	68.4925574
97.5% Chebyshev (Mean, Sd) UCL	88.2078215
99% Chebyshev (Mean, Sd) UCL	126.93464

Attachment B2
ProUCL Summary Sheet
Recreational Receptors Exposed to Sediment
Benzo(a)pyrene

Raw Statistics	
Number of Valid Samples	14
Number of Unique Samples	14
Minimum	0.55
Maximum	100
Mean	17.4307143
Median	9.35
Standard Deviation	26.3432296
Variance	693.965746
Coefficient of Variation	1.51131096
Skewness	2.70376924

Gamma Statistics	
k hat	0.65214075
k star (bias corrected)	0.56001535
Theta hat	26.7284545
Theta star	31.1254224
nu hat	18.2599409
nu star	15.6804298
Approx.Chi Square Value (.05)	7.73597097
Adjusted Level of Significance	0.03122
Adjusted Chi Square Value	7.00681364

Log-transformed Statistics	
Minimum of log data	-0.597837
Maximum of log data	4.60517019
Mean of log data	1.92219322
Standard Deviation of log data	1.52250724
Variance of log data	2.31802831

RECOMMENDATION

Data follow gamma distribution (0.05)

Use Approximate Gamma UCL

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.65195202
Shapiro-Wilk 5% Critical Value	0.874
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	29.8990125

Gamma Distribution Test	
A-D Test Statistic	0.32923925
A-D 5% Critical Value	0.77948605
K-S Test Statistic	0.1655493
K-S 5% Critical Value	0.23917141
Data follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	35.331194
Adjusted Gamma UCL	39.0079008

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.97232859
Shapiro-Wilk 5% Critical Value	0.874
Data are lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	106.125691
95% Chebyshev (MVUE) UCL	56.3133475
97.5% Chebyshev (MVUE) UCL	72.5609065
99% Chebyshev (MVUE) UCL	104.47609

95% Non-parametric UCLs	
CLT UCL	29.0113462
Adj-CLT UCL (Adjusted for skewness)	34.447491
Mod-t UCL (Adjusted for skewness)	30.7469412
Jackknife UCL	29.8990125
Standard Bootstrap UCL	28.6046458
Bootstrap-t UCL	47.0828333
Hall's Bootstrap UCL	76.7685501
Percentile Bootstrap UCL	29.2321429
BCA Bootstrap UCL	34.8592857
95% Chebyshev (Mean, Sd) UCL	48.119648
97.5% Chebyshev (Mean, Sd) UCL	61.3987742
99% Chebyshev (Mean, Sd) UCL	87.4830462

Attachment B2
ProUCL Summary Sheet
Recreational Receptors Exposed to Sediment
Benzo(b)fluoranthene

Raw Statistics	
Number of Valid Samples	14
Number of Unique Samples	14
Minimum	0.55
Maximum	50
Mean	9.87428571
Median	7.9
Standard Deviation	12.6075873
Variance	158.951257
Coefficient of Variation	1.27681006
Skewness	2.75964323

Gamma Statistics	
k hat	0.92191425
k star (bias corrected)	0.77198025
Theta hat	10.7106336
Theta star	12.7908528
nu hat	25.813599
nu star	21.6154469
Approx.Chi Square Value (.05)	12.0488545
Adjusted Level of Significance	0.03122
Adjusted Chi Square Value	11.1104025

Log-transformed Statistics	
Minimum of log data	-0.597837
Maximum of log data	3.91202301
Mean of log data	1.657689
Standard Deviation of log data	1.23817055
Variance of log data	1.5330663

RECOMMENDATION

Data follow gamma distribution (0.05)

Use Approximate Gamma UCL

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.66426898
Shapiro-Wilk 5% Critical Value	0.874
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	15.8414796

Gamma Distribution Test	
A-D Test Statistic	0.37934663
A-D 5% Critical Value	0.76328958
K-S Test Statistic	0.13831269
K-S 5% Critical Value	0.23589836
Data follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	17.7143063
Adjusted Gamma UCL	19.2105641

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.9610049
Shapiro-Wilk 5% Critical Value	0.874
Data are lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	34.0841658
95% Chebyshev (MVUE) UCL	26.9698228
97.5% Chebyshev (MVUE) UCL	34.1522217
99% Chebyshev (MVUE) UCL	48.2606537

95% Non-parametric UCLs	
CLT UCL	15.416652
Adj-CLT UCL (Adjusted for skewness)	18.0720969
Mod-t UCL (Adjusted for skewness)	16.2556753
Jackknife UCL	15.8414796
Standard Bootstrap UCL	15.047661
Bootstrap-t UCL	22.2010389
Hall's Bootstrap UCL	37.9712731
Percentile Bootstrap UCL	16.3107143
BCA Bootstrap UCL	17.9857143
95% Chebyshev (Mean, Sd) UCL	24.5616804
97.5% Chebyshev (Mean, Sd) UCL	30.9169278
99% Chebyshev (Mean, Sd) UCL	43.4005808

Attachment B2
ProUCL Summary Sheet
Recreational Receptors Exposed to Sediment
Benzo(k)fluoranthene

Raw Statistics	
Number of Valid Samples	14
Number of Unique Samples	14
Minimum	0.55
Maximum	71
Mean	11.4678571
Median	7.65
Standard Deviation	17.9251715
Variance	321.311772
Coefficient of Variation	1.56307942
Skewness	3.19916331

Gamma Statistics	
k hat	0.77553213
k star (bias corrected)	0.65696572
Theta hat	14.7870819
Theta star	17.4557922
nu hat	21.7148998
nu star	18.3950403
Approx.Chi Square Value (.05)	9.67556297
Adjusted Level of Significance	0.03122
Adjusted Chi Square Value	8.84669176

Log-transformed Statistics	
Minimum of log data	-0.597837
Maximum of log data	4.26267988
Mean of log data	1.67111738
Standard Deviation of log data	1.32083296
Variance of log data	1.74459971

RECOMMENDATION

Data follow gamma distribution (0.05)

Use Approximate Gamma UCL

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.56474135
Shapiro-Wilk 5% Critical Value	0.874
Data not normal at 5% significance level	
95% UCL (Assuming Normal Distribution)	
Student's-t UCL	19.9518732

Gamma Distribution Test	
A-D Test Statistic	0.48827144
A-D 5% Critical Value	0.76953131
K-S Test Statistic	0.18506335
K-S 5% Critical Value	0.23733876
Data follow gamma distribution	
at 5% significance level	
95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	21.802524
Adjusted Gamma UCL	23.8452633

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.96187675
Shapiro-Wilk 5% Critical Value	0.874
Data are lognormal at 5% significance level	
95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	43.6927954
95% Chebyshev (MVUE) UCL	31.2473629
97.5% Chebyshev (MVUE) UCL	39.7930088
99% Chebyshev (MVUE) UCL	56.5792756

95% Non-parametric UCLs	
CLT UCL	19.3478633
Adj-CLT UCL (Adjusted for skewness)	23.724617
Mod-t UCL (Adjusted for skewness)	20.6345583
Jackknife UCL	19.9518732
Standard Bootstrap UCL	19.1206592
Bootstrap-t UCL	33.9409925
Hall's Bootstrap UCL	49.2351952
Percentile Bootstrap UCL	20.1571429
BCA Bootstrap UCL	25.2071429
95% Chebyshev (Mean, Sd) UCL	32.35005
97.5% Chebyshev (Mean, Sd) UCL	41.3857915
99% Chebyshev (Mean, Sd) UCL	59.134756

Attachment B2
ProUCL Summary Sheet
Recreational Receptors Exposed to Sediment
Chrysene

Raw Statistics	
Number of Valid Samples	14
Number of Unique Samples	14
Minimum	0.55
Maximum	130
Mean	21.0435714
Median	8.35
Standard Deviation	33.9146242
Variance	1150.20173
Coefficient of Variation	1.61163823
Skewness	2.88652682

Gamma Statistics	
k hat	0.59330521
k star (bias corrected)	0.51378743
Theta hat	35.4683746
Theta star	40.9577393
nu hat	16.6125459
nu star	14.3860479
Approx.Chi Square Value (.05)	6.83533599
Adjusted Level of Significance	0.03122
Adjusted Chi Square Value	6.15658763

Log-transformed Statistics	
Minimum of log data	-0.597837
Maximum of log data	4.86753445
Mean of log data	2.0034529
Standard Deviation of log data	1.6050159
Variance of log data	2.57607603

RECOMMENDATION

Data follow gamma distribution (0.05)

Use Approximate Gamma UCL

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.6194604
Shapiro-Wilk 5% Critical Value	0.874
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	37.0954243

Gamma Distribution Test	
A-D Test Statistic	0.39465164
A-D 5% Critical Value	0.78481655
K-S Test Statistic	0.17083931
K-S 5% Critical Value	0.24012219
Data follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	44.2895313
Adjusted Gamma UCL	49.1723412

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.96766288
Shapiro-Wilk 5% Critical Value	0.874
Data are lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	153.193852
95% Chebyshev (MVUE) UCL	70.4463139
97.5% Chebyshev (MVUE) UCL	91.1431488
99% Chebyshev (MVUE) UCL	131.798073

95% Non-parametric UCLs	
CLT UCL	35.9526309
Adj-CLT UCL (Adjusted for skewness)	43.4242545
Mod-t UCL (Adjusted for skewness)	38.2608466
Jackknife UCL	37.0954243
Standard Bootstrap UCL	35.3128895
Bootstrap-t UCL	61.3569265
Hall's Bootstrap UCL	91.7370915
Percentile Bootstrap UCL	36.7428571
BCA Bootstrap UCL	44.7357143
95% Chebyshev (Mean, Sd) UCL	60.5529129
97.5% Chebyshev (Mean, Sd) UCL	77.6486366
99% Chebyshev (Mean, Sd) UCL	111.229875

Attachment B2
ProUCL Summary Sheet
Recreational Receptors Exposed to Sediment
Dibenz(a,h)anthracene

Raw Statistics	
Number of Valid Samples	13
Number of Unique Samples	12
Minimum	0.27
Maximum	21
Mean	6.51692308
Median	2.6
Standard Deviation	6.94134159
Variance	48.1822231
Coefficient of Variation	1.0651256
Skewness	0.98896565

Gamma Statistics	
k hat	0.84060794
k star (bias corrected)	0.69790355
Theta hat	7.75263086
Theta star	9.33785638
nu hat	21.8558065
nu star	18.1454922
Approx.Chi Square Value (.05)	9.49470805
Adjusted Level of Significance	0.03009
Adjusted Chi Square Value	8.61551324

Log-transformed Statistics	
Minimum of log data	-1.30933332
Maximum of log data	3.04452244
Mean of log data	1.17291144
Standard Deviation of log data	1.36401318
Variance of log data	1.86053194

RECOMMENDATION

Data follow gamma distribution (0.05)

Use Approximate Gamma UCL

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.81918783
Shapiro-Wilk 5% Critical Value	0.866
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	9.94815043

Gamma Distribution Test	
A-D Test Statistic	0.50761744
A-D 5% Critical Value	0.76563101
K-S Test Statistic	0.19917884
K-S 5% Critical Value	0.24484069
Data follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	12.4545985
Adjusted Gamma UCL	13.7255638

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.93993383
Shapiro-Wilk 5% Critical Value	0.866
Data are lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	32.916206
95% Chebyshev (MVUE) UCL	20.56488
97.5% Chebyshev (MVUE) UCL	26.3126171
99% Chebyshev (MVUE) UCL	37.6029336

95% Non-parametric UCLs	
CLT UCL	9.6835653
Adj-CLT UCL (Adjusted for skewness)	10.2478025
Mod-t UCL (Adjusted for skewness)	10.03616
Jackknife UCL	9.94815043
Standard Bootstrap UCL	9.59742658
Bootstrap-t UCL	11.0630621
Hall's Bootstrap UCL	9.72107064
Percentile Bootstrap UCL	9.62692308
BCA Bootstrap UCL	10.0076923
95% Chebyshev (Mean, Sd) UCL	14.9085959
97.5% Chebyshev (Mean, Sd) UCL	18.5396794
99% Chebyshev (Mean, Sd) UCL	25.6722398

Attachment B2
ProUCL Summary Sheet
Recreational Receptors Exposed to Sediment
Fluoranthene

Raw Statistics	
Number of Valid Samples	14
Number of Unique Samples	14
Minimum	0.55
Maximum	330
Mean	48.5285714
Median	16.4
Standard Deviation	87.8828432
Variance	7723.39412
Coefficient of Variation	1.81095055
Skewness	2.89813645

Gamma Statistics	
k hat	0.46321025
k star (bias corrected)	0.41156996
Theta hat	104.765755
Theta star	117.910869
nu hat	12.969887
nu star	11.5239588
Approx.Chi Square Value (.05)	4.91420112
Adjusted Level of Significance	0.03122
Adjusted Chi Square Value	4.35501819

Log-transformed Statistics	
Minimum of log data	-0.597837
Maximum of log data	5.79909265
Mean of log data	2.49440606
Standard Deviation of log data	1.88215503
Variance of log data	3.54250754

RECOMMENDATION

Data follow gamma distribution (0.05)

Use Adjusted Gamma UCL

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.58872497
Shapiro-Wilk 5% Critical Value	0.874
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	90.1236799

Gamma Distribution Test	
A-D Test Statistic	0.51803936
A-D 5% Critical Value	0.80039985
K-S Test Statistic	0.18828555
K-S 5% Critical Value	0.24276496
Data follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	113.801052
Adjusted Gamma UCL	128.413071

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.96178963
Shapiro-Wilk 5% Critical Value	0.874
Data are lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	729.936725
95% Chebyshev (MVUE) UCL	189.050603
97.5% Chebyshev (MVUE) UCL	247.369993
99% Chebyshev (MVUE) UCL	361.927143

95% Non-parametric UCLs	
CLT UCL	87.1623635
Adj-CLT UCL (Adjusted for skewness)	106.601426
Mod-t UCL (Adjusted for skewness)	93.1557807
Jackknife UCL	90.1236799
Standard Bootstrap UCL	86.5014704
Bootstrap-t UCL	157.520324
Hall's Bootstrap UCL	216.95736
Percentile Bootstrap UCL	91.2214286
BCA Bootstrap UCL	112.521429
95% Chebyshev (Mean, Sd) UCL	150.908985
97.5% Chebyshev (Mean, Sd) UCL	195.209072
99% Chebyshev (Mean, Sd) UCL	282.228015

Attachment B2
ProUCL Summary Sheet
Recreational Receptors Exposed to Sediment
Fluorene

Raw Statistics	
Number of Valid Samples	13
Number of Unique Samples	13
Minimum	0.047
Maximum	500
Mean	52.1228462
Median	6.3
Standard Deviation	137.063719
Variance	18786.463
Coefficient of Variation	2.62962844
Skewness	3.39656537

Gamma Statistics	
k hat	0.30883013
k star (bias corrected)	0.28884369
Theta hat	168.77513
Theta star	180.453468
nu hat	8.02958349
nu star	7.50993602
Approx.Chi Square Value (.05)	2.45397817
Adjusted Level of Significance	0.03009
Adjusted Chi Square Value	2.06231289

Log-transformed Statistics	
Minimum of log data	-3.05760768
Maximum of log data	6.2146081
Mean of log data	1.73131953
Standard Deviation of log data	2.31764557
Variance of log data	5.37148096

RECOMMENDATION

Data are lognormal (0.05)

Use 99% Chebyshev (MVUE) UCL

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.42821179
Shapiro-Wilk 5% Critical Value	0.866
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	119.875855

Gamma Distribution Test	
A-D Test Statistic	0.99913568
A-D 5% Critical Value	0.8280406
K-S Test Statistic	0.30277191
K-S 5% Critical Value	0.2560517
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	159.512111
Adjusted Gamma UCL	189.805942

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.97179348
Shapiro-Wilk 5% Critical Value	0.866
Data are lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	3292.02179
95% Chebyshev (MVUE) UCL	197.882837
97.5% Chebyshev (MVUE) UCL	262.304404
99% Chebyshev (MVUE) UCL	388.848099

95% Non-parametric UCLs	
CLT UCL	114.651358
Adj-CLT UCL (Adjusted for skewness)	152.916167
Mod-t UCL (Adjusted for skewness)	125.844392
Jackknife UCL	119.875855
Standard Bootstrap UCL	112.589835
Bootstrap-t UCL	648.59573
Hall's Bootstrap UCL	571.249687
Percentile Bootstrap UCL	125.726692
BCA Bootstrap UCL	170.019231
95% Chebyshev (Mean, Sd) UCL	217.824802
97.5% Chebyshev (Mean, Sd) UCL	289.524171
99% Chebyshev (Mean, Sd) UCL	430.363697

Attachment B2
ProUCL Summary Sheet
Recreational Receptors Exposed to Sediment
Indeno(1,2,3-c,d)pyrene

Raw Statistics	
Number of Valid Samples	14
Number of Unique Samples	14
Minimum	0.55
Maximum	21
Mean	6.83928571
Median	4.2
Standard Deviation	6.41510173
Variance	41.1535302
Coefficient of Variation	0.93797832
Skewness	1.05478622

Gamma Statistics	
k hat	1.12453416
k star (bias corrected)	0.9311816
Theta hat	6.08188345
Theta star	7.34473886
nu hat	31.4869566
nu star	26.0730849
Approx.Chi Square Value (.05)	15.4338227
Adjusted Level of Significance	0.03122
Adjusted Chi Square Value	14.3559514

Log-transformed Statistics	
Minimum of log data	-0.597837
Maximum of log data	3.04452244
Mean of log data	1.41617493
Standard Deviation of log data	1.14828172
Variance of log data	1.3185509

RECOMMENDATION

Data follow gamma distribution (0.05)

Use Approximate Gamma UCL

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.86169925
Shapiro-Wilk 5% Critical Value	0.874
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	9.87556496

Gamma Distribution Test	
A-D Test Statistic	0.26681732
A-D 5% Critical Value	0.75764117
K-S Test Statistic	0.12709919
K-S 5% Critical Value	0.23453224
Data follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	11.5539281
Adjusted Gamma UCL	12.4214183

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.9481869
Shapiro-Wilk 5% Critical Value	0.874
Data are lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	21.0812355
95% Chebyshev (MVUE) UCL	18.3760388
97.5% Chebyshev (MVUE) UCL	23.1124419
99% Chebyshev (MVUE) UCL	32.4161885

95% Non-parametric UCLs	
CLT UCL	9.65940051
Adj-CLT UCL (Adjusted for skewness)	10.1758411
Mod-t UCL (Adjusted for skewness)	9.95611925
Jackknife UCL	9.87556496
Standard Bootstrap UCL	9.56287961
Bootstrap-t UCL	10.9573601
Hall's Bootstrap UCL	10.0570228
Percentile Bootstrap UCL	9.71785714
BCA Bootstrap UCL	10.05
95% Chebyshev (Mean, Sd) UCL	14.3126531
97.5% Chebyshev (Mean, Sd) UCL	17.5463851
99% Chebyshev (Mean, Sd) UCL	23.8984255

Attachment B2
ProUCL Summary Sheet
Recreational Receptors Exposed to Sediment
Methylene Chloride

Data File I:\misc_N_T\Risk 2007\NSP\List of Comments_0907\Sed Variable: Methylene chloride

Raw Statistics	
Number of Valid Samples	10
Number of Unique Samples	10
Minimum	0.08
Maximum	36.8
Mean	6.494
Median	0.18
Standard Deviation	13.1721735
Variance	173.506154
Coefficient of Variation	2.02836056
Skewness	1.96334612

Gamma Statistics	
k hat	0.2787514
k star (bias corrected)	0.26179265
Theta hat	23.2967437
Theta star	24.8058913
nu hat	5.57502807
nu star	5.23585298
Approx.Chi Square Value (.05)	1.26226685
Adjusted Level of Significance	0.0267
Adjusted Chi Square Value	0.95812419

Log-transformed Statistics	
Minimum of log data	-2.52572864
Maximum of log data	3.60549785
Mean of log data	-0.63267893
Standard Deviation of log data	2.34834347
Variance of log data	5.51471706

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

Recommended UCL exceeds the maximum observation

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.56547569
Shapiro-Wilk 5% Critical Value	0.842
Data not normal at 5% significance level	
95% UCL (Assuming Normal Distribution)	
Student's-t UCL	14.1296609

Gamma Distribution Test	
A-D Test Statistic	1.4005857
A-D 5% Critical Value	0.82218956
K-S Test Statistic	0.32135347
K-S 5% Critical Value	0.28916591
Data do not follow gamma distribution	
at 5% significance level	
95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	26.9369582
Adjusted Gamma UCL	35.4877058

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.78677383
Shapiro-Wilk 5% Critical Value	0.842
Data not lognormal at 5% significance level	
95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	1112.19222
95% Chebyshev (MVUE) UCL	18.2178963
97.5% Chebyshev (MVUE) UCL	24.2266189
99% Chebyshev (MVUE) UCL	36.0295911

95% Non-parametric UCLs	
CLT UCL	13.3454848
Adj-CLT UCL (Adjusted for skewness)	16.1088274
Mod-t UCL (Adjusted for skewness)	14.5606865
Jackknife UCL	14.1296609
Standard Bootstrap UCL	12.9869571
Bootstrap-t UCL	149.223641
Hall's Bootstrap UCL	192.620878
Percentile Bootstrap UCL	13.6485
BCA Bootstrap UCL	15.147
95% Chebyshev (Mean, Sd) UCL	24.6505882
97.5% Chebyshev (Mean, Sd) UCL	32.5069584
99% Chebyshev (Mean, Sd) UCL	47.9392763

Attachment B2
ProUCL Summary Sheet
Recreational Receptors Exposed to Sediment
Naphthalene

Raw Statistics	
Number of Valid Samples	14
Number of Unique Samples	14
Minimum	0.0295
Maximum	2710
Mean	266.388536
Median	7.05
Standard Deviation	732.214977
Variance	536138.773
Coefficient of Variation	2.74867301
Skewness	3.32090264

Gamma Statistics	
k hat	0.19575392
k star (bias corrected)	0.2014257
Theta hat	1360.83373
Theta star	1322.51514
nu hat	5.48110973
nu star	5.63991955
Approx.Chi Square Value (.05)	1.45824198
Adjusted Level of Significance	0.03122
Adjusted Chi Square Value	1.19669039

Log-transformed Statistics	
Minimum of log data	-3.52336502
Maximum of log data	7.90470391
Mean of log data	1.81296362
Standard Deviation of log data	3.32670186
Variance of log data	11.0669453

RECOMMENDATION

Data are lognormal (0.05)

Use Hall's Bootstrap UCL

Recommended UCL exceeds the maximum observation

In case Hall's Bootstrap method yields
an erratic, unreasonably large UCL value,
use 99% Chebyshev (Mean, Sd) UCL

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.42823952
Shapiro-Wilk 5% Critical Value	0.874
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	612.947207

Gamma Distribution Test	
A-D Test Statistic	0.95183983
A-D 5% Critical Value	0.8749222
K-S Test Statistic	0.2537704
K-S 5% Critical Value	0.25312305
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	1030.28848
Adjusted Gamma UCL	1255.47086

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.98131328
Shapiro-Wilk 5% Critical Value	0.874
Data are lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	1526473.93
95% Chebyshev (MVUE) UCL	1778.02329
97.5% Chebyshev (MVUE) UCL	2387.34361
99% Chebyshev (MVUE) UCL	3584.23539

95% Non-parametric UCLs	
CLT UCL	588.274357
Adj-CLT UCL (Adjusted for skewness)	773.861177
Mod-t UCL (Adjusted for skewness)	641.895001
Jackknife UCL	612.947207
Standard Bootstrap UCL	572.817223
Bootstrap-t UCL	4594.53884
Hall's Bootstrap UCL	4594.7676
Percentile Bootstrap UCL	607.688821
BCA Bootstrap UCL	836.283857
95% Chebyshev (Mean, Sd) UCL	1119.39317
97.5% Chebyshev (Mean, Sd) UCL	1488.48896
99% Chebyshev (Mean, Sd) UCL	2213.50616

Attachment B2
ProUCL Summary Sheet
Recreational Receptors Exposed to Sediment
Pyrene

Raw Statistics	
Number of Valid Samples	14
Number of Unique Samples	14
Minimum	0.55
Maximum	410
Mean	61.9535714
Median	23.9
Standard Deviation	109.05356
Variance	11892.679
Coefficient of Variation	1.76024655
Skewness	2.85149591

Gamma Statistics	
k hat	0.4274993
k star (bias corrected)	0.38351136
Theta hat	144.920871
Theta star	161.54299
nu hat	11.9699805
nu star	10.738318
Approx.Chi Square Value (.05)	4.4074168
Adjusted Level of Significance	0.03122
Adjusted Chi Square Value	3.88336328

Log-transformed Statistics	
Minimum of log data	-0.597837
Maximum of log data	6.01615716
Mean of log data	2.60344703
Standard Deviation of log data	2.06434141
Variance of log data	4.26150546

RECOMMENDATION

Data follow gamma distribution (0.05)

Use Adjusted Gamma UCL

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.60451941
Shapiro-Wilk 5% Critical Value	0.874
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	113.568819

Gamma Distribution Test	
A-D Test Statistic	0.56435123
A-D 5% Critical Value	0.80732063
K-S Test Statistic	0.21037847
K-S 5% Critical Value	0.24386665
Data follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	150.944915
Adjusted Gamma UCL	171.314684

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.92739146
Shapiro-Wilk 5% Critical Value	0.874
Data are lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	1804.49989
95% Chebyshev (MVUE) UCL	296.00595
97.5% Chebyshev (MVUE) UCL	389.560086
99% Chebyshev (MVUE) UCL	573.329072

95% Non-parametric UCLs	
CLT UCL	109.89413
Adj-CLT UCL (Adjusted for skewness)	133.627804
Mod-t UCL (Adjusted for skewness)	117.270792
Jackknife UCL	113.568819
Standard Bootstrap UCL	110.091174
Bootstrap-t UCL	187.532624
Hall's Bootstrap UCL	276.476066
Percentile Bootstrap UCL	113.028571
BCA Bootstrap UCL	137.296429
95% Chebyshev (Mean, Sd) UCL	188.997125
97.5% Chebyshev (Mean, Sd) UCL	243.968972
99% Chebyshev (Mean, Sd) UCL	351.95052

Attachment B2
ProUCL Summary Sheet
Recreational Receptors Exposed to Sediment
Xylenes (total)

Data File I:\misc_N_T\Risk 2007\NSP\List of Comments_0907\Sed Variable: Xylenes (total)

Raw Statistics	
Number of Valid Samples	14
Number of Unique Samples	14
Minimum	0.0025
Maximum	37.5
Mean	5.00878571
Median	0.085
Standard Deviation	12.4737212
Variance	155.59372
Coefficient of Variation	2.4903683
Skewness	2.34257536

Gamma Statistics	
k hat	0.20821916
k star (bias corrected)	0.21121982
Theta hat	24.0553547
Theta star	23.7136166
nu hat	5.83013645
nu star	5.91415483
Approx.Chi Square Value (.05)	1.59566565
Adjusted Level of Significance	0.03122
Adjusted Chi Square Value	1.31805613

Log-transformed Statistics	
Minimum of log data	-5.99146455
Maximum of log data	3.62434093
Mean of log data	-1.90094715
Standard Deviation of log data	2.69199338
Variance of log data	7.24682835

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.45069878
Shapiro-Wilk 5% Critical Value	0.874
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	10.9126205

Gamma Distribution Test	
A-D Test Statistic	2.20685132
A-D 5% Critical Value	0.87008234
K-S Test Statistic	0.37953599
K-S 5% Critical Value	0.25253589
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	18.5644996
Adjusted Gamma UCL	22.4745621

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.86590703
Shapiro-Wilk 5% Critical Value	0.874
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	543.309044
95% Chebyshev (MVUE) UCL	11.2901095
97.5% Chebyshev (MVUE) UCL	15.0550772
99% Chebyshev (MVUE) UCL	22.4506271

95% Non-parametric UCLs	
CLT UCL	10.4923037
Adj-CLT UCL (Adjusted for skewness)	12.7224943
Mod-t UCL (Adjusted for skewness)	11.2604852
Jackknife UCL	10.9126205
Standard Bootstrap UCL	10.2940471
Bootstrap-t UCL	406.886757
Hall's Bootstrap UCL	413.847532
Percentile Bootstrap UCL	10.3238929
BCA Bootstrap UCL	12.616
95% Chebyshev (Mean, Sd) UCL	19.5402311
97.5% Chebyshev (Mean, Sd) UCL	25.8279992
99% Chebyshev (Mean, Sd) UCL	38.1791019

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

Recommended UCL exceeds the maximum observation

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
1,2,4 Tirchlorobenzene

Raw Statistics	
Number of Valid Samples	16
Number of Unique Samples	10
Minimum	9.7
Maximum	120000
Mean	15438.7313
Median	75
Standard Deviation	35798.064
Variance	1281501389
Coefficient of Variation	2.318718
Skewness	*2.49148923*

Gamma Statistics	
k hat	0.20110297
k star (bias corrected)	0.20506283
Theta hat	76770.2796
Theta star	75287.8093
nu hat	6.43529505
nu star	6.56201056
Approx.Chi Square Value (.05)	1.93294156
Adjusted Level of Significance	0.03348
Adjusted Chi Square Value	1.66084376

Log-transformed Statistics	
Minimum of log data	2.27212589
Maximum of log data	11.695247
Mean of log data	5.98835297
Standard Deviation of log data	3.04419978
Variance of log data	9.26715229

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.50395508
Shapiro-Wilk 5% Critical Value	0.887
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	31127.6815

Gamma Distribution Test	
A-D Test Statistic	1.95354211
A-D 5% Critical Value	*0.87788341*
K-S Test Statistic	0.32143776
K-S 5% Critical Value	0.2375486
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	52411.8885
Adjusted Gamma UCL	60998.584

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.82160296
Shapiro-Wilk 5% Critical Value	0.887
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	7050860.16
95% Chebyshev (MVUE) UCL	68392.4182
97.5% Chebyshev (MVUE) UCL	91529.122
99% Chebyshev (MVUE) UCL	136976.697

95% Non-parametric UCLs	
CLT UCL	30159.3751
Adj-CLT UCL (Adjusted for skewness)	36115.7084
Mod-t UCL (Adjusted for skewness)	32056.7491
Jackknife UCL	31127.6815
Standard Bootstrap UCL	29615.3507
Bootstrap-t UCL	92006.2739
Hall's Bootstrap UCL	*116072.498*
Percentile Bootstrap UCL	30890.625
BCA Bootstrap UCL	38025.6063
95% Chebyshev (Mean, Sd) UCL	54448.7671
97.5% Chebyshev (Mean, Sd) UCL	71328.4408
99% Chebyshev (Mean, Sd) UCL	104485.291

RECOMMENDATION

Data are Non-parametric (0.05)

Use Hall's Bootstrap UCL

In case Hall's Bootstrap method yields
an erratic, unreasonably large UCL value,
use 99% Chebyshev (Mean, Sd) UCL

All units are micrograms per kilogram (µg/kg).

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
Xylenes (total)

Raw Statistics	
Number of Valid Samples	18
Number of Unique Samples	12
Minimum	2.5
Maximum	320000
Mean	23333.4722
Median	75
Standard Deviation	76138.688
Variance	5797099805
Coefficient of Variation	3.2630672
Skewness	3.9083269

Gamma Statistics	
k hat	0.17309823
k star (bias corrected)	0.18128556
Theta hat	134799.02
Theta star	128711.143
nu hat	6.23153639
nu star	6.52628032
Approx.Chi Square Value (.05)	1.91391131
Adjusted Level of Significance	0.03574
Adjusted Chi Square Value	1.68414896

Log-transformed Statistics	
Minimum of log data	0.91629073
Maximum of log data	12.6760763
Mean of log data	5.71079356
Standard Deviation of log data	3.00525922
Variance of log data	9.03158299

Normal Distribution Test	
Shapiro-Wilk Test Statisitic	0.352647
Shapiro-Wilk 5% Critical Value	0.897
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	54552.556

Gamma Distribution Test	
A-D Test Statistic	2.73643723
A-D 5% Critical Value	0.89462174
K-S Test Statistic	0.35384223
K-S 5% Critical Value	0.22636765
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	79565.2234
Adjusted Gamma UCL	90420.0187

Lognormal Distribution Test	
Shapiro-Wilk Test Statisitic	0.85812424
Shapiro-Wilk 5% Critical Value	0.897
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	2626284.2
95% Chebyshev (MVUE) UCL	50761.8636
97.5% Chebyshev (MVUE) UCL	67851.9237
99% Chebyshev (MVUE) UCL	101422.037

95% Non-parametric UCLs	
CLT UCL	52852.1155
Adj-CLT UCL (Adjusted for skewness)	70516.7307
Mod-t UCL (Adjusted for skewness)	57307.8789
Jackknife UCL	54552.556
Standard Bootstrap UCL	52191.8814
Bootstrap-t UCL	449034.248
Hall's Bootstrap UCL	405604.69
Percentile Bootstrap UCL	55345.0556
BCA Bootstrap UCL	79602.2222
95% Chebyshev (Mean, Sd) UCL	101558.538
97.5% Chebyshev (Mean, Sd) UCL	135406.586
99% Chebyshev (Mean, Sd) UCL	201894.523

RECOMMENDATION

Data are Non-parametric (0.05)

Use Hall's Bootstrap UCL

Recommended UCL exceeds the maximum observation

In case Hall's Bootstrap method yields
an erratic, unreasonably large UCL value,

All units are micrograms per kilogram (µg/kg).

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
1-Methylnaphthalene

Raw Statistics	
Number of Valid Samples	86
Number of Unique Samples	40
Minimum	16
Maximum	3800000
Mean	75314.2849
Median	80
Standard Deviation	425433.441
Variance	1.81E+11
Coefficient of Variation	5.64877489
Skewness	8.21143718

Gamma Statistics	
k hat	0.13984998
k star (bias corrected)	0.14272343
Theta hat	538536.256
Theta star	527693.907
nu hat	24.0541967
nu star	24.5484301
Approx.Chi Square Value (.05)	14.2647374
Adjusted Level of Significance	0.0472093
Adjusted Chi Square Value	14.1309043

Log-transformed Statistics	
Minimum of log data	2.77258872
Maximum of log data	15.1505116
Mean of log data	5.67802069
Standard Deviation of log data	3.42879438
Variance of log data	11.7566309

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are micrograms per kilogram (µg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.42975737
Lilliefors 5% Critical Value	0.09553984
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	151604.518

Gamma Distribution Test	
A-D Test Statistic	12.1759612
A-D 5% Critical Value	0.96900799
K-S Test Statistic	0.29658775
K-S 5% Critical Value	0.10911735
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	129609.639
Adjusted Gamma UCL	130837.165

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.20931677
Lilliefors 5% Critical Value	0.09553984
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	729417.889
95% Chebyshev (MVUE) UCL	277693.798
97.5% Chebyshev (MVUE) UCL	367606.839
99% Chebyshev (MVUE) UCL	544223.597

95% Non-parametric UCLs	
CLT UCL	150773.044
Adj-CLT UCL (Adjusted for skewness)	194177.344
Mod-t UCL (Adjusted for skewness)	158374.711
Jackknife UCL	151604.518
Standard Bootstrap UCL	150117.257
Bootstrap-t UCL	412995.886
Hall's Bootstrap UCL	420271.63
Percentile Bootstrap UCL	163021.343
BCA Bootstrap UCL	217259.837
95% Chebyshev (Mean, Sd) UCL	275281.685
97.5% Chebyshev (Mean, Sd) UCL	361807.738
99% Chebyshev (Mean, Sd) UCL	531771.416

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
Dibenzofuran

Raw Statistics	
Number of Valid Samples	86
Number of Unique Samples	28
Minimum	16
Maximum	280000
Mean	4672.98256
Median	31.5
Standard Deviation	30331.4648
Variance	919997759
Coefficient of Variation	6.49081491
Skewness	9.01497844

Gamma Statistics	
k hat	0.19628373
k star (bias corrected)	0.19718856
Theta hat	23807.2841
Theta star	23698.0406
nu hat	33.7608018
nu star	33.9164328
Approx.Chi Square Value (.05)	21.5954344
Adjusted Level of Significance	0.0472093
Adjusted Chi Square Value	21.4277916

Log-transformed Statistics	
Minimum of log data	2.77258872
Maximum of log data	12.5425449
Mean of log data	4.68932109
Standard Deviation of log data	2.37974998
Variance of log data	5.66320996

RECOMMENDATION
Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are micrograms per kilogram (µg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.43898764
Lilliefors 5% Critical Value	0.09553984
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	10112.1283

Gamma Distribution Test	
A-D Test Statistic	13.0312486
A-D 5% Critical Value	0.91454265
K-S Test Statistic	0.29058965
K-S 5% Critical Value	0.10695256
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	7339.09289
Adjusted Gamma UCL	7396.51112

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.2336248
Lilliefors 5% Critical Value	0.09553984
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	4955.26804
95% Chebyshev (MVUE) UCL	4717.08324
97.5% Chebyshev (MVUE) UCL	6046.90728
99% Chebyshev (MVUE) UCL	8659.08911

95% Non-parametric UCLs	
CLT UCL	10052.848
Adj-CLT UCL (Adjusted for skewness)	13450.1961
Mod-t UCL (Adjusted for skewness)	10642.046
Jackknife UCL	10112.1283
Standard Bootstrap UCL	9931.35628
Bootstrap-t UCL	50641.6186
Hall's Bootstrap UCL	29951.7628
Percentile Bootstrap UCL	11090.2442
BCA Bootstrap UCL	15809.3895
95% Chebyshev (Mean, Sd) UCL	18929.7465
97.5% Chebyshev (Mean, Sd) UCL	25098.6597
99% Chebyshev (Mean, Sd) UCL	37216.2951

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
1,2,4 Trimethylbenzene

Raw Statistics	
Number of Valid Samples	87
Number of Unique Samples	40
Minimum	7.5
Maximum	100000
Mean	3773.70115
Median	20
Standard Deviation	12722.3847
Variance	161859072
Coefficient of Variation	3.37132809
Skewness	5.94609737

Gamma Statistics	
k hat	0.18820512
k star (bias corrected)	0.18937813
Theta hat	20051.0014
Theta star	19926.8059
nu hat	32.7476912
nu star	32.9517938
Approx.Chi Square Value (.05)	20.8261396
Adjusted Level of Significance	0.04724138
Adjusted Chi Square Value	20.6636737

Log-transformed Statistics	
Minimum of log data	2.01490302
Maximum of log data	11.5129255
Mean of log data	4.28859053
Standard Deviation of log data	2.93236233
Variance of log data	8.59874884

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are micrograms per kilogram (µg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.38360384
Lilliefors 5% Critical Value	0.09498917
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	6041.68426

Gamma Distribution Test	
A-D Test Statistic	11.6424504
A-D 5% Critical Value	0.92252184
K-S Test Statistic	0.3033161
K-S 5% Critical Value	0.10664816
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	5970.8724
Adjusted Gamma UCL	6017.81773

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.2751946
Lilliefors 5% Critical Value	0.09498917
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	22618.1504
95% Chebyshev (MVUE) UCL	14724.2426
97.5% Chebyshev (MVUE) UCL	19255.1254
99% Chebyshev (MVUE) UCL	28155.1672

95% Non-parametric UCLs	
CLT UCL	6017.25322
Adj-CLT UCL (Adjusted for skewness)	6946.3516
Mod-t UCL (Adjusted for skewness)	6186.60483
Jackknife UCL	6041.68426
Standard Bootstrap UCL	6063.3595
Bootstrap-t UCL	9233.2206
Hall's Bootstrap UCL	15982.2445
Percentile Bootstrap UCL	6278.67241
BCA Bootstrap UCL	7502.98276
95% Chebyshev (Mean, Sd) UCL	9719.16438
97.5% Chebyshev (Mean, Sd) UCL	12291.7711
99% Chebyshev (Mean, Sd) UCL	17345.1588

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
Dibenzo(a,h)anthracene

Raw Statistics	
Number of Valid Samples	102
Number of Unique Samples	35
Minimum	0.11
Maximum	42500
Mean	2028.36382
Median	85
Standard Deviation	6526.34835
Variance	42593222.8
Coefficient of Variation	3.21754326
Skewness	4.44694852

Gamma Statistics	
k hat	0.25523599
k star (bias corrected)	0.25426499
Theta hat	7947.0135
Theta star	7977.36174
nu hat	52.0681411
nu star	51.8700585
Approx.Chi Square Value (.05)	36.32539
Adjusted Level of Significance	0.04764706
Adjusted Chi Square Value	36.1393352

Log-transformed Statistics	
Minimum of log data	-2.20727491
Maximum of log data	10.6572594
Mean of log data	4.84140618
Standard Deviation of log data	2.31398326
Variance of log data	5.35451852

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are micrograms per kilogram (µg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.38103036
Lilliefors 5% Critical Value	0.08772707
Data not normal at 5% significance level	
95% UCL (Assuming Normal Distribution)	
Student's-t UCL	3101.11563

Gamma Distribution Test	
A-D Test Statistic	10.5019022
A-D 5% Critical Value	0.88765917
K-S Test Statistic	0.24541219
K-S 5% Critical Value	0.09741064
Data do not follow gamma distribution	
at 5% significance level	
95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	2896.35845
Adjusted Gamma UCL	2911.26967

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.18961505
Lilliefors 5% Critical Value	0.08772707
Data not lognormal at 5% significance level	
95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	4298.62982
95% Chebyshev (MVUE) UCL	4518.33405
97.5% Chebyshev (MVUE) UCL	5745.14138
99% Chebyshev (MVUE) UCL	8154.96682

95% Non-parametric UCLs	
CLT UCL	3091.2761
Adj-CLT UCL (Adjusted for skewness)	3395.30341
Mod-t UCL (Adjusted for skewness)	3148.53774
Jackknife UCL	3101.11563
Standard Bootstrap UCL	3117.11929
Bootstrap-t UCL	3675.9417
Hall's Bootstrap UCL	3389.9698
Percentile Bootstrap UCL	3160.78431
BCA Bootstrap UCL	3506.90196
95% Chebyshev (Mean, Sd) UCL	4845.10515
97.5% Chebyshev (Mean, Sd) UCL	6063.91137
99% Chebyshev (Mean, Sd) UCL	8458.02019

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
Chrysene

Raw Statistics	
Number of Valid Samples	102
Number of Unique Samples	57
Minimum	0.11
Maximum	470000
Mean	14012.0697
Median	195
Standard Deviation	57268.9765
Variance	3279735669
Coefficient of Variation	4.08711759
Skewness	6.12229268

Gamma Statistics	
k hat	0.19397996
k star (bias corrected)	0.19481062
Theta hat	72234.6247
Theta star	71926.6229
nu hat	39.5719121
nu star	39.7413657
Approx.Chi Square Value (.05)	26.296233
Adjusted Level of Significance	0.04764706
Adjusted Chi Square Value	26.1396157

Log-transformed Statistics	
Minimum of log data	-2.20727491
Maximum of log data	13.060488
Mean of log data	5.73580665
Standard Deviation of log data	2.84668314
Variance of log data	8.10360489

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are micrograms per kilogram (µg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.41312969
Lilliefors 5% Critical Value	0.08772707
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	23425.5111

Gamma Distribution Test	
A-D Test Statistic	10.1689041
A-D 5% Critical Value	0.91889393
K-S Test Statistic	0.24543105
K-S 5% Critical Value	0.098749
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	21176.371
Adjusted Gamma UCL	21303.2507

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.16476803
Lilliefors 5% Critical Value	0.08772707
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	61312.4768
95% Chebyshev (MVUE) UCL	48270.4556
97.5% Chebyshev (MVUE) UCL	62732.5163
99% Chebyshev (MVUE) UCL	91140.4341

95% Non-parametric UCLs	
CLT UCL	23339.1688
Adj-CLT UCL (Adjusted for skewness)	27012.1081
Mod-t UCL (Adjusted for skewness)	23998.4154
Jackknife UCL	23425.5111
Standard Bootstrap UCL	23381.7897
Bootstrap-t UCL	34662.7052
Hall's Bootstrap UCL	28368.579
Percentile Bootstrap UCL	24102.6972
BCA Bootstrap UCL	27689.9188
95% Chebyshev (Mean, Sd) UCL	38729.0913
97.5% Chebyshev (Mean, Sd) UCL	49424.1662
99% Chebyshev (Mean, Sd) UCL	70432.57

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
Benzo(k)fluoranthene

Raw Statistics	
Number of Valid Samples	102
Number of Unique Samples	49
Minimum	0.11
Maximum	170000
Mean	6353.42265
Median	175
Standard Deviation	22993.6038
Variance	528705816
Coefficient of Variation	3.61908928
Skewness	5.4668445

Gamma Statistics	
k hat	0.21411395
k star (bias corrected)	0.21435243
Theta hat	29673.0905
Theta star	29640.0776
nu hat	43.679246
nu star	43.7278956
Approx.Chi Square Value (.05)	29.5608907
Adjusted Level of Significance	0.04764706
Adjusted Chi Square Value	29.3941606

Log-transformed Statistics	
Minimum of log data	-2.20727491
Maximum of log data	12.0435537
Mean of log data	5.35628767
Standard Deviation of log data	2.62925368
Variance of log data	6.91297494

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are micrograms per kilogram (µg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.40177987
Lilliefors 5% Critical Value	0.08772707
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	10132.9374

Gamma Distribution Test	
A-D Test Statistic	10.6187184
A-D 5% Critical Value	0.90603949
K-S Test Statistic	0.23639681
K-S 5% Critical Value	0.0982279
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	9398.28927
Adjusted Gamma UCL	9451.59843

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.16968536
Lilliefors 5% Critical Value	0.08772707
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	19551.3397
95% Chebyshev (MVUE) UCL	17658.9695
97.5% Chebyshev (MVUE) UCL	22769.2192
99% Chebyshev (MVUE) UCL	32807.3152

95% Non-parametric UCLs	
CLT UCL	10098.2708
Adj-CLT UCL (Adjusted for skewness)	11415.0835
Mod-t UCL (Adjusted for skewness)	10338.3336
Jackknife UCL	10132.9374
Standard Bootstrap UCL	10115.5606
Bootstrap-t UCL	14479.4674
Hall's Bootstrap UCL	24782.4459
Percentile Bootstrap UCL	10585.354
BCA Bootstrap UCL	11639.7549
95% Chebyshev (Mean, Sd) UCL	16277.3542
97.5% Chebyshev (Mean, Sd) UCL	20571.4473
99% Chebyshev (Mean, Sd) UCL	29006.3617

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
2-Methylnaphthalene

Raw Statistics	
Number of Valid Samples	102
Number of Unique Samples	54
Minimum	7.6
Maximum	3300000
Mean	80467.8588
Median	155
Standard Deviation	377603.218
Variance	1.43E+11
Coefficient of Variation	4.69259682
Skewness	7.02102006

Gamma Statistics	
k hat	0.14370387
k star (bias corrected)	0.14601323
Theta hat	559956.101
Theta star	551099.766
nu hat	29.3155895
nu star	29.7866996
Approx.Chi Square Value (.05)	18.3232146
Adjusted Level of Significance	0.04764706
Adjusted Chi Square Value	18.1942963

Log-transformed Statistics	
Minimum of log data	2.02814825
Maximum of log data	15.009433
Mean of log data	5.91402741
Standard Deviation of log data	3.44975772
Variance of log data	11.9008283

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are micrograms per kilogram (µg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.41627245
Lilliefors 5% Critical Value	0.08772707
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	142535.421

Gamma Distribution Test	
A-D Test Statistic	12.8946293
A-D 5% Critical Value	0.97356193
K-S Test Statistic	0.2711651
K-S 5% Critical Value	0.10075833
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	130810.668
Adjusted Gamma UCL	131737.546

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.1776005
Lilliefors 5% Critical Value	0.08772707
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	835817.052
95% Chebyshev (MVUE) UCL	385874.298
97.5% Chebyshev (MVUE) UCL	509653.359
99% Chebyshev (MVUE) UCL	752793.357

95% Non-parametric UCLs	
CLT UCL	141966.123
Adj-CLT UCL (Adjusted for skewness)	169738.698
Mod-t UCL (Adjusted for skewness)	146867.382
Jackknife UCL	142535.421
Standard Bootstrap UCL	143909.586
Bootstrap-t UCL	267123.072
Hall's Bootstrap UCL	350916.773
Percentile Bootstrap UCL	150154.031
BCA Bootstrap UCL	192256.888
95% Chebyshev (Mean, Sd) UCL	243439.636
97.5% Chebyshev (Mean, Sd) UCL	313957.654
99% Chebyshev (Mean, Sd) UCL	452476.646

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
Indeno(1,2,3-c,d)pyrene

Raw Statistics	
Number of Valid Samples	102
Number of Unique Samples	51
Minimum	0.11
Maximum	100000
Mean	3435.305
Median	152.5
Standard Deviation	12055.5904
Variance	145337259
Coefficient of Variation	3.5093217
Skewness	6.02350484

Gamma Statistics	
k hat	0.23708966
k star (bias corrected)	0.23665238
Theta hat	14489.4761
Theta star	14516.2493
nu hat	48.3662911
nu star	48.2770864
Approx.Chi Square Value (.05)	33.325814
Adjusted Level of Significance	0.04764706
Adjusted Chi Square Value	33.1480864

Log-transformed Statistics	
Minimum of log data	-2.20727491
Maximum of log data	11.5129255
Mean of log data	5.12034117
Standard Deviation of log data	2.52026574
Variance of log data	6.35173941

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are micrograms per kilogram (µg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.38784273
Lilliefors 5% Critical Value	0.08772707
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	5416.9118

Gamma Distribution Test	
A-D Test Statistic	9.06655717
A-D 5% Critical Value	0.89577003
K-S Test Statistic	0.22358559
K-S 5% Critical Value	0.09777128
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	4976.5181
Adjusted Gamma UCL	5003.20032

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.16274335
Lilliefors 5% Critical Value	0.08772707
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	10775.0093
95% Chebyshev (MVUE) UCL	10318.412
97.5% Chebyshev (MVUE) UCL	13245.1486
99% Chebyshev (MVUE) UCL	18994.156

95% Non-parametric UCLs	
CLT UCL	5398.73605
Adj-CLT UCL (Adjusted for skewness)	6159.44406
Mod-t UCL (Adjusted for skewness)	5535.56688
Jackknife UCL	5416.9118
Standard Bootstrap UCL	5342.58579
Bootstrap-t UCL	7884.17795
Hall's Bootstrap UCL	11329.6251
Percentile Bootstrap UCL	5667.09314
BCA Bootstrap UCL	6391.32353
95% Chebyshev (Mean, Sd) UCL	8638.44124
97.5% Chebyshev (Mean, Sd) UCL	10889.8425
99% Chebyshev (Mean, Sd) UCL	15312.2842

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
Benzo(b)fluoranthene

Raw Statistics	
Number of Valid Samples	102
Number of Unique Samples	52
Minimum	0.11
Maximum	410000
Mean	13028.2805
Median	225
Standard Deviation	51339.5281
Variance	2635747147
Coefficient of Variation	3.94062195
Skewness	5.85351393

Gamma Statistics	
k hat	0.20235075
k star (bias corrected)	0.20293521
Theta hat	64384.6408
Theta star	64199.2125
nu hat	41.2795534
nu star	41.3987823
Approx.Chi Square Value (.05)	27.6492801
Adjusted Level of Significance	0.04764706
Adjusted Chi Square Value	27.4884

Log-transformed Statistics	
Minimum of log data	-2.20727491
Maximum of log data	12.9239124
Mean of log data	5.84465178
Standard Deviation of log data	2.81601405
Variance of log data	7.92993511

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are micrograms per kilogram (µg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.39983865
Lilliefors 5% Critical Value	0.08772707
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	21467.084

Gamma Distribution Test	
A-D Test Statistic	9.35448051
A-D 5% Critical Value	0.91129728
K-S Test Statistic	0.22627139
K-S 5% Critical Value	0.09846168
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	19507.016
Adjusted Gamma UCL	19621.1837

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.16219699
Lilliefors 5% Critical Value	0.08772707
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	61158.3514
95% Chebyshev (MVUE) UCL	49175.2176
97.5% Chebyshev (MVUE) UCL	63842.1004
99% Chebyshev (MVUE) UCL	92652.3514

95% Non-parametric UCLs	
CLT UCL	21389.6813
Adj-CLT UCL (Adjusted for skewness)	24537.7832
Mod-t UCL (Adjusted for skewness)	21958.1243
Jackknife UCL	21467.084
Standard Bootstrap UCL	21279.0204
Bootstrap-t UCL	31696.5218
Hall's Bootstrap UCL	25173.9592
Percentile Bootstrap UCL	21964.7543
BCA Bootstrap UCL	25633.1089
95% Chebyshev (Mean, Sd) UCL	35186.1799
97.5% Chebyshev (Mean, Sd) UCL	44773.9207
99% Chebyshev (Mean, Sd) UCL	63607.181

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
Benzo(a)anthracene

Raw Statistics	
Number of Valid Samples	102
Number of Unique Samples	60
Minimum	0.11
Maximum	520000
Mean	14629.1187
Median	185
Standard Deviation	61110.6485
Variance	3734511365
Coefficient of Variation	4.17732945
Skewness	6.50750966

Gamma Statistics	
k hat	0.18974593
k star (bias corrected)	0.19070112
Theta hat	77098.4572
Theta star	76712.2859
nu hat	38.7081704
nu star	38.9030282
Approx.Chi Square Value (.05)	25.6142916
Adjusted Level of Significance	0.04764706
Adjusted Chi Square Value	25.4598639

Log-transformed Statistics	
Minimum of log data	-2.20727491
Maximum of log data	13.1615841
Mean of log data	5.68051942
Standard Deviation of log data	2.90296804
Variance of log data	8.42722343

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are micrograms per kilogram (µg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.41583461
Lilliefors 5% Critical Value	0.08772707
Data not normal at 5% significance level	
95% UCL (Assuming Normal Distribution)	
Student's-t UCL	24674.0251

Gamma Distribution Test	
A-D Test Statistic	10.1711132
A-D 5% Critical Value	0.92349782
K-S Test Statistic	0.22070527
K-S 5% Critical Value	0.09891821
Data do not follow gamma distribution	
at 5% significance level	
95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	22218.7295
Adjusted Gamma UCL	22353.498

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.16999402
Lilliefors 5% Critical Value	0.08772707
Data not lognormal at 5% significance level	
95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	71394.5492
95% Chebyshev (MVUE) UCL	53993.4165
97.5% Chebyshev (MVUE) UCL	70299.4863
99% Chebyshev (MVUE) UCL	102329.603

95% Non-parametric UCLs	
CLT UCL	24581.8909
Adj-CLT UCL (Adjusted for skewness)	28747.8208
Mod-t UCL (Adjusted for skewness)	25323.826
Jackknife UCL	24674.0251
Standard Bootstrap UCL	24587.4167
Bootstrap-t UCL	36041.1992
Hall's Bootstrap UCL	50235.7273
Percentile Bootstrap UCL	25548.1982
BCA Bootstrap UCL	30773.2658
95% Chebyshev (Mean, Sd) UCL	41004.1879
97.5% Chebyshev (Mean, Sd) UCL	52416.7014
99% Chebyshev (Mean, Sd) UCL	74834.3743

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
1,3,5 Trimethylbenzene

Raw Statistics	
Number of Valid Samples	103
Number of Unique Samples	42
Minimum	7.9
Maximum	560000
Mean	6883.45049
Median	8
Standard Deviation	55232.5551
Variance	3050635142
Coefficient of Variation	8.02396345
Skewness	10.040856

Gamma Statistics	
k hat	0.15774659
k star (bias corrected)	0.15962452
Theta hat	43636.1292
Theta star	43122.7642
nu hat	32.4957971
nu star	32.8826509
Approx.Chi Square Value (.05)	20.771109
Adjusted Level of Significance	0.0476699
Adjusted Chi Square Value	20.6345063

Log-transformed Statistics	
Minimum of log data	2.06686276
Maximum of log data	13.2356921
Mean of log data	4.00040688
Standard Deviation of log data	2.69598699
Variance of log data	7.26834583

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are micrograms per kilogram (µg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.46216997
Lilliefors 5% Critical Value	0.08730017
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	15917.162

Gamma Distribution Test	
A-D Test Statistic	18.5814806
A-D 5% Critical Value	0.95865705
K-S Test Statistic	0.30005855
K-S 5% Critical Value	0.09991481
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	10897.1601
Adjusted Gamma UCL	10969.3005

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.31532909
Lilliefors 5% Critical Value	0.08730017
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	6288.11897
95% Chebyshev (MVUE) UCL	5490.77338
97.5% Chebyshev (MVUE) UCL	7096.09623
99% Chebyshev (MVUE) UCL	10249.4421

95% Non-parametric UCLs	
CLT UCL	15835.1146
Adj-CLT UCL (Adjusted for skewness)	21588.3092
Mod-t UCL (Adjusted for skewness)	16814.544
Jackknife UCL	15917.162
Standard Bootstrap UCL	15736.0769
Bootstrap-t UCL	107719.153
Hall's Bootstrap UCL	78289.1572
Percentile Bootstrap UCL	17320.232
BCA Bootstrap UCL	27860.5282
95% Chebyshev (Mean, Sd) UCL	30605.5609
97.5% Chebyshev (Mean, Sd) UCL	40870.137
99% Chebyshev (Mean, Sd) UCL	61032.9092

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
Lead

Raw Statistics	
Number of Valid Samples	103
Number of Unique Samples	80
Minimum	1.95
Maximum	4000
Mean	90.4841748
Median	12
Standard Deviation	401.369119
Variance	161097.17
Coefficient of Variation	4.43579355
Skewness	9.26783713

Gamma Statistics	
k hat	0.40483978
k star (bias corrected)	0.39952082
Theta hat	223.506136
Theta star	226.48175
nu hat	83.3969946
nu star	82.3012892
Approx.Chi Square Value (.05)	62.3910171
Adjusted Level of Significance	0.0476699
Adjusted Chi Square Value	62.1460266

Log-transformed Statistics	
Minimum of log data	0.66782937
Maximum of log data	8.29404964
Mean of log data	2.88289228
Standard Deviation of log data	1.54044012
Variance of log data	2.37295577

RECOMMENDATION

Data are Non-parametric (0.05)

Use 97.5% Chebyshev (Mean, Sd) UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.4127096
Lilliefors 5% Critical Value	0.08730017
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	156.131198

Gamma Distribution Test	
A-D Test Statistic	8.58089262
A-D 5% Critical Value	0.84189085
K-S Test Statistic	0.22422141
K-S 5% Critical Value	0.09495711
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	119.359558
Adjusted Gamma UCL	119.830094

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.11877746
Lilliefors 5% Critical Value	0.08730017
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	89.07606
95% Chebyshev (MVUE) UCL	110.160205
97.5% Chebyshev (MVUE) UCL	133.115702
99% Chebyshev (MVUE) UCL	178.207331

95% Non-parametric UCLs	
CLT UCL	155.534969
Adj-CLT UCL (Adjusted for skewness)	194.124147
Mod-t UCL (Adjusted for skewness)	162.15033
Jackknife UCL	156.131198
Standard Bootstrap UCL	154.313773
Bootstrap-t UCL	341.309599
Hall's Bootstrap UCL	374.005873
Percentile Bootstrap UCL	165.560194
BCA Bootstrap UCL	209.737087
95% Chebyshev (Mean, Sd) UCL	262.870235
97.5% Chebyshev (Mean, Sd) UCL	337.46182
99% Chebyshev (Mean, Sd) UCL	483.982547

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
Arsenic

Raw Statistics	
Number of Valid Samples	103
Number of Unique Samples	67
Minimum	0.325
Maximum	23.4
Mean	4.2968932
Median	2.6
Standard Deviation	4.7711225
Variance	22.7636099
Coefficient of Variation	1.11036562
Skewness	2.02998779

Gamma Statistics	
k hat	1.10320604
k star (bias corrected)	1.07754632
Theta hat	3.89491449
Theta star	3.98766449
nu hat	227.260445
nu star	221.974542
Approx.Chi Square Value (.05)	188.484257
Adjusted Level of Significance	0.0476699
Adjusted Chi Square Value	188.049638

Log-transformed Statistics	
Minimum of log data	-1.1239301
Maximum of log data	3.15273602
Mean of log data	0.94050246
Standard Deviation of log data	1.05005753
Variance of log data	1.10262083

RECOMMENDATION

Data are lognormal (0.05)

Use H-UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.24880911
Lilliefors 5% Critical Value	0.08730017
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	5.07724718

Gamma Distribution Test	
A-D Test Statistic	1.94611803
A-D 5% Critical Value	0.78026484
K-S Test Statistic	0.13573728
K-S 5% Critical Value	0.09113771
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	5.06037435
Adjusted Gamma UCL	5.07206985

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.06502621
Lilliefors 5% Critical Value	0.08730017
Data are lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	5.61609152
95% Chebyshev (MVUE) UCL	6.82986355
97.5% Chebyshev (MVUE) UCL	7.87740173
99% Chebyshev (MVUE) UCL	9.93508764

95% Non-parametric UCLs	
CLT UCL	5.07015973
Adj-CLT UCL (Adjusted for skewness)	5.17063454
Mod-t UCL (Adjusted for skewness)	5.09291922
Jackknife UCL	5.07724718
Standard Bootstrap UCL	5.06576353
Bootstrap-t UCL	5.2547403
Hall's Bootstrap UCL	5.19658575
Percentile Bootstrap UCL	5.12451456
BCA Bootstrap UCL	5.23873786
95% Chebyshev (Mean, Sd) UCL	6.34606682
97.5% Chebyshev (Mean, Sd) UCL	7.23274588
99% Chebyshev (Mean, Sd) UCL	8.9744552

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
Benzo(a)pyrene

Raw Statistics	
Number of Valid Samples	104
Number of Unique Samples	58
Minimum	0.11
Maximum	340000
Mean	12269.0684
Median	185
Standard Deviation	45893.2982
Variance	2106194818
Coefficient of Variation	3.74056911
Skewness	5.3295129

Gamma Statistics	
k hat	0.19962646
k star (bias corrected)	0.20027826
Theta hat	61460.1314
Theta star	61260.1109
nu hat	41.5223033
nu star	41.6578779
Approx.Chi Square Value (.05)	27.8613553
Adjusted Level of Significance	0.04769231
Adjusted Chi Square Value	27.7029764

Log-transformed Statistics	
Minimum of log data	-2.20727491
Maximum of log data	12.7367009
Mean of log data	5.72727454
Standard Deviation of log data	2.87575935
Variance of log data	8.26999184

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are micrograms per kilogram (µg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.39460506
Lilliefors 5% Critical Value	0.08687945
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	19738.4324

Gamma Distribution Test	
A-D Test Statistic	9.35173076
A-D 5% Critical Value	0.91279828
K-S Test Statistic	0.2203425
K-S 5% Critical Value	0.09795179
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	18344.5258
Adjusted Gamma UCL	18449.4021

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.16018748
Lilliefors 5% Critical Value	0.08687945
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	66678.8633
95% Chebyshev (MVUE) UCL	52106.7409
97.5% Chebyshev (MVUE) UCL	67752.5165
99% Chebyshev (MVUE) UCL	98485.613

95% Non-parametric UCLs	
CLT UCL	19671.252
Adj-CLT UCL (Adjusted for skewness)	22184.2021
Mod-t UCL (Adjusted for skewness)	20130.4019
Jackknife UCL	19738.4324
Standard Bootstrap UCL	19512.4402
Bootstrap-t UCL	27003.4589
Hall's Bootstrap UCL	22023.7207
Percentile Bootstrap UCL	20243.6154
BCA Bootstrap UCL	23185.5406
95% Chebyshev (Mean, Sd) UCL	31885.0209
97.5% Chebyshev (Mean, Sd) UCL	40372.8592
99% Chebyshev (Mean, Sd) UCL	57045.574

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
Benzene

Raw Statistics	
Number of Valid Samples	104
Number of Unique Samples	51
Minimum	2.5
Maximum	230000
Mean	8403.40865
Median	21
Standard Deviation	35849.7272
Variance	1285202937
Coefficient of Variation	4.26609351
Skewness	5.38434468

Gamma Statistics	
k hat	0.16547953
k star (bias corrected)	0.16711633
Theta hat	50782.1657
Theta star	50284.7835
nu hat	34.4197412
nu star	34.7601974
Approx.Chi Square Value (.05)	22.2706254
Adjusted Level of Significance	0.04769231
Adjusted Chi Square Value	22.1301791

Log-transformed Statistics	
Minimum of log data	0.91629073
Maximum of log data	12.3458346
Mean of log data	4.45856292
Standard Deviation of log data	3.01719981
Variance of log data	9.10349468

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are micrograms per kilogram (µg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.4201761
Lilliefors 5% Critical Value	0.08687945
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	14238.1312

Gamma Distribution Test	
A-D Test Statistic	14.3061145
A-D 5% Critical Value	0.95048666
K-S Test Statistic	0.27288784
K-S 5% Critical Value	0.09932218
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	13116.1177
Adjusted Gamma UCL	13199.3574

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.23331177
Lilliefors 5% Critical Value	0.08687945
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	31955.0551
95% Chebyshev (MVUE) UCL	22478.0629
97.5% Chebyshev (MVUE) UCL	29356.9151
99% Chebyshev (MVUE) UCL	42869.0882

95% Non-parametric UCLs	
CLT UCL	14185.653
Adj-CLT UCL (Adjusted for skewness)	16168.8497
Mod-t UCL (Adjusted for skewness)	14547.4698
Jackknife UCL	14238.1312
Standard Bootstrap UCL	14290.2354
Bootstrap-t UCL	24385.2601
Hall's Bootstrap UCL	13317.0997
Percentile Bootstrap UCL	14816.9856
BCA Bootstrap UCL	17251.0529
95% Chebyshev (Mean, Sd) UCL	23726.4857
97.5% Chebyshev (Mean, Sd) UCL	30356.7934
99% Chebyshev (Mean, Sd) UCL	43380.749

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
Napthalene

Raw Statistics	
Number of Valid Samples	123
Number of Unique Samples	69
Minimum	0.24
Maximum	2900000
Mean	121182.88
Median	155
Standard Deviation	438376.525
Variance	1.92E+11
Coefficient of Variation	3.61747901
Skewness	4.38812972

Gamma Statistics	
k hat	0.13908531
k star (bias corrected)	0.14111304
Theta hat	871284.508
Theta star	858764.551
nu hat	34.2149874
nu star	34.7138089
Approx.Chi Square Value (.05)	22.2334506
Adjusted Level of Significance	0.04804878
Adjusted Chi Square Value	22.1151148

Log-transformed Statistics	
Minimum of log data	-1.42711636
Maximum of log data	14.8802213
Mean of log data	6.1187798
Standard Deviation of log data	3.68062326
Variance of log data	13.5469876

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are micrograms per kilogram (µg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.43710762
Lilliefors 5% Critical Value	0.07988793
Data not normal at 5% significance level	
95% UCL (Assuming Normal Distribution)	
Student's-t UCL	186696.613

Gamma Distribution Test	
A-D Test Statistic	14.6354677
A-D 5% Critical Value	0.98944431
K-S Test Statistic	0.2587821
K-S 5% Critical Value	0.09504729
Data do not follow gamma distribution	
at 5% significance level	
95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	189206.768
Adjusted Gamma UCL	190219.195

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.14719929
Lilliefors 5% Critical Value	0.07988793
Data not lognormal at 5% significance level	
95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	2385481.8
95% Chebyshev (MVUE) UCL	1074901.33
97.5% Chebyshev (MVUE) UCL	1421954.66
99% Chebyshev (MVUE) UCL	2103673.74

95% Non-parametric UCLs	
CLT UCL	186199.141
Adj-CLT UCL (Adjusted for skewness)	202910.126
Mod-t UCL (Adjusted for skewness)	189303.189
Jackknife UCL	186696.613
Standard Bootstrap UCL	186152.866
Bootstrap-t UCL	213473.412
Hall's Bootstrap UCL	197999.395
Percentile Bootstrap UCL	191306.097
BCA Bootstrap UCL	205520.671
95% Chebyshev (Mean, Sd) UCL	293477.428
97.5% Chebyshev (Mean, Sd) UCL	368029.416
99% Chebyshev (Mean, Sd) UCL	514472.361

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
Thallium

Raw Statistics	
Number of Valid Samples	86
Number of Unique Samples	15
Minimum	0.485
Maximum	1.8
Mean	0.7827907
Median	0.8
Standard Deviation	0.19129528
Variance	0.03659389
Coefficient of Variation	0.24437603
Skewness	2.26028677

Gamma Statistics	
k hat	20.3037995
k star (bias corrected)	19.6032794
Theta hat	0.0385539
Theta star	0.03993162
nu hat	3492.25352
nu star	3371.76406
Approx.Chi Square Value (.05)	3237.80829
Adjusted Level of Significance	0.0472093
Adjusted Chi Square Value	3235.59545

Log-transformed Statistics	
Minimum of log data	-0.72360639
Maximum of log data	0.58778666
Mean of log data	-0.26971796
Standard Deviation of log data	0.21797996
Variance of log data	0.04751526

RECOMMENDATION

Data are Non-parametric (0.05)

Use Student's-t UCL

All units are milligrams per kilogram (mg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.13289503
Lilliefors 5% Critical Value	0.09553984
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	0.81709445

Gamma Distribution Test	
A-D Test Statistic	1.17369572
A-D 5% Critical Value	0.75002122
K-S Test Statistic	0.10682634
K-S 5% Critical Value	0.09611176
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	0.81517659
Adjusted Gamma UCL	0.8157341

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.10002258
Lilliefors 5% Critical Value	0.09553984
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	0.81416722
95% Chebyshev (MVUE) UCL	0.8626845
97.5% Chebyshev (MVUE) UCL	0.89771257
99% Chebyshev (MVUE) UCL	0.96651845

95% Non-parametric UCLs	
CLT UCL	0.81672058
Adj-CLT UCL (Adjusted for skewness)	0.82209275
Mod-t UCL (Adjusted for skewness)	0.8179324
Jackknife UCL	0.81709445
Standard Bootstrap UCL	0.81668538
Bootstrap-t UCL	0.82629393
Hall's Bootstrap UCL	0.83222823
Percentile Bootstrap UCL	0.81843023
BCA Bootstrap UCL	0.82348837
95% Chebyshev (Mean, Sd) UCL	0.87270563
97.5% Chebyshev (Mean, Sd) UCL	0.9116119
99% Chebyshev (Mean, Sd) UCL	0.98803572

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
Fluorene

Raw Statistics	
Number of Valid Samples	101
Number of Unique Samples	48
Minimum	1.5
Maximum	1200000
Mean	25354.6386
Median	80
Standard Deviation	127257.536
Variance	1.62E+10
Coefficient of Variation	5.01910271
Skewness	8.23418037

Gamma Statistics	
k hat	0.16241625
k star (bias corrected)	0.16419267
Theta hat	156108.997
Theta star	154420.04
nu hat	32.8080835
nu star	33.1669193
Approx.Chi Square Value (.05)	20.9974529
Adjusted Level of Significance	0.04762376
Adjusted Chi Square Value	20.8572809

Log-transformed Statistics	
Minimum of log data	0.40546511
Maximum of log data	13.9978321
Mean of log data	5.46354438
Standard Deviation of log data	3.09704847
Variance of log data	9.59170921

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are micrograms per kilogram (µg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.42503871
Lilliefors 5% Critical Value	0.0881603
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	46377.515

Gamma Distribution Test	
A-D Test Statistic	12.7259191
A-D 5% Critical Value	0.95288855
K-S Test Statistic	0.26044619
K-S 5% Critical Value	0.10029315
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	40049.3933
Adjusted Gamma UCL	40318.5466

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.19437959
Lilliefors 5% Critical Value	0.0881603
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	122270.608
95% Chebyshev (MVUE) UCL	78650.6857
97.5% Chebyshev (MVUE) UCL	103017.828
99% Chebyshev (MVUE) UCL	150882.36

95% Non-parametric UCLs	
CLT UCL	46182.759
Adj-CLT UCL (Adjusted for skewness)	57268.4539
Mod-t UCL (Adjusted for skewness)	48106.6594
Jackknife UCL	46377.515
Standard Bootstrap UCL	45877.4788
Bootstrap-t UCL	88351.3758
Hall's Bootstrap UCL	112757.117
Percentile Bootstrap UCL	48005.0891
BCA Bootstrap UCL	63005.4307
95% Chebyshev (Mean, Sd) UCL	80549.6239
97.5% Chebyshev (Mean, Sd) UCL	104432.538
99% Chebyshev (Mean, Sd) UCL	151345.899

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
Acenaphthene

Raw Statistics	
Number of Valid Samples	101
Number of Unique Samples	46
Minimum	0.59
Maximum	670000
Mean	21781.7831
Median	80
Standard Deviation	93551.8787
Variance	8751954002
Coefficient of Variation	4.29495962
Skewness	5.53385981

Gamma Statistics	
k hat	0.16441975
k star (bias corrected)	0.16613665
Theta hat	132476.687
Theta star	131107.633
nu hat	33.2127885
nu star	33.5596034
Approx.Chi Square Value (.05)	21.3105329
Adjusted Level of Significance	0.04762376
Adjusted Chi Square Value	21.1692374

Log-transformed Statistics	
Minimum of log data	-0.52763274
Maximum of log data	13.415033
Mean of log data	5.37707325
Standard Deviation of log data	3.0512915
Variance of log data	9.31037981

Normal Distribution Test	
Lilliefors Test Statisitic	0.4129247
Lilliefors 5% Critical Value	0.0881603
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	37236.5031

Gamma Distribution Test	
A-D Test Statistic	13.0802753
A-D 5% Critical Value	0.95072643
K-S Test Statistic	0.28742878
K-S 5% Critical Value	0.10021325
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	34301.7232
Adjusted Gamma UCL	34530.6723

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.18677118
Lilliefors 5% Critical Value	0.0881603
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	93617.3933
95% Chebyshev (MVUE) UCL	62589.7253
97.5% Chebyshev (MVUE) UCL	81875.941
99% Chebyshev (MVUE) UCL	119759.977

95% Non-parametric UCLs	
CLT UCL	37093.3305
Adj-CLT UCL (Adjusted for skewness)	42570.2922
Mod-t UCL (Adjusted for skewness)	38090.7984
Jackknife UCL	37236.5031
Standard Bootstrap UCL	36417.4189
Bootstrap-t UCL	53172.7999
Hall's Bootstrap UCL	38031.1665
Percentile Bootstrap UCL	38611.2187
BCA Bootstrap UCL	45844.5207
95% Chebyshev (Mean, Sd) UCL	62357.7265
97.5% Chebyshev (Mean, Sd) UCL	79914.9697
99% Chebyshev (Mean, Sd) UCL	114402.774

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are micrograms per kilogram (µg/kg).

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
Fluoranthene

Raw Statistics	
Number of Valid Samples	103
Number of Unique Samples	59
Minimum	0.11
Maximum	1400000
Mean	36737.3457
Median	310
Standard Deviation	166074.145
Variance	2.76E+10
Coefficient of Variation	4.52058094
Skewness	6.64112099

Gamma Statistics	
k hat	0.16959013
k star (bias corrected)	0.1711231
Theta hat	216624.318
Theta star	214683.73
nu hat	34.9355664
nu star	35.2513589
Approx.Chi Square Value (.05)	22.6646114
Adjusted Level of Significance	0.0476699
Adjusted Chi Square Value	22.521433

Log-transformed Statistics	
Minimum of log data	-2.20727491
Maximum of log data	14.1519828
Mean of log data	6.06107056
Standard Deviation of log data	3.11724059
Variance of log data	9.7171889

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are micrograms per kilogram (µg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.42880139
Lilliefors 5% Critical Value	0.08730017
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	63900.0563

Gamma Distribution Test	
A-D Test Statistic	10.9288739
A-D 5% Critical Value	0.94568205
K-S Test Statistic	0.24019213
K-S 5% Critical Value	0.09944048
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	57139.3587
Adjusted Gamma UCL	57502.6181

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.16374744
Lilliefors 5% Critical Value	0.08730017
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	236852.885
95% Chebyshev (MVUE) UCL	152281.224
97.5% Chebyshev (MVUE) UCL	199490.766
99% Chebyshev (MVUE) UCL	292224.769

95% Non-parametric UCLs	
CLT UCL	63653.355
Adj-CLT UCL (Adjusted for skewness)	75094.951
Mod-t UCL (Adjusted for skewness)	65684.7141
Jackknife UCL	63900.0563
Standard Bootstrap UCL	63620.3118
Bootstrap-t UCL	106612.82
Hall's Bootstrap UCL	155213.696
Percentile Bootstrap UCL	66805.37
BCA Bootstrap UCL	79026.2233
95% Chebyshev (Mean, Sd) UCL	108065.373
97.5% Chebyshev (Mean, Sd) UCL	138929.068
99% Chebyshev (Mean, Sd) UCL	199554.819

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
Phenanthrene

Raw Statistics	
Number of Valid Samples	104
Number of Unique Samples	62
Minimum	5.4
Maximum	3700000
Mean	79449.124
Median	210
Standard Deviation	397946.57
Variance	1.58E+11
Coefficient of Variation	5.00882263
Skewness	7.83018993

Gamma Statistics	
k hat	0.15149008
k star (bias corrected)	0.15353043
Theta hat	524451.012
Theta star	517481.294
nu hat	31.509936
nu star	31.9343289
Approx.Chi Square Value (.05)	20.0178796
Adjusted Level of Significance	0.04769231
Adjusted Chi Square Value	19.8852839

Log-transformed Statistics	
Minimum of log data	1.68639895
Maximum of log data	15.1238434
Mean of log data	6.21669134
Standard Deviation of log data	3.29835212
Variance of log data	10.8791267

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are micrograms per kilogram (µg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.43655719
Lilliefors 5% Critical Value	0.08687945
Data not normal at 5% significance level	

95% UCL (Assuming Normal Distribution)	
Student's-t UCL	144216.921

Gamma Distribution Test	
A-D Test Statistic	12.3185734
A-D 5% Critical Value	0.96592698
K-S Test Statistic	0.25674116
K-S 5% Critical Value	0.0998836
Data do not follow gamma distribution	
at 5% significance level	

95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	126744.416
Adjusted Gamma UCL	127589.551

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.13946337
Lilliefors 5% Critical Value	0.08687945
Data not lognormal at 5% significance level	

95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	576899.85
95% Chebyshev (MVUE) UCL	317464.384
97.5% Chebyshev (MVUE) UCL	417735.394
99% Chebyshev (MVUE) UCL	614698.375

95% Non-parametric UCLs	
CLT UCL	143634.391
Adj-CLT UCL (Adjusted for skewness)	175648.701
Mod-t UCL (Adjusted for skewness)	149210.506
Jackknife UCL	144216.921
Standard Bootstrap UCL	143814.545
Bootstrap-t UCL	268689.36
Hall's Bootstrap UCL	355687.791
Percentile Bootstrap UCL	148754.409
BCA Bootstrap UCL	199131.017
95% Chebyshev (Mean, Sd) UCL	249541.519
97.5% Chebyshev (Mean, Sd) UCL	323140.634
99% Chebyshev (Mean, Sd) UCL	467711.845

Attachment B2
ProUCL Summary Sheet
Residential Receptor Exposed to Surface Soil
Pyrene

Raw Statistics	
Number of Valid Samples	104
Number of Unique Samples	65
Minimum	0.11
Maximum	2000000
Mean	49180.2174
Median	345
Standard Deviation	223506.869
Variance	5.00E+10
Coefficient of Variation	4.54464987
Skewness	7.1505907

Gamma Statistics	
k hat	0.16698555
k star (bias corrected)	0.16857892
Theta hat	294517.797
Theta star	291734.089
nu hat	34.7329952
nu star	35.0644152
Approx.Chi Square Value (.05)	22.514573
Adjusted Level of Significance	0.04769231
Adjusted Chi Square Value	22.3733005

Log-transformed Statistics	
Minimum of log data	-2.20727491
Maximum of log data	14.5086577
Mean of log data	6.2728456
Standard Deviation of log data	3.26273151
Variance of log data	10.6454169

RECOMMENDATION

Data are Non-parametric (0.05)

Use 99% Chebyshev (Mean, Sd) UCL

All units are micrograms per kilogram (µg/kg).

Normal Distribution Test	
Lilliefors Test Statisitic	0.41702997
Lilliefors 5% Critical Value	0.08687945
Data not normal at 5% significance level	
95% UCL (Assuming Normal Distribution)	
Student's-t UCL	85557.0793

Gamma Distribution Test	
A-D Test Statistic	9.91793671
A-D 5% Critical Value	0.94882444
K-S Test Statistic	0.22202217
K-S 5% Critical Value	0.09926174
Data do not follow gamma distribution	
at 5% significance level	
95% UCLs (Assuming Gamma Distribution)	
Approximate Gamma UCL	76593.7495
Adjusted Gamma UCL	77077.3879

Lognormal Distribution Test	
Lilliefors Test Statisitic	0.15775445
Lilliefors 5% Critical Value	0.08687945
Data not lognormal at 5% significance level	
95% UCLs (Assuming Lognormal Distribution)	
95% H-UCL	525527.201
95% Chebyshev (MVUE) UCL	299162.511
97.5% Chebyshev (MVUE) UCL	393313.477
99% Chebyshev (MVUE) UCL	578254.819

95% Non-parametric UCLs	
CLT UCL	85229.9013
Adj-CLT UCL (Adjusted for skewness)	101650.155
Mod-t UCL (Adjusted for skewness)	88118.3071
Jackknife UCL	85557.0793
Standard Bootstrap UCL	85601.6533
Bootstrap-t UCL	132906.312
Hall's Bootstrap UCL	199388.733
Percentile Bootstrap UCL	89491.0577
BCA Bootstrap UCL	110803.539
95% Chebyshev (Mean, Sd) UCL	144712.687
97.5% Chebyshev (Mean, Sd) UCL	186049.663
99% Chebyshev (Mean, Sd) UCL	267248.148

Attachment C

Toxicological Profiles

Attachment C1

Oak Ridge National Laboratory
Toxicological Profiles

TOXICOLOGICAL PROFILE FOR CESIUM

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry

April 2004

DISCLAIMER

The use of company or product name(s) is for identification only and does not imply endorsement by the Agency for Toxic Substances and Disease Registry.

UPDATE STATEMENT

A Toxicological Profile for Cesium, Draft for Public Comment was released in July 2001. This edition supersedes any previously released draft or final profile.

Toxicological profiles are revised and republished as necessary. For information regarding the update status of previously released profiles, contact ATSDR at:

Agency for Toxic Substances and Disease Registry
Division of Toxicology/Toxicology Information Branch
1600 Clifton Road NE,
Mailstop E-32
Atlanta, Georgia 30333

FOREWORD

This toxicological profile is prepared in accordance with guidelines* developed by the Agency for Toxic Substances and Disease Registry (ATSDR) and the Environmental Protection Agency (EPA). The original guidelines were published in the *Federal Register* on April 17, 1987. Each profile will be revised and republished as necessary.

The ATSDR toxicological profile succinctly characterizes the toxicologic and adverse health effects information for the hazardous substance described therein. Each peer-reviewed profile identifies and reviews the key literature that describes a hazardous substance's toxicologic properties. Other pertinent literature is also presented, but is described in less detail than the key studies. The profile is not intended to be an exhaustive document; however, more comprehensive sources of specialty information are referenced.

The focus of the profiles is on health and toxicologic information; therefore, each toxicological profile begins with a public health statement that describes, in nontechnical language, a substance's relevant toxicological properties. Following the public health statement is information concerning levels of significant human exposure and, where known, significant health effects. The adequacy of information to determine a substance's health effects is described in a health effects summary. Data needs that are of significance to protection of public health are identified by ATSDR and EPA.

Each profile includes the following:

(A) The examination, summary, and interpretation of available toxicologic information and epidemiologic evaluations on a hazardous substance to ascertain the levels of significant human exposure for the substance and the associated acute, subacute, and chronic health effects;

(B) A determination of whether adequate information on the health effects of each substance is available or in the process of development to determine levels of exposure that present a significant risk to human health of acute, subacute, and chronic health effects; and

(C) Where appropriate, identification of toxicologic testing needed to identify the types or levels of exposure that may present significant risk of adverse health effects in humans.

The principal audiences for the toxicological profiles are health professionals at the Federal, State, and local levels; interested private sector organizations and groups; and members of the public.

This profile reflects ATSDR's assessment of all relevant toxicologic testing and information that has been peer-reviewed. Staff of the Centers for Disease Control and Prevention and other Federal scientists have also reviewed the profile. In addition, this profile has been peer-reviewed by a nongovernmental panel and was made available for public review. Final responsibility for the contents and views expressed in this toxicological profile resides with ATSDR.

Julie Louise Gerberding, M.D., M.P.H.
Administrator
Agency for Toxic Substances and
Disease Registry

Background Information

The toxicological profiles are developed by ATSDR pursuant to Section 104(i) (3) and (5) of the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA or Superfund) for hazardous substances found at Department of Energy (DOE) waste sites. CERCLA directs ATSDR to prepare toxicological profiles for hazardous substances most commonly found at facilities on the CERCLA National Priorities List (NPL) and that pose the most significant potential threat to human health, as determined by ATSDR and the EPA. ATSDR and DOE entered into a Memorandum of Understanding on November 4, 1992 which provided that ATSDR would prepare toxicological profiles for hazardous substances based upon ATSDR's or DOE's identification of need. The current ATSDR priority list of hazardous substances at DOE NPL sites was announced in the Federal Register on July 24, 1996 (61 FR 38451).

QUICK REFERENCE FOR HEALTH CARE PROVIDERS

Toxicological Profiles are a unique compilation of toxicological information on a given hazardous substance. Each profile reflects a comprehensive and extensive evaluation, summary, and interpretation of available toxicologic and epidemiologic information on a substance. Health care providers treating patients potentially exposed to hazardous substances will find the following information helpful for fast answers to often-asked questions.

Primary Chapters/Sections of Interest

Chapter 1: Public Health Statement: The Public Health Statement can be a useful tool for educating patients about possible exposure to a hazardous substance. It explains a substance's relevant toxicologic properties in a nontechnical, question-and-answer format, and it includes a review of the general health effects observed following exposure.

Chapter 2: Relevance to Public Health: The Relevance to Public Health Section evaluates, interprets, and assesses the significance of toxicity data to human health.

Chapter 3: Health Effects: Specific health effects of a given hazardous compound are reported by type of health effect (death, systemic, immunologic, reproductive), by route of exposure, and by length of exposure (acute, intermediate, and chronic). In addition, both human and animal studies are reported in this section.

NOTE: Not all health effects reported in this section are necessarily observed in the clinical setting. Please refer to the Public Health Statement to identify general health effects observed following exposure.

Pediatrics: Four new sections have been added to each Toxicological Profile to address child health issues:

Section 1.6 How Can (Chemical X) Affect Children?
Section 1.7 How Can Families Reduce the Risk of Exposure to (Chemical X)?
Section 3.8 Children's Susceptibility
Section 6.6 Exposures of Children

Other Sections of Interest:

Section 3.9 Biomarkers of Exposure and Effect
Section 3.12 Methods for Reducing Toxic Effects

ATSDR Information Center

Phone: 1-888-42-ATSDR or (404) 498-0110 ***Fax:*** (770) 488-4178
E-mail: atsdric@cdc.gov ***Internet:*** http://www.atsdr.cdc.gov

The following additional material can be ordered through the ATSDR Information Center:

Case Studies in Environmental Medicine: Taking an Exposure History—The importance of taking an exposure history and how to conduct one are described, and an example of a thorough exposure history is provided. Other case studies of interest include *Reproductive and Developmental Hazards; Skin Lesions and Environmental Exposures; Cholinesterase-Inhibiting Pesticide Toxicity*; and numerous chemical-specific case studies.

Managing Hazardous Materials Incidents is a three-volume set of recommendations for on-scene (prehospital) and hospital medical management of patients exposed during a hazardous materials incident. Volumes I and II are planning guides to assist first responders and hospital emergency department personnel in planning for incidents that involve hazardous materials. Volume III—*Medical Management Guidelines for Acute Chemical Exposures*—is a guide for health care professionals treating patients exposed to hazardous materials.

Fact Sheets (ToxFAQs) provide answers to frequently asked questions about toxic substances.

Other Agencies and Organizations

The National Center for Environmental Health (NCEH) focuses on preventing or controlling disease, injury, and disability related to the interactions between people and their environment outside the workplace. Contact: NCEH, Mailstop F-29, 4770 Buford Highway, NE, Atlanta, GA 30341-3724 • Phone: 770-488-7000 • FAX: 770-488-7015.

The National Institute for Occupational Safety and Health (NIOSH) conducts research on occupational diseases and injuries, responds to requests for assistance by investigating problems of health and safety in the workplace, recommends standards to the Occupational Safety and Health Administration (OSHA) and the Mine Safety and Health Administration (MSHA), and trains professionals in occupational safety and health. Contact: NIOSH, 200 Independence Avenue, SW, Washington, DC 20201 • Phone: 800-356-4674 or NIOSH Technical Information Branch, Robert A. Taft Laboratory, Mailstop C-19, 4676 Columbia Parkway, Cincinnati, OH 45226-1998 • Phone: 800-35-NIOSH.

The National Institute of Environmental Health Sciences (NIEHS) is the principal federal agency for biomedical research on the effects of chemical, physical, and biologic environmental agents on human health and well-being. Contact: NIEHS, PO Box 12233, 104 T.W. Alexander Drive, Research Triangle Park, NC 27709 • Phone: 919-541-3212.

Radiation Emergency Assistance Center/Training Site (REAC/TS) provides support to the U.S. Department of Energy, the World Health Organization, and the International Atomic Energy Agency in the medical management of radiation accidents. A 24-hour emergency response program at the Oak Ridge Institute for Science and Education (ORISE), REAC/TS trains, consults, or assists in the response to all kinds of radiation accidents. Contact: Oak Ridge Institute for Science and Education, REAC/TS, PO Box 117, MS 39, Oak Ridge, TN 37831-0117 • Phone 865-576-3131 • FAX 865-576-9522 • 24-Hour Emergency Phone 865-576-1005 (ask for REAC/TS) • e-mail: cooleyp@orau.gov • website (including emergency medical guidance): http://www.orau.gov/reacts/default.htm

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Referrals

The Association of Occupational and Environmental Clinics (AOEC) has developed a network of clinics in the United States to provide expertise in occupational and environmental issues. Contact: AOEC, 1010 Vermont Avenue, NW, #513, Washington, DC 20005 • Phone: 202-347-4976 • FAX: 202-347-4950 • e-mail: AOEC@AOEC.ORG • Web Page: http://www.aoec.org/.

The American College of Occupational and Environmental Medicine (ACOEM) is an association of physicians and other health care providers specializing in the field of occupational and environmental medicine. Contact: ACOEM, 55 West Seegers Road, Arlington Heights, IL 60005 • Phone: 847-818-1800 • FAX: 847-818-9266.

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THE PROFILE HAS UNDERGONE THE FOLLOWING ATSDR INTERNAL REVIEWS:

1. Health Effects Review. The Health Effects Review Committee examines the health effects chapter of each profile for consistency and accuracy in interpreting health effects and classifying end points.

2. Minimal Risk Level Review. The Minimal Risk Level Workgroup considers issues relevant to substance-specific Minimal Risk Levels (MRLs), reviews the health effects database of each profile, and makes recommendations for derivation of MRLs.

3. Data Needs Review. The Research Implementation Branch reviews data needs sections to assure consistency across profiles and adherence to instructions in the Guidance.

PEER REVIEW

A peer review panel was assembled for cesium. The panel consisted of the following members:

1. Ranjan Bose, M.B.B.S., M.D., New Substances Assessment and Controls Bureau, Health Canada, Ottawa, Ontario, Canada;

2. Darrell Fisher, Ph.D., Senior Scientist, Pacific Northwest National Laboratory, Richland, Washington;

3. Richard Leggett, Ph.D., Life Sciences Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee;

4. Bruce Muggenburg, D.V.M., Ph.D., Senior Scientist and Veterinary Physiologist, Toxicology Division, Lovelace Respiratory Research Institute, Albuquerque, New Mexico;

5. Herman Cember, C.H.P., Ph.D., P.E., Adjunct Professor, School of Health Sciences, Purdue University, Lafayette, Indiana; and

6. Marvin Goldman, Ph.D., Professor Emeritus of Radiation Biology, Department of Surgical and Radiological Sciences, University of California, Davis, California.

These experts collectively have knowledge of cesium's physical and chemical properties, toxicokinetics, key health end points, mechanisms of action, human and animal exposure, and quantification of risk to humans. All reviewers were selected in conformity with the conditions for peer review specified in Section 104(I)(13) of the Comprehensive Environmental Response, Compensation, and Liability Act, as amended.

Scientists from the Agency for Toxic Substances and Disease Registry (ATSDR) have reviewed the peer reviewers' comments and determined which comments will be included in the profile. A listing of the peer reviewers' comments not incorporated in the profile, with a brief explanation of the rationale for their exclusion, exists as part of the administrative record for this compound. A list of databases reviewed and a list of unpublished documents cited are also included in the administrative record.

The citation of the peer review panel should not be understood to imply its approval of the profile's final content. The responsibility for the content of this profile lies with the ATSDR.

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1. PUBLIC HEALTH STATEMENT

This public health statement tells you about cesium and the effects of exposure.

The Environmental Protection Agency (EPA) identifies the most serious hazardous waste sites in the nation. These sites make up the National Priorities List (NPL) and are the sites targeted for long-term federal cleanup activities. Stable (not radioactive) cesium (^{133}Cs) has been identified in at least 8 of the 1,636 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL) (HazDat 2003). It was reported that ^{134}Cs (radioactive) has been found in at least 3 of the 1,636 current or former NPL sites and ^{137}Cs (radioactive) has been detected in at least 23 of the 1,636 current or former NPL sites. However, the number of sites evaluated for cesium is not known. As more sites are evaluated, the sites at which cesium is found may increase. This information is important because exposure to this substance may harm you and because these sites may be sources of exposure.

When a substance is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment. This release does not always lead to exposure. You are exposed to a substance when you come in contact with it. You may be exposed by breathing, eating, or drinking the substance, or by skin contact. If the substance is radioactive, you may also be exposed to radiation if you are near it.

External exposure to radiation may occur from natural or man-made sources. Naturally occurring sources of radiation are cosmic radiation from space or radioactive materials in soil or building materials. Man-made sources of radioactive materials are found in consumer products, industrial equipment, atom bomb fallout, and to a smaller extent from hospital waste, medical devices, and nuclear reactors.

If you are exposed to cesium, many factors determine whether you'll be harmed. These factors include the dose (how much), the duration (how long), and how you come in contact with it. You must also consider the other chemicals you're exposed to and your age, sex, diet, family traits, lifestyle, and state of health.

1.1 WHAT IS CESIUM?

Cesium is a naturally-occurring element found in rocks, soil, and dust at low concentrations. Granites contain an average cesium concentration of about 1 part of cesium in a million parts of granite (ppm) and sedimentary rocks contain about 4 ppm. Natural cesium is present in the environment in only one stable form, as the isotope ^{133}Cs. Pure cesium metal is silvery white in color and very soft, but pure cesium is not expected to be found in the environment. Pure cesium metal reacts violently with air and water, resulting in an explosion-like reaction. Cesium compounds do not react violently with air or water and are generally very soluble in water. The most important source of commercial cesium is a mineral known as pollucite, which usually contains about 5–32% cesium oxide (Cs_2O). No known taste or odor is associated with cesium compounds. Cesium is not mined or produced in the United States and very little is imported from other countries. There are relatively few commercial uses for cesium metal and its compounds. Sometimes cesium is used as a getter for residual gas impurities in vacuum tubes and as a coating in tungsten filaments or cathodes of the tubes. Crystalline cesium iodide and cesium fluoride are used in scintillation counters, which convert energy from ionizing radiation into pulses of visible light for radiation detection and spectroscopy. Cesium is also used in highly accurate atomic clocks. For more information on the physical and chemical properties and on the production and use of cesium, see Chapters 4 and 5.

Radioactive forms of cesium are produced by the fission of uranium in fuel elements (fuel rods) during the normal operation of nuclear power plants, or when nuclear weapons are exploded. Radioactive forms of cesium are unstable and eventually change into other more stable elements through the process of radioactive decay. The two most important radioactive isotopes of cesium are ^{134}Cs and ^{137}Cs. Radioactive isotopes are constantly decaying or changing into different isotopes by giving off radiation. Each atom of ^{134}Cs changes into either xenon 134 (^{134}Xe) or barium 134 (^{134}Ba), neither of which is radioactive, while each atom of ^{137}Cs decays to barium 137 (^{137}Ba), which is also not radioactive. As ^{134}Cs and ^{137}Cs decay, beta particles and gamma radiation are given off. The half-life is the time it takes for half of that cesium isotope to give off

its radiation and change into a different element. The half-life of ^{134}Cs is about 2 years and the half-life of ^{137}Cs is about 30 years.

Quantities of radioactive cesium, as well as other radioactive elements, are measured in units of mass (grams) or radioactivity (curies or becquerels). Both the curie (Ci) and the becquerel (Bq) describe the rate of decay and tell us how much a radioactive material decays every second. The becquerel is a new international unit known as the SI unit, and the curie is an older, traditional unit; both are currently used. A becquerel is the amount of radioactive material in which 1 atom transforms every second. One curie is the amount of radioactive material in which 37 billion atoms transform every second; this is approximately equivalent to the radioactivity of 1 gram of radium.

1.2 WHAT HAPPENS TO CESIUM WHEN IT ENTERS THE ENVIRONMENT?

Naturally-occurring cesium occurs in the environment mostly from the erosion and weathering of rocks and minerals. The mining and milling of certain ores can also release cesium to the air, water, and soil. Radioactive cesium is released to the environment during the normal operation of nuclear power plants, explosion of nuclear weapons, and accidents involving nuclear power plants or nuclear powered satellites or submarines.

Nonradioactive (stable) cesium can be neither created nor destroyed under typical environmental conditions, but can react with other compounds found in the environment and change into different cesium compounds. Radioactive decay decreases the concentration of ^{134}Cs and ^{137}Cs. Both stable and radioactive cesium are the same element and behave in a similar manner chemically and in the body. Cesium compounds can travel long distances in the air before being brought back to the earth by rainfall and gravitational settling. In water and moist soils, most cesium compounds are very soluble. Cesium binds strongly to most soils and does not travel far below the surface of the soil. Consequently, cesium is not readily available for uptake by vegetation through roots. However, radiocesium can enter plants upon falling onto the surface of leaves.

1.3 HOW MIGHT I BE EXPOSED TO CESIUM?

You can be exposed to stable or radioactive cesium by breathing air, drinking water, or eating food containing cesium. The level of cesium in air and water is generally very low. The concentration of natural cesium in air is generally less than 1 nanogram (1 nanogram equals 1/1,000,000,000 of a gram) per cubic meter of air (ng/m^3). The amount of cesium in drinking water is ordinarily about 1 microgram (1 microgram equals 1/1,000,000 of a gram) per liter of water (μg/L). On average, a person swallows about 10 μg of stable cesium per day in food and water, and breathes about 0.025 μg per day. Plants and animals contain cesium at concentrations of about 1–300 ng/g.

Radioactive cesium has been detected in surface water and in many types of food. This includes breast milk and pasteurized milk. The amount of radioactive cesium in food and milk is highly dependent upon several factors. The most important factor is whether or not there has been recent fallout from a nuclear explosion such as a weapons test or an accident that has occurred at a nuclear power plant. However, atmospheric testing of nuclear weapons was halted many years ago, and there have only been two major reactor accidents at nuclear plants where radiocesium was released in significant amounts. The two accidents occurred in Windscale, England in 1957 and Chernobyl, Russia in 1986. You should understand that cesium only contributed a small fraction of the total radioactivity released following these events. The radiological impacts in Europe from ^{137}Cs and ^{134}Cs released from the Chernobyl accident, however, were great. These included environmental dispersion of radiocesium and uptake in reindeer, caribou, and livestock. Furthermore, the consequences of external exposure to gamma radiation and beta particles are not unique to ^{137}Cs and ^{134}Cs, but are very similar for all gamma and beta emitting radionuclides. People who work in industries that process or use natural cesium or cesium compounds can be exposed to higher-than-normal levels of cesium. An estimated 16,461 workers (4,276 of these are female) are potentially exposed to natural cesium and cesium compounds in the United States. If you work in the nuclear power industry, you may also be exposed to higher-than-normal levels of radioactive cesium, but there are many precautionary measures taken at these facilities to minimize this exposure.

1.4 HOW CAN CESIUM ENTER AND LEAVE MY BODY?

Stable and radioactive cesium can enter your body from the food you eat or the water you drink, from the air you breathe, or from contact with your skin. When you eat, drink, breathe, or touch things containing cesium compounds that can easily be dissolved in water, cesium enters your blood and is carried to all parts of your body. Cesium is like potassium; it enters cells and helps to maintain a balance of electrical charges between the inside and the outside of cells so that cells can perform tasks that depend on those electrical charges. Cells like muscle cells and nerve cells require changing electrical charges in order to function properly and allow you to think and move.

Once cesium enters your body, your kidneys begin to remove it from the blood; some cesium is quickly released from your body in the urine. A small portion is also released in the feces. Some of the cesium that your body absorbs can remain in your body for weeks or months, but is slowly eliminated from your body through the urine and feces.

1.5 HOW CAN CESIUM AFFECT MY HEALTH?

To protect the public from the harmful effects of toxic chemicals and to find ways to treat people who have been harmed, scientists use many tests.

One way to see if a chemical will hurt people is to learn how the chemical is absorbed, used, and released by the body. In the case of a radioactive chemical, it is also important to gather information concerning the radiation dose and dose rate to the body. For some chemicals, animal testing may be necessary. Animal testing may also be used to identify health effects such as cancer or birth defects. Without laboratory animals, scientists would lose a basic method to get information needed to make wise decisions to protect public health. Scientists have the responsibility to treat research animals with care and compassion. Laws today protect the welfare of research animals, and scientists must comply with strict animal care guidelines.

You are not likely to experience any health effects that could be related to stable cesium itself. Animals given very large doses of cesium compounds have shown changes in behavior, such as increased activity or decreased activity, but it is highly unlikely that you would breathe, eat, or drink amounts of stable cesium large enough to cause similar effects. If you were to breathe, eat, drink, touch, or come close to large amounts of radioactive cesium, cells in your body could become damaged from the radiation that might penetrate your entire body, much like x-rays, even if you did not touch the radioactive cesium. You would probably experience similar effects if you were exposed to any other substance with similar radioactivity. You might also experience acute radiation syndrome, which includes such effects as nausea, vomiting, diarrhea, bleeding, coma, and even death. A number of people in Brazil, who handled radioactive cesium that was scavenged from a medical machine used for radiation therapy, became sick from exposure to the radiation; a few of them died. But people exposed to radioactive cesium that has been widely dispersed in air, water, soil, or foods following nuclear bombings or accidents have not been exposed to amounts large enough to cause the same effects.

1.6 HOW CAN CESIUM AFFECT CHILDREN?

This section discusses potential health effects from exposures during the period from conception to maturity at 18 years of age in humans.

Children can be affected by cesium in the same ways as adults. Stable cesium is not likely to affect the health of children, but large amounts of gamma radiation, from sources such as radioactive cesium, could damage cells and might also cause cancer. Short exposure to extremely large amounts of radiation might cause nausea, vomiting, diarrhea, bleeding, coma, and even death. In addition, if babies were to be exposed to enough radiation while in their mother's womb during the time when their nervous system is rapidly developing, they could experience changes in their brains that could result in changes in behavior or decreased mental abilities. However, it is unlikely that children or babies would be exposed to enough gamma radiation from a radioactive cesium source to do such damage to their bodies.

1.7 HOW CAN FAMILIES REDUCE THE RISK OF EXPOSURE TO CESIUM?

If your doctor finds that you have been exposed to significant amounts of cesium, ask whether your children might also be exposed. Your doctor might need to ask your state health department to investigate.

Since cesium is naturally found in the environment, we cannot avoid being exposed to it. However, the relatively low concentrations of stable cesium do not warrant any immediate steps to reduce exposure. You are unlikely to be exposed to high levels of radioactive cesium unless there is a fuel meltdown and accidental release at a nuclear power plant or a nuclear weapon has been detonated. In such cases, follow the advice of public health officials who will publish guidelines for reducing exposure to radioactive material when necessary.

1.8 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO CESIUM?

Everyone has small amounts of cesium in their body. Laboratories use special techniques to measure the amount of cesium in body fluids such as blood and urine, as well as in feces or other human samples. This can give an indication of whether a person has been exposed to levels of cesium that are higher than those normally found in food, water, or air. Special radiation detectors can be used to detect if a person has absorbed radioactive cesium. It is difficult to determine if a person has been exposed only to external radiation from radioactive cesium. Health professionals examining people who have health problems similar to those resulting from radiation exposure would need to rely on additional information, such as the testing of blood samples for cell counts and chromosomal damage, in order to establish if such people had been affected by being near a source of radioactivity.

1.9 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

The federal government develops regulations and recommendations to protect public health. Regulations can be enforced by law. Federal agencies that develop regulations for toxic

substances include the Environmental Protection Agency (EPA), the Occupational Safety and Health Administration (OSHA), the Food and Drug Administration (FDA), and the U.S. Nuclear Regulatory Commission (USNRC).

Recommendations provide valuable guidelines to protect public health but cannot be enforced by law. Federal organizations that develop recommendations for toxic substances include the Agency for Toxic Substances and Disease Registry (ATSDR), the National Institute for Occupational Safety and Health (NIOSH), and the FDA.

Regulations and recommendations can be expressed in not-to-exceed levels in air, water, soil, or food that are usually based on levels that affect animals; they are then adjusted to help protect people. Sometimes these not-to-exceed levels differ among federal organizations because of different exposure times (an 8-hour workday or a 24-hour day), the use of different animal studies, or other factors.

Recommendations and regulations are also periodically updated as more information becomes available. For the most current information, check with the federal agency or organization that provides it. Some regulations and recommendations for cesium include the following:

There are few guidelines for compounds of stable cesium. Based on eye irritation, the NIOSH has established a recommended exposure limit (REL) for cesium hydroxide of 2 mg/m^3 as a time-weighted average (TWA) for up to a 10-hour workday and a 40-hour workweek. The American Conference of Governmental Industrial Hygienists (ACGIH) has assigned cesium hydroxide a threshold limit value (TLV) of 2 mg/m^3 as a TWA for a normal 8-hour workday and a 40-hour workweek, based on respiratory and eye irritation.

The NRC established guidelines for radioactive cesium that include occupational inhalation exposure Derived Air Concentrations (DACs) of 0.00000004 μCi/mL ($4x10^{-8}$ μCi/mL) for ^{134}Cs and 0.00000006 μCi/mL ($6x10^{-8}$ μCi/mL) for ^{137}Cs. Annual Limits on Intake (ALIs) for on-the-job exposure are 100 μCi ($1x10^2$ μCi) for ^{134}Cs and 200 μCi ($2x10^2$ μCi) for ^{137}Cs.

More information on regulations and guidelines is available in Chapter 8.

1.10 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns, please contact your community or state health or environmental quality department, your regional Nuclear Regulatory Commission office, or contact ATSDR at the address and phone number below.

ATSDR can also tell you the location of occupational and environmental health clinics. These clinics specialize in recognizing, evaluating, and treating illnesses resulting from exposure to hazardous substances.

Toxicological profiles are also available on-line at www.atsdr.cdc.gov and on CD-ROM. You may request a copy of the ATSDR ToxProfiles CD-ROM by calling the information and technical assistance toll-free number at 1-888-42ATSDR (1-888-422-8737), by email at atsdric@cdc.gov, or by writing to:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE
Mailstop E-32
Atlanta, GA 30333
Fax: 1-770-488-4178

For-profit organizations may request a copy of final profiles from the following:

National Technical Information Service (NTIS)
5285 Port Royal Road
Springfield, VA 22161
Phone: 1-800-553-6847 or 1-703-605-6000
Web site: http://www.ntis.gov/

2. RELEVANCE TO PUBLIC HEALTH

2.1 BACKGROUND AND ENVIRONMENTAL EXPOSURES TO CESIUM IN THE UNITED STATES

Naturally-occurring cesium exists as the stable isotope (^{133}Cs) in the earth's crust at an average concentration of about 1 ppm in granites and 4 ppm in sedimentary rocks. The most important source of commercial cesium is the mineral pollucite, which usually contains about 5–32% Cs_2O by weight. The largest deposits of pollucite are located in Manitoba, Canada and account for about two-thirds of the world's known supply. Cesium has very low mobility in soil surfaces. Clay minerals and soils rich in exchangeable potassium adsorb cesium by binding the cations to interlayer positions of the clay particles. The low hydration energy of cesium cations is primarily responsible for their selective sorption and fixation by clays, which can result in limited uptake of cesium by grass and plant material. Continental dust and soil erosion are the main emission sources of naturally-occurring cesium that is present in the environment. As a result of human activities, however, cesium is released into the environment globally in small amounts. Cesium has also been detected in the fly ash of hazardous waste incinerators and coal burning power plants. Cesium is deposited on plants and trees by wet and dry deposition and can be absorbed into the flora through its foliage. The deposited cesium can make its way to soil through litter decomposition. Due to its low relative abundance, limited use in industry, and relatively low level of toxicity observed in animal studies, exposure to stable cesium is not considered to be a significant public health concern.

Radioactive isotopes of cesium (^{134}Cs and ^{137}Cs) are formed during nuclear fission, in commercial applications such as the generation of electricity at nuclear power plants. However, high levels of ^{134}Cs and ^{137}Cs have been released to the environment, as a result of atmospheric nuclear weapons testing (which has been discontinued for many years) or underground weapons testing and the accident at the Chernobyl, Ukraine nuclear reactor site in 1986. Once released, these radioactive cesium isotopes persist in the environment, with the potential for adverse health effects. Following release to the atmosphere, radioactive cesium can travel thousands of miles before settling to earth, and is removed by wet and dry deposition. Radioactive cesium can also be released to soil or water in liquid effluents from spent fuel and fuel reprocessing plants.

The total amount of ^{137}Cs released from weapons testing through 1980 was estimated as $2.6x10^7$ Ci ($9.6x10^{17}$ Bq), 76% of which was released in the northern hemisphere and 24% in the southern hemisphere.

The accident at the Chernobyl nuclear power plant resulted in the release of an estimated $5.4x10^5$ Ci ($2.0x10^{16}$ Bq) of ^{134}Cs and $1.1x10^6$ Ci ($4.0x10^{16}$ Bq) of ^{137}Cs into the atmosphere over Europe (see Agency for Toxic Substances and Disease Registry 1999 for details of other accidental releases). Routine activities at nuclear power plants and fuel-reprocessing stations also release ^{137}Cs and ^{134}Cs to the environment on a regular basis, but these are at such levels as to be considered insignificant.

Since the half-life for some radioactive isotopes of cesium is long (the half-life of ^{137}Cs is about 30 years and the half-life of ^{134}Cs is about 2 years), the general population is exposed to ^{137}Cs and ^{134}Cs for long periods of time after it is released from a nuclear accident or weapons test, with the greatest exposure occurring near the source. Although inhalation and dermal exposure is possible, oral ingestion of contaminated food items is the greatest source of internal exposure for both naturally-occurring and radioactive cesium. Workers employed in the mining and milling of pollucite ores and the production of cesium compounds are exposed to cesium through oral, dermal, and inhalation routes. Similar routes of exposure to ^{137}Cs and ^{134}Cs are experienced by workers employed in the nuclear industry. External exposure to beta and gamma radiation can also occur for workers employed in the nuclear industry as well as for the general population following an accidental release or weapons test.

As discussed in Appendix A, the average annual effective dose of ionizing radiation (including ^{134}Cs and ^{137}Cs) from anthropogenic sources to the U.S. population is very small in comparison to natural sources.

2.2 SUMMARY OF HEALTH EFFECTS

Information regarding health effects in humans that can be associated with exposure to higher-than-normal levels of stable cesium is restricted to an account of decreased appetite, nausea, and diarrhea in a man who repeatedly ingested experimental amounts of cesium chloride and reports of prolonged QT syndrome and associated cardiac arrhythmias in patients who ingested cesium chloride as a component of homeopathic remedies.

Exposure to radioisotopes of cesium is of much greater human health concern. Energy released by radioactive isotopes can result in significant damage to living cells. Both ^{134}Cs and ^{137}Cs emit beta

particles and gamma rays, which may ionize molecules within cells penetrated by these emissions and result in tissue damage and disruption of cellular function. The most important exposure routes for radioisotopes of cesium are external exposure to the radiation released by the radioisotopes and ingestion of radioactive cesium-contaminated food sources. Inhalation and dermal exposure routes may also present a health hazard. The hazards of external exposure to ^{134}Cs and ^{137}Cs are similar to those of other gamma- and beta-emitting radionuclides.

Radiation absorbed doses are expressed in terms of the amount of energy absorbed per unit mass, in units called rad or gray (Gy) (see Appendix D and Agency for Toxic Substances and Disease Registry 1999 for a complete description of principles of ionizing radiation). Generally, acute radiation doses below 15 rad (0.15 Gy) do not result in observable adverse health effects. At doses in the range of 15–50 rad (0.15–0.5 Gy), subclinical responses such as chromosomal breaks and transient changes in formed elements of the blood may be seen in sensitive individuals. Symptoms of acute radiation syndrome are observed at radiation doses above 50 rad (0.5 Gy), characterized by transient hematopoietic manifestations, nausea and vomiting, and moderate leukopenia at doses near 100 rad (1 Gy), progressing through more serious hematopoietic symptoms, clinical signs, and gastrointestinal symptoms with increasing dose (100–800 rad or 1–8 Gy), and usually death in persons receiving total doses ≥1,000 rad (10 Gy). Other health effects from acute or continued exposure to ionizing radiation may include reproductive, developmental, and latent cancer effects.

Signs and symptoms of acute toxicity from external and internal exposure to high levels of radiation from ^{134}Cs or ^{137}Cs are typical of those observed in cases of high exposure to ionizing radiation in general. Depending on the radiation dose, symptoms may include those typical of acute radiation syndrome (vomiting, nausea, and diarrhea), skin and ocular lesions, neurological signs, chromosomal abnormalities, compromised immune function, and death.

Acute or repeated exposure of humans or animals to ionizing radiation (from radioisotopes of cesium or other radioactive elements) may result in reduced male fertility, abnormal neurological development following exposure during critical stages of fetal development, and genotoxic effects such as increased frequencies of chromosomal aberrations, T-lymphocyte point mutations, dominant lethal mutations, and reciprocal translocations.

Due to the ionizing properties of radionuclides such as ^{134}Cs and ^{137}Cs, increased cancer risk would be expected among exposed individuals. However, studies of increased cancer risk specifically associated

with exposure of humans to radioactive cesium isotopes were not located. The only documented reports of health effects in humans exposed to cesium as the source of radiation are derived from accidental exposure to a ^{137}Cs source in 1987 in Goiânia, Brazil, and during 1996 and 1997 in Russia. Long-term cancer studies on exposed individuals have not been completed to date.

Animal studies indicate increased risk of cancer following external or internal exposure to relatively high doses of radiation from ^{137}Cs sources. Increased lifetime risk of mammary tumors was noted in female rats acutely exposed to whole-body radiation. Intravenous injection of ^{137}Cs (as cesium chloride) in dogs resulted in long-term increased risk of all cancers combined in males, and all cancers combined (excluding mammary cancer) in females.

Immunological and Lymphoreticular Effects. Humans who were accidentally exposed externally and internally to ^{137}Cs that resulted in estimated radiation absorbed doses of 100–700 rad (1–7 Gy), exhibited severe bone marrow depression. Similar effects were seen in dogs exposed to ^{137}Cs by intravenous injection, resulting in estimated bone marrow doses of 700–2,400 rad (7–24 Gy).

Reproductive Effects. Exposure to radioisotopes of cesium may result in reduced fertility in males, as evidenced by reduced concentrations of spermatozoa in men who had been exposed externally and internally to $^{137}CsCl$ approximately 1 month prior to testing. Reduced fertility, including sterility, was reported in male mice exposed to gamma radiation from ^{137}Cs either by total-body external radiation, which resulted in a total radiation dose of 300 rad (3 Gy) over a 19.5-day exposure period, or by single or repeated oral dosing, which resulted in estimated total testicular radiation doses of 300–385 rad (3–3.85 Gy), measured at 5 weeks post-treatment. No significant reduction in male fertility was seen from total testicular radiation doses in the range of 10–100 rad (0.1–1 Gy). Persistent germinal epithelium damage and azoospermia were reported in all long-term surviving dogs that had been administered ^{137}Cs (as cesium chloride) by intravenous injection at activity levels resulting in long-term total whole-body doses ranging from 742 to 1,640 rad (7.42–16.40 Gy).

Developmental Effects. Developmental effects such as reduced post-natal body weight, impaired motor activity, morphological changes in the brain, reduced head size, and retarded odontogenesis and palatal closure have been reported in rats that had been exposed to radioactive cesium sources (^{137}Cs) *in utero* via whole-body external exposure of dams; effects were of largest magnitude when exposure occurred around gestational day 15. Reported developmental effects in similarly-exposed mice included significantly decreased brain weight and increased aggressive behavior. Atomic bomb survivors of

Hiroshima and Nagasaki, exposed to high levels of ionizing radiation *in utero* during weeks 8–15 or 16–25 post-ovulation, exhibited later signs of impaired cognitive function. Radiation-induced developmental effects would be expected in humans or animals exposed to similar levels of ionizing radiation from any ionizing radiation source, including a radiocesium source. Resulting adverse health effects would be due to the external, penetrating gamma radiation, not cesium *per se*.

Neurological Effects. Excess exposure to stable cesium appears to result in central nervous system effects. A man who voluntarily ingested cesium chloride daily for 36 days reported neurological signs that included feelings of euphoria, heightened sense perception, and tingling sensations within 15 minutes of dosing, in the absence of apparent adverse mental or motor skills. In animal studies, administration of cesium chloride has been reported to trigger stimulant and depressant central nervous system responses.

Since radioisotopes of cesium such as ^{134}Cs and ^{137}Cs emit beta particles and gamma rays capable of ionizing cells, acute radiation doses >3,000 rad (30 Gy) in humans would be expected to result in symptoms indicative of central nervous system syndrome that include immediate onset of violent nausea and vomiting, diarrhea, irrational behavior, circulatory system collapse, and neuromuscular incoordination, followed by convulsions, coma, and death within 48 hours (see Agency for Toxic Substances and Disease Registry 1999 for more detailed information on health effects from exposure to ionizing radiation).

Cancer. Studies that assess the risk of cesium-induced cancer are restricted to radioactive isotopes, not stable cesium. No human studies were located in which cancer incidence was specifically associated with exposure to radioisotopes of cesium. Due to the nature of ionizing radiation in general, carcinogenic effects similar to those observed in Japanese survivors of the 1945 atomic bombing incidents might be expected among individuals acutely exposed to high levels of radiation from a radioactive cesium source (see Agency for Toxic Substances and Disease Registry 1999 for a detailed discussion of the carcinogenic effects of ionizing radiation). However, it is unlikely that levels of ionizing radiation as high as those experienced by the survivors of the atomic bombing incidents would be experienced by individuals who might be exposed to a radiocesium source. Exceptions are reports of accidental human exposures to radioactive cesium sources in Goiânia, Brazil, in 1987 and Lilo, Georgia (Russia) in 1996 and 1997. However, the incidents are too recent for meaningful carcinogenicity data. The EPA Office of Radiation and Indoor Air (ORIA) has classified all radionuclides, including radioisotopes of cesium, as known human carcinogens.

Animal studies indicate increased risk of cancer following external or internal exposure to relatively high doses of radiation from ^{137}Cs sources. Increased lifetime risk of mammary tumors was noted in female rats acutely exposed to whole-body radiation. There were no significant differences between age groups irradiated at 8, 12, 16, 22, or 36 weeks of age, but irradiation at 64 weeks yielded fewer carcinomas than unirradiated controls. In lifetime studies of dogs administered single intravenous doses of $^{137}CsCl$, which resulted in average initial body burdens ranging from 36.4 to 147 MBq/kg (1 to 4 mCi/kg), benign and malignant neoplasms were found in a variety of tissues and organs, with no apparent single target organ of toxicity.

2.3 MINIMAL RISK LEVELS

Inhalation MRLs

No acute-, intermediate-, or chronic-duration inhalation MRLs were derived for cesium due to the lack of suitable human or animal data regarding health effects following inhalation exposure to stable or radioactive cesium. Available information, considered relevant to inhalation exposure, is limited to two studies of dogs intravenously administered $^{137}CsCl$ (Nikula et al. 1995, 1996; Redman et al. 1972). Adverse health effects included depressed blood factors, severe bone marrow depression, germinal cell damage, early death, and increased incidences of benign and malignant neoplasms in a variety of tissues and organs. Striking similarities in the biokinetics, observed in laboratory animals exposed to $^{137}CsCl$ via either parenteral injection or inhalation or oral routes, indicate that adverse health effects might be common to all three routes of exposure (Boecker et al. 1969a; Melo et al. 1996, 1997; Nikula et al. 1995, 1996). However, extrapolation of data across exposure routes was not considered to be a valid basis for the derivation of inhalation MRLs.

Oral MRLs

No acute-, intermediate-, or chronic-duration oral MRLs were derived for stable cesium due to the lack of suitable human or animal data regarding health effects following oral exposure to stable or radioactive cesium. Reports of health effects following oral exposure to stable cesium are limited. Daily ingestion of approximately 68 mg Cs/kg (as cesium chloride) for up to 36 days resulted in decreased appetite, nausea, and diarrhea, as well as neurological signs within 15 minutes following ingestion (Neulieb 1984). Prolonged QT syndrome and associated cardiac arrhythmias were reported in patients who ingested cesium chloride as a component of homeopathic remedies (Bangh et al. 2001; Harik et al. 2002; Saliba et al. 2001). Animal studies regarding oral exposure to stable cesium are limited to LD_{50} studies that

indicate relatively low toxicity for stable cesium compounds (Ghosh et al. 1990; Johnson et al. 1975; Khosid 1967). Information regarding human exposure to radioactive cesium is inadequate because no human data were available on health effects from oral exposure to radioactive cesium. Oral data regarding health effects in animals exposed to radioactive cesium are restricted to a single study in which only reproductive and genotoxic end points were reported (Ramaiya et al. 1994).

Due to striking similarities in the biokinetics, observed in laboratory animals exposed to ^{137}CsCl via either parenteral injection or inhalation or oral routes, it has been suggested that adverse health effects might be common to all three routes of exposure (Boecker et al. 1969a; Melo et al. 1996, 1997; Nikula et al. 1995, 1996). Depressed blood factors, severe bone marrow depression, germinal cell damage, early death, and increased incidences of benign and malignant neoplasms in a variety of tissues and organs were observed in dogs intravenously administered ^{137}CsCl (Nikula et al. 1995, 1996; Redman et al. 1972). However, extrapolation of data across exposure routes was not considered to be a valid basis for the derivation of oral MRLs.

MRLs for External Exposure to Cesium Isotopes

Two MRLs have been derived for ionizing radiation (Agency for Toxic Substances and Disease Registry 1999) and are applicable to external exposure to radioisotopes of cesium:

- An MRL of 400 mrem (4.0 mSv) has been derived for acute-duration external exposure to ionizing radiation (14 days or less).

The acute MRL is based on results of a study by Schull et al. (1988) in which neurological effects of radiation, measured by intelligence test scores, were evaluated in children 10–11 years of age who had been exposed at critical stages of fetal development (gestation weeks 8–15) during the atomic bombing of Hiroshima and Nagasaki. When IQ scores were regressed on radiation dose estimates, IQ diminished linearly with increasing dose, resulting in an estimated decrease in IQ score of approximately 25 points per 100 rad (100 rem in dose equivalent) or 0.25 points/rem (25 points/Sv). To derive the MRL of 400 mrem (4.0 mSv), the Agency for Toxic Substances and Disease Registry (1999) divided the dose associated with a predicted change of 0.25 IQ points (1 rem) by an uncertainty factor of 3 (for human variability/sensitive population). The Agency for Toxic Substances and Disease Registry (1999) noted that a change in IQ points of 0.25 is less than the reported difference of 0.3 IQ points between separated and unseparated identical twins (Burt 1966).

The NRC set a radiation exposure limit of 500 mrem (5 mSv) for pregnant working women over the full gestational period (USNRC 1991). For the critical gestational period of 8–15 weeks, ATSDR believes that the acute MRL of 400 mrem (4 mSv) is consistent with the NRC limit and could be applied to either acute (0–14-day) or intermediate (15–365-day) exposure periods.

- An MRL of 100 mrem/year (1.0 mSv/year) above background has been derived for chronic-duration external exposure to ionizing radiation (365 days or more).

The MRL is based on the BEIR V (1990) report that the average annual effective ionizing radiation dose to the U.S. population is 360 mrem/year (3.6 mSv/year) from all background sources of radiation (earth, cosmic rays, building materials, etc.), a dose not expected to produce adverse health effects. This dose is obtained mainly by naturally-occurring radiation from external sources, medical uses of radiation, and radiation from consumer products. An uncertainty factor of 3 (for human variability) was applied to the NOAEL.

3. HEALTH EFFECTS

3.1 INTRODUCTION

The primary purpose of this chapter is to provide public health officials, physicians, toxicologists, and other interested individuals and groups with an overall perspective on the toxicology of cesium, existing naturally as the stable (nonradioactive) isotope ^{133}Cs and in the form of radioactive isotopes produced during nuclear fission, the most abundant of which are ^{137}Cs and ^{134}Cs. This chapter contains descriptions and evaluations of toxicological studies and epidemiological investigations and provides conclusions, where possible, on the relevance of toxicity and toxicokinetic data to public health. Section 3.2 contains a discussion of the chemical toxicity of stable cesium; radiation toxicity associated with exposure to radiocesium (primarily ^{137}Cs and ^{134}Cs) is discussed in Section 3.3. The chemical properties of stable and radioactive cesium isotopes are identical and are described in Chapter 4.

A glossary and list of acronyms, abbreviations, and symbols can be found at the end of this profile.

Since the average concentration of stable cesium in the earth's crust is low (on average about 1 ppm) and stable cesium is used only in small quantities in electronic and energy production industries, the risk of significant exposure to stable cesium via inhalation, oral, or dermal routes is expected to be small. Limited information is available on monitoring (or detection) of stable cesium in the environment and on health effects from exposure to stable cesium.

Decreased appetite, nausea, and diarrhea were reported in a man who ingested about 34 mg Cs/kg (as cesium chloride) after morning and evening meals for 36 days; this man also experienced apparent neurological changes within 15 minutes of dosing (Neulieb 1984). Prolonged QT syndrome and associated cardiac arrhythmias have been described in patients who have ingested cesium chloride as a component of homeopathic remedies (Bangh et al. 2001; Harik et al. 2002; Saliba et al. 2001).

Animal studies indicate that cesium is of relatively low toxicity. Acute oral LD_{50} values for rats and mice range from 800 to 2,000 mg Cs/kg, cesium hydroxide being more toxic than cesium iodide or cesium chloride. Single oral doses of cesium chloride, administered to female mice at dose levels ranging from 125 to 500 mg/kg, have been shown to result in significant increases in chromosomal breaks in bone marrow cells (Ghosh et al. 1990, 1991).

No reports were located regarding adverse effects in humans or animals following acute-, intermediate-, or chronic-duration inhalation or dermal exposure to stable cesium.

Radioactive isotopes of cesium are a greater health concern than stable cesium. The most important exposure routes are external exposure to the radiation emitted by the radioisotope and ingestion of radioactive cesium-contaminated food sources. Vascular plants do not accumulate large levels of cesium through root uptake because cesium is strongly adsorbed to soils. However, the deposition of radioactive debris on flora with large surface areas such as lichens or moss is significant. Animals that feed on this vegetation, such as reindeer and caribou, may ingest large quantities of radiocesium (and other radionuclides found in fallout). Human consumption of meat from such animals results in the internalization of these radionuclides (see Section 6.7 for more detailed information on the lichen-caribou-human food chain). Radioactive cesium particles may be found in the air following the release of nuclear fission products; however, no reports were located in which adverse health effects from inhalation of radioactive cesium were discussed. Dermal absorption has been observed in rats, as evidenced by traces of ^{137}Cs in the blood a few minutes following dermal application of $^{137}CsCl$ (Pendic and Milivojevic 1966), but no data were located on relative amounts and absorption rates.

Limited human data are available regarding health effects that can be exclusively associated with exposure to radioactive cesium sources such as ^{137}Cs and ^{134}Cs. These radionuclides are products of either neutron activation or nuclear fission and may, therefore, be released from sites where nuclear fission occurs, from radioactive material removed from such sites, or from leakage of radioactive cesium sources. Both ^{137}Cs and ^{134}Cs emit beta radiation (that travels short distances and can penetrate the skin and superficial body tissues) and gamma radiation (that penetrates the entire body). The radiation dose from these radionuclides can be classified as either external (if the source is outside the body) or internal (if the source is inside the body).

The external dose from cesium radionuclides arises primarily from the penetrating gamma rays that travel great distances in air. Beta radiation emitted outside the body is normally of little health concern unless the radioactive material contacts the skin. Skin contact can allow the beta radiation to pass through the epidermis to live dermal tissue where it becomes a major contributor to a radiocesium-generated radiation dose to the skin. At very high doses, the beta and gamma radiation can cause such adverse effects as erythema, ulceration, or even tissue necrosis.

Once radioactive cesium is internalized, it is absorbed, distributed, and excreted in the same manner as stable cesium. The internal radiation dose from cesium is a measure of the amount of energy that the beta and gamma emissions deposit in tissue. The short-range beta radiation produces a localized dose while the more penetrating gamma radiation contributes to a whole body dose. Molecular damage results from the direct ionization of atoms that are encountered by beta and gamma radiation and by interactions of resulting free radicals with nearby atoms. Tissue damage results when the molecular damage is extensive and not sufficiently repaired in a timely manner.

In radiation biology, the term *absorbed dose* is the amount of energy deposited by radiation per unit mass of tissue, expressed in units of rad or gray (Gy) (see Appendix D for a detailed description of principles of ionizing radiation. The term *dose equivalent* refers to the biologically significant dose, which is determined by multiplying the absorbed dose by radiation weighting factors for the type and energy of the radiations involved. Dose equivalent is expressed in units of rem or sievert (Sv). The radiation weighting factors are considered to be unity for the beta and gamma radiation emitted from ^{134}Cs and ^{137}Cs, so for these radionuclides, the absorbed dose (in rad or Gray) is equal to the dose equivalent (in rem or sievert). The dose equivalent from internalized cesium radionuclides is estimated using the quantity of material entering the body (via ingestion or inhalation), the biokinetic parameters for cesium (retention, distribution, and excretion), the energies and intensities of the beta and gamma radiation emitted, and parameters describing the profile of absorbed radiation energy within the body. If, for example, a person ingests a given activity of radiocesium (measured in curies [Ci] or becquerels [Bq]), the tissues of the body will absorb some of the energy of the emitted beta and gamma radiation in a pattern reflecting the kinetics of distribution and elimination of the ingested radiocesium, the rate at which the radioactive isotope decays to a stable form, and the age of the person at the time of ingestion, which affects both the biokinetics of the radiocesium as well as the potential length of time over which the tissues can be exposed to the radiation. The biodistribution of cesium will vary somewhat for uptake in muscle, fat, various organs, and the skeleton. Therefore, each tissue may receive a different dose equivalent. The total dose equivalent for the body will reflect the integration of the dose equivalents for the various tissues using a weighting scheme for the relative sensitivities of tissues and organs to short- and long-term effects.

The EPA has published a set of internal dose conversion factors for standard persons of various ages (newborn; 1, 5, 10, or 15 years of age; and adult) in its Federal Guidance Report No. 13 supplemental CD (EPA 2000). For example, the EPA has estimated that the dose equivalent following ingestion of 1 Bq of ^{137}Cs is 1.30×10^{-8} Sv (assuming an integration time of 50 years for an adult following the initial

exposure). Age-specific dose coefficients for inhalation and ingestion of any of the radioactive isotopes of cesium by the general public can be found in ICRP publications 71 (ICRP 1995) and 72 (ICRP 1996), respectively. Dose coefficients for inhalation, ingestion, and submersion in a cloud of cesium radionuclides can be found in U.S. EPA Federal Guidance Report No. 11 (EPA, 1993a). Dose coefficients for external exposure to radioisotopes of cesium in air, surface water, or soil contaminated to various depths can be found in U.S. EPA Federal Guidance Report No. 12 (EPA, 1993b).

Adverse health effects resulting from external exposure to beta or gamma emissions from radioisotopes of cesium would be the same as those from other radioactive elements that release beta or gamma radiation, and would not be the result of exposure to cesium *per se* (refer to Section 2.2 for a discussion of dose-related symptoms of acute radiation exposure). Developmental and carcinogenic effects have been reported in Japanese survivors of acute high-dose external radiation from the atomic bombs detonated over Hiroshima and Nagasaki (see Agency for Toxic Substances and Disease Registry 1999 for a detailed description of the health effects related to ionizing radiation in general). Exposure to lower levels of radiation would be expected to result in decreased health risk. Although developmental and carcinogenic effects would be expected in individuals subjected to high radiation doses from any source of gamma radiation, non-accidental exposure to a level of significant health risk via a radiocesium source is not likely to occur.

Symptoms typical of cutaneous radiation syndrome (initial dermal erythema and subsequent ulceration) occurred among Russian military recruits who were accidentally exposed to a sealed source of ^{137}Cs (Gottlöber et al. 2000). Some of the exposed men also described symptoms of nausea, vomiting, and headache, which occurred at the onset of the dermal effects.

A number of individuals in Goiânia, Brazil, who experienced mixed external, dermal, and oral exposure to an opened $^{137}CsCl$ source, exhibited classic symptoms of acute radiation syndrome including vomiting, diarrhea, and nausea, as well as skin lesions from radiation burns, orofacial lesions, ocular injury, hematological effects (bone marrow aplasia, leukopenia, thrombocytopenia, lymphopenia, neutropenia), mild elevations of some liver enzymes, reduced sperm counts, and death (in four cases, attributed to infections resulting from reduced resistance) (Brandão-Mello et al. 1991; Gomes et al. 1990). External exposure was estimated based on frequencies of chromosomal aberrations in lymphocytes of exposed individuals at various times following exposure, while internal doses were estimated based on whole-body radiation counting and excretory levels of ^{137}Cs. The adverse effects were the result of beta and gamma radiation, not cesium *per se*.

Information regarding adverse effects in animals exposed to radioactive isotopes of cesium via natural routes (inhalation, oral, or dermal) is limited to observations of significantly reduced fertility and temporary sterility in male mice following single or repeated oral administration of radioactive cesium nitrate (Ramaiya et al. 1994). Postmating embryo mortality was associated with increased frequency of dominant lethal mutations. Studies of animals exposed to radiocesium via natural routes are not usually performed because levels great enough to cause significant adverse health effects would also pose a severe health risk to investigators. However, it has been shown that distribution patterns of ^{137}Cs are similar in animals exposed to relatively nontoxic levels of ^{137}CsCl by parenteral injection, inhalation exposure, or oral administration (Boecker et al. 1969a; Stara 1965). Therefore, the occurrence of depressed blood factors, severe bone marrow depression, germinal cell damage, early death, and increased incidences of benign and malignant neoplasms in a variety of tissues in dogs exposed to ^{137}CsCl via intravenous injection provide the most reasonable indication of health effects that would be expected in animals exposed to ^{137}CsCl by inhalation or oral exposure (Nikula et al. 1995, 1996).

Adverse neurological, developmental, reproductive, genotoxic, and cancer effects have been observed in animal studies employing external exposure to sealed radioactive cesium sources, and are the result of the external gamma radiation, not the cesium *per se*. Impaired motor activity, decreased thickness of cortical layers of the brain, and increased aggressive behavior were observed after the birth of rats that had been briefly exposed *in utero* to relatively high levels of external radiation from a ^{137}Cs source (Minamisawa et al. 1992; Norton and Kimler 1987, 1988). The most vulnerable developmental period was around gestational days 14–15. In another study, adverse developmental effects in fetal rats irradiated on gestational day 12 included reduced litter size, smaller head size, retarded odontogenesis, and cleft palate when examined on gestational day 18 (Saad et al. 1991, 1994). Significant increases in the formation rate of micronuclei were seen in blood cells of other fetal rats following irradiation of pregnant dams via a sealed ^{137}Cs source on gestational day 14 (Koshimoto et al. 1994). Significantly reduced fertility (including temporary sterility) was reported in male mice exposed to an external ^{137}Cs source for almost 20 days; an increased frequency of dominant lethal mutations was also indicated by increased postmating embryo mortality (Ramaiya et al. 1994). Increased lifetime risk of mammary tumors was noted in female rats that were exposed, between the ages of 8 and 36 weeks, to single whole-body doses of radiation from a ^{137}Cs source (Bartstra et al. 1998). Irradiation at 64 weeks, however, yielded fewer carcinomas than unirradiated controls.

3.2 DISCUSSION OF HEALTH EFFECTS OF STABLE CESIUM BY ROUTE OF EXPOSURE

Section 3.2 discusses the chemical toxicity of cesium. Radiation toxicity resulting from exposure to radiocesium is discussed in Section 3.3.

To help public health professionals and others address the needs of persons living or working near hazardous waste sites, the information in this section is organized first by route of exposure (inhalation, oral, and dermal) and then by health effect (death, systemic, immunological, neurological, reproductive, developmental, genotoxic, and carcinogenic effects). These data are discussed in terms of three exposure periods: acute (14 days or less), intermediate (15–364 days), and chronic (365 days or more).

Levels of significant exposure for each route and duration are presented in tables and illustrated in figures. The points in the figures showing no-observed-adverse-effect levels (NOAELs) or lowest-observed-adverse-effect levels (LOAELs) reflect the actual doses (levels of exposure) used in the studies. LOAELs have been classified into "less serious" or "serious" effects. "Serious" effects are those that evoke failure in a biological system and can lead to morbidity or mortality (e.g., acute respiratory distress or death). "Less serious" effects are those that are not expected to cause significant dysfunction or death, or those whose significance to the organism is not entirely clear. ATSDR acknowledges that a considerable amount of judgment may be required in establishing whether an end point should be classified as a NOAEL, "less serious" LOAEL, or "serious" LOAEL, and that in some cases, there will be insufficient data to decide whether the effect is indicative of significant dysfunction. However, the Agency has established guidelines and policies that are used to classify these end points. ATSDR believes that there is sufficient merit in this approach to warrant an attempt at distinguishing between "less serious" and "serious" effects. The distinction between "less serious" effects and "serious" effects is considered to be important because it helps the users of the profiles to identify levels of exposure at which major health effects start to appear. LOAELs or NOAELs should also help in determining whether or not the effects vary with dose and/or duration, and place into perspective the possible significance of these effects to human health.

The significance of the exposure levels shown in the Levels of Significant Exposure (LSE) tables and figures may differ depending on the user's perspective. Public health officials and others concerned with appropriate actions to take at hazardous waste sites may want information on levels of exposure associated with more subtle effects in humans or animals (LOAELs) or exposure levels below which no

adverse effects (NOAELs) have been observed. Estimates of levels posing minimal risk to humans (Minimal Risk Levels or MRLs) may be of interest to health professionals and citizens alike.

Estimates of exposure levels posing minimal risk to humans (Minimal Risk Levels or MRLs) have been made for cesium. An MRL is defined as an estimate of daily human exposure to a substance that is likely to be without an appreciable risk of adverse effects (noncarcinogenic) over a specified duration of exposure. MRLs are derived when reliable and sufficient data exist to identify the target organ(s) of effect or the most sensitive health effect(s) for a specific duration within a given route of exposure. MRLs are based on noncancerous health effects only and do not consider carcinogenic effects. MRLs can be derived for acute, intermediate, and chronic duration exposures for inhalation and oral routes. Appropriate methodology does not exist to develop MRLs for dermal exposure.

Although methods have been established to derive these levels (Barnes and Dourson 1988; EPA 1990b), uncertainties are associated with these techniques. Furthermore, ATSDR acknowledges additional uncertainties inherent in the application of the procedures to derive less than lifetime MRLs. As an example, acute inhalation MRLs may not be protective for health effects that are delayed in development or are acquired following repeated acute insults, such as hypersensitivity reactions, asthma, or chronic bronchitis. As these kinds of health effects data become available and methods to assess levels of significant human exposure improve, these MRLs will be revised.

A User's Guide has been provided at the end of this profile (see Appendix B). This guide should aid in the interpretation of the tables and figures for Levels of Significant Exposure and the MRLs.

3.2.1 Inhalation Exposure

No reports were located regarding the following health effects in humans or animals following inhalation exposure to stable cesium:

3.2.1.1 Death

3.2.1.2 Systemic Effects

3.2.1.3 Immunological and Lymphoreticular Effects

3.2.1.4 Neurological Effects

3.2.1.5 Reproductive Effects

3.2.1.6 Developmental Effects

3.2.1.7 Cancer

3.2.2 Oral Exposure

Limited information is available regarding health effects following oral exposure of humans to stable cesium compounds. Symptoms of decreased appetite, nausea, diarrhea, and cardiac arrhythmia have been associated with consumption of cesium chloride (Bangh et al. 2001; Harik et al. 2002; Neulieb 1984; Saliba et al. 2001). Death was reported laboratory animals following oral administration of large doses of cesium compounds (Ghosh et al. 1990; Johnson et al. 1975; Khosid 1967).

3.2.2.1 Death

No reports were located regarding death in humans following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

No studies were located regarding death in animals following intermediate- or chronic-duration oral exposure to stable cesium. However, acute oral administration of cesium at high dose levels has resulted in observed mortality in rats and mice. In female mice administered cesium chloride, reported oral LD_{50} values range from 2,300 to 2,500 mg/kg (Ghosh et al. 1990; Khosid 1967). An acute oral LD_{50} value for cesium iodide is 2,386 mg/kg in rats (Johnson et al. 1975). Cesium hydroxide appears to be more highly toxic to rats than cesium chloride and cesium iodide, as evidenced by a lower LD_{50} value of 1,026 mg/kg (Johnson et al. 1975). Some of the toxic effects can be attributed to the strong alkaline nature of cesium hydroxide. Khosid (1967) estimated an acute oral LD_{50} value of 800 mg/kg for mice that were administered cesium hydroxide. No information was located regarding mortality in animals following oral administration of other compounds of stable cesium.

3.2.2.2 Systemic Effects

Decreased appetite, nausea, and diarrhea were reported in a man who ingested about 34 mg Cs/kg (as stable cesium chloride) after morning and evening meals for 36 days; this man also experienced apparent neurological changes within 15 minutes of dosing (Neulieb 1984). Prolonged QT syndrome and associated cardiac arrhythmias have been described in patients who have ingested cesium chloride as a

component of homeopathic remedies (Bangh et al. 2001; Harik et al. 2002; Saliba et al. 2001). No additional information was located regarding systemic effects in humans following oral exposure to stable cesium.

No animal studies were located in which the systemic effects that were observed following oral administration of compounds of stable cesium could be attributed to the presence of cesium. Gastrointestinal and respiratory effects noted in rats administered acute oral doses of cesium hydroxide may have been due to the alkaline properties of the compound rather than the biochemical behavior of cesium.

No data were located regarding, hematological, musculoskeletal, endocrine, dermal, ocular, or metabolic effects in humans or animals following oral exposure to stable cesium.

Respiratory Effects. No reports were located regarding respiratory effects in humans following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

No reports were located regarding respiratory effects in animals following intermediate- or chronic-duration exposure to stable cesium. Congested, cyanotic lungs with petechial hemorrhages were observed in rats following oral treatment with single cesium iodide doses large enough to cause death. A bloody nasal exudate was seen in some relatively high-dose rats (Johnson et al. 1975).

Cardiovascular Effects. Individual case reports describe prolonged QT syndrome and associated cardiac arrhythmia in patients who consumed cesium chloride as a component of homeopathic remedies for cancer prevention (Saliba et al. 2001), tumor reduction (Harik et al. 2002), and cyst reduction (Bangh et al. 2001. No reports were located regarding cardiovascular effects in animals following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

Gastrointestinal Effects. *No reports were located regarding gastrointestinal effects in humans* following acute- or chronic-duration oral exposure to stable cesium, and only one report of intermediate-duration exposure was located. An investigator who voluntarily ingested about 34 mg Cs/kg (as cesium chloride, assuming 70 kg body weight) after each morning and evening meal (68 mg Cs/kg/day) for 36 days reported gradually decreased appetite, prenausea feelings, and diarrhea. The observations were self-described and effects were correlated to dietary habits during the course of the study (Neulieb 1984).

No reports were located regarding gastrointestinal effects in animals following intermediate- or chronic-duration oral exposure to stable cesium. In an acute lethality study of rats, administration of cesium hydroxide or cesium iodide at dose levels up to 910 or 1,217 mg Cs/kg, respectively, resulted in stomach and intestinal hemorrhage, bloody fluid exudate within the peritoneal cavity, and adhesions of abdominal organs. Rats receiving lethal doses of cesium iodide exhibited fluid-filled stomach (Johnson et al. 1975).

Hepatic Effects. No reports were located regarding hepatic effects in humans following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

No reports were located regarding hepatic effects in animals following acute- or chronic-duration oral exposure to stable cesium. No significant effect on maternal liver weight was noted in rats consuming 115 mg Cs/kg/day (as cesium chloride in the drinking water) during gestation and 40 mg Cs/kg/day during lactation (Messiha 1988b).

Renal Effects. No reports were located regarding renal effects in humans following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

No reports were located regarding renal effects in animals following acute- or chronic-duration oral intake of stable cesium, and one report was located regarding intermediate-duration intake. In that study, no significant effect on maternal kidney weight was noted in mice consuming 115 mg Cs/kg/day (as cesium chloride in the drinking water) during gestation and 40 mg Cs/kg/day during lactation (Messiha 1988b).

Body Weight Effects. No reports were located regarding body weight effects in humans following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

No reports were located regarding body weight effects in animals following acute- or chronic-duration oral exposure to stable cesium. One intermediate-duration study found no significant effect on maternal body weight in mice consuming 115 mg Cs/kg/day (as cesium chloride in the drinking water) during gestation and 40 mg Cs/kg/day during lactation (Messiha 1988b).

3.2.2.3 Immunological and Lymphoreticular Effects

No reports were located regarding immunological or lymphoreticular effects in humans following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

No reports were located regarding immunological or lymphoreticular effects in animals following acute- or chronic-duration oral exposure to stable cesium. In one study of intermediate-duration, no significant effect on maternal spleen weight was noted in mice consuming 115 mg Cs/kg/day (as cesium chloride in the drinking water) during gestation and 40 mg Cs/kg/day during lactation (Messiha 1988b).

3.2.2.4 Neurological Effects

No reports were located regarding neurological effects in humans following acute- or chronic-duration oral exposure to stable cesium. In one study of intermediate-duration exposure, an investigator, who voluntarily ingested about 34 mg Cs/kg/day (as stable cesium chloride) after morning and evening meals for 36 days, experienced general feelings of well-being, heightened sense perception, and tingling sensations in lips, cheeks, hands, and feet within 15 minutes of intake (Neulieb 1984). No self-reported adverse effects were noted in performance of mathematical tasks or in automobile driving skill.

No reports were located regarding neurological effects in animals following intermediate- or chronic-duration oral exposure to stable cesium. Rats administered unspecified acute gavage doses of cesium hydroxide exhibited initial signs of hyperexcitability followed by apathy and weakness during the course of 14 days of observation after dosing (Johnson et al. 1975).

3.2.2.5 Reproductive Effects

No reports were located regarding reproductive effects in humans or animals following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

3.2.2.6 Developmental Effects

No reports were located regarding developmental effects in humans following acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

No reports were located regarding developmental effects in animals following acute- or chronic-duration oral exposure to stable cesium. In intermediate-duration studies, one investigator reported reduced body weight in offspring of mouse dams consuming approximately 115 mg Cs/kg/day during gestation and

40 mg Cs/kg/day during lactation (Messiha 1988b). Other observations included slight, but significant, changes in some organ weights and slight differences in activity of some hepatic enzymes among offspring of treated dams, relative to controls. Similar results were reported in the offspring of female mice consuming approximately 40 mg Cs/kg/day only during lactation (Messiha 1989b). However, gross and histopathologic examinations of the offspring, typical of well-designed developmental toxicity studies, were not performed in either of these studies, making them of little value for assessment of the developmental toxicity potential of cesium.

3.2.2.7 Cancer

No reports were located in which cancer in humans or animals could be associated with acute-, intermediate-, or chronic-duration oral exposure to stable cesium.

3.2.3 Dermal Exposure

3.2.3.1 Death

No reports were located regarding death in humans or animals following acute-, intermediate-, or chronic-duration dermal exposure to stable cesium.

3.2.3.2 Systemic Effects

No reports were located in which respiratory, cardiovascular, gastrointestinal, hematological, musculo-skeletal, hepatic, renal, endocrine, body weight, or metabolic effects in humans or animals could be associated with dermal exposure to stable cesium.

Dermal Effects. No reports were located regarding dermal effects in humans following acute-, intermediate-, or chronic-duration dermal exposure to stable cesium.

No reports were located regarding dermal effects in animals following intermediate- or chronic-duration dermal exposure to stable cesium. Cesium hydroxide was considered a nonirritant on intact skin and a mild irritant on abraded skin of rabbits 24 and 48 hours, respectively, following closed-patch application of a 5% solution; similar application of cesium iodide resulted in no observed irritation (Johnson et al. 1975).

Ocular Effects. No reports were located regarding eye irritation in humans resulting from ocular contact with stable cesium.

A 5% solution of cesium hydroxide was extremely irritating and caustic to the rabbit eye (Johnson et al. 1975). It is likely that this effect was the result of the caustic nature of the hydroxide rather than an effect due to cesium *per se*. Similar treatment with cesium iodide resulted in no evidence of ocular irritation.

3.2.3.3 Immunological and Lymphoreticular Effects

No reports were located regarding immunological or lymphoreticular effects in humans following acute-, intermediate-, or chronic-duration dermal exposure to stable cesium.

No reports were located regarding immunological or lymphoreticular effects in animals following acute- or chronic-duration dermal exposure to stable cesium. There was no indication of a sensitization response in guinea pigs following repeated intracutaneous injections of 0.1% solutions of cesium hydroxide or cesium iodide (Johnson et al. 1975).

No reports were located regarding the following health effects in humans or animals following dermal exposure to stable cesium:

3.2.3.4 Neurological Effects

3.2.3.5 Reproductive Effects

3.2.3.6 Developmental Effects

3.2.3.7 Cancer

3.2.4 Other Routes of Exposure

3.2.4.1 Death

No data were located regarding death in humans or animals following exposure to stable cesium via routes other than inhalation, oral, or dermal exposure.

3.2.4.2 Systemic Effects

No data were located regarding respiratory, gastrointestinal, hematological, musculoskeletal, hepatic, renal, endocrine, dermal, ocular, body weight, or metabolic effects in humans or animals following exposure to stable cesium via routes other than inhalation, oral, or dermal exposure.

Cardiovascular Effects. Cardiac arrhythmias were elicited in experimental animals following the injection of stable cesium chloride directly into the circulatory system (Brachmann et al. 1983; Levine et al. 1985; Murakawa et al. 1997; Patterson et al. 1990; Senges et al. 2000).

No reports were located in which the following health effects in humans or animals could be associated with exposure to stable cesium via routes other than inhalation, oral, or dermal exposure:

3.2.4.3 Immunological and Lymphoreticular Effects

3.2.4.4 Neurological Effects

3.2.4.5 Reproductive Effects

3.2.4.6 Developmental Effects

3.2.4.7 Cancer

3.3 DISCUSSION OF HEALTH EFFECTS OF RADIOACTIVE CESIUM BY ROUTE OF EXPOSURE

Section 3.3 discusses radiation toxicity associated with exposure to radionuclides of cesium and is organized in the same manner as that of Section 3.2, first by route of exposure (inhalation, oral, and external) and then by health effect (death, systemic, immunological, neurological, reproductive, developmental, and carcinogenic effects). These data are discussed in terms of three exposure periods: acute (14 days or less), intermediate (15–364 days), and chronic (365 days or more).

Levels of significant exposure for each route and duration are presented in tables and illustrated in figures. The points in the figures showing NOAELs or LOAELs reflect the actual dose (levels of exposure) used in the studies. Refer to Section 3.2 for detailed discussion of the classification of endpoints as a NOAEL, less serious LOAEL, or serious LOAEL.

Refer to Appendix B for a User's Guide, which should aid in the interpretation of the tables and figures for Levels of Significant Exposure.

3.3.1 Inhalation Exposure

During and after nuclear accidents, such as the steam explosion that occurred at the Chernobyl nuclear power plant in 1986, significant amounts of ^{137}Cs (and to a lesser extent ^{134}Cs) may be released to the atmosphere in suspended particles and be widely dispersed through the air. Although radioactive cesium, suspended in the air following such accidents or re-suspended later from ground-deposited fallout (USNRC 1998), may be internalized via inhalation, there was no indication that inhalation was a significant route of exposure to radioactive cesium among individuals exposed externally by either being in the vicinity of a release or in areas receiving substantial ground-deposited fallout, or those exposed by ingestion of radioactive cesium-contaminated food following the Chernobyl accident (Balonov 1993).

No reports were located regarding health effects in humans or animals following inhalation exposure to radioactive cesium. Available human case reports and animal studies involving inhaled radioisotopes of cesium deal exclusively with biokinetics. Parenteral injection of $^{137}CsCl$ in laboratory animals has resulted in distribution patterns and tissue doses of ^{137}Cs that are similar to those resulting from inhalation or oral exposure (Boecker et al. 1969a; Stara 1965). For these reasons, it has been proposed that adverse health effects, related to a soluble and readily absorbed compound such as $^{137}CsCl$, should be similar across the three routes of exposure (Melo et al. 1996, 1997; Nikula et al. 1995, 1996). Therefore, depressed blood factors, severe bone marrow depression, germinal cell damage, early death, and increased incidences of benign and malignant neoplasms in a variety of tissues and organs, effects that have been observed in dogs following intravenous administration of $^{137}CsCl$ (Nikula et al. 1995, 1996; Redman et al. 1972), would be expected to occur following inhalation exposure to air concentrations of $^{137}CsCl$ that would result in comparable ^{137}Cs blood concentrations (see Section 3.3.4 for additional information regarding exposure other than inhalation, oral, dermal, or external exposure).

3.3.1.1 Death

Although no studies were located regarding death in humans or animals following acute-, intermediate-, or chronic-duration inhalation exposure to radioactive cesium, dose-related decreased survival was observed in beagle dogs that had received single intravenous injections of $^{137}CsCl$ in amounts resulting in average initial body burdens of 64–147 MBq/kg (1.7–4.0 mCi/kg) (Nikula et al. 1995, 1996). Similar

effects would be expected in animals exposed to air concentrations of ^{137}CsCl that would result in similar body burdens (see Section 3.3.4 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

3.3.1.2 Systemic Effects

No data were located regarding systemic effects in humans or animals following acute-, intermediate-, or chronic-duration inhalation exposure to radioactive cesium. However, hematological effects similar to those observed in dogs that had received single intravenous injections of ^{137}CsCl (Nikula et al. 1995, 1996; Redman et al. 1972) would be expected in animals exposed to air concentrations of ^{137}CsCl that would result in body burdens similar to those attained via intravenous injection.

Hematological Effects. Depressed blood cell counts and platelet levels, reduced packed-cell volume, and bone marrow aplasia were observed in dogs that had been administered single intravenous injections of ^{137}CsCl, which resulted in average initial body burdens ranging from 36.4 to 141.0 MBq/kg (1.0 to 3.8 mCi/kg) (Nikula et al. 1995; Redman et al. 1972). Severely depressed blood cell counts were observed in 23 dogs that died within 52 days following single intravenous administration of ^{137}CsCl at levels resulting in average initial body burdens in the range of 64–147 MBq/kg (1.7–4.0 mCi/kg) (Nikula et al. 1996). Similar effects would be expected in dogs exposed to air concentrations of ^{137}CsCl that would result in body burdens similar to those attained via intravenous injection (see Section 3.3.4 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

3.3.1.3 Immunological and Lymphoreticular Effects

No data were located regarding immunological or lymphoreticular effects in humans or animals following acute-, intermediate-, or chronic-duration inhalation exposure to radioactive cesium. However, severe bone marrow depression was observed in dogs exposed to ^{137}CsCl by intravenous injection at activity levels resulting in estimated total bone marrow doses of 7–24 Gy (700–2,400 rad) (Nikula et al. 1995). Similar effects would be expected in dogs exposed to air concentrations of ^{137}CsCl that would result in body burdens similar to those attained via intravenous injection (see Section 3.3.4 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

3.3.1.4 Neurological Effects

No data were located regarding neurological effects in humans or animals following acute-, intermediate-, or chronic-duration inhalation exposure to radioactive cesium.

3.3.1.5 Reproductive Effects

No data were located regarding reproductive effects in humans or animals following acute-, intermediate-, or chronic-duration inhalation exposure to radioactive cesium. However, germinal epithelium damage and azoospermia were reported in dogs that had been administered ^{137}CsCl by intravenous injection at activity levels resulting in long-term total whole-body doses ranging from 7.42 to 16.40 Gy (742 to 1,640 rad) (Nikula et al. 1995, 1996). Similar effects would be expected in dogs exposed to air concentrations of ^{137}CsCl that would result in body burdens similar to those attained via intravenous injection (see Section 3.3.4 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

3.3.1.6 Developmental Effects

No data were located regarding developmental effects in humans or animals following acute-, intermediate-, or chronic-duration inhalation exposure to radioactive cesium.

3.3.1.7 Cancer

No data were located in which cancer in humans or animals could be associated with acute-, intermediate-, or chronic-duration inhalation exposure to radioactive cesium. However, benign and malignant neoplasms were found in a variety of tissues and organs of dogs administered single intravenous doses of ^{137}CsCl, which resulted in average initial body burdens ranging from 37 to 147 MBq/kg (1.0 to 4.0 mCi/kg) (Nikula et al. 1995, 1996). Similar effects would be expected in dogs exposed to air concentrations of ^{137}CsCl that would result in body burdens similar to those attained via intravenous injection (see Section 3.3.4 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

3.3.2 Oral Exposure

No reports were located regarding health effects in humans or animals that could be exclusively associated with oral exposure to radioisotopes of cesium. Available human case reports and animal studies involving ingested radioisotopes of cesium deal with biokinetics. Parenteral injection of ^{137}CsCl in laboratory animals has resulted in distribution patterns and tissue doses of ^{137}Cs that are similar to those resulting from inhalation or oral exposure (Boecker et al. 1969a; Stara 1965). For these reasons, it has been proposed that adverse health effects, related to a soluble and readily absorbed compound such as ^{137}CsCl, should be similar across the three routes of exposure (Melo et al. 1996, 1997; Nikula et al. 1995, 1996). Therefore, depressed blood factors, severe bone marrow depression, germinal cell damage, early death, and increased incidences of benign and malignant neoplasms in a variety of tissues and organs, effects that have been observed in dogs following intravenous administration of ^{137}CsCl (Nikula et al. 1995, 1996; Redman et al. 1972), would be expected to occur following oral administration of ^{137}CsCl in amounts that would result in comparable ^{137}Cs blood concentrations (see Section 3.3.4 for additional information regarding exposure other than inhalation, oral, dermal, or external exposure).

3.3.2.1 Death

No reports were located regarding death in humans that could be exclusively associated with acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium. In an event involving mixed external, dermal, and oral exposure of adults and children to an opened ^{137}CsCl source, significant short-term morbidity was followed in 50 patients and 4 deaths were reported within a few weeks among individuals with estimated radiation doses ranging from 4.5 to 6 Gy (450 to 600 rad) (Brandão-Mello et al. 1991).

Although no studies were located regarding death in humans or animals following acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium, decreased survival was observed in young adult beagle dogs that had received single intravenous injections of ^{137}CsCl in amounts resulting in average initial body burdens of 64–147 MBq/kg (1.7–4.0 mCi/kg) (Nikula et al. 1995, 1996). Similar results would be expected in animals following oral exposure to ^{137}CsCl levels that would result in similar body burdens (see Section 3.3.4 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

3.3.2.2 Systemic Effects

Acute radiation syndrome, characterized by nausea, vomiting, and diarrhea was reported in a number of individuals following mixed external, dermal, and oral exposure to an opened 50.9 TBq (1,375 Ci) ^{137}CsCl source in Goiânia, Brazil. Other adverse effects included skin lesions, ocular lesions, severe bone marrow depression, and mild elevations in the activities of some liver enzymes (Brandão-Mello et al. 1991; Gomes et al. 1990; Rosenthal et al. 1991). Hematological effects similar to those observed in dogs that had received single intravenous injections of ^{137}CsCl (Nikula et al. 1995, 1996; Redman et al. 1972) would be expected in animals following oral administration of ^{137}CsCl at levels that would result in body burdens similar to those attained via intravenous injection.

No data were located regarding, respiratory, cardiovascular, musculoskeletal, renal, endocrine, body weight, or metabolic effects in humans or animals following oral exposure to radioactive cesium.

Gastrointestinal Effects. Vomiting, diarrhea, and nausea were observed among eight patients treated for acute radiation exposure to an opened ^{137}CsCl source, both via external exposure and internal exposure (Brandão-Mello et al. 1991). These and other symptoms were classic symptoms of acute radiation syndrome. No reports were located regarding gastrointestinal effects in humans following intermediate- or chronic-duration oral exposure to radioactive cesium

No reports were located for animals regarding gastrointestinal effects following acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium.

Hematological Effects. No reports were located for humans regarding hematological effects following intermediate- or chronic-duration oral exposure to radioactive cesium. In the 1987 incident of overexposure to a scavenged medical radiation source containing 50.9 TBq (1,375 Ci) ^{137}CsCl, approximately 250 persons were contaminated externally, many of whom also were contaminated internally. Twenty individuals developed the acute radiation syndrome, 14 of whom developed bone marrow failure after having received whole-body radiation doses ranging from 1 to 7.0 Gy (100 to 700 rad). Four of these 14 heavily contaminated individuals died. These effects are typical signs and symptoms of the hemopoietic (blood forming) syndrome in which the blood forming cells in the bone marrow are killed. This results in sharp decreases of all the blood cells and consequent impairment of the immune system and anemia (Brandão-Mello et al. 1991; Gomes et al. 1990).

No reports were located for animals regarding hematological effects following acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium. However, depressed blood cell counts and platelet levels, reduced packed-cell volume, and bone marrow aplasia were observed in dogs that had been administered single intravenous injections of ^{137}CsCl, which resulted in average initial body burdens ranging from 36.4 to 141.0 MBq/kg (1.0 to 3.8 mCi) (Nikula et al. 1995; Redman et al. 1972). Severely depressed blood cell counts were observed in 23 dogs that died from 20 to 52 days following single intravenous administration of ^{137}CsCl at levels resulting in initial body burdens in the range of 122–164 MBq/kg (3.3–4.4 mCi/kg) (Nikula et al. 1996) and in 11 other dogs (Nikula et al. 1995) that died from 19 to 81 days following intravenous administration of ^{137}CsCl at levels resulting in initial body burdens in the range of 72–141 MBq/kg (1.9–3.8 mCi/kg). Similar effects would be expected in dogs following oral administration of ^{137}CsCl at levels that would result in body burdens similar to those attained via intravenous injection (see Section 3.3.4 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

Hepatic Effects. No reports were located that associate acute-duration oral exposure to radioactive cesium with hepatic effects. Mild elevations of aminotransferases (ALT/AST) were seen in a few patients hospitalized following radiation exposure to an opened ^{137}CsCl source (Brandão-Mello et al. 1991). Exposures were both external and internal. No reports were located for humans regarding hepatic effects following intermediate- or chronic-duration oral exposure to radioactive cesium.

No reports were located for animals regarding hepatic effects following acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium.

Dermal Effects. Reports of dermal effects are restricted to the accidental mixed external, dermal, and oral exposure of a number of individuals to an opened ^{137}CsCl source in which orofacial lesions, including oral bleeding and associated oral rash, mouth ulcers, acute oral candidiasis, and radiation dermatitis and depigmentation, were observed in 21 patients who had been acutely exposed at estimated radiation doses ranging from 1 to 7 Gy (100 to 700 rad) (Gomes et al. 1990). Some individuals exposed in the same incident exhibited typical radiation-induced skin lesions; the forearm was amputated in one individual with severe radiation injury (Brandão-Mello et al. 1991; Gomes et al. 1990).

No reports were located regarding dermal effects in animals following acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium.

Ocular Effects. No reports were located regarding ocular effects in humans following intermediate- or chronic-duration oral exposure to radioactive cesium. Among 20 patients hospitalized following mixed external, dermal, and oral exposure to an opened ^{137}CsCl source, a few patients complained of lacrimation, hyperemia and edema of the conjunctiva, and ocular pain (Brandão-Mello et al. 1991). A few cases of protracted reduction in visual capacity were also reported, among which retinal injury was documented. In these cases, there was no change in lens transparency. These effects were due to the radiation, not to cesium *per se*.

No reports were located regarding ocular effects in animals following acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium.

3.3.2.3 Immunological and Lymphoreticular Effects

No reports were located that associate immunological or lymphoreticular effects in humans with intermediate- or chronic-duration oral exposure to radioactive cesium. Severe bone marrow depression, characterized by a low white blood cell count and consequent immunodeficiency, developed in 14 patients hospitalized following mixed external, dermal, and oral exposure to an opened ^{137}CsCl source resulting in estimated absorbed doses ranging from 1 to 7 Gy (100 to 700 rad) (Brandão-Mello et al. 1991).

No reports were located regarding immunological or lymphoreticular effects in animals following acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium. However, severe bone marrow depression was observed in dogs exposed to ^{137}CsCl by intravenous injection at activity levels resulting in estimated total bone marrow doses of 7–24 Gy (700–2,400 rad) (Nikula et al. 1995). Similar effects would be expected in dogs following oral exposure to ^{137}CsCl levels that would result in body burdens similar to those attained via intravenous injection (see Section 3.3.4 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

3.3.2.4 Neurological Effects

No reports were located for humans or animals that associate neurological effects with acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium.

3.3.2.5 Reproductive Effects

No reports were located that associate reproductive effects in humans with intermediate- or chronic-duration oral exposure to radioactive cesium. Spermatozoa were reduced or absent in the semen of nine males examined approximately 1 month following presumed acute radiation doses on the order of several hundred rad from an opened ^{137}CsCl source (Brandão-Mello et al. 1991). These individuals may have experienced mixed external, dermal, and oral exposure.

No reports were located regarding reproductive effects in animals following intermediate- or chronic-duration oral exposure to radioactive cesium. Significantly reduced fertility, expressed as the percentage of matings resulting in pregnancy (percent effective matings), was noted in male mice following a single oral administration of ^{137}Cs (as cesium nitrate) at activity levels that resulted in a total dose to the testis of approximately 3 Gy (300 rad) (Ramaiya et al. 1994). Complete, though temporary, sterility was evident at week 6 after dosing. By week 17, there were no significant differences in fertility between treatment and control groups. No significant reduction in male fertility was observed at activity levels resulting in cumulative doses to the testes ranging from 0.1 to 1 Gy (10 to 100 rad). Significantly reduced fertility was also evident in male mice administered daily oral doses of ^{137}Cs (as cesium nitrate) for 2 weeks that resulted in total testicular radiation doses of about 3.85 Gy (385 rad), measured at 5 weeks after treatment (Ramaiya et al. 1994).

Germinal epithelium damage and azoospermia were reported in dogs that had been administered ^{137}Cs (as cesium chloride) by intravenous injection at activity levels resulting in long-term total whole-body doses ranging from 7.42 to 16.40 Gy (742 to 1,640 rad) (Nikula et al. 1995, 1996). Similar effects would be expected in dogs following oral exposure to ^{137}CsCl levels that would result in body burdens similar to those attained via intravenous injection (see Section 3.3.4 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

The highest NOAEL values and all reliable LOAEL values for reproductive effects from oral exposure to radioactive cesium are presented in Table 3-1 and plotted in Figure 3-1.

Table 3-1 Levels of Significant Exposure to Cesium - Radiation Toxicity - Oral

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (rad)	LOAEL Less Serious (rad)	LOAEL Serious (rad)	Reference Chemical Form
	ACUTE EXPOSURE						
	Reproductive						
1	Mouse (Hybrid)	Once (G)		200 M		300 M (temporary sterility)	Ramaiya et al. 1994 Cesium 137(as Cesium Nitrate)
2	Mouse (Hybrid)	2 wk 1x/d (G)		154 M		385 M (reduced fertility)	Ramaiya et al. 1994 Cesium 137(as Cesium Nitrate)

a The number corresponds to entries in Figure 3-1.

d=day(s); (G) = gavage; LOAEL = lowest-observed-adverse-effect level; M = male; NOAEL = no-observed-adverse-effect level; wk = week(s)

Figure 3-1. Levels of Significant Exposure to Cesium - Radiation Toxicity - Oral

Acute (≤14 day)

3.3.2.6 Developmental Effects

No reports were located that associate developmental effects in humans or animals with acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium.

3.3.2.7 Cancer

No reports were located in which cancer in humans or animals could be associated with acute-, intermediate-, or chronic-duration oral exposure to radioactive cesium. However, benign and malignant neoplasms were found in a variety of tissues and organs of dogs administered single intravenous doses of ^{137}CsCl, which resulted in initial body burdens ranging from 37 to 147 MBq/kg (1.0 to 4.0 mCi/kg) (Nikula et al. 1995, 1996). Similar effects would be expected in dogs following oral exposure to ^{137}CsCl levels that would result in body burdens similar to those attained via intravenous injection (see Section 3.3.4 for more detailed information regarding health effects in animals exposed to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure).

3.3.3 External Exposure

This section contains information regarding health effects related to external exposure to radioactive cesium sources. Radionuclides of cesium, such as ^{137}Cs and ^{134}Cs, emit both beta particles and gamma rays, which are a health hazard in living organisms because they are capable of ionizing atoms that they encounter. Beta particles can travel appreciable distances in air, but travel only a few millimeters in tissues. External exposure to beta particles may result in damage to skin and superficial body tissues, but is not a threat to internal organs unless the radiation source is internalized. Gamma radiation, on the other hand, can easily pass completely through the human body and cause ionization of atoms in its path. Several feet of concrete or a few inches of lead or other high-density shielding are required for protection from gamma rays. Because it is so highly penetrating, external gamma radiation emitted by radionuclides such as cesium is a radiation hazard to internal organs (Agency for Toxic Substances and Disease Registry 1999; EPA 1998).

The purpose of this section is to provide information regarding health effects associated with external exposure to a radioactive cesium source. External exposure to radioactive cesium is simply exposure to beta and gamma radiation; there is nothing unique to cesium *per se*. The same hazards exist from

external exposure to any source of beta and gamma radiation. Refer to Agency for Toxic Substances and Disease Registry (1999) for a detailed description of health effects from external exposure to ionizing radiation in general.

3.3.3.1 Death

There are no reports of deaths in humans that could be exclusively associated with acute-, intermediate-, or chronic-duration external exposure to radioactive cesium. Death was noted within a few weeks following mixed external, dermal, and oral exposure to an opened ^{137}CsCl source that resulted in estimated radiation doses ranging from 4 to 6 Gy (400 to 600 rad) (Brandão-Mello et al. 1991). See Section 3.3.2.1 for more detailed information.

Significantly reduced survival was noted in rat fetuses following whole-body irradiation (via a ^{137}Cs source) of pregnant dams on gestational day 14 at acute radiation doses $\leq$4 Gy (400 rad); an LD_{50} value was about 5 Gy (500 rad) (Koshimoto et al. 1994). No reports were located regarding death in animals following intermediate- or chronic-duration external exposure to radioactive cesium.

3.3.3.2 Systemic Effects

Symptoms typical of cutaneous radiation syndrome (initial dermal erythema and subsequent ulceration) occurred among Russian military recruits who were accidentally exposed to a sealed source of ^{137}Cs during training in 1996 and 1997 (Gottlöber et al. (2000). Some of the exposed men also described symptoms of acute radiation syndrome (nausea, vomiting, and headache), which occurred at the onset of the dermal effects. Nausea, vomiting, and diarrhea, were reported in a number of individuals following mixed external, dermal, and oral exposure to an opened ^{137}CsCl source in Goiânia, Brazil; other adverse effects included skin lesions, ocular lesions, severe bone marrow depression, and mild elevations in the activities of some liver enzymes (Brandão-Mello et al. 1991; Gomes et al. 1990; Rosenthal et al. 1991). See Section 3.3.2.2 for a more detailed description of systemic effects following mixed exposure to radioactive cesium.

No reports were located regarding respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, hepatic, renal, endocrine, ocular, body weight, metabolic, or other systemic effects in humans or animals that could be exclusively associated with external exposure to radioactive cesium.

Dermal Effects. Initial dermal erythema progressed to ulceration within a few weeks following initial accidental exposure of Russian military recruits to a sealed external source of ^{137}Cs gamma radiation (Gottlöber et al. 2000). However, there was no information regarding radiation doses to these individuals.

3.3.3.3 Immunological and Lymphoreticular Effects

No reports were located that associate immunological or lymphoreticular effects in humans with intermediate- or chronic-duration external exposure to radioactive cesium. Severe bone marrow depression, characterized by neutropenia and thrombocytopenia, developed in 14 patients hospitalized following mixed external, dermal, and oral exposure to an opened $^{137}CsCl$ source resulting in estimated absorbed doses ranging from 1 to 7 Gy (100 to 700 rad) (Brandão-Mello et al. 1991). This effect was the result of the radiation, not the presence of cesium *per se*.

No reports were located regarding immunological or lymphoreticular effects in animals following acute-, intermediate-, or chronic-duration external exposure to radioactive cesium.

3.3.3.4 Neurological Effects

No reports were located that associate neurological effects in humans or animals with postnatal acute-, intermediate-, or chronic-duration external exposure to radioactive cesium. Neurological effects associated with *in utero* exposure are discussed in Section 3.3.3.6, Developmental Effects.

3.3.3.5 Reproductive Effects

No reports were located that associate reproductive effects in humans with intermediate- or chronic-duration external exposure to radioactive cesium. Spermatozoa were reduced or absent in the semen of nine males examined approximately 1 month following presumed acute exposure to an opened $^{137}CsCl$ source (Brandão-Mello et al. 1991). These individuals may have experienced mixed external, dermal, and oral exposure.

No reports were located regarding reproductive effects in animals following acute- or chronic-duration external exposure to radioactive cesium. Significantly reduced fertility, expressed as the percentage of matings resulting in pregnancy (% effective matings), was noted in male mice following external

exposure to a ^{137}Cs source 23 hours a day for 19.5 days at a dose rate of 0.675 mGy/hour (6.75 mrad/hour), resulting in a total dose of 3Gy (300 rad) (Ramaiya et al. 1994). Complete sterility was evident in males by the third week following exposure termination. During weeks 1 and 2 after the cessation of treatment, significantly increased total and postimplantation embryo mortality was noted. These effects were the result of the radiation, not the presence of cesium *per se*.

All reliable LOAEL values for reproductive effects from external exposure to radioactive cesium are presented in Table 3-2 and plotted in Figure 3-2.

3.3.3.6 Developmental Effects

No reports were located that associate developmental effects in humans with acute-, intermediate-, or chronic-duration external exposure to radioactive cesium. Cells of the developing central nervous system are among the most sensitive to the effects of ionizing radiation in the developing fetus (Agency for Toxic Substances and Disease Registry 1999). Schull and Otake (1999) cite numerous reports in which impaired cognitive function was observed in atomic bomb survivors of Hiroshima and Nagasaki prenatally exposed (during weeks 8–15 or 16–25 after ovulation) to ionizing radiation from the bombs. The small number (38) of mentally-impaired survivors makes it difficult to generalize a dose-response relationship. However, the data are compatible with either a threshold dose of 20–40 rad (0.2–0.4 Gy) or zero threshold linear response. Although such effects would be expected in individuals exposed to similar levels of external radiation from any source of gamma radiation, it is unlikely that such high levels would be achieved from any radiocesium source under situations of normal use.

No reports were located regarding developmental effects in animals following intermediate- or chronic-duration external exposure to radioactive cesium. Significantly reduced postnatal body weight, impaired motor activity, and decreased thickness within cortical layers of the brain were observed in young rats (7–21 days postpartum) exposed during gestation by whole-body radiation of pregnant dams via a sealed ^{137}Cs gamma radiation source at a rate of approximately 0.5 Gy/minute (50 rad/minute) for a total radiation dose of 0.75 or 1 Gy (75 or 100 rad) (Norton and Kimler 1987, 1988). The effects were of larger magnitude when the fetal rats were exposed on gestation day 15 rather than earlier (gestation days 11 or 13) or later (gestation day 17). Significantly smaller litter size, smaller head size, and retarded odontogenesis were observed in fetuses of pregnant mice exposed on gestation day 12 to whole-body irradiation from a sealed ^{137}Cs gamma radiation source for 4.9 minutes (resulting in a total radiation dose

Table 3-2 Levels of Significant Exposure to Cesium - Radiation Toxicity - External Radiation

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (rad)	LOAEL Less Serious (rad)	LOAEL Serious (rad)	Reference Chemical Form
	ACUTE EXPOSURE						
	Developmental						
1	Rat (Wistar)	Once Gd 13, 14, or 15			50 (increased micronuclei in hematocytes)	400 (31.5% fetal mortality)	Koshimoto et al. 1994 Cesium 137
2	Rat (Sprague-Dawley)	Once Gd 11 or 17				100 (altered motor function, decreased cortical thickness)	Norton and Kimler 1987 Cesium 137
3	Rat (Sprague-Dawley)	Once Gd 13, 15, or 17				75 (altered motor function, decreased cortical thickness, most prominent when exposed on Gd 15)	Norton and Kimler 1988 Cesium 137
4	Mouse (C57BL/6)	Once Gd 14			100 M (6% lower body weight, 9% lower brain weight, reduced brain size)		Minamisawa et al. 1990 Cesium 137
5	Mouse (C57BL/6)	Once Gd 14			100 M (16% lower body weight, increased aggressive behavior)		Minamisawa et al. 1992 Cesium 137
6	Mouse Swiss albino	Once Gd 12			400 (retarded odontogenesis)		Saad et al. 1991 Cesium 137

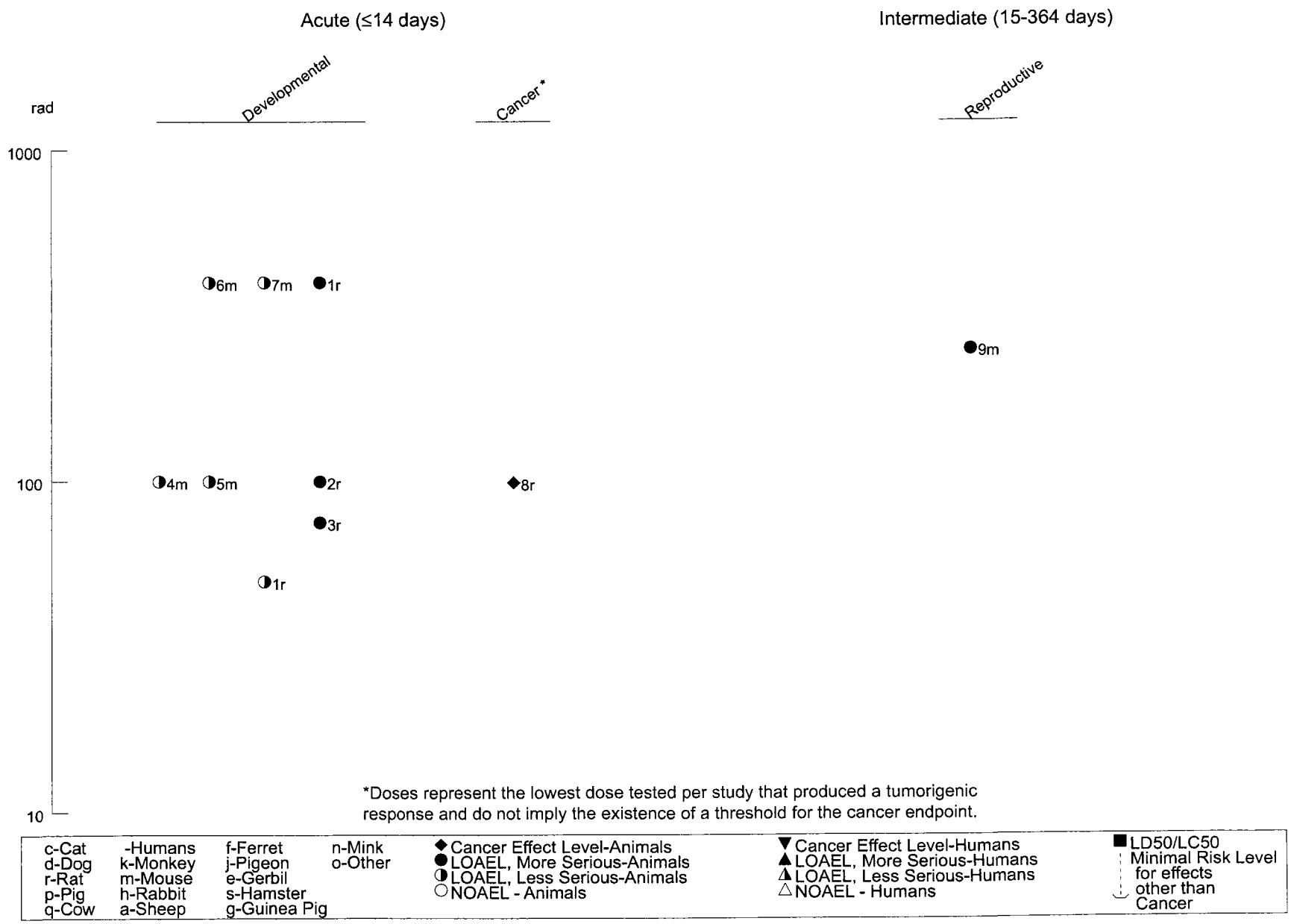
Table 3-2 Levels of Significant Exposure to Cesium - Radiation Toxicity - External Radiation (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (rad)	LOAEL Less Serious (rad)	LOAEL Serious (rad)	Reference Chemical Form
7	Mouse Swiss albino	Once Gd 12			400 (smaller litter size, smaller head size, delayed palatogenesis)		Saad et al. 1994 Cesium 137
	Cancer						
8	Rat WAG/Rij	Once at 8, 12, 16, 22, 36, or 64 wk of age				100 F (mammary carcinoma in 57/200 rats)	Bartstra et al. 1998 Cesium 137
	INTERMEDIATE EXPOSURE						
	Reproductive						
9	Mouse (Hybrid)	19.5 d 23 hr/d				300 M (reduced fertility)	Ramaiya et al. 1994 Cesium 137

a The number corresponds to entries in Figure 3-2.

d=day(s); F = Female; Gd = gestational day; hr = hour(s); LOAEL = lowest-observed-adverse-effect level; M = male; NOAEL = no-observed-adverse-effect level; wk = week(s).

Figure 3-2. Levels of Significant Exposure to Cesium - Radiation Toxicity - External Radiation



of 4 Gy or 400 rad) and examined on gestation day 18. Additionally, all exposed fetuses exhibited cleft palate on examination while normal closure was observed in all unexposed control fetuses (Saad et al. 1991, 1994). Significantly reduced survival was observed in rat fetuses following whole-body irradiation (via a sealed ^{137}Cs gamma radiation source) of pregnant dams on gestation day 14 at acute radiation doses of 4 Gy (400 rad) or greater. An LD_{50} value for fetuses was about 5 Gy (500 rad) (Koshimoto et al. 1994). Aggressive behavior was studied in male mice (100–135 days of age) exposed *in utero* on gestation day 14 through whole-body irradiation of pregnant dams via an external sealed ^{137}Cs gamma radiation source at total radiation doses of 1 or 2 Gy (100 or 200 rad) (Minamisawa et al. 1992). Incidences of aggressive behavior were significantly higher among irradiated groups, relative to untreated controls. The intensity of aggressive behavior was significantly higher only in the 2 Gy (200 rad) exposure group. Minamisawa et al. (1990) found dose-related significantly decreased brain weight in 6-month-old mice that had been irradiated on gestation day 14 at doses of 1–3 Gy (100–300 rad). In each of these studies (Koshimoto et al. 1994; Minamisawa et al. 1990, 1992; Norton and Kimler 1987, 1988), the observed developmental effects were the result of radiation exposure, not the presence of cesium *per se*.

Kusama and Hasegawa (1993) designed a study to examine the relationship between fetal developmental stage at the time of external exposure to a sealed ^{137}Cs gamma radiation source and the occurrence of external malformations and growth retardation. Groups of pregnant ICR mice were irradiated once with 1.5 Gy (150 rad) at a dose rate of 0.2 Gy/minute (20 rad/minute) on 6-hour intervals during the period of organogenesis (gestation days 6.5–14). Fetuses were examined on gestation day 18. The authors reported peaks in the occurrence of exencephaly among fetuses irradiated during gestation days 6.5–8.75 and 10.25–10.75, and the highest peak occurred at gestation day 7.5. Peaks in the occurrence of cleft palate were seen in fetuses irradiated at gestation days 8.75 and 10.75. The most apparent reduction in body weight of irradiated fetuses, relative to controls, occurred in groups irradiated between gestation days 9.75 and 11. The observed developmental effects were the result of radiation exposure, not the presence of cesium *per se*.

All reliable LOAEL values for developmental effects from external exposure to radioactive cesium are presented in Table 3-2 and plotted in Figure 3-2.

3.3.3.7 Cancer

No reports were located regarding cancer in humans following acute-, intermediate-, or chronic-duration external exposure to radioactive cesium in particular. Due to the nature of ionizing radiation in general, carcinogenic effects similar to those observed in Japanese survivors of the 1945 atomic bombing incidents might be expected among individuals acutely exposed to high levels of radiation from a radioactive cesium source. In the only available reports of adverse health effects in humans following exposure to radioactive cesium (^{137}Cs, the accidental human exposures in Goiânia, Brazil in 1987 and Russia in 1996 and 1997), the incidents are too recent for meaningful data on the potential for carcinogenicity. No reports were located in which increased cancer risk could be associated with long-term exposure to low levels of ionizing radiation.

No reports were located regarding cancer in animals following intermediate- or chronic-duration external exposure to radioactive cesium. Increased lifetime risk of mammary tumors was observed in female WRG/Rij rats exposed to single whole-body doses of 1 or 2 Gy (100 or 200 rad) of ^{137}Cs gamma radiation at a dose rate of 75 rad/minute (0.75 Gy/minute) between the ages of 8 and 36 weeks (Bartstra et al. 1998). The excess normalized risk values for carcinoma were 0.9 and 2.2 for 1 and 2 Gy (100 and 200 rad) doses, respectively, with no significant differences between the age groups of 8, 12, 16, 22, or 36 weeks. Irradiation at 64 weeks, however, yielded fewer carcinomas than unirradiated controls. The excess normalized risk values were found to be -0.7 and -0.3 for 1 and 2 Gy (100 and 200 rad) doses, respectively. These effects were the result of the gamma radiation, not the presence of cesium *per se*.

3.3.4 Other Routes of Exposure

Although parenteral injection is not considered to be an exposure route of concern for the general population, it has been considered to be a good indicator of adverse health effects that would be expected in laboratory animals following the absorption of $^{137}CsCl$ into the blood if such animals were to be exposed via inhalation or oral routes (Boecker et al. 1969a; Melo et al. 1996, 1997; Nikula et al. 1995, 1996). This argument is based on the results of biokinetic studies in dogs (Boecker et al. 1969a) in which intravenous injections of $^{137}CsCl$ resulted in temporal and tissue distribution patterns and tissue doses of ^{137}Cs that were similar to those resulting from inhalation exposure (Boecker et al. 1969a). Similar tissue distribution and retention kinetics were also shown in guinea pigs whether exposure to $^{137}CsCl$ had been via intraperitoneal, inhalation, or oral routes (Stara 1965).

In dogs, intravenous administration of soluble ^{137}CsCl has resulted in depression of a number of blood factors, severe bone marrow depression, germinal cell damage (males), and early death (Nikula et al. 1995, 1996; Redman et al. 1972). Long-term surviving dogs exhibited increased incidences of benign and malignant neoplasms in a variety of tissues and organs, with no apparent single target organ of toxicity.

3.3.4.1 Death

Dose-related decreased survival was observed in young adult beagle dogs that had received single intravenous injections of ^{137}CsCl in amounts resulting in average initial body burdens of 71.7–141 MBq/kg (1.9–3.8 mCi/kg) (Nikula et al. 1995). All six dogs in the highest exposure group died between 19 and 33 days following injection. The total whole body radiation dose to death in this group of dogs averaged 11.8 Gy (1,180 rad). Deaths were attributed to severe pancytopenia resulting from hematopoietic cell damage. In a study of 63 other beagle dogs, intravenous injection of ^{137}CsCl resulted in initial body burdens of approximately 64–147 MBq/kg (1.7–4.0 mCi/kg) (Nikula et al. 1996). These dogs, grouped according to age, were juveniles (142–151 days old), young adults (388–427 days old), or middle-aged adults (1,387–2,060 days old) at the time of injection. Early mortality, within 52 days following injection, was noted in 10/10 middle-aged dogs, 10/38 young adults, and 3/15 juveniles. Average initial ^{137}Cs body burdens among the early deaths were in the range of 133–147 MBq/kg (3.6–4.0 mCi/kg). The middle-aged dogs died significantly earlier ($p=0.002$) than the juvenile or young adult dogs, and middle-aged female dogs died significantly earlier ($p=0.002$) than middle-aged male dogs.

3.3.4.2 Systemic Effects

No data were located regarding respiratory, cardiovascular, gastrointestinal, musculoskeletal, hepatic, renal, endocrine, dermal, ocular, body weight, or metabolic effects in humans or animals following exposure to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure.

Hematological Effects. Hematological dyscrasia, characterized by severe thrombocytopenia and leukopenia, and death within 81 days were observed in 11 of 54 dogs that had been administered single intravenous injections of ^{137}CsCl (Nikula et al. 1995; Redman et al. 1972). The early deaths occurred in groups with average initial body burdens ranging from 71.7 to 141 MBq/kg (1.9 to 3.8 mCi) and cumulative doses to death in the range of 11.8–14.0 Gy (1,180–1,400 rad). Moderately to severely depressed blood cell counts were observed among 25 surviving dogs, some of which had been injected with lower levels of ^{137}CsCl that resulted in average initial body burdens of 36.4 or 51.7 MBq/kg (1.0 or

1.4 mCi/kg). Other treatment-related hematological effects included bone marrow aplasia, decreased platelet levels, and reduced packed-cell volume. In long-term surviving dogs, depressed blood values returned toward normal within during the first year after radiocesium injection. Severely depressed blood cell counts were observed in 23 dogs that died within 52 days following single intravenous administration of ^{137}CsCl at levels resulting in average initial body burdens in the range of 64–162 MBq/kg (1.7–4.4 mCi/kg) (Nikula et al. 1996).

3.3.4.3 Immunological and Lymphoreticular Effects

Severe bone marrow depression was observed in dogs administered ^{137}CsCl by intravenous injection at activity levels resulting in estimated total bone marrow doses of 7–24 Gy (700–2,400 rad) (Nikula et al. 1995).

3.3.4.4 Neurological Effects

No data were located regarding neurological effects in humans or animals following exposure to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure.

3.3.4.5 Reproductive Effects

Persistent germinal epithelium damage and azoospermia were reported in all long-term surviving dogs that had been administered ^{137}CsCl by intravenous injection at activity levels resulting in long-term total whole-body doses ranging from 7.42 to 16.40 Gy (742 to 1,640 rad) (Nikula et al. 1995, 1996).

3.3.4.6 Developmental Effects

No data were located regarding developmental effects in humans or animals following exposure to radioactive cesium via routes other than inhalation, oral, dermal, or external exposure.

3.3.4.7 Cancer

Benign and malignant neoplasms were found in a variety of tissues and organs of beagle dogs that had been administered single intravenous injections of ^{137}CsCl (Nikula et al. 1995, 1996). Fifty-four young adult dogs at the Inhalation Toxicology Research Institute (ITRI) received amounts of ^{137}CsCl that resulted in average initial body burdens ranging from 37 to 141 MBq/kg (1.0 to 3.8 mCi/kg) (Nikula et al. 1995). In a study initiated at the Argonne National Laboratory (ANL), 63 beagle dogs, grouped according to age at study initiation (juveniles, 142–151 days old; young adults, 388–427 days old; and middle-aged adults, 1,387–2,060 days old), were administered single intravenous injections of ^{137}CsCl that resulted in initial body burdens of approximately 64–147 MBq/kg (1.7–4.0 mCi/kg) (Nikula et al. 1996). In both studies, dose-related increased incidences were observed for malignant neoplasms, malignant neoplasms excluding mammary neoplasms, all sarcomas considered as a group, all nonmammary carcinomas considered as a group, and malignant liver neoplasms. An increased risk for malignant thyroid neoplasms was seen in the ANL male dogs, but not in the ITRI males or females. In the ITRI (but not ANL) dogs, an increased relative risk for benign neoplasms excluding mammary neoplasms was observed. The occurrence of neoplasms in a diversity of tissues and organs results from the widespread distribution of cesium in the body.

3.4 GENOTOXICITY

Genotoxicity data for stable and radioactive cesium are summarized in Tables 3-3 and 3-4, respectively.

Evidence for genotoxic effects of stable cesium is limited to studies in which cesium chloride induced significantly increased incidences of chromosomal aberrations in human lymphocytes *in vitro* (Ghosh et al. 1993) (see Table 3-3) and mouse bone marrow *in vivo* (Ghosh et al. 1990, 1991; Santos-Mello et al. 2001) (see Table 3-4). Cesium sulfate was not mutagenic in *Escherichia coli (E. coli)* tester strains PQ37 and PQ35 either with or without metabolic activation in the SOS Chromotest (a bacterial colorimetric assay) at doses up to those resulting in significant toxicity (Olivier and Marzin 1987).

Increased frequency of point mutations in T-lymphocytes was observed in individuals in Goiânia, Brazil who had been exposed to an opened ^{137}CsCl source approximately 2.5 years prior to testing (Skandalis et al. 1997). The estimated dose from external radiation was 1.7 Gy (170 rad). The authors estimated internal doses based on whole-body counts and measured activity in urine and feces; however, realistic estimates were not reported. Among individuals exposed in the same incident, frequencies of

Table 3-3. Genotoxicity of Cesium *In Vitro*

Species (test system)	End point	Result: With activation	Result: Without activation	Reference
Stable Cesium				
Mammalian cells:				
Human lymphocytes	Chromosomal aberrations	No data	+	Ghosh et al. 1993
Human lymphocytes	Micronuclei	No data	–	Santos-Mello et al. 1999
Prokaryotic organisms:				
Escherichia coli (PQ 37 and PQ 35)	Mutations	–	–	Olivier and Marzin 1987
Radioactive Cesium				
Mammalian cells:				
Human peripheral blood lymphocytes	Micronuclei	No data	+	Balasem and Ali 1991
Human peripheral blood lymphocytes	Chromosomal aberrations	No data	+	Doggett and McKenzie 1983
Human peripheral blood lymphocytes	Chromosomal aberrations	No data	+	Hintenlang 1993
Human peripheral blood lymphocytes	Chromosomal aberrations	No data	+	Iijima and Morimoto 1991
Human peripheral blood lymphocytes	Sister chromatid exchange	No data	–	Iijima and Morimoto 1991
Human spermatozoa	Chromosomal aberrations	No data	+	Mikamo et al. 1990, 1991
Human spermatozoa and zona-free hamster oocytes fertilization system	Micronuclei	No data	+	Kamiguchi et al. 1991
Mouse (BALB/c, SC3T3/W, Scid/St cells)	DNA double-strand breaks	No data	+	Biedermann et al. 1991
Chinese hamster ovary cells	Chromosomal aberrations, sister chromatid exchange	No data	+	Arslan et al. 1986

DNA = deoxyribonucleic acid; – = negative results; + = positive results

Table 3-4. Genotoxicity of Cesium *In Vivo*

Species (test system)	End point	Results	Reference
Stable Cesium			
Mammalian cells:			
Mouse bone marrow	Chromosomal aberrations	+	Ghosh et al. 1990, 1991
Mouse bone marrow	Micronuclei	+	Santos-Mello et al. 2001
Mouse bone marrow	Micronuclei	–	Santos-Mello et al. 1999
Radioactive Cesium			
Mammalian cells:			
Human peripheral blood lymphocytes	Chromosomal aberrations	+	Natarajan et al. 1998
Human peripheral blood lymphocytes	Chromosomal aberrations	+	Padovani et al. 1993
Monkey germ cells (male)	Reciprocal translocations	+	Tobari et al. 1988
Monkey germ cells (male)	Reciprocal translocations	+	Ramaiya et al. 1994
Mouse germ cells (male)	Reciprocal translocations	+	Ramaiya et al. 1994
Mouse germ cells (male)	Dominant lethal mutations	+	Ramaiya et al. 1994

+ = positive results; – = negative results

chromosomal aberrations were used to estimate external radiation doses (Natarajan et al. 1998). No human reports were located in which genotoxic effects could be associated with specific radiation exposure levels, nor was there any information regarding potential for route-specific differences in observed genotoxic effects related to radioactive cesium exposure. Five years after the initial exposure to radioactive fallout from the Chernobyl accident of 1986, slightly greater frequencies of chromosomal aberrations were observed in peripheral blood lymphocytes of three groups of Byelorussian children (41 total) living in areas with ground contamination from ^{137}Cs fallout than in those of an Italian control group of 10 children (Padovani et al. 1993). Whole-body counts found an internally deposited ^{137}Cs activity range of 0.46–2.8 kBq (12-75 nCi) in children from Navrovl'a, an area (70 km from Chernobyl) exhibiting ^{137}Cs contamination of 550-1,500 GBq/km^2 (15–40 Ci/km^2). Internally deposited ^{137}Cs activity ranges of 0.044–0.4 kBq (1.2-10.8 nCi) and 7.7–32.3 kBq (208–872 nCi) were reported for children evacuated from the Chernobyl area soon after the accident to areas 200–300 km from Chernobyl with ^{137}Cs ground contamination of 40-400 GBq/km^2 (1–10 Ci/km^2), and children living in the Stolin area (250 km from Chernobyl) with ^{137}Cs ground contamination of 40–550 GBq/km^2 (1–5 Ci/km^2), respectively. Internalized activity was presumably from the consumption of ^{137}Cs-contaminated food. Although a small increase in the frequency of chromosomal aberrations in lymphocytes was observed, no pathology was apparent. These genotoxic effects were the result of the radiation, not the presence of cesium *per se*.

A dose-related increased frequency of micronuclei was observed in human peripheral blood lymphocytes exposed *in vitro* to gamma radiation from a sealed ^{137}Cs source at doses ranging from 0.05 to 6.00 Gy (5 to 600 rad) (Balasem and Ali 1991). This effect was the result of the radiation, not the presence of cesium *per se*.

In mice, genotoxic effects resulting from repeated oral exposure (daily administration for 2 weeks) to ^{137}Cs (as cesium nitrate) were compared with those elicited from external whole-body irradiation (23 hours/day for 19.5 days) using a ^{137}Cs source (Ramaiya et al. 1994). At comparable total radiation doses (approximately 3–4 Gy or 300–400 rad), both exposure scenarios resulted in similar increases in dominant lethal mutations among exposed male mice. Significant increases in the frequency of reciprocal translocations have been reported in spermatogonia of mice orally administered single doses of ^{137}Cs (as cesium chloride) that resulted in absorbed total body doses of approximately 3 Gy (300 rad) (Ramaiya et al. 1994).

Significant (dose-related) increases in the formation rate of micronuclei were seen in blood cells of fetal rats following irradiation of pregnant dams to total radiation doses of 0.5–4 Gy (50–400 rad), from a sealed ^{137}Cs gamma source, on gestation day 14 (Koshimoto et al. 1994). In crab-eating monkeys exposed to gamma radiation from an external ^{137}Cs source, increases in reciprocal translocations in spermatogonia were dose-related through the total absorbed dose range of 0.3–1.5 Gy (30–150 rad); it was also noted that the induction rate of translocations after acute high-dose-rate (0.25 Gy/minute or 25 rad/minute) exposure was about 10 times higher than that resulting from longer-term low-dose-rate (1.8×10^{-7} Gy/minute or 1.8×10^{-5} rad/minute) exposure (Tobari et al. 1988). These effects were the result of the radiation, not the presence of cesium *per se*.

Additional assays, performed *in vitro*, have also indicated that radioisotopes of cesium are genotoxic; specific end points include chromosomal aberrations and breaks, sister chromatid exchanges, and micronuclei (Arslan et al. 1986; Biedermann et al. 1991; Doggett and McKenzie 1983; Hintenlang 1993; Iijima and Morimoto 1991; Kamiguchi et al. 1991; Mikamo et al. 1990, 1991). Refer to Agency for Toxic Substances and Disease Registry (1999) for more information on the genotoxic effects of ionizing radiation.

3.5 TOXICOKINETICS

Numerous biokinetic studies have been performed in animals exposed internally to small amounts of the radioisotope ^{137}Cs. The biokinetic behavior of cesium has also been studied in humans either given tracer amounts of radiocesium or accidentally exposed to larger amounts.

Fractional absorption of inhaled or ingested cesium to the blood decreases with decreasing solubility of the carrier. Cesium taken into the body in soluble form is almost completely absorbed to blood. Cesium entering the respiratory or gastrointestinal tract as relatively insoluble particulates is mostly excreted in the feces. Cesium that comes into contact with the skin may be absorbed to some extent through the skin.

Following uptake by the blood, widespread distribution of cesium to all major soft tissues is observed in humans and animals. Cesium levels are slightly higher in skeletal muscle than other tissues. Distribution patterns in animals have been shown to be similar following exposure to soluble cesium compounds by inhalation, oral, and parenteral routes of exposure. Cesium crosses the placenta to the fetus. Cesium is also found in breast milk of a mother with an internal deposition.

Cesium is a close chemical analogue of potassium. Cesium has been shown to compete with potassium for transport through potassium channels and can also substitute for potassium in activation of the sodium pump and subsequent transport into the cell.

Excretion rates for ^{137}Cs have been studied in numerous populations exposed to nuclear fallout following incidents such as the Chernobyl accident. Biologically based pharmacokinetic models have been developed to describe relationships between intake and elimination. Experimental human studies have also been performed using tracer amounts of ^{134}Cs and ^{137}Cs. Urinary excretion is the primary route of elimination of cesium, and is independent of the route of exposure. Urinary to fecal ratios for cesium in humans have been found to range from 2.5:1 to 10:1. Radiocesium excretion rates were higher in males with muscular dystrophy than in normal, age-matched controls, and higher in pregnant women than in others who were not pregnant.

The elimination of cesium in humans appears to be age and sex related; long-term retention is principally a function of muscle mass. Elimination half-times are shorter in children than in adults, and shorter in women than in men.

3.5.1 Absorption

3.5.1.1 Inhalation Exposure

Inhalation exposure to relatively soluble cesium compounds will result in absorption of cesium in humans, although no reports were located that measured absorption of cesium following inhalation exposure. Evidence for absorption was presented by Miller (1964) via whole-body counts of ^{137}Cs (taken periodically for up to 285 days) in two men following occupational exposure to ^{137}Cs (as cesium sulfate) that was presumed to have been by inhalation. Distribution of ^{137}Cs was relatively uniform throughout the body, and steadily decreasing whole-body counts indicated that ^{137}Cs was eliminated from the body with a biological half-time of approximately 73–84 days. Additional indirect evidence of absorption was reported for an adult male who had been accidentally exposed to airborne ^{137}Cs twice in a 13-month period (Hölgye and Malý 2002). In this case, biological half-times for elimination of ^{137}Cs were 72 and 73 days.

Inhaled soluble cesium compounds are readily absorbed and distributed systemically in animals. Approximately 80% cesium absorption was observed in dogs acutely exposed to small amounts of aerosolized ^{137}Cs (as cesium chloride) (Boecker 1969a, 1969b). Deposition and distribution of cesium

following inhalation exposure to radiolabeled cesium chloride was also observed in rats (Lie 1964; Stara and Thomas 1963) and guinea pigs (Stara 1965). Absorption was rapid following inhalation exposure.

3.5.1.2 Oral Exposure

It is generally accepted that cesium ingested as soluble cesium compounds is well absorbed by the gastrointestinal tract of humans and animals. Observations indicating that soluble cesium compounds were absorbed after ingestion by humans include: (1) low fecal excretory rates, (2) urinary excretory rates 4–10 times higher than those of fecal excretion, and (3) elimination half-times ranging from 45 to 147 days (Henrichs et al. 1989; Iinuma et al. 1965; Richmond et al. 1962; Rosoff et al. 1963). Henrichs et al. (1989) estimated an average cesium absorption of 78% in a group of 10 adult (5 male, 5 female) volunteers ingesting a meal of venison that was highly contaminated with ^{137}Cs and ^{134}Cs. Results from other controlled studies on human subjects indicate that absorption represented >90% of the cesium ingested in soluble form (Rosoff et al. 1963; Rundo 1964; Yamataga et al. 1966).

Absorption of ^{137}Cs from ingestion of radioactive fallout particles was found to be in the range of only 3%, indicating that such particles are relatively insoluble in biological fluids (LeRoy et al. 1966). Measurable amounts of ^{137}Cs were found in breast milk of women living in areas contaminated with radioactive fallout from the Chernobyl nuclear accident (Johansson et al. 1998). Based on whole body measurements of radioactivity in mothers and nursing infants and measured radioactivity in breast milk samples, it was estimated that 15% of the mothers' daily ^{137}Cs intake from contaminated food was transferred to the infant.

Animal studies support the findings in humans. Rapid absorption and widespread distribution of cesium was reported in guinea pigs administered single oral doses of soluble ^{137}Cs (as cesium chloride); fractional absorption data were not reported (Stara 1965). In rats orally administered single doses of highly insoluble irradiated fuel particles (mean diameter of 0.93 μm) containing ^{137}Cs and other radioactive elements, absorption of ^{137}Cs was found to be <10% (Talbot et al. 1993).

3.5.1.3 Dermal Exposure

Dermal absorption has been demonstrated in rats (Pendic and Milivojevic 1966). Traces of ^{137}Cs were observed in the blood of rats within a few minutes following the dermal application of $^{137}CsCl$ in aqueous

solution. Approximately 3% of $^{137}CsCl$ applied to a surface area of several cm^2 was absorbed within 6 hours.

3.5.2 Distribution

3.5.2.1 Inhalation Exposure

Once absorbed, cesium is rapidly distributed throughout the body. Separate measurements of radioactivity from head, chest, upper abdomen, lower abdomen, thighs, legs, and feet indicated that ^{137}Cs was widely distributed throughout the bodies of two men who were occupationally exposed to ^{137}Cs (Miller 1964). Proportions of radioactivity in these body segments remained relatively constant from days 9 to 285 after exposure, indicating that ^{137}Cs was not likely to have been selectively accumulating in a particular region.

Animal studies also indicate a relatively uniform distribution of cesium following inhalation exposure to soluble cesium compounds (Boecker 1969a, 1969b; Stara 1965). Within 2 hours following exposure to aerosols of ^{137}Cs (as cesium chloride), up to 60% of the total body burden of ^{137}Cs was found in tissues other than respiratory or gastrointestinal tracts of dogs (Boecker 1969a). At 32 days post exposure, concentrations of ^{137}Cs in skeletal muscles, diaphragm, kidneys, and mandibular salivary gland were slightly higher than the whole-body average; concentrations in lung, skin, bone (femur, ribs), fat, and blood were somewhat lower (Boecker 1969b). Other tissues exhibited concentrations approximating the whole-body average. A relatively uniform distribution of ^{137}Cs in numerous body tissues, with the highest concentrations in skeletal muscle, was also observed in guinea pigs and rats exposed by inhalation (Lie 1964; Stara 1965; Stara and Thomas 1963).

3.5.2.2 Oral Exposure

Widespread distribution of cesium was observed in humans following oral exposure to soluble cesium compounds. In two human subjects orally administered ^{137}Cs (as cesium chloride), whole blood levels of ^{137}Cs within the first hour after administration amounted to approximately 2–3% of the amount administered, indicating that ^{137}Cs was rapidly absorbed and well distributed via the circulation (Rosoff et al. 1963).

Animal studies also showed relatively uniform distribution following oral exposure to soluble cesium compounds. Guinea pigs exhibited ^{137}Cs in numerous body tissues after receiving single oral doses of ^{137}Cs (as cesium chloride). The highest concentrations were found in skeletal muscle (Stara 1965). After the first day post administration, no significant differences in ^{137}Cs distribution patterns were observed between groups of guinea pigs exposed by inhalation, oral administration, or intraperitoneal injection of ^{137}Cs (as cesium chloride) (Stara 1965). Dogs and mice exhibited relatively uniform distribution of cesium throughout body tissues following chronic oral administration of ^{137}Cs (as cesium chloride) (Furchner et al. 1964). Cesium also crossed the placenta of animals and was found in breast milk. Newborn lambs exhibited lower tissue levels of ^{134}Cs than their mothers following oral administration of radiolabeled cesium chloride during pregnancy (Vandecasteele et al. 1989). The concentrations of ^{134}Cs in nursing lambs eventually exceeded the levels in their mothers.

3.5.2.3 Dermal Exposure

One report was located regarding distribution of cesium in animals following dermal exposure (Pendic and Milivojevic 1966). The investigators found widespread distribution of ^{137}Cs in rats within a few minutes following application of $^{137}CsCl$ solution to the skin. Although cesium was distributed throughout the body, it was deposited mainly in the kidneys, muscular tissues (particularly cardiac muscle), and liver.

3.5.2.4 Other Routes of Exposure

Comparative human and animal studies have shown that parenteral exposure to cesium compounds results in cesium distribution patterns similar to those observed following inhalation or oral exposure (Rosoff et al. 1963; Stara 1965). By 3 days following administration, ^{137}Cs was found to be distributed relatively uniformly among the body tissues of five cancer patients who died at various times following intravenous injection of a single tracer dose of ^{137}Cs (as cesium chloride). Time-related decreases were observed in ^{137}Cs retention by all tissues surveyed (Rosoff et al. 1963). Transfer of ^{137}Cs from pregnant dam to fetus has been shown in rats following intraperitoneal injection of radiolabeled cesium chloride (Mahlum and Sikov 1969).

3.5.3 Metabolism

Absorbed cesium behaves in a manner similar to that of potassium (Rundo 1964; Rundo et al. 1963). Both potassium and cesium are alkali metals that distribute throughout the body as cations, and are incorporated into intracellular fluids by active transport mechanisms. Cesium has been shown to compete with potassium for transport through potassium channels and can also substitute for potassium in activation of the sodium pump and subsequent transport into the cell (Cecchi et al. 1987; Edwards 1982; Hodgkin 1947; Latorre and Miller 1983; Sjodin and Beauge 1967). In both types of transport, movement of cesium is sluggish compared with that of potassium (Blatz and Magleby 1984; Coronado et al. 1980; Cukierman et al. 1985; Edwards 1982; Gay and Stanfield 1978; Gorman et al. 1982; Hille 1973; Reuter and Stevens 1980); that is, transport mechanisms generally favor potassium over cesium. Discrimination between potassium and cesium generally is greater for passive transport out of cells (selectivity ratios of Cs:K for a variety of tissues ranging from <0.02 to approximately 0.2) than for active transport into cells (selective Cs:K ratio approximating 0.25) (Leggett et al. 2003). This results in a greater residence time of cesium than potassium in muscle cells and hence in the whole body, since skeletal muscle contains most of the body's potassium or cesium at equilibrium. However, cesium appears to compete somewhat more favorably with potassium during transport out of red blood cells (Forth et al. 1963) or across or between epithelial cells (Cereijido et al. 1981; Greger 1981; Wright 1972).

3.5.4 Elimination and Excretion

3.5.4.1 Inhalation Exposure

Urinary excretion is the major route of elimination of cesium taken into the body in soluble form. In dogs exposed to ^{137}Cs (as cesium chloride) by inhalation, rates of excretion of ^{137}Cs in the urine and feces were highest in the first 3 days after exposure, accounting for approximately 12 and 3% of the initial body burden, respectively. Urinary and fecal excretion of ^{137}Cs continued at lower rates through 130 days of analysis. Rates of elimination were determined by measuring the cesium remaining in the organs of rats sacrificed at 9 time points during 120 days following treatment. The elimination rates for specific tissues (muscle, kidney, liver, and lung) were similar to the whole-body rates, indicating that cesium did not selectively accumulate in any particular tissues (Boecker 1969b). Within 2.5 days following inhalation exposure to ^{137}Cs (as cesium chloride), guinea pigs had eliminated approximately 50% of the initial ^{137}Cs body burden in the urine and feces (Stara 1965). The urinary to fecal ratio for excretion was approximately 3:1 throughout 60 days of post-exposure measurements, by which time, virtually all of the initial ^{137}Cs body burden had been eliminated. The urinary to fecal ratio for elimination of cesium in rats

was about 3.5:1, with a biological half-time of elimination of approximately 4 days; 99% had been eliminated within 65 days following exposure (Stara and Thomas 1963).

No reports were located regarding routes of elimination and excretion of cesium in humans following inhalation exposure. However, kinetics of ^{137}Cs elimination in two adult males, accidentally exposed to ^{137}Cs (as cesium sulfate), were studied by whole-body measurements of gamma emission. The measured biological half-times were 73 and 84 days (Miller 1964). In the case of another adult male, accidentally exposed to airborne ^{137}Cs twice in a 13-month period, biological half-times for elimination were 72 and 73 days (Hölgye and Malý 2002).

In dogs exposed by inhalation, elimination rates of ^{137}Cs from specific tissues were similar to the rate of whole-body elimination, indicating that ^{137}Cs did not selectively accumulate in certain tissues, but was relatively uniformly eliminated from the body with a half-time of approximately 36–42 days (Boecker 1969b). Elimination rates of ^{137}Cs in guinea pigs and rats exposed by inhalation also did not vary significantly according to tissue type, although much shorter half-times of ^{137}Cs elimination (2.5 and 4 days) were observed for guinea pigs and rats, respectively (Stara 1965; Stara and Thomas 1963).

Relatively insoluble inhaled particles containing cesium were not absorbed in significant amounts and were more slowly eliminated from the lungs.

3.5.4.2 Oral Exposure

Urinary excretion is the primary route of elimination for cesium in humans. Among seven cancer or pulmonary patients who were administered single oral doses of ^{137}Cs (as cesium chloride), 7-day cumulative excretion of ^{137}Cs ranged from 7.0 to 17.3% of the administered activity. The urinary to fecal excretion ratio ranged from 2.5:1 to 10:1 (Rosoff et al. 1963). In a study of four Japanese volunteers orally administered single doses of ^{137}Cs (as cesium chloride), urinary to fecal excretion ratios ranging from 4.57:1 to 8.75:1 were calculated for excretory data collected after day 4 post administration. During the first 4 days after administration, excretory rates were consistently higher and the urinary:fecal excretory ratio was also somewhat higher (Iinuma et al. 1965). Based on the results of numerous reports of urinary and fecal excretion of Cs in human subjects, Leggett et al. (2003) reported an average urinary fraction (i.e., the ratio of cumulative urinary Cs to cumulative Cs in urine plus feces) of 0.86. Other sources of information on excretion rates of ^{137}Cs include numerous studies of populations exposed via fallout following atmospheric testing of nuclear weapons and from the Chernobyl accident, and

mathematical models, such as those described in Section 3.5.5, have been developed to describe the relationships among intake, retention, and elimination of cesium.

Animal data support the findings in humans of urinary excretion as the major route of elimination of cesium following oral administration of soluble cesium compounds. Guinea pigs had eliminated approximately 50% of the initial ^{137}Cs body burden in the urine and feces within 2.5 days following administration (Stara 1965). The urinary to fecal ratio was within the range of 2–3:1 throughout 60 days of post exposure measurements, by which time, virtually all of the initial ^{137}Cs body burden had been eliminated.

Elimination half-times for cesium in the whole body, sometimes expressed in terms of whole-body radioactivity retention, have been reported by some investigators (Henrichs et al. 1989; Iinuma et al. 1967; Lloyd et al. 1973; Melo et al. 1997; Richmond et al. 1962; Rundo 1964). For example, among 10 volunteers who consumed ^{134}Cs- and ^{137}Cs-contaminated food, approximately 6% of the initial body burden was rapidly eliminated (average half-time of elimination of 0.3 days); the remaining 94% was eliminated more slowly (average half-time of elimination of 90 days) (Henrichs et al. 1989). In another oral study of four adult males, a mean elimination half-time was 135 days for ^{134}Cs and ^{137}Cs (Richmond et al. 1962).

Elimination rates for ^{137}Cs appear to be age- and sex-dependent, decreasing with age and lower in adult males than adult females. Results of studies of populations that consumed food containing ^{137}Cs from weapons testing fallout showed elimination half-times that varied from 15±5 days in infants to 100±50 days in adults (McCraw 1965). Similar studies after the Chernobyl accident found similar elimination half-times, which ranged from about 8 days for 1-year-old infants to about 110 days for adults (IAEA 1991). A 4-year study of 110 persons comprising a cross-section within an unspecified population indicated that children, 5–14 years of age, had the shortest elimination half-times (20 days, with no significant difference between males and females) (Boni 1969b). The elimination half-times in older groups were significantly longer (47 days for adolescent and adult females, 67 days in 15-year-old males, and 93 days in males 30–50 years of age). Melo et al. (1994) also reported age- and sex-related differences in elimination rates among individuals internally contaminated by $^{137}CsCl$ in the Goiânia, Brazil accident (see Agency for Toxic Substances and Disease Registry 1999 for a complete description of the incident). Elimination half-times for girls 1–4 years of age averaged 24 days. For 7–10-year-old girls and boys, the average elimination half-time was 37 days. Elimination half-times of 58 and 83 days were estimated for adolescent and adult males, respectively; compared with 46 and 66 days for adolescent

and adult females, respectively. In the study of Melo et al. (1994), a high correlation was found between biological half-time for ^{137}Cs and weight for all age groups and sexes, except adult females.

Elimination rates of cesium may be altered by potassium intake. Following the intraperitoneal injection of ^{137}Cs in rats, a basal diet supplemented with 8–11% potassium resulted in cesium clearance of 60 days compared to about 120 days for rats receiving the unsupplemented basal diet that contained 1% potassium (Richmond and Furchner 1961). After 20 days on the diets, rats receiving supplemental potassium had body burdens of ^{137}Cs that were one-half those of the rats not receiving supplemental potassium. This finding shows that supplemental potassium reduces the uptake and increases the elimination of ingested ^{137}Cs.

Retention of cesium was lower in males suffering from muscular dystrophy than in age-matched controls. Older males with advanced signs of muscular dystrophy had lower retention than younger males exhibiting earlier stages of the disease (Lloyd et al. 1973).

Cesium crosses the placenta from mother to fetus. Measurable amounts of ^{137}Cs have been detected in human placenta and fetal tissue (Toader et al. 1996; Yoshioka et al. 1976). Cesium concentrations are higher in older fetuses than in younger ones (Toader et al. 1996). Pregnancy may increase the removal of cesium from the mother, as indicated by shorter elimination half-times during pregnancy relative to measurements taken before or after pregnancy or among nonpregnant controls (Bengtsson et al. 1964; Rundo and Turner 1966; Thornberg and Mattsson 2000; Zundel et al. 1969). Cesium has also been detected in human breast milk (Thornberg and Mattsson 2000).

3.5.4.3 Dermal Exposure

No reports were located regarding elimination and excretion of cesium in humans or animals following dermal exposure.

3.5.4.4 Other Routes of Exposure

Evidence for age-related differences in cesium elimination rates was observed in rats injected with ^{134}Cs or ^{137}Cs (as cesium chloride) at various ages. Retention of radiocesium from a single intraperitoneal injection of 0.5 μCi of carrier-free $^{134}CsCl$ increased with increasing age of rats at the time of dosing (Lengemann 1970). For example, the amount of radiocesium retained at 49 days after dosing was

>14 times greater in rats dosed at 4 months of age than in rats dosed at 1 month of age. Retention in rats injected at 21 months of age was approximately 1.9 times greater than that of the rats dosed at 4 months of age. Long-term retention also appears to be age related in dogs injected intravenously with $^{137}CsCl$; puppies 3–5 months of age exhibited elimination half-times that were shorter than those of adult dogs (Melo et al. 1997). Age-related increases in cesium retention rates were also observed in other young dogs from 61 to approximately 300 days old, after which cesium retention reached a plateau; the increases in cesium retention were similar to growth curves (Tyler et al. 1969). There is some indication, however, that retention of cesium is higher in neonatal rats than in weanling or adult rats (Lengemann 1969, 1970; Mahlum and Sikov 1969).

3.5.5 Physiologically Based Pharmacokinetic (PBPK)/Pharmacodynamic (PD) Models

Physiologically based pharmacokinetic (PBPK) models use mathematical descriptions of the uptake and disposition of chemical substances to quantitatively describe the relationships among critical biological processes (Krishnan et al. 1994). PBPK models are also called biologically based tissue dosimetry models. PBPK models are increasingly used in risk assessments, primarily to predict the concentration of potentially toxic moieties of a chemical that will be delivered to any given target tissue following various combinations of route, dose level, and test species (Clewell and Andersen 1985). Physiologically based pharmacodynamic (PBPD) models use mathematical descriptions of the dose-response function to quantitatively describe the relationship between target tissue dose and toxic end points.

PBPK/PD models refine our understanding of complex quantitative dose behaviors by helping to delineate and characterize the relationships between: (1) the external/exposure concentration and target tissue dose of the toxic moiety, and (2) the target tissue dose and observed responses (Andersen and Krishnan 1994; Andersen et al. 1987). These models are biologically and mechanistically based and can be used to extrapolate the pharmacokinetic behavior of chemical substances from high to low dose, from route to route, between species, and between subpopulations within a species. The biological basis of PBPK models results in more meaningful extrapolations than those generated with the more conventional use of uncertainty factors.

The PBPK model for a chemical substance is developed in four interconnected steps: (1) model representation, (2) model parameterization, (3) model simulation, and (4) model validation (Krishnan and Andersen 1994). In the early 1990s, validated PBPK models were developed for a number of toxicologically important chemical substances, both volatile and nonvolatile (Krishnan and Andersen

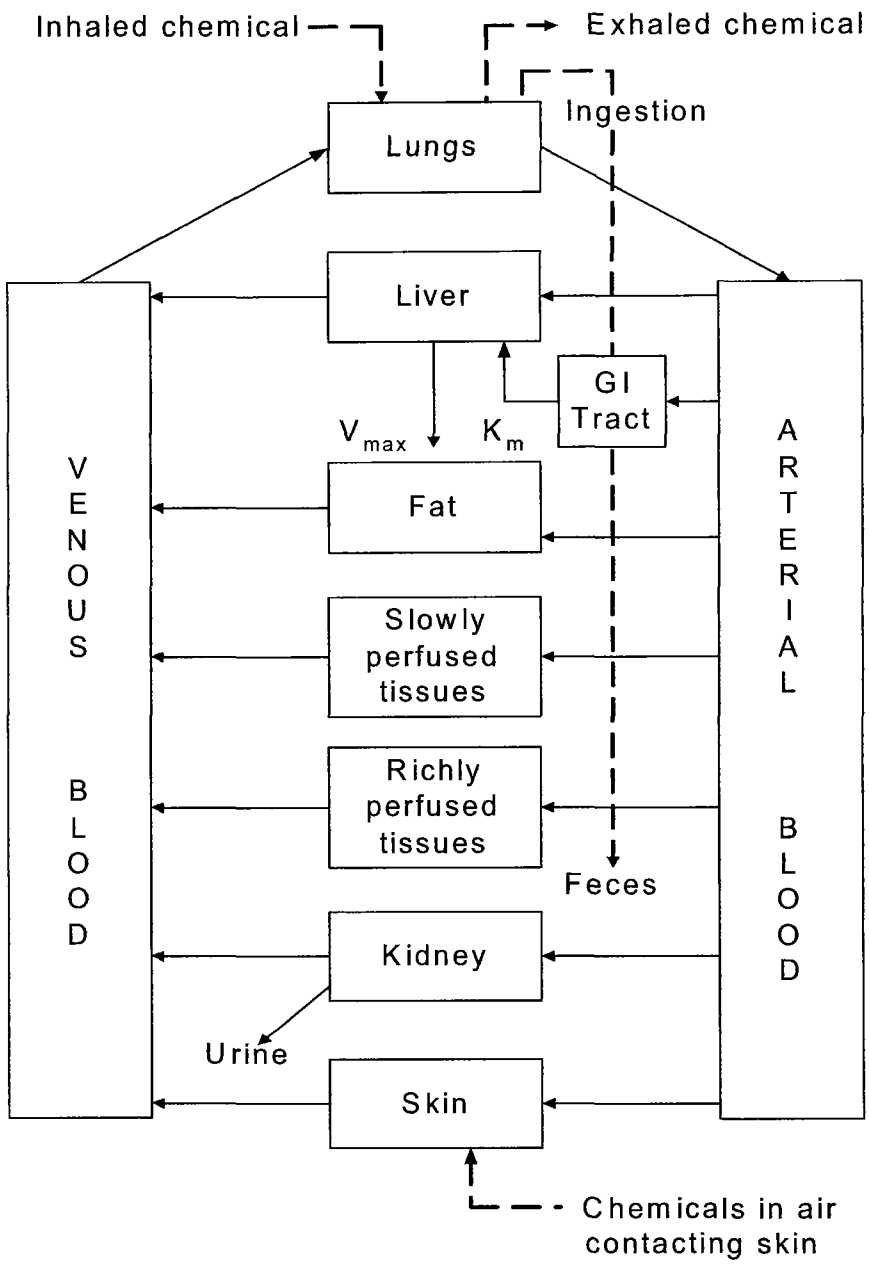
1994; Leung 1993). PBPK models for a particular substance require estimates of the chemical substance-specific physicochemical parameters, and species-specific physiological and biological parameters. The numerical estimates of these model parameters are incorporated within a set of differential and algebraic equations that describe the pharmacokinetic processes. Solving these differential and algebraic equations provides the predictions of tissue dose. Computers then provide process simulations based on these solutions.

The structure and mathematical expressions used in PBPK models significantly simplify the true complexities of biological systems. If the uptake and disposition of the chemical substance(s) is adequately described, however, this simplification is desirable because data are often unavailable for many biological processes. A simplified scheme reduces the magnitude of cumulative uncertainty. The adequacy of the model is, therefore, of great importance, and model validation is essential to the use of PBPK models in risk assessment.

PBPK models improve the pharmacokinetic extrapolations used in risk assessments that identify the maximal (i.e., the safe) levels for human exposure to chemical substances (Andersen and Krishnan 1994). Similar models have been developed for radionuclides. These PBPK models provide a scientifically sound means to predict the target tissue dose of chemicals and radiation in humans who are exposed to environmental levels (for example, levels that might occur at hazardous waste sites) based on the results of studies where doses were higher or were administered in different species. Figure 3-3 shows a conceptualized representation of a PBPK model. Figures 3-4 through 3-7 show models for radionuclides in general or specifically for cesium.

The International Commission on Radiological Protection (ICRP 1994, 1995) developed a Human Respiratory Tract Model for Radiological Protection, which contains respiratory tract deposition and clearance compartmental models for inhalation exposure that may be applied to particulate aerosols of cesium compounds. The ICRP (1979, 1989, 1993) also developed a 2-compartment biokinetic model for human oral exposure that applies to cesium. EPA (1998) adopted the ICRP (1993, 1994, 1995) models for assessment of radiologic risks from cesium exposures. The National Council on Radiation Protection and Measurements (NCRP) also developed a respiratory tract model for inhaled radionuclides (NCRP 1997). At this time, the NCRP recommends the use of the ICRP model for calculating radiation doses for workers and the general public. Readers interested in this topic are referred to NCRP Report No. 125; *Deposition, Retention and Dosimetry of Inhaled Radioactive Substances* (NCRP 1997). In the appendix

Figure 3-3. Conceptual Representation of a Physiologically Based Pharmacokinetic (PBPK) Model for a Hypothetical Chemical Substance



Source: adapted from Krishnan et al. 1994

Note: This is a conceptual representation of a physiologically based pharmacokinetic (PBPK) model for a hypothetical chemical substance. The chemical substance is shown to be absorbed via the skin, by inhalation, or by ingestion, metabolized in the liver, and excreted in the urine or by exhalation.

to the report, NCRP provides the animal testing clearance data and equations fitting the data that supported the development of the human model for cesium.

Human Respiratory Tract Model for Radiological Protection (ICRP 1994)

Deposition. The ICRP (1994) developed a deposition model to describe the behavior of inhaled aerosols and vapors in the respiratory tract. This model was developed to estimate the fractions of radioactivity in breathing air that deposit in each anatomical region of the respiratory tract. ICRP (1994) provides inhalation dose coefficients, which can be used to estimate the committed equivalent and effective doses to organs and tissues throughout the body based on a unit intake of radioactive material. The model applies to three levels of particle solubility, a wide range of particle sizes (approximately 0.0005–100 μm in diameter), and parameter values. The model may be adjusted for various segments of the population (e.g., sex, age, level of physical exertion). This model also allows the evaluation of the bounds of uncertainty in deposition estimates. Uncertainties arise from natural biological variability among individuals. The ICRP model is applicable to particulate aerosols containing cesium, but was developed for a wide variety of radionuclides and their chemical forms.

The ICRP deposition model may be used to estimate the amount of inhaled material that initially enters each compartment (see Figure 3-4). The model was developed with 5 compartments: (1) the anterior nasal passages (ET_1); (2) all other extrathoracic airways (ET_2) (posterior nasal passages, the naso- and oropharynx, and the larynx); (3) the bronchi (BB); (4) the bronchioles (bb); and (5) the alveolar interstitium (AI). Particles deposited in the ET_1 region may be cleared either by dissolution and absorption into the blood or by nose-blowing. Particles deposited in each of the other regions may then be removed from each region and redistributed either upward into the respiratory tree by mucociliary clearance mechanisms, or to the lymphatic system and blood by different particle removal mechanisms.

For extrathoracic deposition of particles, the model is based on experimental data, where deposition is related to particle size and airflow parameters. The model scales deposition for women and children from adult male data. Similarly to the extrathoracic region, experimental data served as the basis for lung (bronchi, bronchioles, and alveoli) aerosol transport and deposition. A theoretical model of gas transport and particle deposition was used to interpret data and to predict deposition for compartments and subpopulations other than adult males. Table 3-5 provides reference respiratory values for the general Caucasian population and for several levels of physical activity.

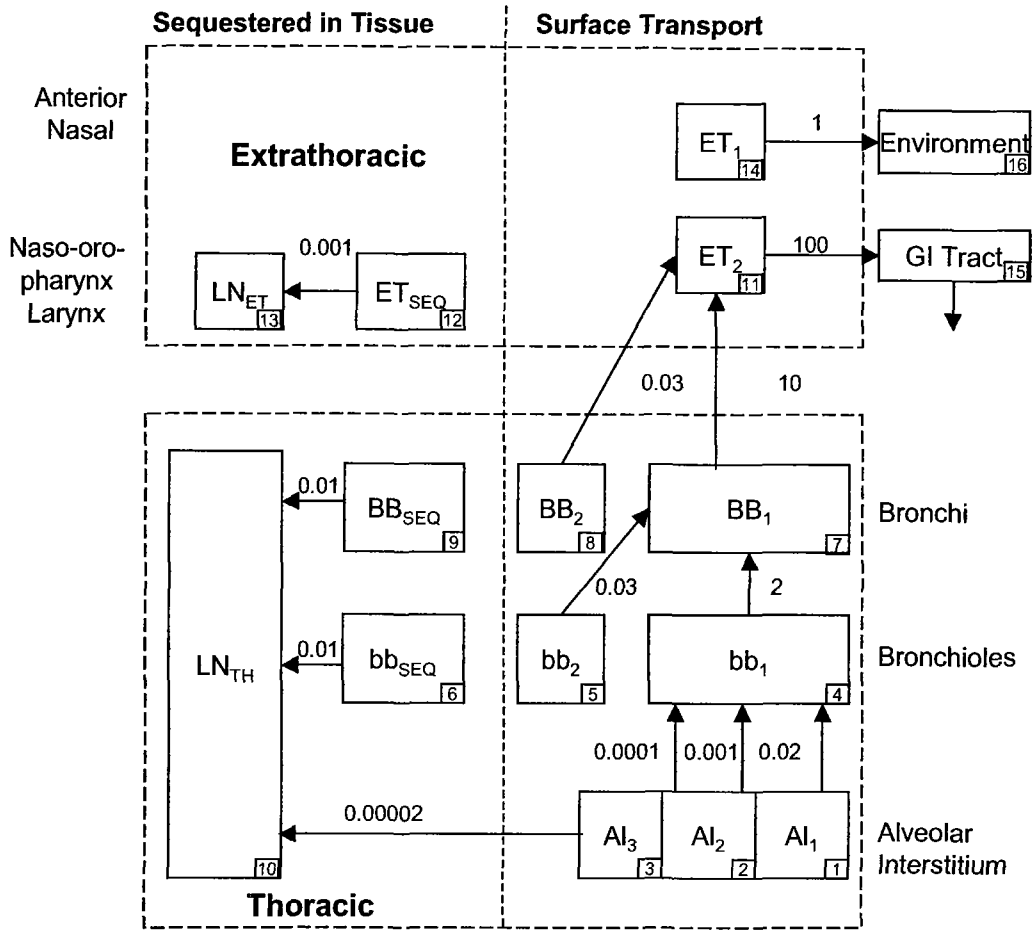
Table 3-5. Reference Respiratory Values for a General Caucasian Population at Different Levels of Activity[a]

Activity:		Resting (sleeping)			Sitting awake			Light exercise			Heavy exercise		
Maximal workload (%):		8			12			32			64		
Breathing parameters:[b]		V_T (L)	B (m^3h^{-1})	f_R (min^{-1})	V_T (L)	B (m^3h^{-1})	f_R (min^{-1})	V_T (L)	B (m^3h^{-1})	f_R (min^{-1})	V_T (L)	B (m^3h^{-1})	f_R (min^{-1})
Age	Sex												
3 months		0.04	0.09	38	N/A	N/A	N/A	0.07	0.19	48	N/A	N/A	N/A
1 year		0.07	0.15	34	0.1	0.22	36	0.13	0.35	46	N/A	N/A	N/A
5 years		0.17	0.24	23	0.21	0.32	25	0.24	0.57	39	N/A	N/A	N/A
10 years	Both:	0.3	0.31	17	0.33	0.38	19	0.58	1.12	32			
	Male:										0.841	2.22	44
	Female:										0.667	1.84	46
15 years	Male:	0.500	0.42	14	0.533	0.48	15	1.0	1.38	23	1.352	2.92	36
	Female:	0.417	0.35	14	0.417	0.40	16	0.903	1.30	24	1.127	2.57	38
Adult	Male:	0.625	0.45	12	0.750	0.54	12	1.25	1.5	20	1.923	3.0	26
	Female:	0.444	0.32	12	0.464	0.39	14	0.992	1.25	21	1.364	2.7	33

[a]See Annex B (ICRP 1994) for data from which these reference values were derived.
[b]B = ventilation rate; f_R = respiration frequency; V_T = tidal volume

H = hour; L = liter; m = meter; min = minute; N/A = not applicable

Figure 3-4. Compartment Model to Represent Particle Deposition and Time-Dependent Particle Transport in the Respiratory Tract*



*Compartment numbers shown in lower right corners are used to define clearance pathways. The clearance rates, half-lives, and fractions by compartment, as well as the compartment abbreviations are presented in Table 3-6.

Source: ICRP 1994

Deposition of inhaled gases and vapors is modeled as a partitioning process that depends on the physiological parameters noted above as well as the solubility and reactivity of compounds in the respiratory tract (see Figure 3-5). The ICRP (1994) model defines three categories of solubility and reactivity: SR-0, SR-1, and SR-2:

- Type SR-0 compounds include insoluble and nonreactive gases (e.g., inert gases such as H_2, He). These compounds do not significantly interact with the respiratory tract tissues and essentially all the inhaled gas is exhaled. Radiation doses from inhalation of SR-0 compounds are assumed to result from the irradiation of the respiratory tract from the air spaces.

- Type SR-1 compounds include soluble or reactive gases and vapors that are expected to be taken up by the respiratory tract tissues and may deposit in any or all of the regions of the respiratory tract, depending on the dynamics of the airways and properties of the surface mucous and airway tissues, as well as the solubility and reactivity of the compound.

- Type SR-2 compounds include soluble and reactive gases and vapors that are completely retained in the extrathoracic regions of the respiratory tract. SR-2 compounds include sulfur dioxide (SO_2) and hydrogen fluoride (HF).

Respiratory Tract Mechanical (Particle) Clearance. The clearance portion of the model identifies the principal clearance pathways within the respiratory tract. The model was developed to predict the retention and clearance of various radioactive materials. The compartmental model is linked to the deposition model (see Figure 3-4) and to reference values presented in Table 3-6. Table 3-6 provides clearance rates by biological processes only, not by radioactive decay, and deposition fractions for each compartment for insoluble particles. The table provides rates of insoluble particle transport for each of the compartments, expressed as a fraction per day and also as half-time. ICRP (1994) also developed modifying factors for some of the parameters, such as age, smoking, and disease status. Parameters of the clearance model are based on human data, although particle retention in airway walls is based on experimental data from animal experiments.

The clearance of particles from the respiratory tract is a dynamic process. The rate of clearance generally changes with time from each region and by each route. Following deposition of large numbers of particles over a short time period (acute exposure), transport rates change as particles are cleared from the various regions. Physical and chemical properties of deposited material determine the rate of dissolution. As particles dissolve, absorption rates also tend to change over time. By creating a model with compartments of different clearance rates within each region (e.g., BB_1, BB_2, BB_{seq}), the ICRP model

Table 3-6. Reference Values of Parameters for the Compartment Model to Represent Time-dependent Particle Transport from the Human Respiratory Tract

Part A: Clearance rates for insoluble particles

Pathway	From	To	Rate (d^{-1})	Half-time[a]
$m_{1,4}$	AI_1	bb_1	0.02	35 days
$m_{2,4}$	AI_2	bb_1	0.001	700 days
$m_{3,4}$	AI_3	bb_1	0.0001	7,000 days
$m_{3,10}$	AI_3	LN_{TH}	0.00002	—
$m_{4,7}$	bb_1	BB_1	2	8 hours
$m_{5,7}$	bb_2	BB_1	0.03	23 days
$m_{6,10}$	bb_{seq}	LN_{TH}	0.01	70 days
$m_{7,11}$	BB_1	ET_2	10	100 minutes
$m_{8,11}$	BB_2	ET_2	0.03	23 days
$m_{9,10}$	BB_{seq}	LN_{TH}	0.01	70 days
$m_{11,15}$	ET_2	GI tract	100	10 minutes
$m_{12,13}$	ET_{seq}	LN_{ET}	0.001	700 days
$m_{14,16}$	ET_1	Environment	1	17 hours

See next page for Part B

Table 3-6. Reference Values of Parameters for the Compartment Model to Represent Time-dependent Particle Transport from the Human Respiratory Tract

Part B: Partition of deposit in each region between compartments[b]

Region or deposition site	Compartment	Fraction of deposit in region assigned to compartment[c]
ET_2	ET_2	0.9995
	ET_{seq}	0.0005
BB	BB_1	$0.993-f_s$
	BB_2	f_s
	BB_{seq}	0.007
bb	bb_1	$0.993-f_s$
	bb_2	f_s
	bb_{seq}	0.007
AI	AI_1	0.3
	AI_2	0.6
	AI_3	0.1

[a]The half-times are approximate since the reference values are specified for the particle transport rates and are rounded in units of d^{-1}. A half-time is not given for the transport rate from AI_3 to LN_{TH}, since this rate was chosen to direct the required amount of material to the lymph nodes. The clearance half-time of compartment AI_3 is determined by the sum of the clearance rates from it.
[b]See paragraph 181, Chapter 5 (ICRP 1994) for default values used for relating f_s to d_{ae}.
[c]It is assumed that f_s is size-dependent. For modeling purposes, f_s is taken to be:

$$f_s = 0.5 \; for \; d_{ae} \leq 2.5\sqrt{\rho / \chi} \; \mu m \; and$$

$$f_s = 0.5e^{0.63(d_{ae}\sqrt{\rho/\chi}-2.5)} \; for \; d_{ae} > 2.5\sqrt{\rho / \chi} \; \mu m$$

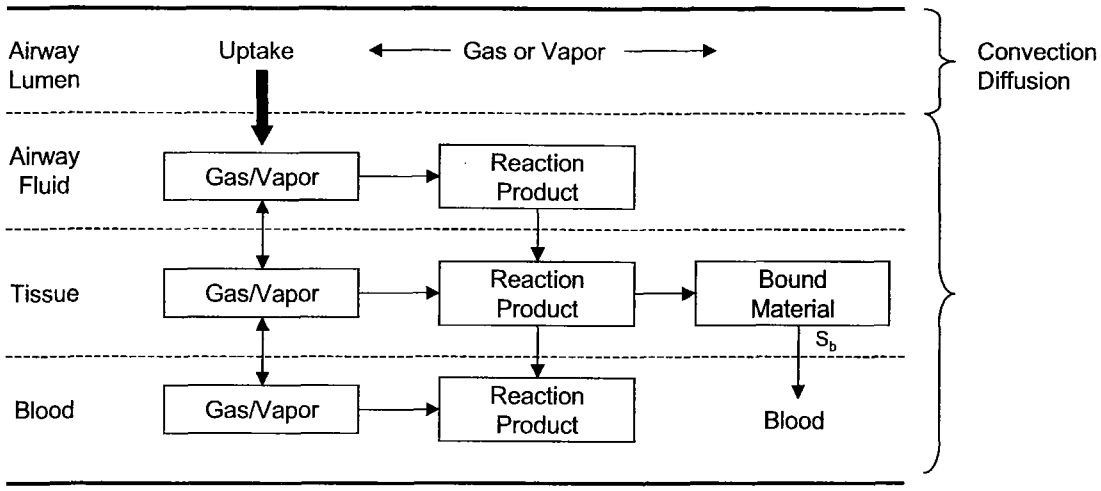
where:

f_s = fraction subject to slow clearance
d_{ae} = aerodynamic particle diameter/(μm)
ρ = particle density (g/cm^3)
χ = particle shape factor

AI = alveolar-interstitial region; BB = bronchial region; bb = bronchiolar region; BB_{seq} = compartment representing prolonged retention in airway walls of small fraction of particles deposited in the bronchial region; bb_{seq} = compartment representing prolonged retention in airway walls of small fraction of particles deposited in the bronchiolar region; d = day(s); ET = extrathoracic region; ET_{seq} = compartment representing prolonged retention in airway tissue of small fraction of particles deposited in the nasal passages; GI tract = gastrointestinal tract; LN_{ET} = lymphatics and lymph nodes that drain the extrathoracic region; LN_{TH} = lymphatics and lymph nodes that drain the thoracic region

Source: ICRP 1994

Figure 3-5. Reaction of Gases or Vapors at Various Levels of the Gas-Blood Interface



Source: ICRP 1994

overcomes problems associated with time-dependent functions. Each compartment clears to other compartments by constant rates for each pathway.

Particle transport from all regions is toward both the lymph nodes and the pharynx. A majority of deposited particles are eventually swallowed. In the front part of the nasal passages (ET_1), nose blowing, sneezing, and wiping remove most of the deposited particles. Particles remain in the nasal passages for about a day. For particles with AMADs of a few micrometers or greater, the ET_1 compartment is probably the largest deposition site. A majority of particles deposited at the back of the nasal passages and in the larynx (ET_2) are removed quickly by the mucous fluids that cover the airways. In this region, particle clearance is completed within 15 minutes.

Ciliary action removes deposited particles from the bronchi and bronchioles. Though mucociliary action rapidly transports most particles deposited here toward the pharynx, a fraction of these particles is cleared more slowly. Evidence for this clearance is found in human studies. For humans, retention of particles deposited in the lungs (BB and bb) is apparently biphasic. The "slow" action of the cilia may remove as many as half of the bronchi- and bronchiole-deposited particles. In human bronchi and bronchiole regions, mucus movement is influenced by location. Movement is slower in areas closer to alveoli. It takes about 2 days for particles to travel from the bronchioles to the bronchi and 10 days from the bronchi to the pharynx. The second (slower) compartment is assumed to have approximately equal fractions deposited between BB_2 and bb_2 and both with clearance half-times estimated at 20 days. Particle size is a primary determinant of the fraction deposited in this slow thoracic compartment. A small fraction of particles deposited in the BB and bb regions may be retained in the airway wall for even longer periods (BB_{seq} and bb_{seq}).

If particles reach and become deposited in the alveoli, they tend to stay imbedded in the fluid on the alveolar surface or move into the lymph nodes. The mechanism by which particles are physically resuspended and removed from the AI region is coughing. For modeling purposes, the AI region is divided into three subcompartments representing different slow clearance rates, all of which are slow.

Particle clearance from the alveolar-interstitial region has been measured in human subjects. The ICRP model uses 2 half-times to represent clearance. About 30% of the particles have a 30-day half-time, and the remaining 70% are given a half-time of several hundred days. Over time, AI particle transport diminishes and some insoluble particles may remain in lungs 10–50 years after exposure.

Absorption into Blood. The ICRP model assumes that absorption into blood occurs at equivalent rates in all parts of the respiratory tract, except in the anterior nasal passages (ET_1), where no absorption occurs. Absorption is essentially a 2-stage process, as shown in Figure 3-6. First, there is dissociation (dissolution) of particles. The dissolved molecules or ions then diffuse across capillary walls and are taken up by the blood. Immediately following dissolution, rapid absorption takes place. For some elements, rapid absorption does not occur because of binding to respiratory-tract components. In the absence of specific data for specific compounds, the model uses the following default absorption rate values for those specific compounds that are classified as Types F (fast), M (medium), S (slow), and V (instantaneous):

- For Type F, there is rapid 100% absorption within 10 minutes of the material deposited in the BB, bb, and AI regions, and 50% of material deposited in ET2. Thus, for nose breathing, there is rapid absorption of approximately 25% of the deposit in ET and 50% for mouth breathing.

- For Type M, about 70% of the deposit in AI reaches the blood eventually. There is rapid absorption of about 10% of the deposit in BB and bb, and 5% of material deposited in ET2. Thus, there is rapid absorption of approximately 2.5% of the deposit in ET for nose breathing, and 5% for mouth breathing.

- For Type S, 0.1% is absorbed within 10 minutes and 99.9% is absorbed within 7,000 days, so there is little absorption from ET, BB, or bb, and about 10% of the deposit in AI reaches the blood eventually.

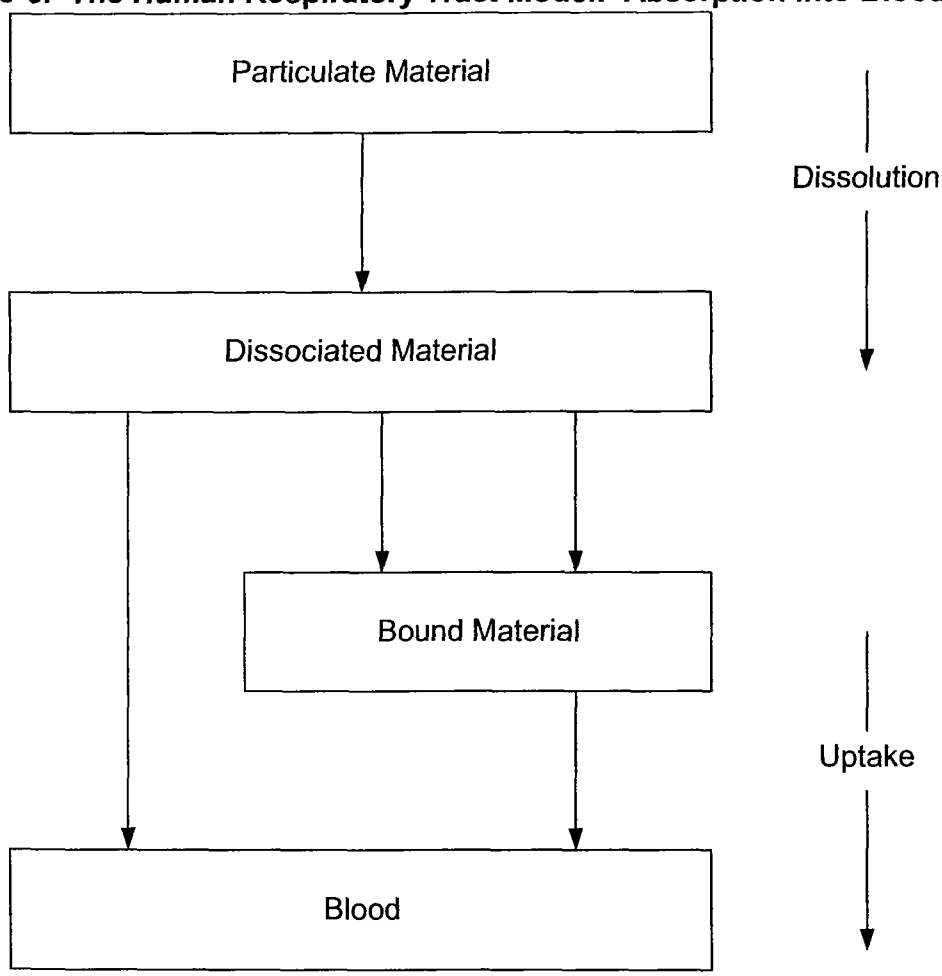
- For Type V, complete absorption (100%) is considered to occur instantaneously.

ICRP (1995) considers the experimental and human data to support the following classifications: cesium chloride and nitrate, Type F; cesium in irradiated fuel fragments, Type F or M; and cesium in fused aluminosilicate particles, M or S. ICRP (1995) recommends assigning all cesium aerosols to Type F in the absence of specific information supporting an alternative classification.

ICRP (1993) Cesium Biokinetic Model

Description of the Model. ICRP (1979, 1989, 1993) developed a 2-compartment model of the kinetics of ingested cesium in humans (Figure 3-6) that is applicable to infants, children, adolescents, and adults. The model is based on an age- and gender-specific model of Leggett (1986) in which compartment sizes and retention half-times were expressed as a function of the mass of potassium in the body. Ingested cesium is assumed to be completely absorbed into blood. Absorbed cesium is then assumed to distribute

Figure 3-6. The Human Respiratory Tract Model: Absorption into Blood



Source: ICRP 1994

uniformly in the body and to be eliminated from fast and slow elimination pools. The fraction of the total body cesium associated with the fast and slow pools, as well as the elimination half-times from each pool, are assumed to vary with age. The elimination half-times vary for ages 3 months, 1, 5, 10, 15 years, and adult (>15 years). The contribution of the fast pool decreases from 45% at age 5 years to 10% in adults. The elimination half-times of the fast pool decrease from 9.1 days at age 5 years to 2 days in adults, whereas the elimination half-times of the slow pool increase from 13–16 days in infants to 110 days in adults.

Validation of the Model. The extent to which the ICRP model has been validated is not described in ICRP (1993).

Risk Assessment. The ICRP biokinetic model has been used to establish radiation dose equivalents (Sv/Bq) of ingested ^{134}Cs, ^{136}Cs, and ^{137}Cs for ages 3 months to 70 years (ICRP 1993).

Target Tissues. The ICRP model is designed to calculate radiocesium intake limits based on radiation dose to all major organs.

Species Extrapolation. The ICRP model is designed for applications to human dosimetry and cannot be applied to other species without modification.

Interroute Extrapolation. The ICRP model is designed to simulate oral exposure assuming 100% absorption of ingested cesium. The model is applicable to other routes of exposure if the extent of absorption for a particular exposure route is known.

Leggett et al. (2003) Cesium Biokinetics Model

Description of the Model. Leggett et al. (2003) developed a PBPK model of the kinetics of ingested or injected cesium in humans (Figure 3-7). The model includes compartments representing blood (plasma and red blood cells), brain, gastrointestinal tract, heart, kidney, liver, lung, pancreas, skin, and spleen. Transfers between plasma and tissues are simulated as first-order, flow-limited, rate constants (Table 3-7). Flow limitation is imposed by deriving the plasma-to-tissue rate constants in terms of tissue blood flows and empirically-derived arterial-venous extraction fractions (Table 3-8). Rate constants for transfers from tissues to plasma are derived from the product of the plasma-to-tissue rate constants and empirically-derived equilibrium ratios for cesium (fraction of body burden) in plasma and tissue (Table 3-9).

Table 3-7. Transfer Coefficients (d^{-1}) for a Reference Adult Male

Plasma to heart	14.128
Plasma to liver	19.515
Plasma to kidneys	67.108
Plasma to muscle	30.022
Plasma to gastrointestinal tract tissue	52.98
Plasma to stomach contents	4.516
Plasma to small intestine contents	1.0480
Plasma to large intestine contents	0.02
Plasma to spleen	5.298
Plasma to pancreas	1.766
Plasma to brain	0.424
Plasma to red marrow	5.298
Plasma to other skeleton	3.532
Plasma to skin	4.415
Plasma to lungs	4.415
Plasma to adipose tissue	8.83
Plasma to other 1[a]	8.826
Plasma to other 2[b]	0.00353
Plasma to red blood cells	1.8
Heart to plasma	8.073
Liver to plasma	2.204
Liver to small intestine	0.116
Kidneys to urinary bladder contents	1.678
Kidneys to plasma	31.876
Muscle to plasma	0.0751
Gastrointestinal tract tissue to plasma	8.191
Gastrointestinal tract tissue to liver	0.431
Gastrointestinal tract tissue to stomach contents	0.0333
Gastrointestinal tract tissue to small intestine contents	0.108
Gastrointestinal tract tissue to large intestine contents	0.0667
Spleen to plasma	5.033
Spleen to liver	0.265
Pancreas to plasma	1.678
Pancreas to liver	0.0883
Skin to plasma	0.867
Skin to excreta	0.0159
Brain to plasma	0.0848
Red marrow to plasma	0.706
Other skeleton to plasma	0.128
Lungs to plasma	1.472
Adipose tissue to plasma	1.766
Other 1[a] to plasma	0.692

Table 3-7. Transfer Coefficients (d^{-1}) for a Reference Adult Male

Other 2[b] to plasma	0.00141
Red blood cells to plasma	0.257
Urinary bladder contents to urine	12.0
Stomach to small intestine (contents)	40.0
Small intestine contents to plasma	28.215
Small intestine to large intestine (contents)	0.3
Small intestine contents to liver	1.485
Large intestine contents to feces	0.5

[a]Remaining tissues and fluids in the body with a relatively short turnover time.
[b]Small component of the remaining tissues and fluids in the body with a tenacious retention of Cs apparent in some long-term studies.

Source: Leggett et al. 2003

Table 3-8. Tissue-specific Extraction Fractions Assumed for Cesium

Tissues	Extraction fraction
Kidneys, gastrointestinal tract, heart	0.2
Liver, skin	0.05
Brain	0.002
All other tissues	0.1

Source: Leggett et al. 2003

Table 3-9. Tissue Masses, Cesium Equilibrium Distribution, and Tissue Blood Flow for a Reference Adult Male

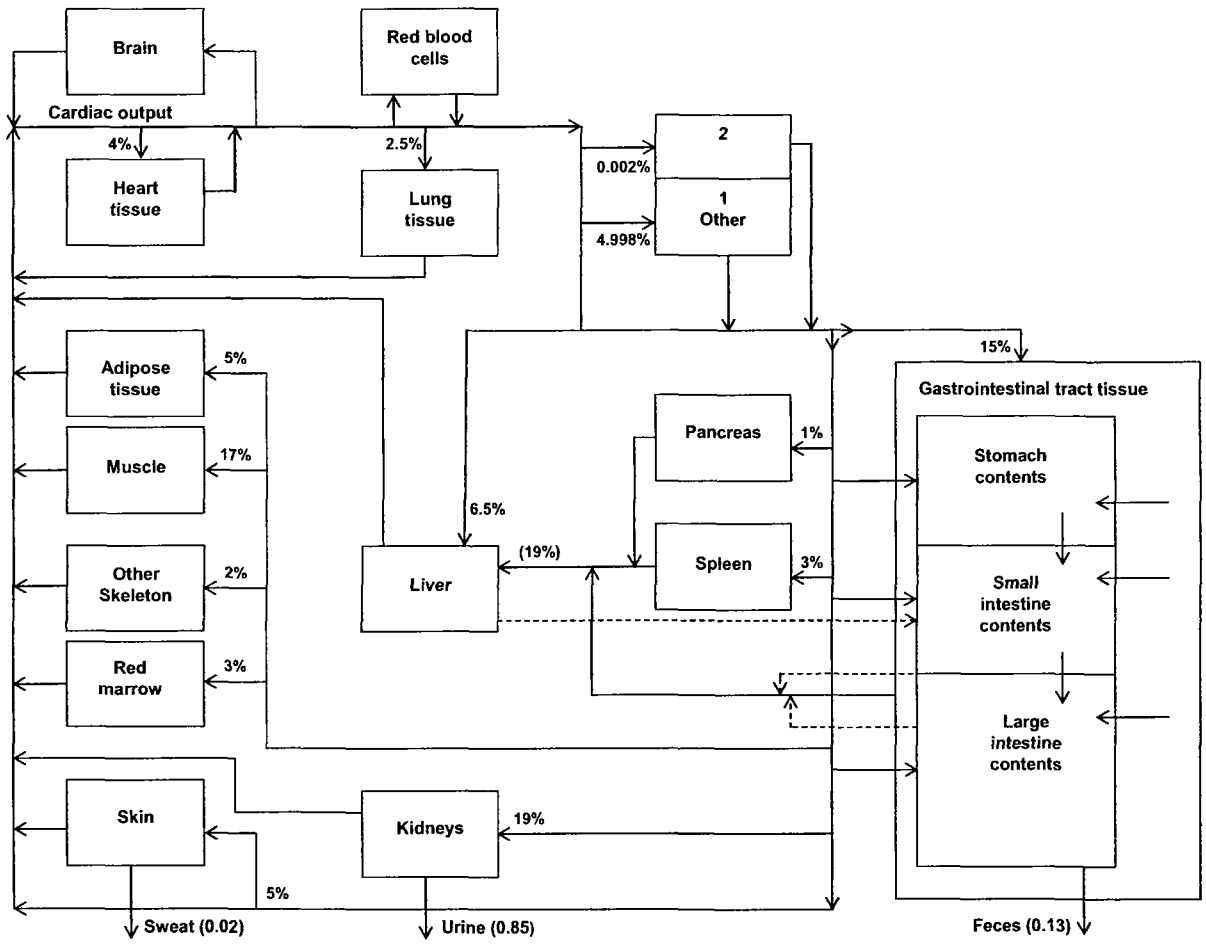
Compartment	Mass[a] (g)	Equilibrium cesium content[a] (fraction of total-body cesium)	Blood flow (FCO)
Adipose tissue[b]	12,000	0.01	0.05
Brain	1,450	0.01	0.12
Gastrointestinal contents	900	0.004	–
Gastrointestinal tract tissue	1,170	0.015	0.15
Heart	330	0.0035	0.04
Kidneys	310	0.004	0.19
Liver	1,800	0.02	0.065 (arterial) (0.19) (portal)
Lungs	500	0.006	0.025
Skeletal muscle	29,000	0.8	0.17
Plasma	3,100	0.002	–
Red blood cells	2,500	0.014	–
Skeleton	10,500	0.07	0.05
Red marrow		(0.015)	(0.03)
Bone and other tissue		(0.055)	(0.02)
Skin	3,300	0.01	0.05
Spleen	150	0.002	0.03
Pancreas	140	0.002	0.01
Other[c]	5,850	0.0305	0.05
Totals	73,000	1.00	1.00

[a]Without blood.
[b]Separable adipose tissue excluding yellow bone marrow.
[c]Remaining tissues and fluids in the body.

FCO = fraction of cardiac output

Source: Leggett et al. 2003

Figure 3-7. Directions of Flow of Cesium



Solid arrows represent plasma flow and broken arrows represent flow not involving plasma. Percentages indicate distribution of carbon monoxide. Numbers besides sweat, urine, and feces are fractions of cumulative excretion.

Source: Leggett et al. (2003).

Excretory pathways represented in the model include feces, sweat, and urine. Transfers between the central plasma flow and the gastrointestinal tract include secretion into the gastrointestinal tract and absorption to plasma.

Validation of the Model. The model has been evaluated for predicting observed blood and plasma cesium concentrations, whole-body retention of cesium, and urinary clearance of cesium in adults, following injections or ingestion of cesium (Leggett et al. 2003). Additional evaluations are reported, based on evaluations in rats and dogs. Model predictions corresponded reasonably well with observations.

Risk Assessment. The model is intended for predicting internal tissue doses to radiation following exposures to cesium for use in radiation risk assessment.

Target Tissues. The model is designed to calculate cesium concentrations in tissues, and includes major sites of accumulation (e.g., kidney, liver, and muscle).

Species Extrapolation. The model is designed for applications to human dosimetry and can be applied to other species (e.g., dog and rat) with modification of physiological parameters.

Interroute Extrapolation. The model is designed to simulate the intravenous or oral exposures to cesium and can be applied to other routes of exposure with the addition of simulations or estimates of the uptake of cesium into the central blood compartment.

3.6 MECHANISMS OF ACTION

3.6.1 Pharmacokinetic Mechanisms

Cesium is rapidly absorbed into blood following inhalation or oral exposure to soluble cesium compounds, as demonstrated by the rapid distribution of cesium activity after inhalation or ingestion. Approximately 80% absorption of ^{137}Cs was observed in dogs exposed to aerosols containing ^{137}Cs (as cesium chloride) (Boecker 1969a, 1969b). Oral ingestion of ^{134}Cs- and ^{137}Cs-contaminated food by volunteers resulted in approximately 78% absorption. Animal studies indicate that absorption rates from orally administered soluble cesium compounds are highest in the duodenum, followed in order by the jejunum, ileum, and colon. Very little absorption occurs in the stomach or caecum (Moore and Comar 1962, 1963). Absorption rates are higher in fasted rats than in fed rats, indicating that stomach contents

may influence the rate of cesium absorption. Relatively insoluble forms of cesium compounds, which may sometimes be associated with irradiated fuel particles, are poorly absorbed by inhalation and oral exposure routes (Boecker et al. 1974, 1977; LeRoy et al. 1966; Talbot et al. 1993). Dermal retention, but not transdermal absorption, has been qualitatively demonstrated in humans (Rundo 1964). Dermal absorption was observed following application of ^{137}CsCl in aqueous solution to the skin of rats (Pendic and Milivojevic 1966). Traces of ^{137}Cs were observed in the blood of rats within a few minutes following application.

Once absorbed, cesium is rapidly distributed throughout the body, becoming incorporated into the intracellular fluid of numerous tissues. Animal studies indicate that distribution patterns are similar following absorption from inhalation or oral exposure and that concentrations of cesium within muscle tissue are somewhat higher than the whole-body average (Stara 1965). Comparative human and animal studies have shown that parenteral exposure to cesium compounds results in cesium distribution patterns similar to those following inhalation or oral exposure (Rosoff et al. 1963; Stara 1965).

Absorbed cesium behaves in a manner similar to that of potassium. Both potassium and cesium are alkali metals that are distributed throughout the body as cations, becoming incorporated into intracellular fluids. Cesium has been shown to compete with potassium for transport through potassium channels and can also substitute for potassium in activation of the sodium pump and subsequent transport into the cell (Cecchi et al. 1987; Edwards 1982; Hodgkin 1947; Latorre and Miller 1983; Sjodin and Beauge 1967). In both types of transport, movement of cesium is sluggish compared with that of potassium (Blatz and Magleby 1984; Coronado et al. 1980; Cukierman et al. 1985; Edwards 1982; Gay and Stanfield 1978; Gorman et al. 1982; Hille 1973; Reuter and Stevens 1980). Discrimination between potassium and cesium generally is greater for passive transport out of cells than for active transport into cells (Leggett et al. 2003). This results in a greater residence time of cesium than potassium in muscle cells and hence in the whole body, since skeletal muscle contains most of the body's potassium or cesium at equilibrium. Although the biokinetics of potassium and cesium may vary somewhat in such characteristics as relative affinity for various cell types and differing retention rates, the similarities allow elimination rates for potassium to be used as an index of elimination rates for cesium (USNRC 1983). Urinary excretion is the major route of elimination of cesium.

3.6.2 Mechanisms of Toxicity

Earth contains relatively small amounts of stable (nonradioactive) cesium. Cesium has few industrial applications. At environmental levels, stable cesium is not chemically toxic in animals. Cesium is not likely to be of toxic concern to humans exposed to cesium by inhalation, oral, or dermal contact. Although a number of investigators have reported cesium-induced alterations in behavior or cardiac activity in animals systems exposed to cesium chloride by parenteral injection, underlying mechanisms are not yet fully understood.

Cesium may have both depressant and antidepressant properties in rodents, as it was shown to decrease the conditioned avoidance response of pole-climbing (Bose and Pinsky 1983b) and to reduce vertical and horizontal motor activity (Bose and Pinsky 1981, 1984; Bose et al. 1981; Pinsky et al. 1980), while enhancing amphetamine-induced hyperactivity and reducing the locomotor depressive action of reserpine (Messiha 1978).

Increased vertical activity (rearing), but not horizontal activity, was observed in mice given repeated injections of cesium chloride (Johnson 1972). Rastogi et al. (1980) found no increase in behavioral activity in rats repeatedly injected with cesium chloride, but noted a number of biochemical changes in the brain that included a statistically significant rise in tyrosine hydroxylase activity that resulted in a slight but statistically significant increase in tyrosine levels, markedly enhanced levels of the neurotransmitters norepinephrine and dopamine, and increased levels of a norepinephrine metabolite (4-hydroxy-3-methoxyphenylglycol). Cesium appeared to block the uptake of norepinephrine by synaptosomes.

Cesium was shown to alter normal cardiac rhythm, triggering short-lived early after depolarizations (EADs) and polymorphic ventricular tachyarrhythmias (VTs) in canine myocardial muscle fibers and Purkinje cells (Brachmann et al. 1983; Levine et al. 1985; Murakawa et al. 1997; Patterson et al. 1990), effects that are similar to those observed in humans with congenital and acquired long QT syndrome (Bonatti et al. 1983). Prolonged QT syndrome and associated cardiac arrhythmia have been observed in patients who consumed cesium chloride as a component of homeopathic remedies (Bangh et al. 2001; Harik et al. 2002; Saliba et al. 2001). Available animal data suggest that cesium-induced EADs and VTs were most likely the result of ionic imbalance due to reduced potassium permeability (Isenberg 1976) and imbalances of intra- and extracellular concentrations of calcium and sodium (Szabo et al. 1987).

Both ^{134}Cs and ^{137}Cs emit gamma radiation, and therefore, radioactive cesium is a health hazard. Highly penetrating gamma rays are the major cause of damage to tissues and internal organs following external overexposure to radioactive cesium. Once radioactive cesium is taken internally, cells of nearby tissues are at highest risk for damage due to the emission of beta particles. Radiation-induced damage in cells may be repaired quickly. Misrepaired damage may lead to permanent DNA changes and the potential for carcinogenesis. Very large acute radiation doses can damage or kill enough cells to cause the disruption of organ systems (acute radiation syndrome), harm to developing fetuses, and even death. Human and animal data indicate that radioactive cesium overexposure can result in adverse effects such as reduced fertility, abnormal neurological development, genotoxicity, and damage to blood-forming organs (Bartstra et al. 1998; Matsuda et al. 1985; Nikula et al. 1995, 1996; Padovani et al. 1993; Ramaiya et al. 1994; Skandalis et al. 1997; Tobari et al. 1988). For a more complete discussion of the mechanisms associated with the toxic effects of ionizing radiation, refer to Chapter 5 of the Toxicological Profile for Ionizing Radiation (Agency for Toxic Substances and Disease Registry 1999).

3.6.3 Animal-to-Human Extrapolations

No data were located to indicate significant interspecies differences in pharmacokinetics or health effects associated with exposure to stable or radioactive cesium.

3.7 TOXICITIES MEDIATED THROUGH THE NEUROENDOCRINE AXIS

Recently, attention has focused on the potential hazardous effects of certain chemicals on the endocrine system because of the ability of these chemicals to mimic or block endogenous hormones. Chemicals with this type of activity are most commonly referred to as *endocrine disruptors*. However, appropriate terminology to describe such effects remains controversial. The terminology *endocrine disruptors*, initially used by Colborn and Clement (1992), was also used in 1996 when Congress mandated the Environmental Protection Agency (EPA) to develop a screening program for "...certain substances [which] may have an effect produced by a naturally occurring estrogen, or other such endocrine effect[s]...". To meet this mandate, EPA convened a panel called the Endocrine Disruptors Screening and Testing Advisory Committee (EDSTAC), which in 1998 completed its deliberations and made recommendations to EPA concerning *endocrine disruptors*. In 1999, the National Academy of Sciences released a report that referred to these same types of chemicals as *hormonally active agents*. The terminology *endocrine modulators* has also been used to convey the fact that effects caused by such chemicals may not necessarily be adverse. Many scientists agree that chemicals with the ability to disrupt

or modulate the endocrine system are a potential threat to the health of humans, aquatic animals, and wildlife. However, others think that endocrine-active chemicals do not pose a significant health risk, particularly in view of the fact that hormone mimics exist in the natural environment. Examples of natural hormone mimics are the isoflavinoid phytoestrogens (Adlercreutz 1995; Livingston 1978; Mayr et al. 1992). These chemicals are derived from plants and are similar in structure and action to endogenous estrogen. Although the public health significance and descriptive terminology of substances capable of affecting the endocrine system remains controversial, scientists agree that these chemicals may affect the synthesis, secretion, transport, binding, action, or elimination of natural hormones in the body responsible for maintaining homeostasis, reproduction, development, and/or behavior (EPA 1997). Stated differently, such compounds may cause toxicities that are mediated through the neuroendocrine axis. As a result, these chemicals may play a role in altering, for example, metabolic, sexual, immune, and neurobehavioral function. Such chemicals are also thought to be involved in inducing breast, testicular, and prostate cancers, as well as endometriosis (Berger 1994; Giwercman et al. 1993; Hoel et al. 1992).

No studies were located regarding endocrine disruptive effects resulting from exposure to stable or radioactive cesium.

3.8 CHILDREN'S SUSCEPTIBILITY

This section discusses potential health effects from exposures during the period from conception to maturity at 18 years of age in humans, when all biological systems will have fully developed. Potential effects on offspring resulting from exposures of parental germ cells are considered, as well as any indirect effects on the fetus and neonate resulting from maternal exposure during gestation and lactation. Relevant animal and *in vitro* models are also discussed.

Children are not small adults. They differ from adults in their exposures and may differ in their susceptibility to hazardous chemicals. Children's unique physiology and behavior can influence the extent of their exposure. Exposures of children are discussed in Section 6.6, Exposures of Children.

Children sometimes differ from adults in their susceptibility to hazardous chemicals, but whether there is a difference depends on the chemical (Guzelian et al. 1992; NRC 1993). Children may be more or less susceptible than adults to health effects, and the relationship may change with developmental age (Guzelian et al. 1992; NRC 1993). Vulnerability often depends on developmental stage. There are critical periods of structural and functional development during both prenatal and postnatal life and a

particular structure or function will be most sensitive to disruption during its critical period(s). Damage may not be evident until a later stage of development. There are often differences in pharmacokinetics and metabolism between children and adults. For example, absorption may be different in neonates because of the immaturity of their gastrointestinal tract and their larger skin surface area in proportion to body weight (Morselli et al. 1980; NRC 1993); the gastrointestinal absorption of lead is greatest in infants and young children (Ziegler et al. 1978). Distribution of xenobiotics may be different; for example, infants have a larger proportion of their bodies as extracellular water and their brains and livers are proportionately larger (Altman and Dittmer 1974; Fomon 1966; Fomon et al. 1982; Owen and Brozek 1966; Widdowson and Dickerson 1964). The infant also has an immature blood-brain barrier (Adinolfi 1985; Johanson 1980) and probably an immature blood-testis barrier (Setchell and Waites 1975). Many xenobiotic metabolizing enzymes have distinctive developmental patterns. At various stages of growth and development, levels of particular enzymes may be higher or lower than those of adults, and sometimes unique enzymes may exist at particular developmental stages (Komori et al. 1990; Leeder and Kearns 1997; NRC 1993; Vieira et al. 1996). Whether differences in xenobiotic metabolism make the child more or less susceptible also depends on whether the relevant enzymes are involved in activation of the parent compound to its toxic form or in detoxification. There may also be differences in excretion, particularly in newborns who all have a low glomerular filtration rate and have not developed efficient tubular secretion and resorption capacities (Altman and Dittmer 1974; NRC 1993; West et al. 1948). Children and adults may differ in their capacity to repair damage from chemical insults. Children also have a longer remaining lifetime in which to express damage from chemicals; this potential is particularly relevant to cancer.

Certain characteristics of the developing human may increase exposure or susceptibility, whereas others may decrease susceptibility to the same chemical. For example, although infants breathe more air per kilogram of body weight than adults breathe, this difference might be somewhat counterbalanced by their alveoli being less developed, which results in a disproportionately smaller surface area for alveolar absorption (NRC 1993).

Soluble cesium compounds are readily absorbed into body fluids and bloodstream and are widely distributed throughout the body (see Section 3.5 for detailed information). PBPK models are used to simulate potential age-related differences in deposition of inhaled cesium, as well as differences in elimination rates for absorbed cesium (see Section 3.5.5 for more information on PBPK models). Although inhalation exposure to environmental levels of stable or radioactive cesium is not considered to be a major health concern, age-related differences in physical properties of the respiratory system and

ventilation patterns could result in differences in absorption rates of inhaled soluble or insoluble cesium compounds. Soluble cesium compounds are assumed to be completely absorbed in the gastrointestinal tract, with no adjustments for age. Available human and animal data do not indicate that age-related differences might exist for absorption and distribution of cesium following oral exposure. Since cesium is principally absorbed and distributed in ionic form (as Cs^+), any age-related differences in absorption following oral exposure would likely be the result of differences in diffusion rates and active transport mechanisms involved in the movement of cesium through extra- and intracellular fluids. Elimination rates for cesium appear to be age-related and may be most closely related to body mass. As described in detail in Section 3.5.4.2, young children exhibit whole-body biological elimination half-times that are much shorter than those of older children and adults (Boni 1969b; Melo et al. 1994). It is not known whether or not these age-related differences may be due to higher retention of cesium in adult tissues and lower rates of excretion.

Measurable amounts of ^{137}Cs have been found in the breast milk of women living in areas contaminated with radioactive fallout. Transfers to newborns and 1-year-old children were estimated to be approximately 40 and 50%, respectively (Johansson et al. 1998). Animal studies have shown that cesium crosses the placental barrier, but cesium is found in lower concentrations in the fetus than in maternal or placental tissues (Mahlum and Sikov 1969; Vandecasteele et al. 1989).

Although no information was located regarding age-related health effects in humans exposed to stable cesium, age-related differences in the pharmacokinetics of stable cesium could conceivably result in age-related differences in health effects. No studies were located regarding age-related differences in toxicity in animals exposed to stable cesium.

Most of the available information regarding age-related health effects from overexposure to cesium concern developmental effects related to *in utero* irradiation of human or animal fetuses from an external source of radiation. Impaired cognitive function was observed in atomic bomb survivors overexposed to ionizing radiation *in utero* during critical stages of neurological development (Schull and Otake 1999). Developmental toxicity studies employing external gamma radiation from a radioactive cesium source (or from any other gamma ray source) indicate that rats and mice are most sensitive to the effects of external radiation around gestation day 14. Effects observed following irradiation during this period include reduced survival, decreased brain size, smaller head size, and retarded odontogenesis (Koshimoto et al. 1994; Minamisawa et al. 1990; Norton and Kimler 1987, 1988; Saad et al. 1991, 1994). When tested as adults, animals irradiated during this developmental period exhibit increased aggressive behavior

(Minamisawa et al. 1992; Norton and Kimler 1987, 1988). Although comparative studies of neurological effects in animals first irradiated as juveniles or adults were not located, it is apparent that there are critical stages of fetal developmental during which there is increased susceptibility to the effects of radiation. In these studies, although cesium was used as the gamma source, the effects were not unique to cesium. Similar results would be elicited by any gamma source.

3.9 BIOMARKERS OF EXPOSURE AND EFFECT

Biomarkers are broadly defined as indicators signaling events in biologic systems or samples. They have been classified as markers of exposure, markers of effect, and markers of susceptibility (NAS/NRC 1989).

Due to a nascent understanding of the use and interpretation of biomarkers, implementation of biomarkers as tools of exposure in the general population is very limited. A biomarker of exposure is a xenobiotic substance or its metabolite(s) or the product of an interaction between a xenobiotic agent and some target molecule(s) or cell(s) that is measured within a compartment of an organism (NAS/NRC 1989). The preferred biomarkers of exposure are generally the substance itself or substance-specific metabolites in readily obtainable body fluid(s), or excreta. However, several factors can confound the use and interpretation of biomarkers of exposure. The body burden of a substance may be the result of exposures from more than one source. The substance being measured may be a metabolite of another xenobiotic substance (e.g., high urinary levels of phenol can result from exposure to several different aromatic compounds). Depending on the properties of the substance (e.g., biologic half-life) and environmental conditions (e.g., duration and route of exposure), the substance and all of its metabolites may have left the body by the time samples can be taken. It may be difficult to identify individuals exposed to hazardous substances that are commonly found in body tissues and fluids (e.g., essential mineral nutrients such as copper, zinc, and selenium). Biomarkers of exposure to cesium are discussed in Section 3.9.1.

Biomarkers of effect are defined as any measurable biochemical, physiologic, or other alteration within an organism that, depending on magnitude, can be recognized as an established or potential health impairment or disease (NAS/NRC 1989). This definition encompasses biochemical or cellular signals of tissue dysfunction (e.g., increased liver enzyme activity or pathologic changes in female genital epithelial cells), as well as physiologic signs of dysfunction such as increased blood pressure or decreased lung capacity. Note that these markers are not often substance specific. They also may not be directly

adverse, but can indicate potential health impairment (e.g., DNA adducts). Biomarkers of effects caused by cesium are discussed in Section 3.9.2.

A biomarker of susceptibility is an indicator of an inherent or acquired limitation of an organism's ability to respond to the challenge of exposure to a specific xenobiotic substance. It can be an intrinsic genetic or other characteristic or a preexisting disease that results in an increase in absorbed dose, a decrease in the biologically effective dose, or a target tissue response. If biomarkers of susceptibility exist, they are discussed in Section 3.11, Populations that are Unusually Susceptible.

3.9.1 Biomarkers Used to Identify or Quantify Exposure to Cesium

Stable or radioactive isotopes of cesium may be measured in samples of urine, blood, feces, or body tissues by a number of methods outlined in Section 7.1. Stable cesium is of little toxicological concern. However, overexposure to radioactive isotopes of cesium may pose a significant health risk. Internal exposure may be quantified by direct counting (*in vivo* measurements) of radioactive emission from the body using whole-body counters capable of distinguishing the gamma emissions that are unique to radioactive isotopes of cesium. Radioactivity can be accurately measured in blood, excrement, and tissue samples using scintillation counting. The biomarkers that may help quantify exposure to stable or radioactive cesium are similar in children and adults.

3.9.2 Biomarkers Used to Characterize Effects Caused by Cesium

There are no known biomarkers of effect for exposure to stable cesium. High-level external or internal exposure to radioactive cesium can result in bone marrow aplasia, reduced white blood cell counts, decreased hemoglobin and platelet levels, and increased frequencies of chromosomal aberrations in lymphocytes. Frequencies of chromosomal aberrations were used to estimate external radiation doses among individuals in Goiânia, Brazil who had been exposed to an opened ^{137}CsCl source (Natarajan et al. 1998). These results are not unique to radioactive cesium. Similar results would be expected following overexposure to any source of ionizing radiation.

3.10 INTERACTIONS WITH OTHER CHEMICALS

No data were located regarding interactions of cesium with other chemicals that might influence the toxicity of cesium.

3.11 POPULATIONS THAT ARE UNUSUALLY SUSCEPTIBLE

A susceptible population will exhibit a different or enhanced response to cesium than will most persons exposed to the same level of cesium in the environment. Reasons may include genetic makeup, age, health and nutritional status, and exposure to other toxic substances (e.g., cigarette smoke). These parameters result in reduced detoxification or excretion of cesium, or compromised function of organs affected by cesium. Populations who are at greater risk due to their unusually high exposure to cesium are discussed in Section 6.7, Populations with Potentially High Exposures.

Increased susceptibility to the toxic effects resulting from exposure to high levels of stable or radioactive cesium might be indicated among individuals with abnormally low potassium intake, those with compromised kidney function, and patients taking stimulant or depressant drugs for the treatment of mental disorders (see Sections 3.5 and 3.6 for detailed information on the toxicokinetics and mechanisms of action of cesium). Individuals with compromised immune function might be more susceptible to the adverse effects of radiation overexposure from a radioactive cesium source (or from any other gamma emitting radioactive source).

Evidence for potential age-related differences in susceptibility to stable or radioactive cesium toxicity is provided by studies of elimination rates for ^{137}Cs in humans (Boni 1969b; Melo et al. 1994; Toader et al. 1996). Elimination rates are higher in young children than adults, and higher in adult females than adult males, indicating that lower elimination rates could result in greater retention and, therefore, increased toxicity for a given intake. Animal studies support these findings (Lengemann 1970; Mahlum and Sikov 1969; Melo et al. 1996, 1997; Tyler et al. 1969). There is some indication, however, that retention of cesium is higher in neonatal rats than in weanling or young adult rats (Lengemann 1969, 1970; Mahlum and Sikov 1969) (see Sections 3.5.4.2 and 3.5.4.4 for more detailed information.

Pregnant women (Zundel et al. 1969) and individuals suffering from muscular dystrophy (Lloyd et al. 1973) exhibited decreased cesium retention, which may decrease susceptibility to stable or radioactive cesium-induced toxicity.

3.12 METHODS FOR REDUCING TOXIC EFFECTS

This section will describe clinical practice and research concerning methods for reducing toxic effects of exposure to cesium. However, because some of the treatments discussed may be experimental and unproven, this section should not be used as a guide for treatment of exposures to cesium. When specific exposures have occurred, poison control centers and medical toxicologists should be consulted for medical advice. The following texts provide specific information about treatment following exposures to cesium:

Ellenhorn MJ, Schonwald S, Ordog G, et al., eds. 1997. Medical toxicology: Diagnosis and treatment of human poisoning. 2nd edition. Baltimore, MD: Williams & Wilkins, 1682-1723.

Haddad LM, Shannon MW, Winchester JF, eds. 1998. Clinical management of poisoning and drug overdose. 3rd edition. Philadelphia, PA: WB Saunders, 413-425.

NCRP Report No. 65. 1980. Management of persons accidentally contaminated with radionuclides. Bethesda MD: National Council on Radiation Protection and Measurements.

3.12.1 Reducing Peak Absorption Following Exposure

Because soluble cesium compounds are rapidly absorbed into blood following inhalation, oral, and dermal exposure, there are no prescribed methods for reducing peak absorption following exposure. Early counter measures that may aid in reducing peak absorption following oral exposure to stable or radioactive cesium include oral administration of insoluble Prussian blue (ferric hexacyanoferrate) that exchanges potassium for cesium, forming an insoluble complex that is eliminated through the feces (Oak Ridge Associated Universities 2003). Animal studies indicate that absorption of cesium may also be reduced by the administration of excess potassium (Richmond and Furchner 1961). Cathartics such as magnesium sulfate, as well as gastric lavage, will shorten the transit time of ingested cesium in the gastrointestinal tract (Ellenhorn et al. 1997; Gerber et al. 1992; Haddad et al. 1998). Countermeasures attempt to reduce the body burden of cesium following inadvertent exposure (see Section 3.12.2).

3.12.2 Reducing Body Burden

Oral administration of Prussian blue (potassium ferricyanoferrate) may enhance the fecal excretion of absorbed cesium (Ducousso et al. 1975; Ellenhorn et al. 1997; Gerber et al. 1992; Haddad et al. 1998;

Melo et al. 1996). Animal studies indicate that excretion of cesium may also be enhanced by the administration of excess potassium (Richmond and Furchner 1961). Higher plasma concentrations of potassium may increase the mobilization of cesium from tissues, and thus increase excretion.

3.12.3 Interfering with the Mechanism of Action for Toxic Effects

No data were located regarding reduction of the toxic effects of cesium through interfering with mechanisms of action.

3.13 ADEQUACY OF THE DATABASE

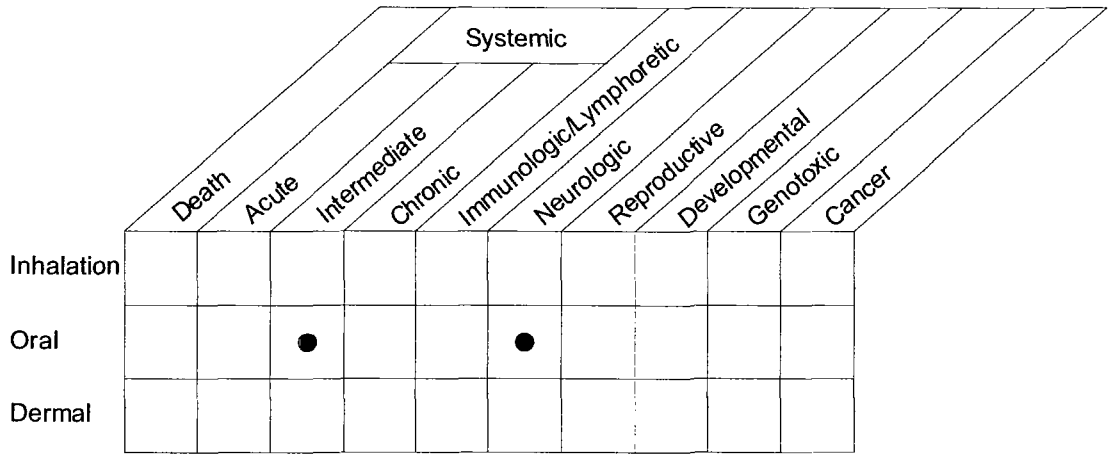
Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of cesium is available. Where adequate information is not available, ATSDR, in conjunction with the National Toxicology Program (NTP), is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of cesium.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

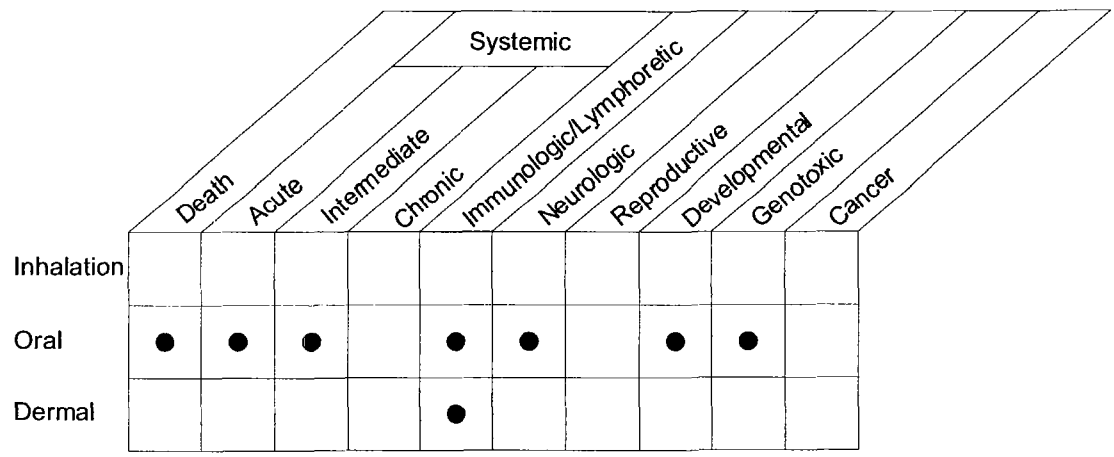
3.13.1 Existing Information on Health Effects of Cesium

The existing data on health effects of inhalation, oral, and dermal exposure of humans and animals to stable and radioactive cesium are summarized in Figure 3-8 and 3-9, respectively. The purpose of these figures is to illustrate the existing information concerning the health effects of cesium. Each dot in the figure indicates that one or more studies provide information associated with that particular effect. The dot does not necessarily imply anything about the quality of the study or studies, nor should missing information in this figure be interpreted as a "data need." A data need, as defined in ATSDR's Decision Guide for Identifying Substance-Specific Data Needs Related to Toxicological Profiles (Agency for Toxic

Figure 3-8. Existing Information on Health Effects of Stable Cesium



Human



Animal

● Existing Studies

Figure 3-9. Existing Information on Health Effects of Radioactive Cesium

	Death	Acute	Intermediate	Chronic	Immunologic/Lymphoretic	Neurologic	Reproductive	Developmental	Genotoxic	Cancer
	Systemic									
Inhalation										
Oral	●	●			●		●		●	
Dermal	●	●			●		●		●	
External	●	●			●		●		●	

Human

	Death	Acute	Intermediate	Chronic	Immunologic/Lymphoretic	Neurologic	Reproductive	Developmental	Genotoxic	Cancer
	Systemic									
Inhalation										
Oral		●					●		●	
Dermal										
External	●					●	●	●	●	●

Animal

● Existing Studies

Substances and Disease Registry 1989), is substance-specific information necessary to conduct comprehensive public health assessments. Generally, ATSDR defines a data gap more broadly as any substance-specific information missing from the scientific literature.

As shown in Figure 3-8, limited information is available regarding health effects in humans following intermediate-duration oral exposure to stable cesium. No information is available regarding health effects in humans following inhalation or dermal exposure to stable cesium. Information is available on the health effects in animals exposed to stable cesium. However, the available information is mostly from acute oral LD_{50} studies, a single intermediate-duration oral study, and a study of male reproductive toxicity.

As shown in Figure 3-9, limited information is available regarding oral and dermal exposure to radioactive cesium. An accidental exposure of a number of individuals in Goiânia, Brazil resulted in adverse health effects that could be attributed to external and internal (oral and dermal) exposure to radiation from a radioactive cesium source. Information is also available for humans externally exposed to a radiocesium source that resulted in adverse dermal effects. Numerous reports are available regarding other cases of external and internal environmental exposure to radioactive isotopes of cesium in humans, especially from areas with significant amounts of radioactive fallout. However, at present, associations between exposure to environmental levels of radioactive cesium and adverse health effects have not been confirmed. Present environmental levels of radiocesium, therefore, might not represent overexposure to radiation.

Reduced sperm counts in mice were found following oral administration of radioactive cesium. Studies of dogs, intravenously administered $^{137}CsCl$, resulted in hematologic dyscrasia as early effects and tumors of various organs as late effects. Given by this route, the tissue distribution of ^{137}Cs is similar to that resulting from oral or inhalation exposure.

3.13.2 Identification of Data Needs

Acute-Duration Exposure. Results from human and animal studies indicate that stable cesium is of little acute oral toxicity concern. Toxicokinetic data regarding the widespread distribution of cesium absorbed following oral exposure indicate that dermal and inhalation exposure to stable cesium would not present a greater health concern than that posed by oral exposure. Acute-duration inhalation and oral MRLs were not derived for stable cesium due to a lack of human or animal data. To generate appropriate

data for deriving acute-duration inhalation and oral MRLs for stable cesium, at least one comprehensive acute inhalation and one acute oral toxicity study would be needed of at least one animal species exposed to several dose levels.

Reports of adverse effects in humans that can be specifically attributed to acute exposure to radioactive cesium are restricted to the accounts of accidental external exposure (Gottlöber et al. 2000) and both external and internal (dermal and oral) exposure (Brandão-Mello et al. 1991) to an opened ^{137}CsCl source. Observed health effects were representative of those resulting from overexposure to other beta- and gamma-emitting sources of ionizing radiation. Animal data regarding acute oral exposure to radioactive cesium are limited to reports of dominant lethal mutations and reduced fertility in mice (Ramaiya et al. 1994). Acute-duration inhalation and oral MRLs were not derived for radioactive cesium due to a lack of human or animal data. To generate appropriate data for deriving acute-duration inhalation and oral MRLs for radioactive cesium, at least one comprehensive acute inhalation study and one acute oral toxicity study of at least one animal species exposed to several dose levels would be needed. Such studies could be designed to also generate data regarding potential age-related differences in toxicity. However, great danger would be posed to investigators considering the exposure of laboratory animals to radioactive cesium at levels great enough to cause significant adverse health effects. Distribution patterns of ^{137}Cs are similar in animals exposed to relatively nontoxic levels of ^{137}CsCl by parenteral injection, inhalation exposure, or oral administration (Boecker et al. 1969a; Stara 1965). Therefore, the results of the intravenous injection studies in dogs (Nikula et al. 1995, 1996) provide the most reasonable indication of health effects that would be expected in animals exposed by inhalation or oral administration. An acute-duration MRL that was derived by Agency for Toxic Substances and Disease Registry (1999) for acute external exposure to ionizing radiation was considered to be appropriate as an acute-duration MRL for external exposure to ionizing radiation from a radioactive cesium source.

Intermediate-Duration Exposure. Limited information regarding intermediate-duration oral exposure in humans indicates that the nervous and cardiovascular systems are the most likely targets of toxicity for high-dose stable cesium (Bangh et al. 2001; Harik et al. 2002; Neulieb 1984; Saliba et al. 2001). Limited data indicate that intermediate-duration oral exposure of pregnant mice to stable cesium may adversely affect developing fetuses (Messiha 1988b, 1989b). Orally administered stable cesium has also been shown to be genotoxic to female mice (Ghosh et al. 1990, 1991). Intermediate-duration inhalation and oral MRLs were not derived for stable cesium due to the paucity of human or animal data. To generate appropriate data for deriving intermediate-duration inhalation and oral MRLs for stable cesium, at least one comprehensive intermediate-duration inhalation and one intermediate-duration oral

toxicity study of at least one animal species exposed to several dose levels would be needed. Such studies could be designed to also generate data regarding potential age-related differences in toxicity.

No human or animal data are available in which intermediate-duration inhalation or oral exposure to radioactive cesium can be associated with adverse human health effects. When humans are exposed to high levels of radioactivity from a radiocesium source, as were the cases of acute exposure in Goiânia, Brazil (Brandão-Mello et al. 1991) and Russia (Gottlöber et al. 2000), such exposures should be of value in assessing potential health hazards. Animal studies could be designed to assess the health effects associated with intermediate-duration exposure to radioactive cesium. Such studies could be designed to also generate data regarding potential age-related differences in toxicity.

Chronic-Duration Exposure and Cancer. Since there are no studies pertaining to noncancer or cancer health effects in humans or animals following chronic-duration inhalation or oral exposure to stable cesium, no chronic-duration inhalation or oral MRLs were derived for stable cesium. Additional data from acute- and intermediate-duration animal studies might be helpful in determining the need for longer-term studies.

There are no data regarding noncancer or cancer health effects in humans following chronic-duration inhalation or dermal exposure to radioactive cesium. Low levels of radioactive cesium are found in the diets of individuals living in areas that have been contaminated with radioactive fallout; however, there is a lack of information regarding dose-response following chronic-duration oral exposure. No chronic-duration inhalation or oral MRLs were derived for radioactive cesium. A chronic-duration MRL that was derived by Agency for Toxic Substances and Disease Registry (1999) for chronic external exposure to ionizing radiation was appropriate as a chronic-duration MRL for external exposure to ionizing radiation from a radioactive cesium source. Long-term research into health effects associated with chronic exposure to radioactive cesium following incidents such as the Chernobyl nuclear accident may help to elucidate long-term noncancer and cancer health risks from chronic exposure to radionuclides of cesium.

Genotoxicity. No genotoxicity studies of *in vivo* exposure of humans to stable cesium compounds were located. Stable cesium (as cesium chloride) induced chromosomal aberrations in human lymphocytes *in vitro* (Ghosh et al. 1993) and chromosomal aberrations and micronuclei in mouse bone marrow *in vivo* (Ghosh et al. 1990, 1991; Santos-Mello et al. 2001). Cesium sulfate was not mutagenic in *E. coli* either with or without metabolic activation (Olivier and Marzin 1987). Studies in mammalian

systems would be useful. Studies of workers exposed to known levels of stable cesium would be useful in establishing whether or not cesium is of genotoxicity concern in humans.

In vivo human data are limited to the findings of increased point mutations in T-lymphocytes and chromosomal aberrations among individuals who had been exposed to beta and gamma radiation from an opened ^{137}CsCl source (Natarajan et al. 1998; Skandalis et al. 1997) and apparently increased frequencies of chromosomal aberrations in lymphocytes of children living in areas contaminated by ^{137}Cs fallout following the Chernobyl nuclear accident (Padovani et al. 1993). *In vitro* exposure of human lymphocytes to a sealed ^{137}Cs gamma source resulted in an increased frequency of micronuclei (Balasem and Ali 1991). *In vivo* oral and external exposure of male mice to a ^{137}Cs gamma source resulted in increases in dominant lethal mutations (Ramaiya et al. 1994). Increases in the frequency of reciprocal translocations in spermatogonia were observed following oral exposure to ^{137}Cs in mice (Ramaiya et al. 1994) and external exposure to a ^{137}Cs source in crab-eating monkeys (Tobari et al. 1988). A number of *in vitro* genotoxicity assays have indicated chromosomal aberrations and breaks, sister chromatid exchanges, and micronuclei in animal cells (Arslan et al. 1986; Biedermann et al. 1991; Doggett and McKenzie 1983; Hintenlang 1993; Iijima and Morimoto 1991; Kamiguchi et al. 1991; Mikamo et al. 1990, 1991).

Human and animal studies show that external and internal exposure to radioactive cesium is a genotoxicity concern. External exposure to any gamma source would be expected to result in genotoxic effects similar to those observed following external exposure to radioisotopes of cesium (see Agency for Toxic Substances and Disease Registry 1999 for more information on ionizing radiation). Whenever possible, additional human studies should focus on exposure levels to establish dose-response relationships.

Reproductive Toxicity. There are no reports of reproductive effects in humans or animals exposed to stable cesium. Although stable cesium appears to be of relatively low toxicity concern, animal studies could be designed to assess the potential for adverse health effects (including reproductive effects) associated with repeated exposure. Human data could be collected from individuals occupationally exposed to significant levels of stable cesium.

Reports of human reproductive effects following exposure to radioactive cesium are limited to the findings of reduced sperm counts following external and internal exposure to an opened ^{137}CsCl source (Brandão-Mello et al. 1991). Mice, exposed to ^{137}Cs either orally or externally, exhibited reduced fertility

(including complete sterility) (Ramaiya et al. 1994). Persistent germinal epithelium damage and azoospermia were observed in all long-term surviving dogs that had been administered intravenous injections of ^{137}Cs (Nikula et al. 1995, 1996). In cases of known human exposure to radioactive cesium, exposure-response relationships should be established when possible. Although reduced fertility has been shown in males, additional animal studies could be designed to assess the potential for reproductive toxicity in females.

Developmental Toxicity. There are no reports of developmental effects in humans exposed to stable cesium. One investigator reported reduced body weight and certain organ weights among offspring, as well as indications of altered activity of some hepatic enzymes among offspring of pregnant mouse dams repeatedly exposed (orally) to stable cesium (Messiha 1988b). The same investigator reported similar results in pups exposed (via their nursing mothers) only during lactation (Messiha 1989b). These studies did not include gross and histopathologic examination of the offspring. Well-designed animal studies could more completely assess the potential for developmental toxicity of stable cesium.

Although there are no reports of developmental effects in humans exposed specifically to radioisotopes of cesium, impaired cognitive function was observed in atomic bomb survivors of Hiroshima and Nagasaki prenatally exposed to high levels of ionizing radiation during critical stages of neural development (Schull and Otake 1999; Schull et al. 1988). External exposure to sufficiently high doses of radiation from a radioactive cesium source would be expected to result in similar effects. *In utero* exposure of rat and mouse fetuses via whole body exposure of dams resulted in developmental effects such as reduced postnatal body weight, impaired motor activity, morphological changes in the brain, increased aggressive behavior, reduced brain and head size, and retarded odontogenesis and palatal closure (Minamisawa et al. 1990, 1992; Norton and Kimler 1987, 1988; Saad et al. 1991, 1994). Continued monitoring of populations known to have been exposed to ionizing radiation (including radioactive cesium sources) should help to refine estimates of dose-response and the relationship to adverse health effects, including developmental toxicity.

Immunotoxicity. There are no reports regarding the immunotoxicity of stable cesium in humans or animals. Animal studies could be designed to assess these parameters, but such studies do not presently seem necessary.

Severe bone marrow depression was observed in individuals exposed externally and internally to a ^{137}Cs source. This effect is typical of individuals exposed to ionizing radiation (see Agency for Toxic

Substances and Disease Registry 1999 for additional information on the effects of ionizing radiation). A similar effect was observed in dogs given an intravenous injection of ^{137}CsCl (Nikula et al. 1995). Data should be collected from individuals known to have been exposed to ionizing radiation (including that from radioactive cesium sources). Additional animal studies could be designed to establish dose-response relationships.

Neurotoxicity. Data regarding neurological effects of stable cesium in humans are restricted to a single case of an investigator reporting feelings of euphoria, heightened sense perception, and tingling sensations within 15 minutes of ingesting oral doses of cesium chloride during a 36-day exposure period. No apparent adverse effects on mental or motor skills were observed (Neulieb 1984). Administration of cesium chloride to animals has triggered stimulant (Johnson 1972; Messiha 1978) and depressant (Bose and Pinsky 1981, 1983b, 1984; Bose et al. 1981; Pinsky et al. 1980) central nervous system responses. Additional animal studies could be designed to elucidate mechanisms responsible for the observed neurological effects.

Although there are no reports of neurotoxicity in humans exposed specifically to radioisotopes of cesium, impaired cognitive function was observed in atomic bomb survivors of Hiroshima and Nagasaki prenatally exposed to high levels of external ionizing radiation during critical stages of neural development (Schull and Otake 1999; Schull et al. 1988). External exposure to sufficiently high doses of radiation from a sealed radioactive cesium gamma source would result in similar effects. *In utero* exposure of rat and mouse fetuses via whole-body exposure of dams resulted in impaired motor activity, morphological changes in the brain, increased aggressive behavior, and reduced brain and head size (Minamisawa et al. 1990, 1992; Norton and Kimler 1987, 1988; Saad et al. 1991, 1994), effects that have been shown to be related to critical developmental stages. Neurotoxic effects, noted in humans suffering from acute radiation syndrome due to ionizing radiation exposure, are well-characterized (see Agency for Toxic Substances and Disease Registry 1999 for more detailed information on the effects of ionizing radiation). Such effects would be expected in humans exposed to sufficiently high doses of radiation from a radioactive cesium source. Additional well-designed animal studies might elucidate mechanisms of neurotoxicity, but do not presently appear to be needed.

Epidemiological and Human Dosimetry Studies. Stable cesium is ubiquitous in the earth's crust, but is found at such low environmental levels that the probability of human intake of toxic amounts of stable cesium is negligible. Although there is no apparent need for specifically designed epidemiological or human dosimetry studies regarding stable cesium, such data, collected from

individuals occupationally exposed to significant amounts of cesium or persons living near toxic waste sites with significant levels of cesium, might be useful.

Due to accidental or intentional releases during nuclear fission, ^{134}Cs and ^{137}Cs can be found in air, soil, water, and food. There is concern for the health of humans living in close proximity to release or storage sites or in areas receiving significant amounts of radioactive fallout. Epidemiological studies of radiation doses typically involve estimates of exposure that are based on whole-body measurements of internally-deposited ^{134}Cs or ^{137}Cs or genotoxic effects such as chromosomal aberrations in peripheral blood lymphocytes. A need remains for epidemiological data that can provide quantitative human dose-response information while supplying additional information on the health effects of exposure to ionizing radiation and radioisotopes of cesium; in particular, for cases of known internal exposure.

Biomarkers of Exposure and Effect.

Exposure. Both stable and radioactive isotopes of cesium may be detected in samples of urine, blood, feces, or body tissues. Due to the relatively long biological half-time of cesium (several months in humans), short-term exposures cannot be readily distinguished from longer-term ones. No new biomarkers of exposure are needed at this time.

Effect. No known biomarkers of effect exist for exposure to stable cesium. Although high radiation doses from internally deposited radioactive cesium can cause bone marrow aplasia, altered blood values, and increased chromosomal aberrations in lymphocytes (Brandão-Mello et al. 1991; Natarajan et al. 1998), these effects are not specific to radioactive cesium.

Absorption, Distribution, Metabolism, and Excretion. Human and animal data show that inhaled or ingested cesium (in soluble compounds) is rapidly absorbed into the blood (Boecker 1969a, 1969b; Henrichs et al. 1989; Lie 1964; Miller 1964; Stara 1965; Stara and Thomas 1963), whereas relatively insoluble forms of cesium are not readily absorbed into blood following inhalation or oral exposure (Boecker et al. 1974, 1977; Leroy et al. 1966; Talbot et al. 1993). Dermal absorption has been qualitatively (but not quantitatively) demonstrated in rats (Pendic and Milivojevic 1966). Additional studies could measure relative absorption rates for a variety of soluble and insoluble cesium compounds. Furthermore, studies could be designed to measure dermal absorption rates. Other studies could further assess the fate of relatively insoluble inhaled particles containing radioisotopes of cesium that may be retained in lung tissue for long periods of time.

Cesium absorbed via inhalation or ingestion has been shown to be rapidly distributed throughout the body in humans and animals (Boecker 1969a, 1969b; Furchner et al. 1964; Lie 1964; Miller 1964; Rosoff et al. 1963; Stara 1965; Stara and Thomas 1963). Once absorbed by pregnant women, cesium can pass the placental barrier and be absorbed by the conceptus. Absorbed cesium can also be found in the milk of lactating women (Thornberg and Mattsson 2000). Once cesium is absorbed into body fluids, distribution patterns in soft tissue are expected to be similar for any route of exposure since cesium is distributed throughout the body as the cation (Cs^+), much like potassium (K^+). Available studies appear to adequately describe the distribution of absorbed cesium. Additional studies could be designed to elucidate mechanisms whereby cesium ions may influence central nervous system activity.

Human and animal studies adequately describe elimination of absorbed cesium, primarily via the urine (Boecker 1969b; Hölgye and Malý 2002; Iinuma et al. 1965; Rosoff et al. 1963; Stara 1965; Stara and Thomas 1963). Age-related differences in elimination rates have been described in humans (Boni 1969b; Melo et al. 1994) and dogs (Melo et al. 1996). In cases of known human exposure to cesium, additional information may help to further assess age-related differences in the toxicokinetics of cesium.

Comparative Toxicokinetics. Available cesium toxicokinetic data in humans and various animal species indicate similar patterns of absorption, distribution, and elimination. The central nervous system appears to be a target for effects in humans (Neulieb 1984) and animals (Bose and Pinsky 1981, 1983b, 1984; Bose et al. 1981; Johnson 1972; Messiha 1978; Pinsky et al. 1980). Additional studies could be designed to elucidate and compare mechanisms responsible for central nervous system effects.

Methods for Reducing Toxic Effects. Oral administration of Prussian blue that exchanges potassium for cesium, cathartics that shorten the transit time of ingested cesium within the gastrointestinal tract, and gastric lavage may aid in reducing peak absorption of ingested cesium, but due to the rapid absorption of cesium from soluble cesium compounds, these measures would only be of potential benefit within a short time following initial exposure. Most countermeasures focus on reducing the body burden of absorbed cesium. The intestinal reabsorption of cesium that is excreted into the small intestine via the bile can be blocked by oral administration of Prussian blue, forming an insoluble cesium complex that is excreted in the feces. Animal studies also indicate that increased plasma concentrations of potassium may increase the mobilization of cesium from tissues, increasing its excretion (Richmond and Furchner 1961). There are no prescribed methods for interfering with mechanisms of action for toxic effects of cesium, since such mechanisms have not been elucidated.

Children's Susceptibility. Available information on age-related differences in health effects comes from *in utero* exposure to radiation from external ionizing radiation sources (including radioisotopes of cesium). Studies have shown neurological effects in humans and animals exposed during critical periods of central nervous system development (Koshimoto et al. 1994; Minamisawa et al. 1990; Norton and Kimler 1987, 1988; Schull and Otake 1999). Comparative studies of neurological effects in animals first irradiated as juveniles or adults are lacking. No information was located regarding age-related health effects in humans or animals exposed to stable cesium.

PBPK models account for potential age-related differences in deposition of inhaled cesium, as well as differences in elimination rates for absorbed cesium (see Section 3.5.5 for more information on PBPK models). Potential differences in absorption rates of inhaled cesium compounds could be due to age-related differences in physical properties of the respiratory system and/or ventilation patterns. PBPK models also could adjust for potential age-related differences in gastrointestinal absorption. If such differences exist, they would likely be the result of differences in diffusion rates and active transport mechanisms. Young children exhibit biological half-times for absorbed cesium that are shorter than those of older children and adults (Boni 1969b; Melo et al. 1994); these age-related differences are associated with body mass and could be the result of age-related differences in tissue retention and excretory rates.

Cesium is found in the breast milk of mothers with an internal cesium burden (Johansson et al. 1998; Thornberg and Mattsson 2000), and can be transferred to nursing infants (Johansson et al. 1998). Cesium has been shown to cross the placental barrier of animals, but concentrations of cesium in the fetal tissues are less than those in corresponding tissues of the mother (Mahlum and Sikov 1969; Vandecasteele et al. 1989). Data collected from areas containing elevated concentrations of radioactive cesium fallout might be of value in further assessing age-related transfer rates.

Biomarkers of exposure or effect are the same in adults and children (see Section 3.9 for detailed information on biomarkers of exposure and effect). There are no data on interactions of cesium with other chemicals in children. No pediatric-specific methods have been found to reduce peak absorption, or body burden, of cesium following exposure, although methods employed to reduce the body burden of cesium in adults are also effective in children.

Child health data needs relating to exposure are discussed in Section 6.8.1, Identification of Data Needs: Exposures of Children.

3.13.3 Ongoing Studies

A single relevant ongoing study was identified in the Federal Research In Progress database (FEDRIP 2002). Dr. R. Albertini, from the University of Vermont, Burlington, Vermont, is quantitatively examining radiation-induced mutation at the hprt gene in human T-lymphocytes. Gamma radiation sources used in the study include ^{137}Cs.

4. CHEMICAL, PHYSICAL, and RADIOLOGICAL INFORMATION

4.1 CHEMICAL IDENTITY

Information regarding the chemical identity of cesium is located in Table 4-1.

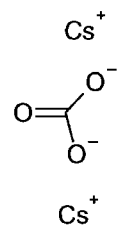
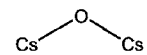
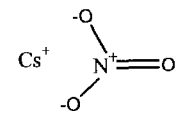
4.2 PHYSICAL, CHEMICAL, AND RADIOLOGICAL PROPERTIES

Information regarding the physical and chemical properties of cesium is located in Table 4-2.

Cesium is a silvery white, soft, ductile metal with only one oxidation state (+1). At slightly above room temperature, cesium exists in the liquid state. Compared to the other stable alkali metals, cesium has the lowest boiling point and melting point, highest vapor pressure, highest density, and lowest ionization potential. These properties make cesium far more reactive than the other members of the alkali metal group. When exposed to air, cesium metal ignites, producing a reddish violet flame, and forms a mixture of cesium oxides. Pure cesium reacts violently with water to form cesium hydroxide, the strongest base known, as well as hydrogen gas. The burning cesium can ignite the liberated hydrogen gas and produce an explosion. Cesium salts and most cesium compounds are generally very water soluble, with the exception of cesium alkyl and aryl compounds, which have low water solubility.

There are several radioactive isotopes of cesium ranging from ^{114}Cs to ^{145}Cs (Helmers 1996). The radioactive isotopes have a wide range of half-lives ranging from about 0.57 seconds (^{114}Cs) to about $3x10^6$ years (^{135}Cs) (Helmers 1996). The radioactive isotopes ^{137}Cs and ^{134}Cs are significant fission products because of their high fission yield, and their relatively long half-lives. The fission yield of ^{137}Cs in nuclear reactions is relatively high; about 6 atoms of ^{137}Cs are produced per 100 fission events (WHO 1983). ^{137}Cs has a radioactive half-life of about 30 years and decays by beta decay either to stable ^{137}Ba or a meta-stable form of barium (^{137m}Ba). The meta-stable isotope (^{137m}Ba) is rapidly converted to stable ^{137}Ba (half-life of about 2 minutes) accompanied by gamma ray emission whose energy is 0.662 MeV (ICRP 1983). Figure 4-1 illustrates this decay scheme. The first beta decay mode that forms ^{137m}Ba accounts for roughly 95% of the total intensity, while the second mode accounts for about 5% (WHO 1983). Radioactive ^{134}Cs primarily decays to stable ^{134}Ba by beta decay accompanied by gamma ray emissions or less frequently to stable ^{134}Xe by electron capture (EC) accompanied by a single gamma ray

Table 4-1. Chemical Identity of Cesium and Compounds

Characteristic	Cesium (metal)	Cesium chloride	Cesium carbonate	Cesium hydroxide	Cesium oxide	Cesium nitrate
Synonym(s)	Caesium	Cesium monochloride	Dicesium salt	Cesium hydrate		
Registered trade name(s)	No data	No data	No data	No data	No data	No data
Chemical formula	Cs	CsCl	Cs_2CO_3	CsOH	Cs_2O	$CsNO_3$
Chemical structure	Cs	Cs—Cl		Cs—OH		
Identification numbers:						
CAS registry	7440-46-2	7647-17-8	534-17-8	21351-79-1	20281-00-9	7789-18-6
NIOSH RTECS	FK9225000	FK9625000	FK9400000	FK9800000	No data	
EPA hazardous waste	No data	No data	No data	No data	No data	No data
OHM/TADS	No data	No data	No data	No data	No data	No data
DOT/UN/NA/ IMCO shipping	No data	No data	No data	No data	No data	No data
HSDB	No data	No data	No data	No data	No data	No data
NCI	No data	No data	No data	No data	No data	No data

CAS = Chemical Abstracts Services; DOT/UN/NA/IMCO = Department of Transportation/United Nations/North America/International Maritime Dangerous Goods Code; EPA = Environmental Protection Agency; HSDB = Hazardous Substances Data Bank; NCI = National Cancer Institute; NIOSH = National Institute for Occupational Safety and Health; OHM/TADS = Oil and Hazardous Materials/Technical Assistance Data System; RTECS = Registry of Toxic Effects of Chemical Substances

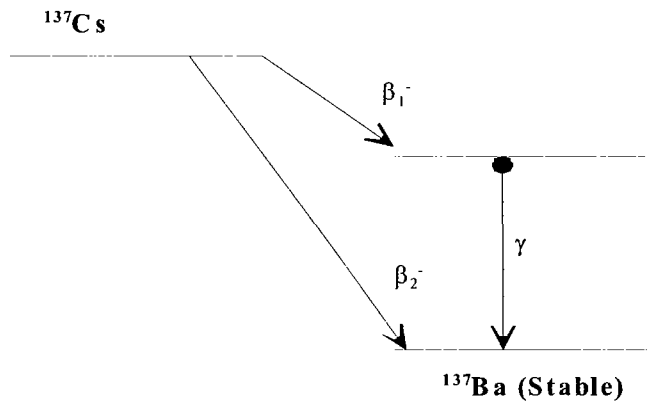
Table 4-2. Physical and Chemical Properties of Cesium and Compounds

Property	Cesium (metal)	Cesium chloride	Cesium carbonate	Cesium hydroxide	Cesium oxide	Cesium nitrate
Molecular weight	132.906	168.36	325.82	149.91	281.81	194.91[b]
Color	Silvery-white	White	White	Colorless	Golden-yellow	White[b]
Physical state	Solid (liquid slightly above room temperature)	Solid	Solid	Solid	Solid	Solid[b]
Melting point	29 °C	646 °C	610 °C	272 °C	490 °C	414 °C[b]
Boiling point	685 °C	1290 °C	No data	No data	No data	No Data
Density, g/cm^3	1.93 (20 °C)[b]	3.988 (20 °C)[b]	4.24 (20 °C)[b]	3.68 (20 °C)[b]	4.65 (20 °C)[b]	3.66 (20 °C)[b]
Odor	No data	No data	No data	No data	No data	No data
Odor threshold:						
Water	No data	No data	No data	No data	No data	No data
Air	No data	No data	No data	No data	No data	No data
Solubility:						
Water	Reacts violently with water	1.87 kg/L (20 °C)	2.1 kg/L	4 kg/L (15 °C)	Very soluble in water[b]	Soluble[b]
Organic-solvent(s)		Soluble in ethanol[b]	Soluble in ethanol and ether[b]	Soluble in ethanol[b]		
Partition coefficients:						
Log K_{ow}	N/A	N/A	N/A	N/A	N/A	N/A
Log K_{oc}	N/A	N/A	N/A	N/A	N/A	N/A
Vapor pressure	0.0075 mmHg at 144.5 °C	No data	No data	No data	No data	No data
Henry's Law constant	N/A	N/A	N/A	N/A	N/A	N/A
Autoignition temperature	No data	No data	No data	No data	No data	
Flashpoint	No data	No data	No data	No data	No data	No data
Flammability limits	No data	No data	No data	No data	No data	No data
Conversion factors	No data	No data	No data	No data	No data	No data
Explosive limits	No data	No data	No data	No data	No data	No data

[a]Data from Burt 1993 unless otherwise specified.
[b]Lide 1998

N/A = not applicable

Figure 4-1. The Decay Scheme of ^{137}Cs



β = beta decay; γ= γ -ray emission

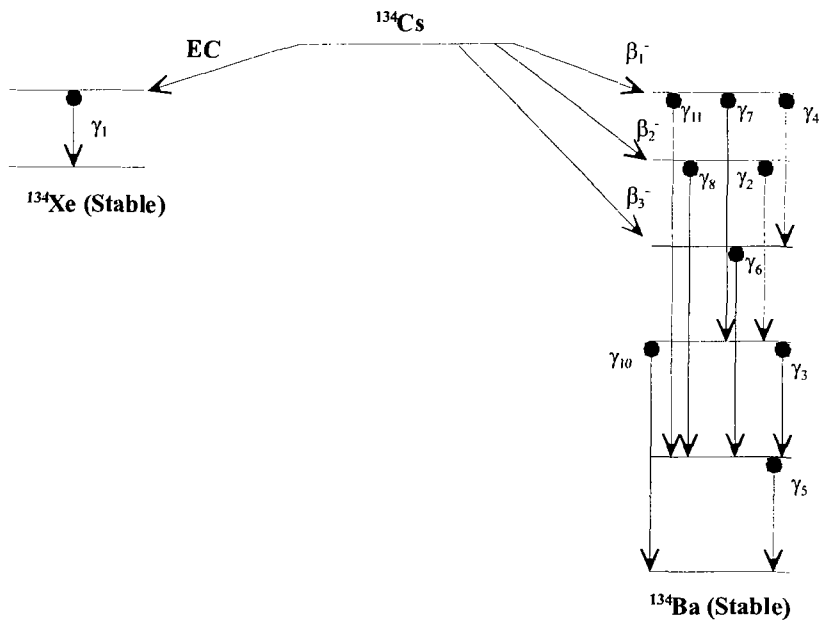
emission as depicted in Figure 4-2. The energy of the various gamma rays are in the range of 0.24–1.4 MeV. The half-life average energy of the beta transitions and intensity of the transitions for both ^{134}Cs and ^{137}Cs are summarized in Table 4-3.

Table 4-3. Decay Properties of the Radioactive Isotopes of Cesium

Isotope	Half-life (years)	Decay mode	Intensity percent	Beta particle energy (MeV)
^{134}Cs	2.062	β_1^-	27	0.02309
		β_2^-	2.5	0.1234
		β_3^-	70	0.2101
^{137}Cs	30	β_1^-	94.6	0.1734
		β_2^-	5.4	0.4246

[a]ICRP 1983
[b]The ^{134}Xe daughter yield from the electron capture decay of ^{134}Cs is approximately $3x10^{-6}$.

Figure 4-2. The Decay Scheme of ^{134}Cs



EC = electron capture; β = beta decay; γ = gamma-ray emission

5. PRODUCTION, IMPORT/EXPORT, USE, AND DISPOSAL

5.1 PRODUCTION

Cesium is the rarest of the naturally occurring alkali metals, ranking 40th in elemental abundance. Although it is widely distributed in the earth's crust, cesium is found at relatively low concentrations. Granites contain cesium at about 1 ppm of cesium and sedimentary rocks contain approximately 4 ppm of cesium. Cesium is found in muscovite, beryl, spodumene, potassium feldspars, leucite, petalite, and other related minerals. The most common commercial source of cesium is pollucite, which contains between 5 and 32% Cs_2O (Burt 1993). There are three basic methods of converting pollucite ore to cesium metal or related compounds: direct reduction with metals; decomposition with bases; and acid digestion. In each method, grinding of the mined ore to approximately 75 μm sized particles precedes chemical conversion.

Direct Reduction With Metals. Pollucite is directly reduced by heating the ore in the presence of calcium to 950 °C in vacuum, or in the presence of either sodium or potassium to 750 °C in an inert atmosphere (Burt 1993). Excessive amounts of reducing metal are required, and the resultant cesium metal is impure, requiring further distillation and purification.

Decomposition With Bases. Alkaline decomposition is carried out by roasting the pollucite ore with either a calcium carbonate-calcium chloride mix at 800–900 °C or with a sodium carbonate-sodium chloride mix at 600–800 °C followed by a water leach of the roasted mass. The resultant cesium chloride solution is separated from the gangue by filtration (Burt 1993).

Acid Digestion. Acid digestion of pollucite is the preferred commercial process for producing pure cesium. Hydrofluoric, hydrobromic, hydrochloric, and sulfuric acid may be employed in this method. Hydrofluoric and hydrobromic acid digestion yield the greatest cesium recovery, but the inherent difficulties of safely handling these acids limit their use. Digestion with hydrochloric acid takes place at elevated temperatures and produces a solution of mixed cesium chlorides, aluminum, and other alkali metals separated from the siliceous residue by filtration. The impure cesium chloride can be purified as cesium chloride double salts that are then recrystallized. The purified double salts are decomposed to cesium chloride by hydrolysis or precipitated with hydrogen sulfide (Burt 1993).

Sulfuric acid digestion is performed at 110 °C with a 35–40% solution of sulfuric acid, followed by a hot water wash and vacuum filtration. Cesium alum is crystallized from the leach filtrate by stage cooling to 50 °C and then to 20 °C and roasted in the presence of 4% carbon. The residue is leached to produce cesium sulfate solution, which can be converted to cesium chloride (Burt 1993).

Radioactive isotopes of cesium such as ^{134}Cs and ^{137}Cs are produced by nuclear fission in fuel rods in nuclear power plants and in fallout from nuclear weapons. Radiocesium can be recovered from fission products by digestion with nitric acid. After filtration to remove the waste, the radioactive cesium phosphotungstate is precipitated using phosphotungstic acid (Burt 1993). Other processes for the removal of ^{134}Cs and ^{137}Cs from radioactive waste involve solvent extraction using macrocyclic polyethers, or crown ethers and coprecipitation with sodium tetraphenylboron (Burt 1993).

5.2 IMPORT/EXPORT

The United States is 100% import-reliant for elemental cesium. No economic data, such as production volume, consumption, or import/export volumes of cesium, are available. Although there is no information regarding the countries shipping cesium or cesium compounds to the United States, it is believed that Canada is the major foreign source of cesium (USGS 1999). Other possible sources of cesium-bearing material include Germany and the United Kingdom.

5.3 USE

There are relatively few commercial uses for cesium metal and its compounds. Cesium is used as a getter (combines chemically with residual gas in partial vacuum to increase the vacuum) for residual gas impurities in vacuum tubes and as a coating to reduce the work function of the tungsten filaments or cathodes of the tubes. Crystalline cesium iodide and cesium fluoride are used in scintillation counters, which convert energy from ionizing radiation into pulses of visible light (Burt 1993). Cesium is also used in magnetohydrodynamic power generators as a plasma seeding agent (Lewis 1997). Recently, cesium compounds have been employed as catalysts in organic synthesis, replacing sodium or potassium salts. One of the most interesting uses of cesium is in the production of highly accurate atomic clocks. When exposed to microwave radiation, the natural vibration of cesium atoms occurs at a frequency of 9,192,631,770 Hz, and 1 second in time is defined as the duration of 9,192,631,770 periods of radiation absorbed or emitted by the transition of ^{133}Cs atoms in two hyperfine levels of their ground state. Radioactive ^{137}Cs has been approved as the gamma ray source for the sterilization of wheat, flour,

potatoes, surgical equipment and other medical supplies, and sewage sludge, and is also used as a calibration source in gamma ray spectroscopy (Lewis 1997). ^{137}Cs is also used in industrial radiography and for imaging of transport containers at border crossings. ^{131}Cs was recently approved by the U.S. FDA as an active source in radioactive seeds used to treat prostate cancer (FDA 2003).

5.4 DISPOSAL

Because of its high reactivity, special precautions are required for the handling and disposal of pure cesium metal. Cesium metal is usually stored and transported in stainless steel containers, which are contained in outer packing, to ensure that the metal remains in a dry, oxygen-free environment.

Most nonradioactive cesium minerals, compounds, and materials do not require special disposal and handling requirements. However, some chemical forms may be classified as hazardous materials if the compound is chemically reactive, flammable, or toxic. Care should be taken to read and understand all of the hazards, precautions, and safety procedures for each specific chemical form. In addition, all federal, state, and local laws and regulations should be investigated and subsequently followed with regard to disposal and handling of the specific chemical form of the cesium mineral, compound, or material.

Radioactive cesium requires special disposal and handling requirements. Radioactive waste containing ^{134}Cs and ^{137}Cs is usually grouped into four categories: low-level waste (LLW), high-level waste (HLW), mixed waste, and spent nuclear fuel.

Low-level waste is all radioactive waste that cannot be classified as either HLW, spent fuel, or mixed waste. Low-level does not necessarily mean low radioactivity or low environmental hazard. Low-level waste types that may be contaminated with ^{134}Cs and ^{137}Cs include both wet and dry wastes. Examples of the physical form of LLW are: spent ion exchange resins, filter sludges, filter cartridges, evaporator bottoms, compactible trash, noncompactible trash, irradiated components, ashes produced from the incineration of combustible material, contaminated detergents or solvents, organic liquids, and discarded contaminated equipment or tools. Of the LLW generated today, approximately 64% of the volume and 70% of the radioactivity is generated as a result of nuclear power plant activities or supporting fuel cycle operations. Other sources of LLW are commercial, academic, and government research laboratories and medical facilities. Radiocesium contamination accounts for only a small fraction of the activity of LLW. Nearly all of the ^{134}Cs and ^{137}Cs produced as a result of fission events remains trapped within the spent nuclear fuel rods (DOE 1996b). Low-level waste from Department of Energy (DOE) sources is currently

disposed of at several DOE facilities across the United States. Only two sites accept non-DOE LLW: Barnwell, South Carolina and Richland, Washington (DOE 1996a). As required by the Federal Low Level Radioactive Waste (LLRW) Policy Act in 1980 and the 1985 amendments, states are required to build facilities to contain LLW generated from sources within its boundaries. The law encourages states to cooperate together in coordinating LLW disposal facilities. Many states have formed "compacts" to collaborate on construction of these LLW facilities. However, other than Barnwell, South Carolina and Richland, Washington, no other facility in the United States is accepting LLW from non-DOE sources (Eisenbud 1987). Over half of the LLW in the eastern United States is disposed of at the Barnwell site (Eisenbud 1987). The method of disposal for LLW has been to package the material in drums or boxes and bury the material in shallow pits and trenches. Approximately 3 million cubic meters of LLW generated has been disposed of in this way (DOE 1996a).

As defined by the Nuclear Waste Policy Act, HLW is "the highly radioactive material resulting from the reprocessing of spent nuclear fuel, including liquid waste produced directly in reprocessing and any solid material derived from such liquid waste that contains fission products in sufficient concentration..." (42 USC 100). Most HLW has been generated from the production of plutonium. A smaller fraction is related to the recovery of enriched uranium from naval reactor fuel. This waste typically contains highly concentrated solutions of relatively long-lived fission by-products such as ^{90}Sr and ^{137}Cs, hazardous chemicals, and toxic heavy metals. Liquid HLW is typically stored in large underground tanks of either stainless steel or carbon steel depending on whether they are acid or alkaline solutions. Approximately 100 million gallons of liquid HLW is stored in underground tanks in Washington, South Carolina, Idaho, and New York. These tanks contain a variety of radioactive liquids, solids, and sludges. Some of the liquid waste has been solidified into glass, ceramic slag, salt cake, and sludge. High-level waste in solid form is stored in underground bins (DOE 1996a).

Mixed waste contains both radioactive and chemically hazardous materials. All HLW is managed as mixed waste and some LLW is classified as mixed waste. Certain hazardous mixed wastes that contain radioactive isotopes have been incinerated in Oak Ridge, Tennessee (DOE 1996a). Spent nuclear fuel, such as fuel elements and irradiated targets used in nuclear reactors, are currently stored at the commercial nuclear power plants and DOE facilities where they were produced. Spent fuel is highly radioactive, due to the large concentration of fission products, and must be stored in special water-cooled pools that shield and cool the material. Nearly all of the DOE spent fuel, about 3,000 metric tons, is stored at four sites: Hanford, Savannah River, Idaho National Environmental and Engineering

Laboratory, and West Valley. Commercial reactors have generated >30,000 metric tons of spent fuel. The spent fuel from these facilities is located at >100 commercial nuclear reactor sites around the country.

The establishment of a HLW and spent fuel repository for both DOE and commercial waste is currently under evaluation at Yucca Flats, Nevada. This commercial waste storage facility will not be ready to accept spent fuel before 2010.

6. POTENTIAL FOR HUMAN EXPOSURE

6.1 OVERVIEW

Cesium has been identified in at least 8 of the 1,636 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL) (HazDat 2003). It was reported that ^{134}Cs has been found in at least 3 of the 1,636 current or former NPL sites and ^{137}Cs has been detected in at least 23 of the 1,636 current or former NPL sites. However, the number of sites evaluated for cesium is not known. The frequency of these sites can be seen in Figures 6-1, 6-2, and 6-3. Of these sites, none are located in the Commonwealth of Puerto Rico.

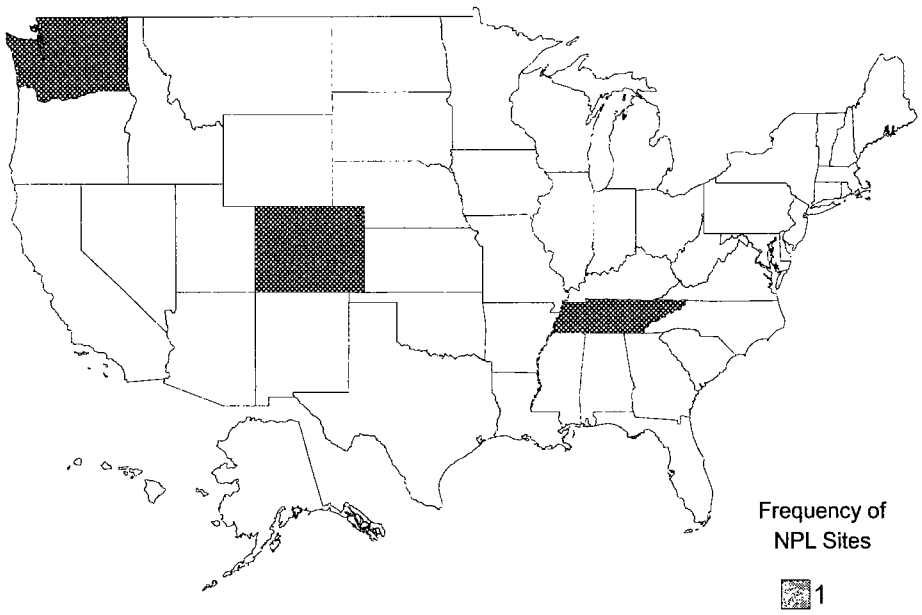
Naturally-occurring cesium and cesium minerals consist of only one stable isotope, ^{133}Cs. Cesium occurs in the earth's crust at low concentrations. Granites contain an average cesium concentration of about 1 ppm and sedimentary rocks contain about 4 ppm (Burt 1993). Higher concentrations are found in lepidolite, carnallite, muscovite, beryl, spodumene, potassium feldspars, leucite, petalite, and related minerals. The most important source of commercial cesium is the mineral pollucite, which usually contains about 5–32% Cs_2O (Burt 1993). The largest deposits of pollucite are located in Manitoba, Canada and account for about two-thirds of the world's known supply. Smaller deposits are located in Zimbabwe, Namibia, Brazil, Scandinavia, Czechoslovakia, and the United States. Continental dust and soil erosion are the main emission sources of naturally occurring cesium present in the environment. Cesium is also released to the environment as a result of human activities. The mining of pollucite ores and the production and use of cesium compounds in the electronic and energy production industries contribute to its direct release to the environment. Cesium has also been detected in the fly ash of hazardous waste incinerators and coal burning power plants (Fernandez et al. 1992; Mumma et al. 1990). Since the production and use of cesium compounds are limited, and since the natural concentration of cesium in the earth's crust is low, stable ^{133}Cs is not often monitored or detected in the environment.

Of much greater concern is the release of radioactive forms of cesium to the environment, such as ^{137}Cs and ^{134}Cs. These and other radioactive isotopes were released to the environment as a result of atmospheric testing of nuclear weapons (carried out from 1945 to 1980) and accidents that occurred at nuclear power plants such as the incident at the Chernobyl nuclear power plant in 1986 and the accident at the Windscale nuclear weapons facility in the United Kingdom in 1957. Small amounts of ^{137}Cs and ^{134}Cs

Figure 6-1. Frequency of NPL Sites with Cesium Contamination

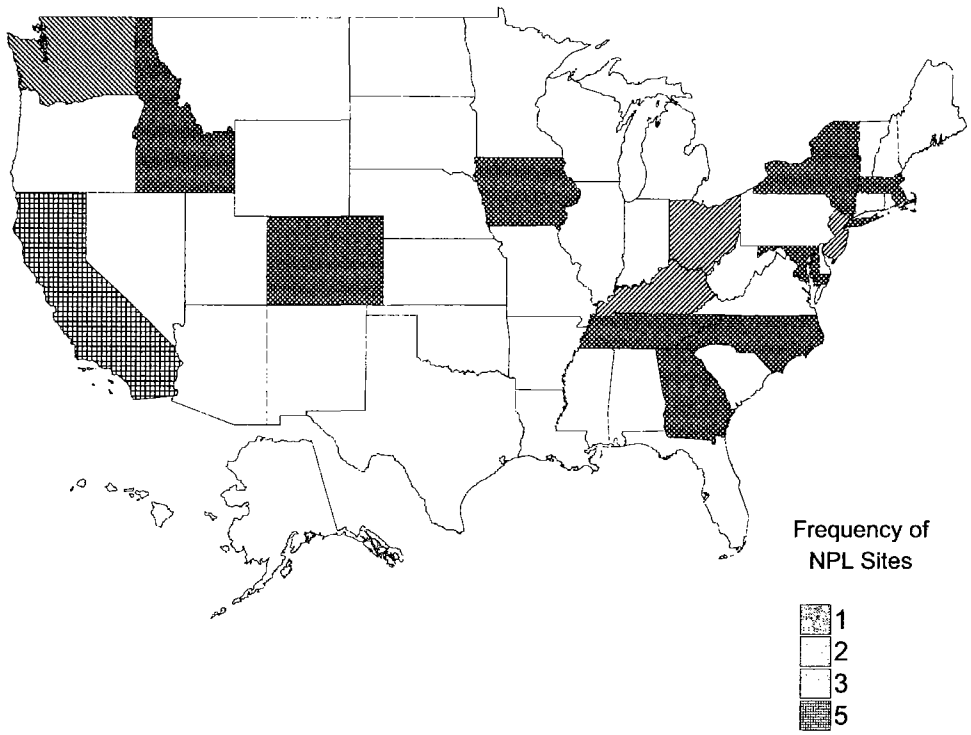
Derived from HazDat 2003

Figure 6-2. Frequency of NPL Sites with ^{134}Cs Contamination



Derived from HazDat 2003

Figure 6-3. Frequency of NPL Sites with ^{137}Cs Contamination



Derived from HazDat 2003

are also released in the airborne and liquid effluents during the normal operation of nuclear power plants. These levels are very low in comparison to the amounts released from weapons tests and accidents at nuclear power plants and are not expected to have a major impact upon human health. For the most part, testing of nuclear weapons has been discontinued by most nations for many years now. However, India and Pakistan have recently (May of 1998) conducted limited underground tests of nuclear weapons (UN 1998).

Radioactive materials are commonly referred to by activity rather than mass. The activity is the number of transformations (disintegrations) that the material undergoes in a given period of time. The most common units of activity are the curie (Ci) or the becquerel (Bq). One curie is equal to 3.7×10^{10} transformations per second (tps) and 1 becquerel is equal to 1 tps. For convenience, picocuries (pCi) are often reported for lower activities; $1 pCi = 1 \times 10^{-12}$ Ci=0.037 Bq.

Radioactive cesium is removed from the air by wet and dry deposition and can travel thousands of miles before settling to earth. Wet deposition is considered the most important pathway for the removal of radioactive cesium from the atmosphere. It is a complex process that depends upon meteorological conditions such as temperature, the microphysical structure of the clouds, and the rainfall rate, as well as the physical and chemical properties of the airborne cesium.

Cesium has very low mobility in soil. In general, it has been reported that cesium usually does not migrate below depths of about 40 cm, with the majority of cesium being retained in the upper 20 cm of the soil surface (Korobova et al. 1998; Takenaka et al. 1998). Clay minerals and soils rich in exchangeable potassium adsorb cesium by binding the cations to interlayer positions of the clay particles (Paasikallio 1999). The low hydration energy of cesium cations is primarily responsible for their selective sorption and fixation by clays. These factors can limit the uptake of cesium in grass and plant material. There are exceptional areas, however, where cesium fixation in soil is much less, resulting in greater transport in the soil and uptake in plants. Regions in Venezuela, Brazil, and Russia have been identified where the mobility of cesium is considerably greater than in other soils (LaBrecque and Rosales 1996; WHO 1983). Cesium is also deposited on plants and trees by wet and dry deposition and is absorbed into the flora through its foliage (Sawidis et al. 1990). The deposited cesium can make its way to soil through decomposition of the contaminated foliage.

Since the half-life for some radioactive isotopes of cesium is long (the half-life of ^{137}Cs is about 30 years and the half-life of ^{134}Cs is about 2 years), the general population is exposed to ^{137}Cs and ^{134}Cs for long

periods of time after it is released from a nuclear accident or weapons test, with the greatest exposure occurring near the source. Although inhalation and dermal exposure is possible, oral ingestion of contaminated food items is the greatest source of internal exposure for both naturally occurring and radioactive cesium. Cesium is uniformly distributed throughout the whole body similar to potassium, and it does not accumulate in any one particular part of the body like iodine (thyroid) or strontium (bones). For this reason, radioactive cesium only poses significant health risks if a large amount has been ingested. Workers employed in the mining and milling of pollucite ores and the production of cesium compounds are exposed to cesium through oral, dermal, and inhalation routes. Similar routes of exposure to ^{137}Cs and ^{134}Cs occurs for workers employed in the nuclear industry. External exposure to gamma radiation can also occur for workers employed in the nuclear industry as well as for the general population. The health consequences of external exposure to gamma radiation are not unique to ^{137}Cs and ^{134}Cs, but are similar for all gamma emitting radionuclides.

6.2 RELEASES TO THE ENVIRONMENT

Throughout this chapter, the units used to express concentration or intakes of cesium are the same units reported by the authors. In most cases, values are expressed in mass units when referring to ^{133}Cs, while radioactive cesium isotopes are expressed in units of activity.

According to the Toxics Release Inventory (TRI), in 2000, there were no reportable releases of ^{133}Cs, or its compounds into the environment by commercial sources (TRI00 2002). The TRI data should be used with caution since only certain types of facilities are required to report. Therefore, it is not an exhaustive list. Facilities are required to report to TRI if they have 10 or more full-time employees, or if the facility is classified under Standard Industrial Classification (SIC) codes 20–39, if the facility manufactures or processes over 25,000 pounds of the chemical, or otherwise uses >10,000 pounds of the chemical in a calendar year.

In the United States, commercial nuclear power plant operators are required to report any detectable quantities of radioactive materials released to the environment (10 CFR 50.36a). Table 6-1 summarizes releases of ^{137}Cs and ^{134}Cs to the atmosphere and water for 1993 from pressurized water reactor (PWR) and boiling water reactor (BWR) nuclear power plants. Nearly all of the radioactive material reported as being released in effluents are from planned releases. Planned releases result from normal plant operation or from anticipated operational occurrences. The latter include unplanned releases of radioactive

Table 6-1. Radiocesium Releases from Nuclear Power Plants for 1993

		Annual total site environmental releases for 1993			
		Water		Air	
Installation	Location[a]	^{134}Cs, Ci	^{137}Cs, Ci	^{134}Cs, Ci	^{137}Cs, Ci
Boiling water reactors					
Browns Ferry[b]	Decatur, AL	0.033	0.18	$3.5x10^{-4}$	$8.9x10^{-4}$
Brunswick[b]	Wilmington, NC	$2.9x10^{-4}$	$1.7x10^{-3}$	No data	$7.0x10^{-4}$
Clinton	Clinton, IL	No data	No data	No data	No data
Cooper	Omaha, NE	$9.3x10^{-3}$	0.052	No data	No data
Dresden[b]	Joliet, IL	$1.2x10^{-6}$	0.025	No data	$1.9x10^{-4}$
Duane Arnold	Cedar Rapids, IA	No data	No data	No data	$1.4x10^{-6}$
Edwin I. Hatch	Baxley, GA	$6.3x10^{-3}$	0.044	No data	$6.2x10^{-5}$
Fermi	Laguna Beach, MI	No data	$8.3x10^{-6}$	No data	No data
Grand Gulf	Vicksburg, MS	$3.5x10^{-4}$	$6.0x10^{-4}$	$2.1x10^{-6}$	$2.3x10^{-6}$
Hope Creek	Wilmington, DE	No data	$4.8x10^{-5}$	No data	No data
Humbolt Bay[b]	Eureka, CA	No data	$8.8x10^{-3}$	No data	$3.2x10^{-5}$
James A. Fitzpatrick	Syracuse, NY	No data	$4.1x10^{-5}$	No data	$6.6x10^{-6}$
LaCrosse[b]	LaCrosse, WI	$3.2x10^{-4}$	0.010	No data	$1.0x10^{-4}$
LaSalle	Ottawa, IL	No data	No data	No data	No data
Limerick	Philadelphia, PA	$2.0x10^{-3}$	$6.0x10^{-3}$	No data	No data
Millstone	New London, CT	0.066	0.224	$6.9x10^{-6}$	$8.9x10^{-5}$
Monticello	St. Cloud, MN	No data	No data	No data	$4.8x10^{-4}$
Nine Mile Point	Oswego, NY	No data	No data	No data	$1.9x10^{-5}$
Oyster Creek	Toms River, NJ	No data	No data	No data	$6.5x10^{-5}$
Peach Bottom	Lancaster, PA	$5.2x10^{-4}$	$1.4x10^{-3}$	$6.1x10^{-5}$	$3.0x10^{-4}$
Perry	Painesville, OH	$8.8x10^{-5}$	$1.6x10^{-4}$	No data	No data
Pilgram	Boston, MA	No data	$9.8x10^{-4}$	$2.4x10^{-5}$	$7.3x10^{-5}$
Quad-Cites	Moline, IL	No data	$3.6x10^{-3}$	No data	$1.8x10^{-4}$
River Bend	Baton Rouge, LA	$2.6x10^{-4}$	$2.2x10^{-3}$	No data	No data
Shoreham	Brookhaven, NY	No data	No data	No data	No data
Susquehanna	Berwick, PA	No data	$2.9x10^{-5}$	No data	No data
Vermont Yankee	Brattleboro, VT	No data	No data	No data	$9.9x10^{-5}$
WNP-2	Richland, WA	$6.3x10^{-3}$	0.019	$6.7x10^{-6}$	$7.2x10^{-6}$
Total		**0.12**	**0.58**	**$4.5x10^{-4}$**	**$3.3x10^{-3}$**
Pressurized water reactors					
Arkansas One	Russellville, AR	0.054	0.11	No data	$3.5x10^{-8}$
Beaver Valley	Shippingport, PA	$1.2x10^{-3}$	$2.3x10^{-3}$	No data	No data
Big Rock Point	Charlevoix, MI	$9.5x10^{-4}$	0.012	$1.1x10^{-5}$	$1.5x10^{-4}$
Braidwood	Joliet, IL	$6.5x10^{-4}$	$1.7x10^{-3}$	No data	No data
Byron	Byron, IL	$5.6x10^{-3}$	$1.5x10^{-3}$	No data	No data
Callaway	Fulton, MO	$5.3x10^{-4}$	$7.8x10^{-4}$	No data	No data
Calvert Cliffs	*Washington, DC*	*0.14*	*0.21*	$3.4x10^{-3}$	$4.2x10^{-3}$

Table 6-1. Radiocesium Releases from Nuclear Power Plants for 1993

		Annual total site environmental releases for 1993			
		Water		Air	
Installation	Location[a]	^{134}Cs, Ci	^{137}Cs, Ci	^{134}Cs, Ci	^{137}Cs, Ci
Catawba	Rock Hill, SC	$1.6x10^{-3}$	$3.9x10^{-3}$	No data	No data
Comanche Peak	Glen Rose, TX	$8.7x10^{-3}$	$8.7x10^{-3}$	No data	No data
Crystal River	Tampa, FL	$2.7x10^{-3}$	$9.3x10^{-3}$	No data	$3.1x10^{-6}$
Davis-Besse	Toledo, OH	$1.4x10^{-3}$	$6.2x10^{-3}$	No data	No data
Diablo Canyon	San Luis Obispo, CA	0.036	0.064	$3.3x10^{-5}$	$2.4x10^{-4}$
Donald C. Cook	St. Joseph, MI	$1.3x10^{-3}$	$6.3x10^{-3}$	No data	$7.3x10^{-7}$
Fort Calhoun	Omaha, NE	$1.3x10^{-3}$	0.012	No data	$1.7x10^{-6}$
H.B. Robinson	Hartsville, SC	$3.6x10^{-4}$	$4.1x10^{-4}$	$5.8x10^{-6}$	$1.8x10^{-5}$
Haddam Neck	Middletown, CT	0.013	0.023	$8.0x10^{-4}$	$8.8x10^{-3}$
Harris	Raleigh, NC	$2.9x10^{-4}$	$3.9x10^{-4}$	No data	No data
Indian Point[b]	Peekskill, NY	$3.0x10^{-3}$	0.10	No data	$7.0x10^{-4}$
Joseph M. Farley	Dothan, AL	$1.5x10^{-3}$	$3.6x10^{-3}$	No data	No data
Kewaunee	Green Bay, WI	No data	$2.6x10^{-6}$	No data	$2.0x10^{-6}$
Maine Yankee	Wicassett, ME	$1.4x10^{-3}$	$8.2x10^{-3}$	No data	$4.2x10^{-5}$
McGuire	Charlotte, NC	$1.6x10^{-3}$	$4.6x10^{-3}$	No data	$1.3x10^{-6}$
North Anna	NW Richmond, VA	$6.2x10^{-3}$	$9.2x10^{-3}$	$3.4x10^{-6}$	$8.2x10^{-5}$
Oconee	Greenville, SC	$2.7x10^{-3}$	0.010	No data	$3.9x10^{-4}$
Palisades	South Haven, MI	$2.4x10^{-4}$	$4.3x10^{-3}$	No data	$1.1x10^{-5}$
Palo Verde	Phoenix, AZ	No data	No data	$2.0x10^{-3}$	$1.7x10^{-3}$
Point Beach	Manitowoc, WI	0.019	0.027	$6.9x10^{-3}$	$6.9x10^{-3}$
Prairie Island	Minneapolis, MN	$2.8x10^{-3}$	$3.8x10^{-3}$	$1.5x10^{-5}$	$1.8x10^{-5}$
R.E. Ginna	Rochester, NY	0.042	0.041	No data	$5.2x10^{-6}$
Rancho Seco[b]	Sacramento, CA	$1.9x10^{-5}$	$3.6x10^{-4}$	No data	No data
Salem	Wilmington, DE	0.81	1.0	No data	$7.0x10^{-7}$
San Onofre[b]	San Clemente, CA	0.49	0.57	$2.5x10^{-5}$	$4.0x10^{-5}$
Seabrook	Portsmouth, NH	No data	$3.3x10^{-5}$	No data	No data
Sequoyah	Daisy, TN	0.086	0.14	No data	No data
South Texas	Bay City, TX	$3.4x10^{-3}$	$5.4x10^{-3}$	No data	No data
St. Lucie	Ft. Pierce, FL	0.055	0.083	$9.2x10^{-6}$	$2.1x10^{-5}$
Summer	Columbia, SC	$1.6x10^{-3}$	$3.3x10^{-3}$	$2.2x10^{-5}$	$2.9x10^{-5}$
Surry	Newport News, VA	$7.6x10^{-5}$	0.011	No data	$7.2x10^{-5}$
Three Mile Island[b]	Harrisburg, PA	0.026	0.030	$1.2x10^{-7}$	$4.7x10^{-6}$
Trojan[b]	Portland, OR	$1.0x10^{-3}$	$4.0x10^{-3}$	No data	No data
Turkey Point	Florida City, FL	$9.4x10^{-4}$	$5.5x10^{-3}$	No data	$9.4x10^{-7}$
Vogtle	Augusta, GA	$5.6x10^{-3}$	$7.3x10^{-3}$	No data	No data
Waterford	New Orleans, LA	0.013	0.016	No data	No data
Wolf Creek	Burlington, KS	0.022	0.024	No data	No data

Table 6-1. Radiocesium Releases from Nuclear Power Plants for 1993

		Annual total site environmental releases for 1993			
		Water		Air	
Installation	Location[a]	^{134}Cs, Ci	^{137}Cs, Ci	^{134}Cs, Ci	^{137}Cs, Ci
Yankee Rowe[b]	Greenfield, MA	4.3×10^{-6}	6.0×10^{-5}	No data	1.0×10^{-7}
Zion	Waukegan, IL	0.014	0.029	2.4×10^{-4}	2.9×10^{-4}
Total		**1.88**	**2.85**	**0.013**	**0.023**

[a]Post office state abbreviations used.
[b]Facilities that are permanently or indefinitely shut down.

materials from miscellaneous actions such as equipment failure, operator error, or procedure error; these releases are not of such consequence as to be considered an accident.

The TRI data should be used with caution because only certain types of facilities are required to report. This is not an exhaustive list.

6.2.1 Air

Stable cesium is introduced into the atmosphere by resuspension of soil, accidental release from mining and milling pollucite, and emissions from hazardous waste incinerators or coal burning plants. These emissions are expected to be low since cesium occurs naturally in the earth's crust at low concentrations and only small amounts of pollucite are mined annually. Cesium was detected at concentrations of 10.8 and 6.11 mg/m^3 in the effluent of a coal-burning power plant in the western United States (Ondov et al. 1989) and has been identified in the fly ash from municipal incinerators (Mumma et al. 1990, 1991). Fly ash from five municipal waste incinerators in the United States contained cesium at concentrations of 2,100–12,000 ppm (EPA 1990a). Stable cesium has been identified in air at one of the eight NPL hazardous waste sites where it was detected in some environmental media (HazDat 2003).

Radioactive isotopes of cesium such as ^{137}Cs and ^{134}Cs have been released to the atmosphere from atmospheric nuclear weapons testing, accidents at nuclear reactors, and nuclear-powered satellites burning up in the atmosphere upon re-entry. The total amount of ^{137}Cs released from weapons testing through 1980 was estimated as $2.6x10^7$ Ci ($9.6x10^{17}$ Bq), 76% of which was released in the northern hemisphere and 24% in the southern hemisphere (WHO 1983). On April 26, 1986, a steam buildup caused an explosion and fire at a nuclear power generating plant in Chernobyl, Russia, releasing an estimated $5.4x10^5$ Ci ($2.0x10^{16}$ Bq) of ^{134}Cs and $1.1x10^6$ Ci ($4.0x10^{16}$ Bq) of ^{137}Cs into the atmosphere over Europe (Watson 1987). Long-range atmospheric transport spread the radionuclides throughout the Northern Hemisphere. No airborne activity from Chernobyl has been reported south of the equator (Eisler 1995). By early May 1986, these radionuclides were readily detectable in environmental samples collected in North America (Huda et al. 1988). More recent estimates have put the total activity of ^{137}Cs released from the Chernobyl power plant as $2.3x10^6$ Ci ($8.5x10^{16}$ Bq) and $1.2x10^6$ Ci ($4.4x10^{16}$ Bq) for ^{134}Cs (Buzulukov and Dobrynin 1993). On January 24, 1978, the Soviet nuclear-powered satellite Cosmos 954 re-entered earth's atmosphere over the Canadian Arctic, releasing an estimated 86 Ci of ^{137}Cs (Barrie et al. 1992). In October 1957, an accident at the Windscale nuclear weapons plant at Sellafield in the United Kingdom resulted in the release of 595 Ci of ^{137}Cs (Agency for Toxic Substances and Disease

Registry 1999). Routine activities at nuclear power plants and fuel-reprocessing stations also release ^{137}Cs and ^{134}Cs to the environment on a regular basis. Radiocesium released in airborne effluents from the normal operation of nuclear power plants is considered low in comparison to releases from past atmospheric weapons tests and the major releases following accidents at nuclear power plants. In 1998, it was reported that $1.3x10^{-4}$ Ci of ^{134}Cs and $5.1x10^{-3}$ Ci of ^{137}Cs were released to the atmosphere from the Savannah River plutonium processing site in South Carolina (DOE 1998b). In 1993, the Nuclear Regulatory Commission (NRC) estimated that 0.013 Ci of ^{134}Cs and 0.023 Ci of ^{137}Cs were released in airborne effluents from 30 PWR nuclear power plants operating in the United States (USNRC 1993). It was also estimated that $4.6x10^{-4}$ Ci of ^{134}Cs and $3.3x10^{-3}$ Ci of ^{137}Cs were released in airborne effluents from 28 BWR nuclear power plants (USNRC 1993). The total airborne and liquid releases of ^{134}Cs and ^{137}Cs from the individual nuclear power plants are summarized in Table 6-1.

Radioactive ^{134}Cs was not identified in air at the 3 NPL hazardous waste sites where it was detected in some environmental media, but ^{137}Cs was identified in air at 5 of the 23 NPL hazardous waste sites where it was detected in some environmental media (HazDat 2003).

6.2.2 Water

Cesium can be released to water surfaces during the mining, milling, and production process of pollucite ore. The natural erosion and weathering of rocks will also lead to cesium's introduction into ground and surface water. Stable cesium was identified in groundwater and surface water at four and one of the eight NPL hazardous waste sites, respectively, where it was detected in some environmental media (HazDat 2003).

The dumping of high and low level radioactive wastes into the Arctic waters by the former Soviet Union has also led to the release of ^{137}Cs and ^{134}Cs as well as other radioactive nuclides into these waters. In the past, the majority of radioactive cesium released to water surfaces in North America arose from deposition following atmospheric nuclear weapons testing conducted by the United States, primarily during the 1960s (Robbins et al. 1990). Radioactive cesium can be introduced to water from nuclear power plants (accidents and normal operation) and at facilities that produce weapons grade plutonium and uranium. During the period of 1961–1973, it was estimated that about 514 Ci of ^{137}Cs was emitted to the Savannah River watershed due to the activities at the Savannah River plutonium processing plant (Olsen et al. 1989). It was further noted that about 18% of this total (92 Ci) drained directly into the Savannah River (Olsen et al. 1989). In 1998, it was reported that $1.0x10^{-4}$ Ci of ^{134}Cs and 0.19 Ci of ^{137}Cs were

released in liquid effluents from the Savannah River plutonium processing site in South Carolina (DOE 1998b). In 1993, the NRC estimated that 1.88 Ci of ^{134}Cs and 2.85 Ci of ^{137}Cs were released in liquid effluents from 30 PWR nuclear power plants operating in the United States (NRC 1993a). It was also estimated that 0.12 Ci of ^{134}Cs and 0.58 Ci of ^{137}Cs were released in liquid effluents from 28 BWR nuclear power plants (NRC 1993a). The EPA reported that the total on-site liquid discharge of ^{137}Cs from containment ponds at the Nevada Test Site was 0.0017 Ci in 1997 (EPA 1999c). It was estimated that 1,622 Ci of ^{137}Cs and 811 Ci of ^{134}Cs were released to the cooling pond surrounding the Chernobyl nuclear power plant following the accident in 1986 (UNSCEAR 1996).

Radioactive ^{134}Cs was not identified in water at the three NPL hazardous waste sites where it was detected in some environmental media (HazDat 2003). However, ^{137}Cs was identified in groundwater and surface water at 5 and 3 of the 23 NPL hazardous waste sites, respectively, where it was detected in some environmental media (HazDat 2003).

6.2.3 Soil

Anthropogenic sources of stable ^{133}Cs releases to soils include the mining, milling, and processing of pollucite ore. It is also found in the ash of coal burning power plants and municipal waste incinerators. Stable cesium was detected at concentrations of 0.44–2.01 ppm in the bottom ash of municipal solid waste incinerators operating in the United States (Mumma et al. 1990) and at concentrations of 3–23 ppm from a municipal waste incinerator operating in Barcelona, Spain (Fernandez et al. 1992). Stable cesium was identified in soil at one of the eight NPL hazardous waste sites and in sediment at one of the eight NPL hazardous waste sites where it was detected in some environmental media (HazDat 2003).

Radioactive cesium has been released to soil surfaces by underground nuclear weapons testing, fallout from the accident at the Chernobyl nuclear power plant, and fallout from atmospheric weapons testing. About 1,400 underground tests have been performed worldwide, with a total explosive yield of about 90 megatons (Agency for Toxic Substances and Disease Registry 1999). Small amounts of ^{137}Cs and ^{134}Cs are also released to soil from the normal operation of nuclear power plants and the storage of spent fuel rods. Not including the 30-km exclusionary zone, an area of approximately 2.4×10^4 km^2 near the Chernobyl nuclear power plant was contaminated with ^{137}Cs at a deposition density $>5.4 \times 10^{-5}$ Ci/m^2 following the accident in 1986 (UNSCEAR 1996). Within the exclusionary zone the contamination density may have been 2 orders of magnitude greater in limited areas (UNSCEAR 1996). The mean deposition density of ^{137}Cs and ^{134}Cs in four different soils in Devoke, United Kingdom for May 1986

were reported as $3.7x10^{-7}$–$5.4x10^{-7}$ Ci/m^2 and $1.0x10^{-7}$–$1.8x10^{-7}$ Ci/m^2, respectively (Hilton et al. 1993). The concentrations of ^{137}Cs in eight sediment cores of the Danube River, Austria were about 540 pCi/kg in April 1985, but increased to approximately 27,000–81,000 pCi/kg in October 1986, following the accident at the Chernobyl nuclear power plant (Rank et al. 1990). The deposition of ^{137}Cs attributed to the accident at the Chernobyl nuclear power plant in sediment at five different sites in Lake Constance, Germany ranged from $2.7x10^{-7}$ to $2.1x10^{-6}$ Ci/m^2, while the concentration attributed to fallout from nuclear weapons testing since 1963 ranged from $1.4x10^{-7}$ to $5.4x10^{-7}$ Ci/m^2 (Richter et al. 1993). It was estimated that 2,973 Ci of ^{137}Cs and 1,622 Ci of ^{134}Cs were released to the sediments in the cooling pond surrounding the Chernobyl nuclear power plant following the accident in 1986 (UNSCEAR 1996). The deposition density of ^{137}Cs in 123 soil cores collected at the Idaho National Engineering and Environmental Laboratory (INEEL), a site for stored transuranic waste in the United States, ranged from $1.6x10^{-8}$ to $3.4x10^{-7}$ Ci/m^2 (DOE 1998a). The deposition density of ^{137}Cs in soils from Idaho, Montana, and Wyoming ranged from $3.0x10^{-9}$ to $1.1x10^{-7}$ Ci/m^2, and it was assumed that its origin was fallout from the Nevada Test Site (DOE 1998a). The mean deposition density of ^{137}Cs in the top layer (0–8 cm) of soils near the Chernobyl nuclear power plant in 1988 was $8.6x10^{-5}$ Ci/m^2 and the mean deposition density of ^{134}Cs was $1.9x10^{-5}$ Ci/m^2 (Mikhaylovskaya et al. 1993).

It was reported that ^{134}Cs was identified in soil at one site, but was not detected in sediment at the three NPL hazardous waste sites where it was detected in some environmental media (HazDat 2003). It was also reported that ^{137}Cs was identified in soil and sediment at 14 and 5 of the 23 NPL hazardous waste sites, respectively, where it was detected in some environmental media (HazDat 2003).

6.3 ENVIRONMENTAL FATE

6.3.1 Transport and Partitioning

The transport and partitioning of particulate matter in the atmosphere is largely dependent upon the physical properties of the matter such as size and density as well as the meteorological conditions such as temperature, the microphysical structure of the clouds, and rainfall rate. The particle size of ^{137}Cs released to the atmosphere following the Chernobyl nuclear accident was in the range of 0.1–10 μm, (Hirose et al. 1993). Particles <5 μm in diameter usually have low deposition velocities and are transported long distances before being removed from the atmosphere. Atmospheric cesium is deposited on land and water via wet and dry deposition and the deposited cesium may be re-suspended to the atmosphere by disturbances that occur on the ground such as vehicular traffic and construction activity.

The wet deposition velocity of ^{137}Cs at Tsukauba, Japan from May 5 to May 30, 1986 ranged from 0.0026 to 0.110 m/second, and the largest value recorded was during a period of heavy rainfall (Hirose et al. 1993). The mean deposition velocity (wet and dry) of ^{137}Cs measured in Prague, Czechoslovakia was reported as 0.08 m/second from 1989–1992 and the mean flux rate was 1,108 pCi/m^2-year (Rybacek et al. 1994).

Since cesium does not volatilize from water, transport of cesium from water to the atmosphere is not considered likely, except by windblown sea sprays. Most of the cesium released to water will adsorb to suspended solids in the water column and ultimately be deposited in the sediment core. Cesium can also bioconcentrate and has been shown to bioaccumulate in both terrestrial and aquatic food chains. Mean bioconcentration factors (BCF) for ^{137}Cs of 146, 124, and 63 were reported for fish, brown macroalgae, and molluscs, respectively (Fisher et al. 1999). Mean BCF values of 92, 58, 39, and 150 were reported for ^{137}Cs in cod, haddock, plaice, and whiting, respectively (Steele 1990). In a study of aquatic organisms inhabiting the Ottawa River, a 4-fold increase of ^{137}Cs levels was observed with each trophic level (Rowan et al. 1998). The levels of ^{137}Cs in lake trout from Great Slave Lake, Canada were consistently higher than levels found in food sources and a biomagnification factor of 1.9 was calculated for lake trout, relative to their food sources. The biomagnification factor was 3.5 for large mature trout populating the lake (Rowan et al. 1998). It was shown that the bioconcentration and bioaccumulation of ^{137}Cs by aquatic organisms is significantly reduced in waters with a large humic content and high levels of potassium cations (Penttila et al. 1993). Because of the high potassium concentration in oceans, the transfer of ^{137}Cs and ^{134}Cs to fish is much greater in freshwater and the activity of freshwater fish may be 100 times that of ocean fish, given the same cesium concentration in the water (WHO 1983).

In soil surfaces, cesium has low mobility in comparison to other metals and usually does not migrate below a depth of 40 cm. The major portion of cesium is retained in the upper 20 cm of the soil surface (Korobova et al. 1998; Ruse and Peart 2000; Takenaka et al. 1998). Vertical migration patterns of ^{137}Cs in four agricultural soils from southern Chile indicated that approximately 90% of the applied cesium was retained in the top 40 cm of soil, and that in one soil, essentially 100% was bound in the upper 10 cm (Schuller et al. 1997). Migration rates of radiocesium were derived from the depth distribution profiles and were in the range of 0.11 to 0.29 cm/year (Schuller et al. 1997). The vertical migration patterns of ^{90}Sr and ^{137}Cs produced from the atomic bomb exploded in Nagasaki, Japan were studied over a 40-year period (Mahara 1993). Over this period, 95% of the cesium remained in the top 10 cm of the soil surface and no cesium was detected below a depth of 40 cm. In contrast, only 70% of ^{90}Sr was located within a depth of 10 cm and a small percentage was detectable below a depth of 200 cm. The *in situ* vertical

migration rate of ^{90}Sr was calculated as 0.42 cm/year and the migration rate of ^{137}Cs was 0.10 cm/year (Mahara 1993). The distribution of ^{137}Cs deposited in eastern France due to the Chernobyl accident was studied recently in four soil types with differing land use. The soils were all high in clay content, organic matter content never exceeded 10% in any of the soils, and none of the soils had been ploughed or tilled since the Chernobyl accident in 1986. In all cases, the majority of ^{137}Cs was detected in the upper 10 cm of soil, with only 1–2% migrating to depths of 40 cm or more (Renaud et al. 2003). Soil adsorption coefficients (K_d) of five radionuclides (^{54}Mn, ^{60}Co, ^{65}Zn, ^{85}Sr, and ^{137}Cs) were measured for 36 agricultural soils collected in Japan. It was determined that ^{137}Cs had the largest median K_d of all five radionuclides, and that a positive correlation was observed between the adsorption coefficient and exchangeable potassium content in the soil (Yasuda et al. 1995). No correlations were observed for other soil properties such as pH, water content, cation exchange capacity, and exchangeable calcium. Other studies have reported that clay and zeolite minerals strongly bind cesium cations and can therefore reduce the bioavailability of cesium and the uptake in plants by irreversibly binding cesium in interlayer positions of the clay particles (Paasikallio 1999). Experiments conducted by growing plants in a peat soil showed that the introduction of zeolites into the soil-plant system decreased the uptake of ^{134}Cs in plants by a factor of 8 (Shenber and Johanson 1992). The low hydration energy of cesium cations is primarily responsible for their selective sorption and fixation by clays and zeolites (Hakem et al. 1997). Soils rich in organic matter adsorb cesium, but the cesium adsorbed in the organic fraction is readily exchangeable and highly available for plant uptake (Sanchez et al. 1999). Regions in Venezuela, Brazil, and Russia have been identified where a great deal of rain is encountered, the soil is peaty or podzolic (a type of forest soil characterized by high leachability), and the mobility of cesium is considerably greater than in other soils (LaBrecque and Rosales 1996; WHO 1983).

The plant/soil concentration ratio (activity/kg of plant/activity/kg of soil) of ^{137}Cs for field crops in southern Finland ranged from 0.01 to 0.26. In northern Finland, this ratio ranged from 0.01 to 2.29, with the lowest values occurring in clay and silt soils and the highest values observed in fine sands (Paasikallio et al. 1994). The plant/soil concentration ratios for a series of vegetables and grains decreased in the following order: lettuce, cabbage>carrot, potato>cereals, onion; for fruits, the order was: black-currant>strawberry>apple (Paasikallio et al. 1994). The mean plant/soil concentration ratios of ^{137}Cs for trees at the Hanford Waste Site in the United States were 0.03 (roots), 0.06 (cores), and 0.02 (leaf/twig) (Landeen and Mitchell 1986).

6.3.2 Transformation and Degradation

6.3.2.1 Air

When pure cesium metal is exposed to air, an explosion-like oxidation occurs, forming a mixture of cesium oxides (Cs_2O, Cs_2O_2, and Cs_2O_3). Cesium compounds released to the atmosphere will eventually settle to earth by wet and dry deposition. Radioactive forms of cesium such as ^{137}Cs and ^{134}Cs are continuously transformed to stable isotopes of barium or xenon by the natural process of radioactive decay. The pathways and mechanisms of these reactions have been described in Chapter 4.

6.3.2.2 Water

When pure cesium metal is released to water, a vigorous reaction occurs yielding cesium hydroxide (CsOH), the strongest base known, and hydrogen gas, which may ignite spontaneously. In general, cesium compounds are very water soluble, in which cesium is present in the ionic form as the Cs^+ cation. Under normal environmental conditions, Cs^+ cations are neither degraded nor transformed, but may adsorb to suspended solids and sediment in the water column, forming insoluble complexes.

6.3.2.3 Sediment and Soil

Cesium salts and most cesium compounds are generally very water soluble, with the exception of cesium alkyl and aryl compounds, which have low water solubility. Cesium cations have low hydration energy and can react with clay minerals, zeolites, or soils with a high percentage of exchangeable potassium, forming insoluble, immobile complexes.

6.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

Reliable evaluation of the potential for human exposure to cesium depends in part on the reliability of supporting analytical data from environmental samples and biological specimens. Radioactive cesium is measured in units of activity, not mass. A great deal of monitoring data for radioactive cesium in environmental samples prior to, and shortly following, the accident at the Chernobyl nuclear power plant on April 26, 1986 has been included in this chapter.

6.4.1 Air

Data reporting the background levels of ^{133}Cs in the atmosphere are limited. Since the amount of cesium mined and milled is small, it is expected that background concentrations in the atmosphere will be low. The concentration of ^{133}Cs measured in the South Pole during 1974–1975 was reported in the range of 0.072–0.14 pg/m^3, with a mean of 0.10 pg/m^3 (Maenhaut et al. 1979). The maximum airborne concentrations of ^{133}Cs at the Tera Nova Bay Station in the Arctic were reported as 20–90 pg/m^3 during the 1989–1990 Italian expedition and 10–60 pg/m^3 for the 1990–1991 expedition (Chiavarini et al. 1994). The average concentration of ^{133}Cs in precipitation collected in Lennox, Massachusetts during the summer of 1984 was reported as 0.0075 μg/L, with a range from 0.0050 to 0.024 μg/L (Dasch and Wolff 1989).

Radioactive ^{134}Cs and ^{137}Cs have been detected at various concentrations (activities) in the atmosphere following the accident at the Chernobyl nuclear power plant on April 26, 1986. The greatest concentrations were observed in locations throughout Russia and Europe, but detectable levels were measured globally, including in North America. The air concentrations of ^{134}Cs above the destroyed reactor were 1,756.8 pCi/m^3 on May 8 and 54.0 pCi/m^3 on May 18 (Buzulukov and Dobrynin 1993). The concentrations of ^{137}Cs in the air above the destroyed reactor were 8,918.9 pCi/m^3 on May 8 and 135.1 pCi/m^3 on May 18 (Buzulukov and Dobrynin 1993). Atmospheric concentrations of ^{134}Cs in Belgrade, Yugoslavia were 5.4×10^{-4} (May 1, 1986), 160.5 (May 2, 1986), 145.0 (May 3, 1986), and 25.4 (May 4, 1986) pCi/m^3, while atmospheric concentrations of ^{137}Cs were 0.001 (May 1, 1986), 324 (May 2, 1986), 276 (May 3, 1986), and 49 (May 4, 1986) pCi/m^3 (Ajdacic and Martic 1990). Cesium concentrations decreased significantly over time as advection, wet deposition, and dry deposition lowered the atmospheric concentrations. The average monthly atmospheric concentrations of ^{137}Cs in Belgrade that were measured during 1991–1996 ranged from 1.3×10^{-4} to 2.0×10^{-3} pCi/m^3, with a pronounced maximum during the winter months (Todorovic et al. 1999). Monitoring data from Prague, Czechoslovakia indicated that there was a gradual decrease in the atmospheric concentration of ^{137}Cs from May 1986 to February 1992. Maximum concentrations were observed immediately following the accident at the Chernobyl nuclear power plant (approximately 0.05 pCi/m^3) and gradually decreased over a 6-year period (Rybacek et al. 1994). The atmospheric concentration of ^{134}Cs decreased more rapidly, presumably due to the shorter half-life of ^{134}Cs. The concentration of ^{137}Cs in the atmosphere of Thessaloniki, Greece ranged from 8.1×10^{-4} to 0.044 pCi/m^3 from July 1987 to December 1988, and the concentration of ^{137}Cs in rainfall ranged from 0.27 to 34 pCi/L from November 1986 to February 1989 (Papastefanou et al. 1989). During a heavy rainfall event occurring on May 6, 1986, ^{137}Cs was detected at a concentration of 46,000 pCi/L (Papastefanou et al. 1989). The concentration of ^{137}Cs in Tsukuba, Japan

during May 1986 ranged from about 0.054 to 1.6 pCi/m^3 (Hirose et al. 1993). The average atmospheric concentrations of ^{134}Cs and ^{137}Cs in eastern Canada were reported as 0.024 and 0.046 pCi/m^3, respectively, during May 10–24, 1986 (Huda et al. 1988). The maximum atmospheric concentration of ^{137}Cs measured in New York City in May 1986 was reported as 0.26 pCi/m^3 (Feely et al. 1988). The average concentrations of ^{137}Cs and ^{134}Cs in Barrow, Alaska were reported as 0.027 and 0.019 pCi/m^3, respectively, for the month of May 1986 (DOE 1986).

6.4.2 Water

The background concentration of ^{133}Cs in fresh water lakes and rivers is ordinarily in the range of 0.01 to 1.2 μg/L, and the concentration in seawater is about 0.5 μg/L (WHO 1983). Stable cesium was detected in streams that feed into the Tamagawa River, Japan at concentrations of 9×10^{-4} to 0.093 μg/L (Tanizaki et al. 1992). Studies from five distinct geochemical areas of the semi-arid endorheic region of the Famatina Range (La Rioja, Argentina) have shown some areas contain high levels of ^{133}Cs in natural waters and sediment (Fernandez-Turiel et al. 1995). The cesium concentration in fresh water systems of this region ranged from 0.58 to 3.69 μg/L (Fernandez-Turiel et al. 1995). The concentrations of 19 trace metals were studied in drinking water and groundwater supplies in southern Nigeria. Stable cesium was detected in groundwater and drinking water at mean concentrations of 0.61 μg/L (range 0.09–3.72 μg/L) and 0.35 μg/L (range 0.05–4.32 μg/L), respectively (Asubiojo et al. 1997). The mean concentration of cesium in drinking water was lower than the mean concentration of any of the other trace elements analyzed.

High and low level radioactive wastes were dumped by the former Soviet Union into remote Arctic waters, leading to the release of radioactive cesium into the Kara and Barents Seas. The level of ^{137}Cs in surface water of the Barents Sea and Kara Sea was 0.14 and 0.16 pCi/L, respectively, and ^{137}Cs was also detected in deep water of the Barents Sea at a concentration of 0.15 pCi/L (Fisher et al. 1999). The concentration of ^{137}Cs in the Black Sea ranged from 2.7 to 8.1 pCi/L for the period of 1991–1996, with the exception of the spring of 1992, when concentrations as high as 43 pCi/L were observed (Strezov et al. 1999). Preliminary measurements taken in the vicinity of the nuclear submarine, the Kursk, which accidentally sank in the Barents Sea in August 2000, indicate that that no leakage of radionuclides from the reactor has occurred and only background levels of radioactive cesium (and other radionuclides) have been observed in water and sediment near the submarine (Amundsen et al. 2002). From 1988 to 1991, the mean concentrations of ^{137}Cs and ^{134}Cs along the Spanish coast of the Mediterranean Sea were 0.13 and 0.0072 pCi/L, respectively (Molero et al. 1999). Due to its shorter half-life, ^{134}Cs was detected in all

14 samples collected in 1988 and 1989, but only in 3 samples collected in 1990 and 1991, suggesting that the ^{134}Cs levels observed in surface Mediterranean waters during this period were due mainly to Chernobyl-related deposition. The ^{137}Cs concentration incorporated into the Mediterranean Sea near the Spanish coast from the post-Chernobyl fallout was about 0.032 pCi/L, which was approximately a 33% increase over previous levels (Molero et al. 1999). Maximum ^{137}Cs and ^{134}Cs levels in the immediate vicinity of nuclear power plants located on the southern Catalan shore of the Mediterranean were 0.57 and 0.059 pCi/L (Molero et al. 1999). Concentrations of ^{137}Cs in lakes and streams in Devoke, United Kingdom decreased exponentially from a maximum concentration of about 8.1 pCi/L on May 6, 1986 to about 0.027 pCi/L 1,200 days later (Hilton et al. 1993). The mean concentration of ^{137}Cs in six lakes located in central Finland ranged from 111 pCi/L in 1987 to 8.1 pCi/L in 1989 (Penttila et al. 1993).

The concentration of ^{137}Cs and ^{134}Cs in groundwater at 18 U.S. Department of Energy (DOE) facilities was reported in the range of 2.7×10^{-3} to 1.83×10^{3} pCi/L (DOE 1992). The concentration of ^{137}Cs measured in groundwater wells at Carlsbad, New Mexico (the site of Project GNOME) ranged from 99 to 6.8×10^{5} pCi/L in 1997 (EPA 1999c). The concentration of ^{137}Cs in groundwater at the Chernobyl nuclear power plant was in the range of 40.5–1,100 pCi/L in 1988 and 29.7–129.7 pCi/L in 1989 (Prister et al. 1990). The mean concentration of ^{137}Cs in drinking water in Poland in 1974 was reported as 0.2 pCi/L (Glowiak et al. 1977b). These concentrations in water may be compared to the federal radiation safety standards. For continuous ingestion over a lifetime, the maximum concentrations of ^{134}Cs and ^{137}Cs in drinking water are limited to 900 and 1,000 pCi/L respectively; these limits assume no other intake of radioactivity. If other radioisotopes are ingested, then the intake limits for each must be adjusted proportionately (USNRC 1999a).

6.4.3 Sediment and Soil

Stable cesium occurs naturally in the earth's crust at low concentrations. Granites contain an average cesium concentration of about 1 ppm and sedimentary rocks contain about 4 ppm (Burt 1993). Others have estimated cesium concentration of granites as high as 5 ppm (WHO 1983). Stable cesium was detected in dust samples from roadside and pedestrian traffic in Nagpur, India at concentrations of 1.53–3.63 μg/g, with the largest value obtained in the vicinity of a metals factory (Chutke et al. 1995). ^{133}Cs was detected at concentrations ranging from 0.9 to 2.2 μg/g in alluvial sediments in the Sava River, Croatia (Vertacnik et al. 1997). The concentration of ^{133}Cs in river sediment from five distinct geochemical areas of the semi-arid endorheic region of the Famatina Range (La Rioja, Argentina) was 2.28–6.20 μg/g (Fernandez-Turiel et al. 1995).

The concentration of ^{137}Cs in soils of Thessaloniki, Greece ranged from 1,440 to 35,324 pCi/kg (average 8,154 pCi/kg) and the concentration of ^{134}Cs ranged from about 270 to 5,676 pCi/kg during the period of August 1986 to February 1989, with most of the fallout attributed to the accident at the Chernobyl nuclear power plant (Papastefanou et al. 1989). The concentration of ^{137}Cs in 10 uncultivated fields from southern England ranged from 0 to 946 pCi/kg, with the highest levels contained in the top 10 cm of the soil surface (Owens et al. 1996). The concentration of ^{137}Cs in five cultivated fields ranged from 0 to 540 pCi/kg, and the concentrations were well distributed from the surface to the plough layer (Owens et al. 1996). The concentration of ^{137}Cs in three soils in Hong Kong receiving a large amount of rainfall ranged from 32 to 201 pCi/kg (Ruse and Peart 2000). The concentration of ^{137}Cs in sediment from the Ribble Estuary, England near the British Nuclear Fuels Laboratory ranged from 270 to 1,351 pCi/kg (Brown et al. 1999). The average concentration of ^{137}Cs in sediment from Lake Ontario was reported as 8,108 pCi/kg, and suspended solids from the Niagara River contained 324 pCi/kg, measured during the 1983–1986 sampling period (Platford and Joshi 1989). The average concentrations of ^{137}Cs in uncultivated soils in northern Poland were reported as 616–4,170 pCi/kg from 1988 to 1991 (Pietrzak-Flis et al. 1994). The mean concentration of ^{137}Cs in surface soil samples from the Los Alamos nuclear laboratory test site during the period of 1974–1996 was reported as 611 pCi/kg (Fresquez et al. 1998). Concentrations of ^{137}Cs around the perimeter of the site and background concentrations off the site were reported as 589 and 419 pCi/kg , respectively. The concentration of ^{137}Cs and ^{134}Cs in soils and sediments at 18 U.S. DOE facilities was reported to range from 20 to 4.69×10^7 pCi/kg (DOE 1992). The concentration of ^{137}Cs in sediment from the Savannah River ranged from 5 to 100 pCi/kg in 1986, while the concentration in suspended solids and particulate matter ranged from 240 to 4.324×10^6 pCi/kg (Olsen et al. 1989). The mean concentration of ^{137}Cs in soil at the Hanford Site in the United States was 4,540 pCi/kg (Landeen and Mitchell 1986). The mean concentration of ^{137}Cs in soils taken from two high-elevation sites in northern Colorado ranged from 4,054 to 7,027 pCi/kg (Ulsh et al. 2000).

The concentration of ^{137}Cs in sediment near the nuclear submarine, the Kursk, which accidentally sank in the Barents Sea in August 2000, ranged from about 18.9 to 40.5 pCi/kg (Amundesen et al. 2002). These levels are considered typical background levels in the Barents Sea and are attributed to releases from the Sellafield reprocessing facility in the United Kingdom, Chernobyl related fallout, and deposition from past nuclear weapons tests.

6.4.4 Other Environmental Media

Information on the concentrations of ^{137}Cs, ^{134}Cs, and ^{133}Cs in various animals is shown in Table 6-2. Concentrations are dependent on the location, date, and level of exposure. For example, the concentrations of ^{137}Cs in bullhead catfish inhabiting an abandoned nuclear reactor reservoir at the Savannah River site in South Carolina were as high as $1.54x10^5$ pCi/kg (McCreedy et al. 1997), but concentrations in various freshwater species of fish in the Ottawa River were in the range of 54 to 351 pCi/kg (Rowan et al. 1998). After the accident at the Chernobyl nuclear power plant, the average concentrations of ^{137}Cs in perch, pike, and roach obtained from 52 freshwater lakes in Finland were 55,811, 66,297, and 15,270 pCi/kg, respectively, in 1988. By contrast, the mean concentrations in 1992 had fallen to 14,324, 18,567, and 5,892 pCi/kg, respectively (Sarkka et al. 1996). The concentration of total ^{137}Cs and ^{134}Cs measured in body tissue from sheep in Ireland from 1989 to 1991 ranged from about 2,700 to 10,811 pCi/kg, with highest levels observed during November of 1989 (McGee et al. 1993).

The concentrations of ^{133}Cs in plants from Britain were studied over a 1-year period. Concentrations of 50–300 ng/g were measured, with the highest levels observed during the summer and fall months. The median concentration of ^{133}Cs in poplar leaves collected in Bulgaria was reported as 75 ng/g, while concentrations in land plants ranged from 30 to 440 ng/g (Djingova et al. 1995). In contrast to the radioactive isotopes of cesium, concentrations of stable ^{133}Cs depend primarily on the root uptake from soil and not on atmospheric deposition. Lichens and mosses have been shown to trap and retain ^{137}Cs and ^{134}Cs more than vascular plants due to their relatively large surface area. Lichens and mosses from northern Greece contained ^{137}Cs levels of $6.6x10^4$–$5.1x10^5$ pCi/kg during the period of 1989–1991 (Papastefanou et al. 1992) and moss samples from Finland collected in 1988–1989 contained $4.3x10^4$–$9.7x10^5$ pCi/kg (Penttila et al. 1993). The mean concentration of ^{137}Cs in three species of lichens collected in August 1986 from Megalopolis, Greece were $2.6x10^4$–$3.3x10^4$ pCi/kg, while the mean concentrations for the same three species of lichens collected in October 1996 had fallen to 3,324–7,892 pCi/kg (Riga-Karandinos and Karandinos 1998). Mushrooms, lichens, and mosses obtained near Manitoba, Canada in August 1986 contained ^{137}Cs at mean concentrations of $6.4x10^5$, $8.6x10^4$, and $8.4x10^4$ pCi/kg, respectively (Mihok et al. 1989). Since caribou and reindeer consume large amounts of this vegetation during the winter months, elevated levels of ^{137}Cs and ^{134}Cs have been detected in these animals.

Stable cesium has been detected infrequently in food products at low concentrations. The average concentration of stable ^{133}Cs in 110 onion samples collected in Denmark was 0.21 μg/kg, with a range of

Table 6-2. Concentration of ^{133}Cs, ^{134}Cs, and ^{137}Cs in Animals

Species	Location and date	^{133}Cs (μg/g)	^{134}Cs (Bq/kg)	^{137}Cs (Bq/kg)	Source
Royal albatross (N=3)	Indian Ocean (1994)	0.009–0.025 (0.014 mean); liver	No data	No data	Kim et al. 1998
Black-footed albatross (N=18)	North Pacific (1985)	0.007–0.049 (0.022 mean); liver	No data	No data	Kim et al. 1998
Black-browed albatross (N=9)	Indian Ocean (1994)	0.013–0.042 (0.022 mean); liver	No data	No data	Kim et al. 1998
White-capped albatross (N=3)	Indian Ocean (1994)	0.011–0.039 (0.029 mean); liver	No data	No data	Kim et al. 1998
Yellow-nosed albatross (N=4)	Indian Ocean (1994)	0.0029–0.079 (0.022 mean); liver	No data	No data	Kim et al. 1998
Grey-headed albatross (N=10)	Indian Ocean (1994)	0.011–0.031 (0.016 mean); liver	No data	No data	Kim et al. 1998
Northern giant petrel (N=6)	Indian Ocean (1994)	0.005–0.034 (0.015 mean); liver	No data	No data	Kim et al. 1998
Northern fulmar (N=17)	North Pacific (1985)	0.008–0.036 (0.016 mean); liver	No data	No data	Kim et al. 1998
Bluefin tuna (N=14)	Newfoundland (1990)	0.08–0.24 (0.13 mean); muscle	No data	No data	Hellou et al. 1992a
Cod (N=12)	Newfoundland (1990)	0.16–0.24 (0.19 mean); muscle	No data	No data	Hellou et al. 1992b
Cod (N=10)	Newfoundland (1990)	0.14–0.36 (0.23 mean); muscle	No data	No data	Hellou et al. 1992b
Pilot whale (N=9)	North Atlantic (1987–1996)	0–0.010 (0.006 mean); liver	No data	No data	Becker et al. 1997
White-sided dolphin (N=4)	North Atlantic (1987–1996)	0.027–0.042 (0.032 mean); liver	No data	No data	Becker et al. 1997
Beluga whale (N=15)	Arctic (1987–1996)	0.021–0.046 (0.031 mean); liver	No data	No data	Becker et al. 1997
Ringed seal (N=13)	Arctic (1987–1996)	0.0045–0.048 (0.020 mean); liver	No data	No data	Becker et al. 1997
Woodcock (N=24)	Ireland (1986)	No data	3.9–206.4; muscle	6.2–565.5; muscle	Pearce 1995
Duck (N=5)	Ireland (1986)	No data	2.2–14.3; muscle	6.4–18.0; muscle	Pearce 1995
Snipe (N=5)	Ireland (1986–1987)	No data	1.0–5.4; muscle	3.6–16.9; muscle	Pearce 1995
Reindeer (N=8)	Northern Sweden (1986–1987)	No data	No data	900 (mean); muscle	Ahman and Ahman 1994
Deer (N=11)	Los Alamos (1991–1998)	No data	No data	2,516 (mean); muscle	Fresquez et al. 1999a

Table 6-2. Concentration of ^{133}Cs, ^{134}Cs, and ^{137}Cs in Animals

Species	Location and date	^{133}Cs (μg/g)	^{134}Cs (Bq/kg)	^{137}Cs (Bq/kg)	Source
Deer (N=11)	Los Alamos (1991–1998)	No data	No data	888 (mean); bone	Fresquez et al. 1999a
Caribou (N=18)	Saskatchewan (1995)	No data	No data	58 (mean); bone	Thomas and Gates 1999
Caribou (N=18)	Saskatchewan (1995)	No data	No data	228 (mean); liver	Thomas and Gates 1999
Caribou (N=18)	Saskatchewan (1995)	No data	No data	367 (mean); muscle	Thomas and Gates 1999
Caribou (N=18)	Saskatchewan (1995)	No data	No data	553 (mean); kidney	Thomas and Gates 1999
Caribou (N=36)	Alaska (1987)	No data	No data	26–232; neck	Allaye-Chan et al. 1990
Caribou (N=36)	Alaska (1987)	No data	No data	28.4–201.1; shoulder	Allaye-Chan et al. 1990
Caribou (N=36)	Alaska (1987)	No data	No data	30.2–166.5; back	Allaye-Chan et al. 1990

not detected to 0.98 μg/kg (Bibak et al. 1998). By comparison, other elements such as calcium and potassium had mean concentrations of $2x10^5$ and $1.6x10^6$ μg/kg, respectively. The concentration range of ^{133}Cs in wheat flour samples collected in Pakistan was 6.7–11.2 ppb, but ^{133}Cs was not detected from wheat flour samples from America (Ahman et al. 1994).

Levels of ^{137}Cs were below detection limits for all foods analyzed for in the U.S. Food and Drug Administration (FDA) Total Diet Study in 1991–1996 with the exception of honey (Capar and Cunningham 2000). The concentration of ^{137}Cs in honey from the 1995 Market Basket Survey was 6.7 Bq/kg (181.1 pCi/kg), which is almost 200 times lower than the regulatory level of ^{137}Cs in foods. The average concentrations of total ^{137}Cs and ^{134}Cs in milk powder, infant milk powder, infant cereal, meat, lentil, wheat, and macaroni samples from Saudi Arabia were 514, 351, 486, 162, 270, 676, and 351 pCi/kg, respectively (Abdul-Majid et al. 1992). For the month of June 1986, the average concentrations of total ^{137}Cs and ^{134}Cs in milk, green vegetables, fruit, lamb, and beef were reported as 3,243, 2,703, 2,703, 8,108, and 1,622 pCi/kg in high deposition areas of the United Kingdom (Cumbria, north Wales, Scotland, northern Ireland, and the Isle of Man) (Mondon and Walters 1990). The concentration of total ^{137}Cs and ^{134}Cs was <676 pCi/kg in each of these food sources in areas of low deposition during this time frame. The maximum concentration of ^{137}Cs in pasteurized milk from 65 cities in the United States was 14 pCi/L in May 1989 (EPA 1989). The concentration of ^{137}Cs in fresh milk from Chester, New York and pasteurized milk samples from New York City in May 1986 ranged from about 5.4 to 18.9 pCi/L (Feely et al. 1988). Using radiological surveys from 1978 and 1985–1986, the concentration of ^{137}Cs in 44 adult food groups from the Rongelap Island and Rongelap Atoll was in the range of 0.52–13,000 pCi/kg (Robinson and Phillips 1989).

6.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

General population exposure to cesium occurs by ingestion of food and drinking water, by inhalation of ambient air, and dermal contact with cesium in soil. Since stable ^{133}Cs is found in low concentrations in the environment, the exposure to the general population is also low. The National Health And Nutritional Examination Survey (NHANES), conducted by the Centers for Disease Control and Prevention (CDC), reported that the geometric mean concentration of ^{133}Cs in the urine of 2,464 U.S. residents in various population groups was as follows: children aged 6–11 years, 4.87 μg/L; adolescents aged 12–19 years, 4.54 μg/L; adults aged 20 or older, 4.26 μg/L (CDC 2003). Based upon gender, males had a higher geometric mean concentration (4.83 μg/L), as compared to females (3.94 μg/L). Occupational exposure to ^{133}Cs occurs primarily through inhalation and dermal contact at workplaces where pollucite is mined or

cesium compounds are manufactured or used. The National Occupational Exposure Survey (NOES) conducted by the National Institute for Occupational Safety and Health (NIOSH) from 1981 to 1983 has estimated that 16,461 workers (4,276 of these were female) were potentially exposed to cesium and cesium compounds in the United States (NOES 1989). The NOES database does not contain information on the frequency, level, or duration of the exposure of workers to any of the chemicals listed therein. They are surveys that only provide estimates of workers potentially exposed to the chemicals. The median concentration of ^{133}Cs in the lungs of metal workers in northern Sweden was 0.016 μg/g and was lower than the median concentration of 0.021 μg/g for a control group that was not occupationally exposed (Hewitt 1988). The range of ^{133}Cs concentrations in lung tissue of coal miners from the United Kingdom was 0.07–0.91 μg/g (Hewitt 1988).

Exposure to radioactive cesium is of greater concern from a health perspective than exposure to stable cesium. The primary source of radioactive cesium in the environment is due to fallout from past atmospheric nuclear weapons tests and the Chernobyl accident. Additional contributions from the normal operation of nuclear power plants and other nuclear facilities are small by comparison. Current exposure of the general U.S. population to ^{134}Cs and ^{137}Cs is expected to be low since atmospheric testing of nuclear weapons was discontinued in 1963 and Chernobyl-related fallout was low in the United States. As discussed in Appendix A, the average annual effective dose of ionizing radiation (including ^{134}Cs and ^{137}Cs) to the U.S. population from anthropogenic sources is negligible in comparison to natural sources, especially radon and its decay products.

The average daily intake (AVDI) of ^{137}Cs and ^{134}Cs was estimated for adult males residing in the Ukraine in 1994, based upon total diet samples. The mean intake of ^{137}Cs was estimated as 109 pCi/day and the mean intake of ^{134}Cs was estimated as 8.1 pCi/day (Shiraishi et al. 1997). Based on dietary patterns and the concentration of radiocesium in food sources, the total dietary intakes of ^{134}Cs and ^{137}Cs for typical adults residing in Croatia for the month of May 1986 were estimated as 2.8×10^4 and 5.9×10^4 pCi, respectively (Lokobauer et al. 1988). The mean monthly levels of ^{137}Cs in human muscle tissue in Graz, Austria were reported as 1,519 (July 1986 to June 1987), 1,049 (July 1987 to June 1988), 340.5 (July 1988 to June 1989), and 202.7 pCi/kg (July 1989 to June 1990), with a maximum value of 9,584 pCi/kg observed in an individual during September 1986 (Rabitsch et al. 1991). The monthly averages for ^{134}Cs were about half of those reported for ^{137}Cs. By comparison, the maximum concentration of ^{137}Cs in muscle tissue from Harwell, England in 1959 was reported as 224 pCi/kg, the mean concentration in muscle tissue from Massachusetts during January 1961 to June 1962 was 100 pCi/kg, and the mean concentration in human muscle tissue obtained from Japan during April to December 1963 was reported

as 119 pCi/kg (Rabitsch et al. 1991). The mean concentrations of ^{137}Cs in the urinary excretion of Italians in northern Italy were 7.3 and 6.2 pCi/day in 1995 and 1996, respectively (Ropolo and Cesana 1997). These values were about two orders of magnitude less than values reported for measurements taken in 1987. The mean concentration of ^{137}Cs in brain, heart, liver, gonads, muscle, bone, and teeth were 0.440, 1.860, 0.490, 2.440, 0.017, 0.106, and 0.23 pCi/g, respectively, for adult cadavers over 34 years of age in Poland during 1975 (Glowiak et al. 1977a). The mean body burdens of ^{137}Cs for adults in Helsinki, Viitasaari, and Ammans, Finland from 1987 to 1994 are given in Table 6-3 (Rahola and Suomela 1998).

Persons employed at nuclear power facilities and waste disposal sites are potentially exposed to higher levels of ^{137}Cs and ^{134}Cs than the is general population. The NOES conducted by NIOSH from 1981 to 1983 estimated that 13,148 workers (1,294 of these were female) were potentially exposed to ^{134}Cs and ^{137}Cs in the United States (NOES 1989).

6.6 EXPOSURES OF CHILDREN

This section focuses on exposures from conception to maturity at 18 years in humans. Differences from adults in susceptibility to hazardous substances are discussed in Section 3.7, Children's Susceptibility.

Children are not small adults. A child's exposure may differ from an adult's exposure in many ways. Children drink more fluids, eat more food, breathe more air per kilogram of body weight, and have a larger skin surface in proportion to their body volume. A child's diet often differs from that of adults.

The developing human's source of nutrition changes with age: from placental nourishment to breast milk or formula to the diet of older children who eat more of certain types of foods than adults. A child's behavior and lifestyle also influence exposure. Children crawl on the floor, put things in their mouths, sometimes eat inappropriate things (such as dirt or paint chips), and spend more time outdoors. Children also are closer to the ground, and they do not use the judgment of adults to avoid hazards (NRC 1993a).

As for adults in the general population, exposures of children to cesium occur from normal ingestion of food and drinking water, inhaling air, and dermal contact with cesium in soil. No information on cesium levels in amniotic fluid, meconium, cord blood, or neonatal blood was available.

Radioactive cesium was detected in several pasteurized milk and breast milk samples worldwide, and since children tend to consume large amounts of milk, this represents an important source of childhood

Table 6-3. The Mean Body Burdens of ^{137}Cs for Adults in Finland from 1987 to 1994[a]

Location	Date	Concentration (pCi/kg)
Helsinki, Finland	1987	649
	1988	568
	1989	405
	1990	297
	1991	246
	1992	214
	1993	195
	1994	181
Viitasaari, Finland	1987	3,514
	1988	1,946
	1989	1,595
	1990	1,081
	1991	838
	1992	676
	1993	649
	1994	514
Ammans, Finland	1987	2,892
	1988	3,108
	1989	2,243
	1990	2,486
	1991	1,568
	1992	1,405
	1993	865
	1994	811

[a]Rahola and Suomela 1998

exposure. The maximum concentration of ^{137}Cs in pasteurized milk from 65 cities in the United States was 14 pCi/L in May 1989 (EPA 1989). Concentrations of ^{137}Cs in human milk samples from several U.S. cities from 1956 to 1961 were <20 pCi/L (Eaman 1986). The overall ^{137}Cs concentrations in milk taken 7 days postpartum from 37 mothers in two Italian hospitals were 5.8–115 pCi/L (Eaman 1986). In a study of females from northern Sweden, ^{137}Cs was detected in breast milk from 8 out of 12 mothers at concentrations of 7.3–178.4 pCi/kg (Johansson et al. 1998). The infants of these mothers who were breast-fed had whole-body levels of ^{137}Cs in the range of 45.9–675.7 pCi/kg (Johansson et al. 1998). Based on dietary patterns and the concentration of radiocesium in food sources, the total dietary intakes of ^{134}Cs and ^{137}Cs for children (10 years old) residing in Croatia for May–June 1986 were estimated as 43,000 and 190,000 pCi, respectively (Lokobauer et al. 1988). For infants (1 year of age), it was estimated that the total intakes of ^{134}Cs and ^{137}Cs were 46,000 and 170,000 pCi, respectively (Lokobauer et al. 1988). The total intakes of ^{134}Cs and ^{137}Cs for adults during this same time period were estimated as 40,000 and 84,000 pCi, respectively (Lokobauer et al. 1988). The higher intakes of ^{134}Cs and ^{137}Cs for infants and children were linked to a greater consumption of contaminated milk.

The tendency of young children to ingest soil, either intentionally through pica or unintentionally through hand-to-mouth activity, is well documented. These behavioral traits can result in ingestion of cesium present in soil and dust. Soil ingestion may be a potentially important exposure pathway in areas that have historically had a great deal of ^{134}Cs and ^{137}Cs deposited onto soil surfaces from the accident at the Chernobyl nuclear power plant or fallout from weapons testing. Playing in contaminated soil could also lead to dermal and external exposure. Ingested cesium is adsorbed strongly to soils and may not be in bioavailable form. In a study in which 102 healthy volunteers were fed ^{134}Cs-contaminated soil pellets, only about 1% of the original amount was absorbed and on average, about 60% of the intake was excreted within 48 hours (LeRoy et al. 1966).

It is unlikely that children whose parents are employed at nuclear power generating plants and facilities that store or handle radioactive waste are exposed to ^{134}Cs and ^{137}Cs from parents' clothing or items removed from the workplace because workers pass through exit monitors at nuclear power plants, and the extensive use of outerware that remains in the plant prevents these types of incidents. It is also unlikely that children are exposed to ^{133}Cs from parents' clothing or items that have been removed from the workplace if the parents are employed in the mining, milling, or processing of pollucite ore. Other home exposures are unlikely since household items or products used in crafts, hobbies, or cottage industries do not contain significant amounts of cesium.

6.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

Few populations are likely to be exposed to high levels of ^{133}Cs. Persons residing in the vicinity of pollucite mines and workers employed in the mining, milling, and production of cesium may be exposed to higher levels than the general population.

Human exposure to ^{134}Cs and ^{137}Cs can be external, due to exposure from a radioactive cloud and contaminated environmental media after deposition, or internal, via inhalation and ingestion of contaminated food or drinking water. In both cases, populations residing near the source are exposed to potentially higher doses than populations far removed from the source. Humans residing near areas where nuclear weapon testing was previously conducted may have been exposed to higher doses of radiation from ^{134}Cs and ^{137}Cs, both internally and externally, than the general population. Populations residing in southern Utah and Nevada were exposed to radioactive cesium (and many other radionuclides) due to testing conducted at the Nevada Test Site (NTS). A total of 100 surface or near-surface tests with a total explosive yield of about 1 megaton were performed at the Nevada test site between 1951 and 1962. The dust from these tests also drifted over the continental United States, producing varying degrees of exposure for remote populations depending upon the meteorological conditions. For example, the greatest non-local fallout from one of the tests was received in New York State in 1953, some 2,000 miles away from the source, due to wet deposition (Agency for Toxic Substances Disease Registry 1999). About 500 underground tests were also performed at the NTS, but underground testing rarely leads to significant off-site contamination (Agency for Toxic Substances Disease Registry 1999). The EPA Office of Radiation and Indoor Air conducts off-site environmental monitoring around former U.S. nuclear test areas. The 1997 report concluded that the current exposure to populations around the NTS from radionuclides, including ^{137}Cs and ^{134}Cs, was negligible in comparison to background levels (EPA 1999c).

Populations residing in the vicinity of nuclear power plants may also be exposed to higher levels of ^{134}Cs and ^{137}Cs than the general population due to airborne and liquid effluents from these plants. Persons employed in these facilities are also likely to be exposed to greater levels than the general population, particularly those employees who must handle radioactive waste material. However, despite the potential for exposure, increased body burdens of radioactive cesium have not been observed among the population of workers in nuclear facilities or among members of the off-site general public.

Human populations that received a large amount of fallout from the Chernobyl nuclear accident were potentially exposed to high levels of radiocesium. These populations were primarily located in the Ukraine and northern Europe that received a great deal of rainfall in the weeks following the Chernobyl

accident. Not including the 30-km exclusionary zone, an area of approximately $2.4x10^4$ km^2 near the plant was contaminated with ^{137}Cs at a deposition density >$5.4x10^{-5}$ Ci/m^2 (UNSCEAR 1996). Within the exclusionary zone, the contamination density may have been a factor of about 100 greater in limited areas (UNSCEAR 1996). The Bryansk-Belarus region about 200 km northeast of the reactor had deposition densities as high as $1.3x10^{-4}$ Ci/m^2, and the Kaluga-Tula-Orel location approximately 500 km northeast of the reactor had deposition densities of about $1.6x10^{-5}$ Ci/m^2 (Agency for Toxic Substances Disease Registry 1999). While cesium is not considerably taken up by the roots of vascular plants, the deposition of radioactive debris on flora with large surface areas such as lichens or moss is significant (see Section 6.4.4). As a result, animals that feed on this vegetation such as caribou or reindeer may ingest large quantities of radiocesium. Nordic or Eskimo populations that use these animals as an important source for food are exposed to larger quantities of ^{137}Cs and ^{134}Cs than the general population (Allaye-Chan et al. 1990; WHO 1983). The average concentration of ^{137}Cs in the skeletal muscle of a herd of caribou from Alaska was in the range of 76–133 Bq/kg in 1987 (Allaye-Chan et al. 1990). According to the National Council on Radiation Protection and Measurements, the maximum nonoccupational radiocesium intake is 300,666 Bq/year ($8.1x10^6$ pCi/year) for adults (NCRP 1977). At the maximum average skeletal muscle concentration (133 Bq/kg), an annual consumption of over 2,260 kg of caribou meat would be required to reach this limit (assuming no other intake sources). Using the mean ^{137}Cs level of 900 Bq/kg in the muscle of reindeer obtained from northern Sweden in 1986–1987 (Ahman and Ahman 1994), over 330 kg of contaminated meat would have to be consumed to reach the maximum intake level. Populations residing in the Marshall Islands were exposed to higher levels of ^{137}Cs than the general population due to nuclear weapons testing conducted by the United States from 1946 to 1958. Using radiological surveys from 1978 and 1985–1986, the AVDI of ^{137}Cs due to food ingestion was estimated for seven age groups residing in the Rongelap Atoll (Robinson and Phillips 1989). The intakes in pCi/day were as follows: 0–3 months of age, 424; 4–8 months of age, 556; 9 months to 1.4 years of age 773; 1.5–3 years of age, 517; 4–11 years of age, 594; 12–17 years of age, 761; over 18 years of age, 1,085 (Robinson and Phillips 1989). This corresponds to an annual intake of $3.96x10^5$ pCi/year for adults over 18 years of age.

6.8 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of cesium is available. Where adequate information is not available, ATSDR, in conjunction with the National Toxicology Program (NTP), is required to assure the

initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of cesium.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

6.8.1 Identification of Data Needs

Physical and Chemical Properties. As shown in Table 4-2, the relevant physical and chemical properties of cesium and its compounds are known (Burt 1993; Lide 1998). In general, cesium compounds are very water soluble and primarily exist in ionic form in aqueous environments. Cesium adsorbs strongly to soils and is not very mobile (Korobova et al. 1998; Paasikallio 1999; Ruse and Peart 2000; Takenaka et al. 1998). The radioactive decay modes of the two most important cesium isotopes, ^{134}Cs and ^{137}Cs, are also well known (ICRP 1983), and no additional physical or chemical data are required to predict the environmental fate of cesium.

Production, Import/Export, Use, Release, and Disposal. Knowledge of a chemical's production volume is important because it may indicate possible environmental contamination and human exposure. If a chemical's production volume is high, there is an increased probability of general population exposure via consumer products and environmental sources such as air, drinking water, and food. Data concerning production volumes, import, and use of commercially significant cesium compounds are not available. No information was found for cesium in the TRI. According to the Emergency Planning and Community Right-to-Know Act of 1986, 42 U.S.C. Section 11023, industries are required to submit chemical release and off-site transfer information to the EPA. This database will be updated yearly and should provide a list of industrial production facilities and emissions. The United States is 100% import reliant for stable ^{133}Cs. No data are available on production volume, consumption, or import/export volumes of cesium. Although there is no information regarding the countries shipping cesium or cesium compounds to the United States, it is believed that Canada is the major source of cesium (USGS 1999). Data regarding the U.S. production (if any) of cesium and its compounds as well as import/export statistics would be useful. The amount of ^{134}Cs and ^{137}Cs released in airborne and liquid effluents from nuclear power plants in the United States is known (USNRC 1993).

Environmental Fate. Information is available to permit the assessment of the environmental fate and transport of cesium in air (Hirose et al. 1993; Rybacek et al. 1994), water (Burt 1993; WHO 1983), and sediment and soil (Korobova et al. 1998; Paasikallio 1999; Ruse and Peart 2000; Takenaka et al. 1998). Cesium compounds are water soluble, and only one oxidation state (+1) is observed under environmental conditions (Burt 1993). Cesium released to the atmosphere may be carried long distances before being deposited onto soil and water surfaces by wet and dry deposition (Hirose et al. 1993). Most of the cesium released to water will adsorb to suspended solids in the water column and ultimately be deposited in the sediment core. In soil surfaces, cesium is strongly adsorbed in the upper layers and generally has very low mobility (Korobova et al. 1998; Paasikallio 1999; Ruse and Peart 2000; Takenaka et al. 1998). This is particularly true for soils with high potassium content or soils rich in clay. The radioactive decay modes of the two most important cesium isotopes, ^{134}Cs and ^{137}Cs, have been described in Chapter 4 (ICRP 1983).

Bioavailability from Environmental Media. The bioavailability of cesium in environmental media is well understood and no data needs are identified at this time. For the most part, cesium is adsorbed strongly in the surface of most soils and is not readily bioavailable (Paasikallio 1999). In a study in which 102 healthy volunteers were fed ^{134}Cs-contaminated soil pellets, only about 1% of the original intake was absorbed, and on average, about 60% of the original amount was excreted within 48 hours (LeRoy et al. 1966). In peaty or podzolic soils with low clay content, cesium is reversibly adsorbed to the organic fraction and is expected to be in bioavailable form (Sanchez et al. 1999). Cesium uptake in vascular plants has been demonstrated (Djingova et al. 1995; Willey and Martin 1995). Increasing the clay or potassium content results in lower uptake by plants (Shenber and Johanson 1992). It has also been shown that fish residing in waters with high concentrations of humic material and potassium, such as oceans, have lower whole-body cesium concentrations than fish in freshwater where the concentration of dissolved potassium is lower for the same cesium concentration in the water (WHO 1983).

Food Chain Bioaccumulation. Cesium bioaccumulates in both aquatic and terrestrial food chains (Rowan et al. 1998; WHO 1983). Mean BCFs for ^{137}Cs of 146, 124, and 63 were reported for fish, brown macroalgae, and molluscs, respectively (Fisher et al. 1999). Cesium accumulates in aquatic organisms from both food sources and from cesium dissolved in the water column or adsorbed to suspended solids and sediments. The lichen-caribou-human food chain is an important route of human exposure and has been well studied (Allaye-Chan et al. 1990; WHO 1983). No additional data are required to assess the potential for human exposure to cesium through food chain bioaccumulation.

Exposure Levels in Environmental Media. Stable cesium and radioactive cesium have been detected in air (Ajdacic and Martic 1990; Chiavarini et al. 1994; Dasch and Wolff 1989; Rybacek et al. 1994; Todorovic et al. 1999), water (Asubiojo et al. 1997; DOE 1992; Fisher et al. 1999; Prister et al. 1990; Strezov et al. 1999; WHO 1983), soil/sediment (Burt 1993; DOE 1992, 1998a; Ruse and Peart 2000; WHO 1983), and food (Ahman et al. 1994; Bibak et al. 1998; Mondon and Walters 1990). Due to the large surface area of lichens and moss, they can collect a great deal of atmospheric nuclear fallout and often have high concentrations of ^{134}Cs and ^{137}Cs (Papastefanou et al. 1992; Penttila et al. 1993). Grazing animals such as reindeer and caribou that feed on large quantities of lichens or moss may potentially ingest large amounts of radioactive cesium, and this cesium may be transferred to humans who consume these animals as a meat source (Allaye-Chan et al. 1990; WHO 1983). Continued monitoring data on ^{134}Cs and ^{137}Cs in environmental media are needed to increase knowledge of human exposure to ^{134}Cs and ^{137}Cs.

Exposure Levels in Humans. Monitoring data are available for the levels of ^{134}Cs and ^{137}Cs in human populations (Glowiak et al. 1977b; Lokobauer et al. 1988; Rabitsch et al. 1991; Rahola and Suomela 1998; Ropolo and Cesana 1997). Most of the current data are for areas of eastern Europe and Russia. Limited data are available regarding the levels of ^{133}Cs in humans (Hewitt 1988). Stable cesium was detected in the urine of U.S. residents at geometric mean concentrations of about 4–5 μg/L depending upon age and gender (CDC 2003). More information regarding the background concentration of ^{133}Cs in human populations would be useful, but given the ubiquitous distribution of cesium at low levels in the environment, background levels are unlikely to approach levels that would reflect cesium toxicity. Since the levels of radioactivity change over time, continued monitoring data on the levels of ^{134}Cs and ^{137}Cs in humans are needed to evaluate potential risks to human health at current exposure levels.

Exposures of Children. Children, like adults, are not expected to be over exposed to ^{133}Cs. Existing data show that children are exposed to ^{134}Cs and ^{137}Cs after major releases. These exposures are primarily related to dietary intake and the intake of contaminated milk (Lokobauer et al. 1988). Dietary consumption patterns suggest that the weight-adjusted intake of radioactive cesium for children may be greater than for adults following a nuclear release (Lokobauer et al. 1988). In a study of females from northern Sweden, ^{137}Cs was detected in breast milk from 8 out of 12 mothers at concentrations of 7.3–178.4 pCi/kg (Johansson et al. 1998). The infants of these mothers who were being breast-fed had whole-body levels of ^{137}Cs in the range of 45.9–675.7 pCi/kg (Johansson et al. 1998). Additional data regarding the transfer of ^{134}Cs and ^{137}Cs from breast milk and contaminated pasteurized milk samples to children, as

well as body burden studies on children, would be useful in assessing the potential risk that these radionuclides would pose following a major release.

Child health data needs relating to susceptibility are discussed in Section 3.13.2, Identification of Data Needs: Children's Susceptibility.

Exposure Registries. Cesium is currently one of the chemicals for which a subregistry has been established in the National Exposure Registry. The information that is amassed in the National Exposure Registry facilitates the epidemiological research needed to assess adverse health outcomes that may be related to cesium exposure.

6.8.2 Ongoing Studies

The database of federal research programs in progress (FEDRIP 2002) indicates several current projects that may fill some existing data gaps and add to the current database of knowledge. Studies are in progress to identify methods for more effective removal of radioactive cesium from contaminated soils and wastes. Researchers at the Universitaet des Saarlandes, Saarbruecken (Germany) are seeking cost-effective methods to remove gamma-emitting radionuclides, such as ^{137}Cs and ^{134}Cs, from radioactive waste commonly produced at nuclear power plants. Researchers at the Oak Ridge National Laboratory, Environmental Sciences Division are studying the environmental fate and behavior of selected radionuclides in terrestrial and aquatic environments. Dr. Michael Blaylock of Phytotech Incorporated is investigating the use of metal-accumulating plant species to extract and remove the radionuclides cesium and strontium from contaminated soils ("phytoextraction"). Rufus L. Chaney from the Beltsville Agricultural Research Center in Beltsville, Maryland is researching the feasibility of developing plants, soil, and plant management practices that can cost-effectively phytoextract radionuclides and heavy metals from contaminated soils. Dr. Heit from the U.S. DOE is conducting measurements regarding the fallout of nuclear debris from both atmospheric weapons tests and accidental atmospheric releases to determine the mechanisms of transport and deposition and to verify and correct fallout models. Work is being performed by Dr. Kinkead at the Los Alamos National Laboratory regarding the separation of cesium and strontium from high-level radioactive waste. Dr. Leon Kochian from the Agricultural Research Center in Ithaca, New York is investigating the bioaccumulation of ^{137}Cs in plants grown in contaminated soils.

7. ANALYTICAL METHODS

The purpose of this chapter is to describe the analytical methods that are available for detecting, measuring, and/or monitoring cesium, its metabolites, and other biomarkers of exposure and effect to cesium. The intent is not to provide an exhaustive list of analytical methods. Rather, the intention is to identify well-established methods that are used as the standard methods of analysis. Many of the analytical methods used for environmental samples are the methods approved by federal agencies and organizations such as EPA and the National Institute for Occupational Safety and Health (NIOSH). Other methods presented in this chapter are those that are approved by groups such as the Association of Official Analytical Chemists (AOAC) and the American Public Health Association (APHA). Additionally, analytical methods are included that modify previously used methods to obtain lower detection limits and/or to improve accuracy and precision.

7.1 BIOLOGICAL MATERIALS

Entry of cesium and its radioisotopes into the human body occurs by ingestion, inhalation, or penetration through skin (IAEA 1988; NCRP 1977, 1985). The amounts of cesium in the body can be assessed by bioassay *(in vivo* and *in vitro)* measurements. *In vivo* measurements are made with whole-body counters. *In vivo* measurement techniques are commonly used to measure body burdens of cesium radioisotopes, but can not be used to measure the body content of the stable isotope of cesium. *In vitro* measurements provide an indirect estimate of internally deposited cesium (both the stable and radioactive isotopes), by techniques that measure cesium in body fluids, feces, or other human samples (Gautier 1983). Examples of these analytical techniques are given in NCRP Report No. 87 (1987) and are also listed in Table 7-1 for stable cesium and Table 7-2 for radioactive cesium.

7.1.1 *In Vivo* Cesium Measurements

In vivo measurement techniques are the most direct and widely used approach for assessing the burden of cesium radioisotopes in the body. The *in vivo* measurement of these radioisotopes within the body is performed with various radiation detectors and associated electronic devices that are collectively known as whole-body counters. These radiation detectors usually employ sodium iodide (NaI), hyperpure germanium, or organic liquid scintillation detectors to measure the 605 (98%) and 796 keV (85%) gamma

Table 7-1. Analytical Methods for Determining Cesium in Biological Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Urine	Sample dried, ashed, and irradiated	INAA	$1x10^{-5}$ μg/g	No data	Oughton and Day 1993
Soft tissue	Sample ashed and then concentrated by precipitation with AMP, extracted with sodium phenylboron	Flame photometry	0.005 μg/g	96–99%	Feldman and Rains 1964
Feces	Sample dried, ashed, and irradiated	INAA	$1x10^{-5}$ μg/g	No data	Oughton and Day 1993

AMP = ammonium molybdophosphate; INAA = instrument neutron activated analysis

Table 7-2. Analytical Methods for Determining Radiocesium in Biological Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Urine	Sample is acidified and concentrated on a KCFC column	γ-spectrometry with NaI detector	6 pCi/L	98%	Boni 1966
Urine	Sample transferred to Marinelli beaker and counted	γ-spectrometry with NaI detector	100 pCi/L	98%	Gautier 1983
Urine	Sample transferred to Marinelli beaker and counted	γ-spectrometry with NaI(Tl) detector	2 pCi/L	No data	Cahill and Wheeler 1968
Soft tissue	Sample wet-ashed	γ-spectrometry	No data	No data	Baratta et al. 1969
Soft tissue	Sample directly counted in detector	γ-spectrometry	5 pCi/g	No data	Rabon and Johnson 1973
Soft tissue	Sample digested in acid, oxidized with $HClO_4$, concentrated by precipitated with AMP, purified by resin column, precipitated with hexachloroplatinic acid	β-counter	0.1 pCi/g	40–85%	Nevissi 1992
Feces	Sample directly counted in detector	γ-spectrometry with NaI detector	0.14 nCi/L	No data	Lipsztein et al. 1991
Human milk	Sample directly counted in detector	γ-spectrometry with NaI detector	0.001 pCi/L	No data	Risica et al. 1994

KCFC = potassium cobalt ferrocyanide

rays from the decay of ^{134}Cs, and 662 keV (89.9%) gamma rays that are emitted from the decay of ^{137}Cs (Palmer et al. 1976). ^{134}Cs and ^{137}Cs distribute uniformly in muscle and soft tissue of the body. The photons emitted by ^{134}Cs and ^{137}Cs are easily detected and quantitated using whole-body counting techniques (NCRP 1987; Palmer et al. 1976; Sun et al. 1997). Many configurations of the whole-body counter and scanning methods have been utilized, ranging from unshielded single-crystal field detectors to shielded, multi-detector scanning detectors (IAEA 1962, 1970, 1972, 1976, 1985; NCRP 1987; Palmer et al. 1976). Where appropriate, shielding of the room that houses the whole-body counter and/or the detector is often used to increase the detection sensitivity of the equipment by minimizing background radiation. Additionally, care must be exercised to ensure that external contamination with radioactive cesium or other gamma-emitting radioisotopes are not present on the clothing or skin of the individual to be scanned. *In vitro* measurements of cesium (see Section 7.1.2) are used in conjunction with whole-body counting when monitoring individuals working with cesium, especially in conjunction with the assessment of individuals who have experienced accidental exposures to cesium (Bhat et al. 1973).

Whole-body counters are calibrated using tissue-equivalent phantoms. These phantoms are constructed to mimic the shape and density of the anatomical structure using tissue-equivalent materials such as water-filled canisters or masonite (Barnaby and Smith 1971; Bhat et al. 1973; Sun et al. 1997). For example, the bottle mannequin absorber (BOMAB) consists of a series of water-filled polyethylene canisters constructed into seated or reclined human forms (Sun et al. 1997). Cesium standards are measured either as point sources along the phantom or dissolved within the water-filled canisters. Comparisons of the actual counts obtained from the phantom to the known activity of the cesium standards are used to determine the efficiency of the counting technique and, thus, to provide the basis for calibrating the counting system.

Assessment of short- and long-term retention of cesium radioisotopes takes into account the turnover rate for cesium within the human body. Although the physical half-life of ^{137}Cs is 30 years, the biological and effective half-life of cesium inside the body is approximately 110 days (NCRP 1987; Rundo and Newton 1964). This relatively high turnover rate for cesium in the body is due to the high solubility of cesium in body fluids that allows for the rapid uptake (e.g., absorption of ingested cesium through the gut) and elimination of cesium into and from the body (e.g., excreted through urine) (NCRP 1987). For acute and chronic exposures to cesium, the estimates of cesium retention are determined from direct, whole-body measurements. Models for cesium in the human body have been developed for estimating the short- and long-term retention of cesium based on whole-body measurement techniques (ICRP 1979, 1989, 1993; NCRP 1987; Sun et al. 1997).

7.1.2 *In Vitro* Cesium Measurements

In vitro analyses of cesium are routinely performed in support of an *in vivo* monitoring program or in situations where direct *in vivo* measurements can not be obtained. Urinalysis is the preferred sample for *in vitro* analyses of cesium, although other sample types, such as feces, tissue, bone, or blood, can also be analyzed. Urinalysis is an optimum method for assessing the clearance of soluble cesium. Fecal analysis is used to assess the clearance of ingested, insoluble cesium (Baratta et al. 1969; Gautier 1983; Ide and McInroy 1975; NCRP 1987).

The *in vitro* analysis of the stable isotope of cesium, ^{133}Cs, in human samples (e.g., urine, tissue, feces) is performed by a number of methods that have the selectivity and sensitivity to measure cesium in biological matrices. These methods include spectrophotometry, instrumental neutron activation analysis (INAA), and inductively coupled plasma mass spectrometry (ICP-MS) (Dreizen et al. 1970; Iyengar and Woittiez 1988; Paschal et al. 1996). Of these methods, the INAA and ICP-MS methods offer the greatest detection sensitivity and are the preferred method of analysis for cesium in human samples (Iyengar and Woittiez 1988).

For the *in vitro* analysis of the cesium radioisotopes ^{134}Cs and ^{137}Cs in human samples, a number of analytical methods can be used to measure the cesium radioisotopes directly in the samples without requiring an extensive sample preparation procedure. In the radiochemical analysis of cesium in urine, a 24-hour urine collection (approximately 2 L) is obtained, followed by the transfer of a 1 L aliquot to a Marinelli beaker for counting in a gamma-ray spectrometer (Gautier 1983). This simple procedure offers high recoveries of cesium (98%) and the minimum detection sensitivity (100 pCi/L) that is required to evaluate individuals for exposures to radioactive cesium (Gautier 1983). Similar methods are also used for the analysis of cesium radioisotopes in tissues, feces, and blood (Table 7-1). Mass spectrometry techniques have also been employed to measure cesium radioisotopes in human samples.

Accuracy of *in vivo* and *in vitro* measurements of cesium is determined through the use of standard, certified solutions or radioactive sources with known concentrations or activities of cesium. National Institute of Standards and Technology (NIST) traceable standards for ^{133}Cs can be obtained through a number of commercial sources. The primary source of certified cesium radioisotope standards is the NIST. Gamma-ray point sources for ^{137}Cs (standard reference material [SRM] 4200, 60,000 Bq [1.6 μCi]

and SRM 4207, 300,000 Bq [8 μCi]) and standard solutions of ^{137}Cs (SRM 4233, 600,000 Bq/g [16 μCi/g]) are available from NIST.

7.2 ENVIRONMENTAL SAMPLES

Two common approaches are available for measuring cesium radioisotopes in the environment. Cesium radioisotopes can either be measured directly in the field (*in situ*) using portable survey instruments, or samples can be procured from the field and returned to the laboratory for quantitation of cesium. However, quantitation of the stable cesium isotope in environmental samples is generally conducted in the laboratory.

7.2.1 Field Measurements of Cesium

In situ measurement techniques are useful for the rapid characterization of radionuclide contamination in the environment, such as soils, sediments, and vegetation, and for monitoring personnel for exposure to radionuclides. The measurement of gamma ray-emitting radionuclides such as ^{134}Cs and ^{137}Cs in the environment is conducted with portable survey instruments such as Gieger-Mueller detectors, sodium iodide scintillation detectors, and gamma-ray spectrometers. The use of gamma-spectrometers in field survey equipment is preferred for measuring cesium in the field because of its energy selectivity and detection sensitivity. The relatively high energy and penetrance of the gamma ray that is emitted during the decay of ^{134}Cs and ^{137}Cs provide an advantage for assessing the level of cesium. These gamma-ray spectrometers are equipped with a high purity germanium detector that is able to separate the 602, 662, and 796 keV gamma rays emitted from ^{134}Cs and ^{137}Cs from the gamma rays emitted from other radionuclides; for example, ^{40}K (USNRC 1997). Minimum detectable activities (MDAs) of 0.005 Bq/g for ^{137}Cs are routinely achieved using p-type germanium gamma spectrometers with 10-minute counting times (USNRC 1997). Computational methods have been derived to aid in determining the concentrations and distributions of ^{134}Cs and ^{137}Cs in different soil types and depths (Fülöp and Ragan 1997; Hillmann et al. 1996; USNRC 1997). The concentrations and distributions of ^{134}Cs and ^{137}Cs that have been derived from the computational analysis of the survey data are often verified by laboratory-based analyses of soil samples procured from the survey area.

7.2.2 Laboratory Analysis of Environmental Samples

Analytical methods for measuring cesium and cesium radioisotopes in environmental samples (e.g. air, water, soil, and biota) are summarized in Tables 7-3 (^{133}Cs) and 7-4 (^{134}Cs, ^{137}Cs). The methods that are commonly used in the analysis of ^{133}Cs are based on instrumental analytical techniques such as spectrophotometry, instrumental neutron activation analysis, and mass spectrometry. The analysis of ^{134}Cs and ^{137}Cs can be determined either as total mass or total activity, depending on the analytical technique that is used. Typically, radiochemical methods of analysis employing gamma spectrometry techniques are used to quantitate ^{134}Cs and ^{137}Cs in environmental samples. However, spectrophotometric and mass spectrometry techniques have been used to determine the total mass of ^{134}Cs and ^{137}Cs in samples. Using the specific activity of ^{137}Cs (89 μCi/μg), it can be deduced that a sample with activity of 1 μCi of ^{137}Cs contains roughly 0.011 μg of ^{137}Cs.

The analysis of cesium in air is based on the measurement of cesium in aerosols or particles that become trapped on cellulose or glass fiber filters after a measured amount of air is pulled through the filters. For the analysis for ^{133}Cs, the filter is solvent extracted and the extracted metals are analyzed by INAA (Gone et al. 2000). Analysis of ^{134}Cs and ^{137}Cs can be performed directly from the filter, or by following some sample preparation (e.g., ashing or solvent extraction), using gamma spectrometry (Kanapilly et al. 1983; Kolb 1971; Krieger et al. 1976).

For the analysis of cesium in water, a broad array of sample preparation and detection methodologies are available (see Tables 7-3 and 7-4). Different standardized methods that can directly measure cesium or its radioactive isotopes within a water sample using INAA or radiochemical techniques with minimal sample preparation and good detection sensitivities (10–20 pCi/L), precision (4–9%), and bias (-5–1%) (ASTM 1999; EPA 1980). Other methods are available that preconcentrate cesium from natural or potable waters when interfering impurities are present or the activity of the cesium radioisotopes are too low (<30 pCi/L) for quantitation by gamma spectrometry (APHA 1998; Frigieri et al. 1980). This preconcentration of cesium can be achieved either through precipitation with molybdate compounds, for example, or through chromatographic techniques using columns packed with resins that specifically bind cesium (EPA 1980; Frigieri et al. 1980; Gaur 1996; Petrow and Levine 1967).

The quantity of cesium and its radioisotopes in soil, sediments, vegetation, and biota is determined using detection methods similar to those described above (Tables 7-3 and 7-4). Analysis of the stable cesium isotope by INAA and ICP-MS requires either digesting the sample in acid or ashing the sample before

Table 7-3. Analytical Methods for Determining Cesium in Environmental Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Water	Sample is purified by passage through Dowex 50-X8 resin, concentrated with NCFC column	Electrothermal-AA	1 μg/L	99% at 10–100 μg/L	Frigieri et al. 1980
River water	Sample concentrated by precipitation with AMP, purified by extraction with sodium tetraphenylboron	Flame photometry	0.010 μg/L	94.5%	Feldman and Rains 1964
River water	Sample is purified by passage through Dowex 50-X8 resin, concentrated with NCFC column	Electrothermal-AA	1 μg/L	99% at 10–100 μg/L	Frigieri et al. 1980
Lake water	Filtered samples were irradiated	INAA	0.010 μg/L	90%	Hakonson and Whicker 1975
Sea water	Sample was precipitated with sodium tetraphenyl-borate, and the precipitate was neutron irradiated	INAA	0.008 μg/L	92%	Taskaev 1987
Sea water	Sample precipitated with AMP, purified by extraction with sodium tetraphenyl-boron	Flame photometry	0.010 μg/L	94.5%	Feldman and Rains 1964
Mineral and thermal waters	Direct aspiration of sample into graphite furnace	Graphite furnace-AA	0.00185 μg/L	92.3–100.9%	Bermejo-Barrera et al. 1989
Groundwater	Sample purified by ultracentrifugation	ICP-MS	0.010 μg/L	No data	Probst et al. 1995
Groundwater	Sample purified by ultracentrifugation	ICP-AES	0.010 μg/L	No data	Probst et al. 1995
Soil	Sample was pre-ashed, digested with acid	Electrothermal-AA	0.09 mg/g	80–85%	Anderson et al. 1996
Soil	Sample was dried, ground, and irradiated	INAA	0.003 ng/g	No data	Oughton and Day 1993
Soil	Sample digested with acid	ICP-MS	0.011 μg/g	95%	Robb et al. 1995
Sediment	Sample was dried and irradiated	INAA	0.010 μg/g	90%	Hakonson and Whicker 1975
Silicate rock	Sample digested in HF/H_2SO_4	Graphite furnace-AA	0.05 μg/L	76%	Grobenski et al. 1983

Table 7-3. Analytical Methods for Determining Cesium in Environmental Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Vegetation	Sample ashed and irradiated	INAA	$1x10^{-5}$ μg/g	No data	Oughton and Day 1993
Vegetation	Sample prepared by microwave digestion	ICP-MS	$2x10^{-5}$ μg/g	95–105%	Dombovári et al. 2000

AA = atomic absorption; AMP = ammonium molybdophosphate; ICP-AES = inductively coupled plasma-atomic emission spectrometry; ICP-MS = inductively coupled plasma-mass spectrometry; INAA = instrumental neutron activation analysis; NCFC = ammonium hexacyanocobalt ferrate

Table 7-4. Analytical Methods for Determining Radiocesium in Environmental Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Air (occup-ational)	Sample filter was solvent extracted	Scintillation counter with NaI detector	No data	95%	Kanapilly et al. 1983
Air (ambient)	Sample filter digested in acid, cesium precipitated with chloroplatinate	γ-spectrometry with Ge/Li detector	0.01 fCi/m^3	80%	Krieger et al. 1976
Drinking water	Direct count of sample	γ-spectrometry with Ge/Li detector	10 pCi/L	No data	EPA 1980
Drinking water	Direct count of sample	γ-spectrometry with Ge detector	<2 pCi/L	92–100%	APHA 1998
Fresh water	Sample concentrated with Dowex 1x8/KCFC mixed ion exchange column	γ-spectrometry with NaI detector	3 pCi/L	99%	Boni 1966
River water	Sample precipitated with AMP, concentrated with Dowex-50 cation exchange column	Scintillation counter with NaI detector	<7 fCi/L	99%	Kahn et al. 1957
River water	Sample concentrated on Dowex 50W-X8 column	γ-spectrometry with Ge(Li) detector	2 pCi/L (50 L sample)	97%	Luetzelschwab 1976
Lake water	Sample concentrated on ACFC column	γ-spectrometry with NaI(Tl) detector	No data	97%	Eyman and Kevern 1975
Water and waste water	Direct count of sample	γ-spectrometry with Ge/Li detector	<2 pCi/L	92–100% at 2–94 pCi/L	ASTM 1999
Sea water	Sample purified by passage through chelating resin, concentrated with KCFC ion exchange column	γ-spectrometry with NaI detector	0.07 pCi/L	98%	Boni 1966
Soil	Sample dried and crushed	γ-spectrometry with Ge(Li) detector	0.05 pCi/g	No data	Arnalds et al. 1989
Soil	Sample mixed with 5% Ag and compressed into disc	GDMS	0.2 pg/g	No data	Betti et al. 1996
Sediment	Sample extracted with acid, concentrated by precipitation with AMP, solvent extracted with sodium tetraphenylboron	γ-spectrometry with Ge(Li) detector	No data	96%	Eyman and Kevern 1975

Table 7-4. Analytical Methods for Determining Radiocesium in Environmental Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Biota	Sample ashed, oxidized with $HClO_4$, concentrated by precipitation with AMP, puriifed by resin column, precipitated with hexa-chloroplatinic acid	β-counter	0.1 pCi/g	No data	Nevissi 1992

ACFC = ammonium hexacyanocobalt ferrate; AMP=ammonium molybdophosphate; ICP-MS = inductively coupled plasma-mass spectrometry; KCFC = potassium cobalt ferrocyanide; GDMS = glow discharge mass spectrometry

analysis. In some cases where interfering compounds or materials may be present, additional sample concentration or purification may be required. For the radioisotopes of cesium, direct detection of ^{134}Cs and ^{137}Cs within the neat sample can be performed using gamma spectrometry detection methods.

The detection limits, accuracy, and precision of any analytical methodology are important parameters in determining the appropriateness of a method to quantitate a specific analyte at the desired level of sensitivity within a particular matrix. The MDA refers to the intrinsic detection capability of a measurement procedure (sampling through data reduction and reporting) (USNRC 1984). Several factors influence the MDA, including background count rates, size or concentration of sample, detector sensitivity, recovery of desired analyte during sample isolation and purification, level of interfering contaminants, and particularly, counting time. Because of these variables, the MDAs may vary between laboratories using the same or similar measurement procedures.

The accuracy of a measurement technique in determining the quantity of a particular analyte in environmental samples is dependent on the reliability of the calibrating technique. Thus, the availability of standard, certified radiation sources with known concentrations of cesium and its radioisotopes is required in order to insure the reliability of the calibration methods and accuracy of cesium measurements in environmental samples. NIST traceable standards for ^{133}Cs can be obtained through a number of commercial sources. The primary source of certified cesium radioisotope standards is the NIST. Gamma-ray point sources for ^{137}Cs (SRM 4200, 60,000 Bq [1.6 μCi] and SRM 4207, 300,000 Bq [8 μCi]) and standard solutions of ^{137}Cs (SRM 4233, 600,000 Bq/g [16 μCi/g]) are available from NIST. SRMs are also available containing the stable (SRM 1944 [sediment], SRM 2710 and 2711 [soil]) and radioactive isotopes of cesium (SRM 4350 [sediment] and SRM 4357 [sediment]).

7.3 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of cesium is available. Where adequate information is not available, ATSDR, in conjunction with the National Toxicology Program (NTP), is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of cesium.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

7.3.1 Identification of Data Needs

Methods for Determining Biomarkers of Exposure and Effect. Analytical methods with satisfactory sensitivity and precision are available to determine the levels of cesium and its radioisotopes in human tissues and body fluids.

Methods for Determining Parent Compounds and Degradation Products in Environmental Media. Analytical methods with the required sensitivity and accuracy are available for measuring cesium, both total and isotopic, in environmental matrices (Tables 7-2 and 7-4). Knowledge of the levels of cesium in various environmental media, along with appropriate modeling (see Chapters 4 and 6), can be used to evaluate potential human exposures through inhalation and ingestion pathways.

Whether in the environment or in the human body, cesium radioisotopes will undergo radioactive decay to nonradioactive isotopes (see Chapter 4). Current analytical methods, such as mass spectrometry, have the necessary resolution and sensitivity to detect and quantitate these decay products.

7.3.2 Ongoing Studies

Current research trends in the quantitation of cesium and its radioisotopes are focused on improving the selectivity and detection sensitivity of cesium in biological and environmental samples. Mass spectrometry approaches, such as double focusing sector field inductively coupled mass spectrometry or time-of-flight selected ion monitoring systems, are being developed further to provide the required selectivity and sensitivity to rapidly measure cesium in the presence of other trace metals in complex environmental samples. Cesium-selective electrodes are being developed into a highly-selective, rapid detection technique for measuring cesium in environmental samples and waste streams. Current efforts are focused on the development of electrode membranes that contain cesium binding agents, such as crown ether derivatives. New cesium-selective resins, for example lanthanum-based resins or montmorillonite clays, are being developed and tested for selectivity and recovery of cesium.

The database of federal research programs in progress (FEDRIP 2002) indicates several current projects that may fill some existing data gaps and add to the current database of knowledge. W.H. Aberth from Antek Incorporated, located in Palo Alto, California is attempting to increase the sensitivity to which cesium can be analyzed in human tissue through the use of a cluster ion gun in liquid dynamic secondary ion mass spectrometry (SIMS).

8. REGULATIONS AND ADVISORIES

The international, national, and state regulations and guidelines regarding stable cesium in air, water, and other media are summarized in Table 8-1. The regulations regarding radioactive cesium are summarized in Table 8-2.

No MRLs were derived for inhalation or oral exposure to stable or radioactive cesium. Two MRLs, derived by the Agency for Toxic Substances and Disease Registry (1999) for external exposure to ionizing radiation, are applicable to external exposure to radioisotopes of cesium. An MRL of 400 mrem (4.0 mSv) was derived for acute-duration external exposure (14 days or less), based on cognitive learning deficit in children who had been exposed to ionizing radiation at critical stages of fetal development (gestation weeks 8–15) during the atomic bombing of Hiroshima and Nagasaki (Schull et al. 1988). An MRL of 100 mrem/year (1.0 mSv/year) above background was derived for chronic-duration external exposure (365 days or more), based on the BEIR V (1990) report that the average annual effective ionizing radiation dose to the U.S. population is 360 mrem/year (3.6 mSv/year), a dose not expected to produce adverse health effects.

The EPA has not derived reference concentrations (RfCs) or reference doses (RfDs) for stable or radioactive cesium (IRIS 2002). The IRIS database does not provide cancer assessments for radio-isotopes of cesium. This function is the responsibility of the EPA Office of Radiation and Indoor Air (ORIA). All radionuclides, including radioisotopes of cesium, are classified as known human (Group A) carcinogens. This classification is based on results of epidemiological studies of Japanese atomic bomb survivors, underground uranium miners, radium dial painters, and patients subjected to a variety of radiation treatments, as well as results of laboratory animal research and mammalian tissue culture studies. ORIA has published cancer slope factors (mortality and morbidity cancer risk estimates) for all known radionuclides, by various exposure routes (inhalation, drinking water ingestion, food ingestion, soil ingestion, immersion in a cloud, and external exposure from contaminated soil) for five age groups and 14 radiogenic cancer sites (EPA 2000). Slope factors for ^{137}Cs and ^{134}Cs are listed in Table 8-2.

Table 8-1. Regulations and Guidelines Applicable to Cesium

Agency	Description	Information	Reference
INTERNATIONAL			
Guidelines:			
IARC		No data	IARC 2000
NATIONAL			
Regulations and Guidelines:			
a. Air			
ACGIH	TLV (8-hour TWA for a 40-hour workweek)		ACGIH 2000, 2001
	Cesium hydroxide (based on upper respiratory tract, skin, and eye irritation)	2 mg/m^3	
EPA		No data	
NIOSH	REL (10-hour TWA for a 40-hour workweek		NIOSH 1992, 2000
	Cesium hydroxide (based on skin, eye, and respiratory irritation)	2 mg/m^3	
OSHA		No data	
b. Water		No data	
c. Food		No data	
d. Other		No data	
STATE		No data	

ACGIH = American Conference of Governmental Industrial Hygienists; EPA = Environmental Protection Agency; IARC = International Agency for Research on Cancer; NIOSH = National Institute for Occupational Safety and Health; OSHA = Occupational Safety and Health Administration; REL = recommended exposure limit; TLV = threshold limit value; TWA = time weighted average

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cesium

Agency	Description	Information	Reference
INTERNATIONAL			
Guidelines:			
IARC		No data	IARC 2000
NATIONAL			
Regulations and Guidelines:			
a. Air			
ACGIH		No data	ACGIH 2000
EPA	Detection limits for man-made beta particle and photon emitters	10 pCi/L	EPA 1999a 40 CFR 141.25
NIOSH		No data	NIOSH 2000
NRC	Effluent concentrations–air		NRC 1999a 10 CFR 20 App B
	^{134}Cs	$2x10^{-10}$ µCi/mL	
	^{137}Cs	$2x10^{-10}$ µCi/mL	
b. Water			NRC 1999a 10 CFR 20 App B
NRC	Effluent concentrations–water		
	^{134}Cs	$9x10^{-7}$ µCi/ml	
	^{137}Cs	$1x10^{-6}$ µCi/ml	
c. Food			FDA 1998
FDA	Derived intervention level[a] (DIL; Bq/kg food) in accidentally-contaminated human food		
	^{134}Cs	930	
	^{137}Cs	1360	
d. Other			
EPA	Concentration levels for environmental compliance		EPA 1999a 40 CFR 61 App E
	^{134}Cs	$0.7x10^{-14}$ Ci/m^3	
	^{137}Cs	$1.9x10^{-14}$ Ci/m^3	
	Carcinogenicity–slope factors[b]		EPA 2002
	Lifetime risk per pCi–ingestion		
	Water		
	^{134}Cs	$4.22x10^{-11}$	
	^{137}Cs	$3.04x10^{-11}$	
	Food		
	^{134}Cs	$5.14x10^{-11}$	
	^{137}Cs	$3.74x10^{-11}$	
	Soil		
	^{134}Cs	$5.81x10^{-11}$	
	^{137}Cs	$4.33x10^{-11}$	
	Lifetime risk per pCi–inhalation		
	^{134}Cs	$1.65x10^{-11}$	
	^{137}Cs	$1.19x10^{-11}$	

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cesium

Agency	Description	Information		Reference
NATIONAL (*cont.*)				
	External exposure–risk/year per pCi/g soil			EPA 2002
	^{134}Cs	7.10×10^{-6}		
	^{137}Cs	5.32×10^{-10}		
	^{137}Cs (plus disintegration products)	2.55×10^{-6}		
NRC	Occupational inhalation exposure			NRC 1999a
	ALIs			10 CFR 20 App B
	^{134}Cs	100 μCi		
	^{137}Cs	200 μCi		
	DACs			
	^{134}Cs	4×10^{-8} μCi/mL		
	^{137}Cs	6×10^{-8} μCi/mL		
	Quantities of licensed material requiring labeling			NRC 1999b 10 CFR App C
	^{134}Cs	10 μCi		
	^{137}Cs	10 μCi		
STATE				
a. Air				
Michigan	Gross beta particle activity			MI Dept Environ Quality 2000
	^{134}Cs	15 pCi/L		
b. Water		No data		
c. Food		No data		
d. Other				
Louisiana	Quantity required for consideration of need for emergency plan for responding to a release	Release fraction	Quantity	LA Dept Environ Quality 2000
	^{134}Cs	0.01	2,000 Ci	
	^{137}Cs	0.01	3,000 Ci	

[a]The FDA-recommended Derived Intervention Level (DIL) for radionuclides of cesium, is defined as the DIL for the most sensitive age group (adults) that was calculated from the most limiting Protective Action Goal (PAG; 5 mSv committed effective whole body dose equivalent).
[b]EPA classifies all radionuclides as Group A (known human) carcinogens. Radionuclide risk coefficients, or slope factors, are calculated by EPA's Office of Radiation and Indoor Air (ORIA) to assist HEAST users with risk-related evaluations and decision-making at various stages of the remediation process. Ingestion and inhalation slope factors are central estimates in a linear model of the age-averaged, lifetime attributable radiation cancer incidence (fatal and nonfatal cancer) risk per unit of activity inhaled or ingested, expressed as risk/picocurie (pCi). Ingestion values are tabulated separately for ingestion of tap water, dietary intakes (food), and incidental soil ingestion. External exposure slope factors are central estimates of the lifetime attributable radiation cancer incidence risk for each year of exposure to external radiation from photon-emitting radionuclides distributed uniformly in a thick layer of soil, and are expressed as risk/year per pCi/gram of soil.

ACGIH = American Conference of Governmental Industrial Hygienists; ALI = annual limitations on intake; CFR = Code of Federal Regulations; DAC = derived air concentrations; EPA = Environmental Protection Agency; FDA = Food and Drug Administration; IARC = International Agency for Research on Cancer; NIOSH = National Institute for Occupational Safety and Health; NRC = Nuclear Regulatory Commission; OSHA = Occupational Safety and Health Administration

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10. GLOSSARY

Some terms in this glossary are generic and may not be used in this profile.

Absorbed Dose, Chemical—The amount of a substance that is either absorbed into the body or placed in contact with the skin. For oral or inhalation routes, this is normally the product of the intake quantity and the uptake fraction divided by the body weight and, if appropriate, the time, expressed as mg/kg for a single intake or mg/kg/day for multiple intakes. For dermal exposure, this is the amount of material applied to the skin, and is normally divided by the body mass and expressed as mg/kg.

Absorbed Dose, Radiation—The mean energy imparted to the irradiated medium, per unit mass, by ionizing radiation. Units: rad (rad), gray (Gy).

Absorbed Fraction—A term used in internal dosimetry. It is that fraction of the photon energy (emitted within a specified volume of material) which is absorbed by the volume. The absorbed fraction depends on the source distribution, the photon energy, and the size, shape and composition of the volume.

Absorption—The process by which a chemical penetrates the exchange boundaries of an organism after contact, or the process by which radiation imparts some or all of its energy to any material through which it passes.

Absorption Coefficient—Fractional absorption of the energy of an unscattered beam of x- or gamma-radiation per unit thickness (linear absorption coefficient), per unit mass (mass absorption coefficient), or per atom (atomic absorption coefficient) of absorber, due to transfer of energy to the absorber. The total absorption coefficient is the sum of individual energy absorption processes (see Compton Effect, Photoelectric Effect, and Pair Production).

Absorption Coefficient, Linear—A factor expressing the fraction of a beam of x- or gamma radiation absorbed in a unit thickness of material. In the expression $I=I_0e^{-\mu x}$, I_0 is the initial intensity, I the intensity of the beam after passage through a thickness of the material x, and μ is the linear absorption coefficient.

Absorption Coefficient, Mass—The linear absorption coefficient per cm divided by the density of the absorber in grams per cubic centimeter. It is frequently expressed as μ/ρ, where μ is the linear absorption coefficient and ρ the absorber density.

Absorption Ratio, Differential—Ratio of concentration of a nuclide in a given organ or tissue to the concentration that would be obtained if the same administered quantity of this nuclide were uniformly distributed throughout the body.

Activation—The process of making a material radioactive by bombardment with neutrons or protons.

Activity—The number of radioactive nuclear transformations occurring in a material per unit time (see Curie, Becquerel). The term for activity per unit mass is specific activity.

Activity Median Aerodynamic Diameter (AMAD)—The diameter of a unit-density sphere with the same terminal settling velocity in air as that of the aerosol particle whose activity is the median for the entire size distribution of the aerosol.

Acute Exposure, Chemical—Exposure to a chemical for a duration of 14 days or less, as specified in the Toxicological Profiles.

Acute Exposure, Radiation—The absorption of a relatively large amount of radiation (or intake of a radioactive material) over a short period of time.

Acute Radiation Syndrome—The symptoms which taken together characterize a person suffering from the effects of intense radiation. The effects occur within hours or days.

Ad libitum—Available in excess and freely accessible.

Adsorption Coefficient (K_{oc})—The ratio of the amount of a chemical adsorbed per unit surface area or per unit weight of organic carbon of a specific particle size in the soil or sediment to the concentration of the chemical in solution at equilibrium.

Adsorption Ratio (K_d)—See Distribution Coefficient

Alpha Particle—A positively charged particle ejected spontaneously from the nuclei of some radioactive elements. It is identical to a helium nucleus, i.e., 2 neutrons and two protons, with a mass number of 4 and an electrostatic charge of +2.

Alpha Track—The track of ionized atoms (pattern of ionization) left in a medium by an alpha particle that has traveled through the medium.

Annihilation (Positron-Electron)—An interaction between a positive and a negative electron in which they both disappear; their rest mass, being converted into electromagnetic radiation (called annihilation radiation) with two 0.51 MeV gamma photons emitted at an angle of 180° to each other.

Annual Limit on Intake (ALI)—The derived limit for the amount of radioactive material taken into the body of an adult worker by inhalation or ingestion in a year. It is the smaller value of intake of a given radionuclide in a year by the reference man that would result in a committed effective dose equivalent of 5 rem or a committed dose equivalent of 50 rem to any organ or tissue.

Atom—The smallest particle of an element that cannot be divided or broken up by chemical means. It consists of a central core called the *nucleus*, which contains *protons* and *neutrons* and an outer shell of *electrons*.

Atomic Mass (u)—The mass of a neutral atom of a nuclide, usually expressed in terms of "atomic mass units." The "atomic mass unit" is one-twelfth the mass of one neutral atom of carbon-12; equivalent to 1.6604×10^{-24} g.

Atomic Mass Number—See Mass Number.

Atomic Number—The number of protons in the nucleus of an atom. The "effective atomic number" is calculated from the composition and atomic numbers of a compound or mixture. An element of this atomic number would interact with photons in the same way as the compound or mixture. (Symbol: Z).

Atomic Weight—The weighted mean of the masses of the neutral isotopes of an element expressed in atomic mass units.

Attenuation—A process by which a beam from a source of radiation is reduced in intensity by absorption and scattering when passing through some material.

10. GLOSSARY

Attenuation Coefficient—The fractional reduction in the intensity of a beam of radiation as it passes through an absorbing medium. It may be expressed as reduction per unit distance, per unit mass thickness, or per atom, and is called the linear, mass, or atomic attenuation coefficient, respectively.

Auger Effect—The emission of an electron from the extranuclear portion of an excited atom when the atom undergoes a transition to a less excited state.

Background Radiation—The amount of radiation to which a member of the general population is exposed from natural sources, such as terrestrial radiation from naturally occurring radionuclides in the soil, cosmic radiation originating from outer space, and naturally occurring radionuclides deposited in the human body.

Becquerel (Bq)—International System of Units unit of activity and equals that quantity of radioactive material in which one transformation (disintegration) occurs per second (see Units).

Terabecquerel (TBq)—One trillion becquerel.
Gigabecquerel (GBq)—One billion becquerel.
Megabecquerel (MBq)—One million becquerel.
Kilobecquerel (kBq))—One thousand becquerel.
Millibecquerel (mBq)—One-thousandth of a becquerel.
Microbecquerel (μBq)—One-millionth of a becquerel.

Beta Particle—An electron that is emitted from the nucleus of an atom during one type of radioactive transformation. A beta particle has a mass and charge equal in magnitude to that of the electron. The charge may be either +1 or -1. Beta particles with +1 charges are called positrons (symbolized β^+), and beta particles with -1 charges are called negatrons (symbolized β^-).

Bioconcentration Factor (BCF)—The quotient of the concentration of a chemical in aquatic organisms at a specific time or during a discrete time period of exposure divided by the concentration in the surrounding water at the same time or during the same period.

Biologic Effectiveness of Radiation—See Relative Biological Effectiveness.

Biological Half-time—The time required for a biological system, such as that of a human, to eliminate by natural process half of the amount of a substance (such as a chemical substance, either stable or radioactive) that has entered it.

Biomarkers—Broadly defined as indicators signaling events in biologic systems or samples. They have been classified as markers of exposure, markers of effect, and markers of susceptibility.

Body Burden, Chemical—The total amount of a chemical found in an animal or human body.

Body Burden, Radioactivity—The amount of radioactive material found in an animal or human body.

Bone Seeker—Any compound or ion which migrates in the body and preferentially deposits into bone.

Branching—The occurrence of two or more modes by which a radionuclide can undergo radioactive decay. For example, ^{214}Bi can undergo alpha or beta minus decay, ^{64}Cu can undergo beta minus, beta plus, or electron capture decay. An individual atom of a nuclide exhibiting branching disintegrates by one mode only. The fraction disintegrating by a particular mode is the "branching fraction" for that mode. The "branching ratio" is the ratio of two specified branching fractions (also called multiple disintegration).

Bremsstrahlung—X rays that are produced when a charged particle accelerates (speeds up, slows down, or changes direction) in the strong field of a nucleus.

Buildup Factor—The ratio of the radiation intensity, including both primary and scattered radiation, to the intensity of the primary (unscattered) radiation.

Cancer Effect Level (CEL)—The lowest dose of chemical or radiation in a study, or group of studies, that produces significant increases in the incidence of cancer (or tumors) between the exposed population and its appropriate control.

Capture, Electron—A mode of radioactive decay involving the capture of an orbital electron by its nucleus. Capture from a particular electron shell, e.g., K or L shells, is designated as "K-electron capture" or "L-electron capture."

Capture, K-Electron—Electron capture from the K shell by the nucleus of the atom. Also loosely used to designate any orbital electron capture process.

Carcinogen—A chemical or radiation that is capable of inducing cancer.

Carcinoma—Malignant neoplasm composed of epithelial cells, regardless of their derivation.

Case-Control Study—A type of epidemiological study which examines the relationship between a particular outcome (disease or condition) and a variety of potential causative agents (such as toxic chemicals). In a case-controlled study, a group of people with a specified and well-defined outcome is identified and compared to a similar group of people without outcome.

Case Report—Describes a single individual with a particular disease or exposure. These may suggest some potential topics for scientific research but are not actual research studies.

Cataract—A clouding of the crystalline lens of the eye which obstructs the passage of light.

Ceiling Value—A concentration of a substance that should not be exceeded, even temporarily.

Charged Particle—A nuclear particle, atom, or molecule carrying a positive or negative charge.

Chronic Exposure—A long-term, continuous exposure to a chemical or radioactive material. For example, exposure to a chemical for 365 days or more, as specified in the Toxicological Profiles.

Cohort Study—A type of epidemiological study of a specific group or groups of people who have had a common insult (e.g., exposure to an agent suspected of causing disease or a common disease) and are followed forward from exposure to outcome. At least one exposed group is compared to one unexposed group.

Collective Dose—The sum of the individual doses received in a given period of time by a specified population from exposure to a specified source of radiation. Collective dose is expressed in units such as man-rem and person-sievert.

Compton Effect—An attenuation process observed for x- or gamma radiation in which an incident photon interacts with an orbital electron of an atom to produce a recoil electron and a scattered photon whose energy is less than the incident photon.

Containment—The confinement of a chemical or radioactive substance in such a way that it is prevented from being dispersed from its container or into the environment, or is released only at a specified rate.

Contamination—Deposition of a stable or radioactive substance in any place where it is not desired.

Cosmic Rays—High-energy particulate and electromagnetic radiations that originate outside the earth's atmosphere and interact with the atmosphere to produce a shower of secondary cosmic rays.

Count (Radiation Measurements)—The external indication of a radiation-measuring device designed to enumerate ionizing events. It refers to a single detected event. The term "count rate" refers to the total number registered in a given period of time. The term is sometimes erroneously used to designate a disintegration, ionizing event, or voltage pulse.

Counter, Gas-flow Proportional (GPC)—An instrument for detecting beta particle radiation. Beta particles are detected by ionization of the counter gas which results in an electrical impulse at an anode wire.

Counter, Geiger-Mueller (GM counter)— Highly sensitive, gas-filled radiation-measuring device that detects (counts) individual photons or particulate radiation.

Counter, Scintillation—The combination of a crystal or phosphor, photomultiplier tube, and associated circuits for counting light emissions produced in the phosphors by ionizing radiation. Scintillation counters generally are more sensitive than GM counters for gamma radiation.

Counting, Cerenkov—Relatively energetic β-particles pass through a transparent medium of high refractive index and a highly-directional, bluish-white light ("Cerenkov" light) is emitted. This light is detected using liquid scintillation counting equipment.

Cross-sectional Study—A type of epidemiological study of a group or groups which examines the relationship between exposure and outcome to a chemical or to chemicals at one point in time.

Curie (Ci)—A unit of radioactivity. One curie equals that quantity of radioactive material in which there are 3.7×10^{10} nuclear transformations per second. The activity of 1 gram of radium is approximately 1 Ci.

Attocurie (aCi)—One-thousandth of a femtocurie (3.7×10^{-8} disintegrations per second).
Femtocurie (fCi)—One-billionth of a microcurie (3.7×10^{-5} disintegrations per second).
Megacurie (MCi)—One million curies (3.7×10^{16} disintegrations per sec).
Microcurie (μCi)—One-millionth of a curie (3.7×10^{4} disintegrations per sec).
Millicurie (mCi)—One-thousandth of a curie (3.7×10^{7} disintegrations per sec).
Nanocurie (nCi)—One-billionth of a curie (3.7×10^{1} disintegrations per sec).
Picocurie (pCi)—One-millionth of a microcurie (3.7×10^{-2} disintegrations per second.

Daughter Products—See Progeny and Decay Product

Decay Chain or Decay Series—A sequence of radioactive decays (transformations) beginning with one nucleus. The initial nucleus, the parent, decays into a daughter or progeny nucleus that differs from the first by whatever particles were emitted during the decay. If further decays take place, the subsequent nuclei are also usually called daughters or progeny. Sometimes, to distinguish the sequence, the daughter of the first daughter is called the granddaughter, etc.

Decay Constant (λ)—The fraction of the number of atoms of a radioactive nuclide which decay in unit time (see Disintegration Constant).

Decay Product, Daughter Product, Progeny—A new nuclide formed as a result of radioactive decay. A nuclide resulting from the radioactive transformation of a radionuclide, formed either directly or as the result of successive transformations in a radioactive series. A decay product (daughter product or progeny) may be either radioactive or stable.

Decay, Radioactive—Transformation of the nucleus of an unstable nuclide by spontaneous emission of radiation, such as charged particles and/or photons (see Disintegration).

Delta Ray—An electron removed from an atom of a medium that is irradiated, or through which radiation passes, during the process of ionization (also called secondary electron). Delta rays cause a track of ionizations along their path.

Derived Air Concentration (DAC)—The concentration of radioactive material in air that, if breathed by the reference man for a working year of 2000 hours under conditions of light work (at a rate of 1.2 liters of air per hour), would result in an intake of one ALI (see Annual Limit on Intake).

Deterministic Effect—A health effect, the severity of which varies with the dose and for which a threshold is believed to exist (also called a non-stochastic effect).

Developmental Toxicity—The occurrence of adverse effects on the developing organism that may result from exposure to a chemical or radiation prior to conception (either parent), during prenatal development, or postnatally to the time of sexual maturation. Adverse developmental effects may be detected at any point in the life span of the organism.

Disintegration Constant—Synonymous with decay constant. The fraction of the number of atoms of a radioactive material that decays per unit time (see Decay Constant.)

Disintegration, Nuclear—A spontaneous nuclear transformation (radioactivity) characterized by the emission of energy and mass from the nucleus. When large numbers of nuclei are involved, the process is characterized by a definite half-life (see Transformation, Nuclear).

Distribution Coefficient (K_d)—Describes the distribution of a chemical between the solid and aqueous phase at thermodynamic equilibrium, is given as follows:

$$K_d = \frac{[C]_s}{[C]_w}, \text{ Units = (L solution)/(kg solid)},$$

where $[C]_s$ is the concentration of the chemical associated with the solid phase in units of (mg)/(kg solid), and $[C]_w$ is the concentration of the chemical in the aqueous phase in units of (mg)/(L solution). As the magnitude of K_d decreases, the potential mobility of the chemical to groundwater systems increases and vice versa.

Dose—A general term denoting the quantity of a substance, radiation, or energy absorbed. For special purposes it must be appropriately qualified. If unqualified, it refers to radiation absorbed dose.

> **Absorbed Dose**—The energy imparted to matter by ionizing radiation per unit mass of irradiated material at the place of interest. The unit of absorbed dose is the rad. One rad equals 100 ergs per gram. In SI units, the absorbed dose is the gray which is 1 J/kg (see Rad).

Cumulative Dose (Radiation)—The total dose resulting from repeated or continuous exposures to radiation.

Dose Assessment—An estimate of the radiation dose to an individual or a population group usually by means of predictive modeling techniques, sometimes supplemented by the results of measurement.

Dose Equivalent (DE)—A quantity used in radiation safety practice to account for the relative biological effectiveness of the several types of radiation. It expresses all radiations on a common scale for calculating the effective absorbed dose. The NRC defines it as the product of the absorbed dose, the quality factor, and all other modifying factors at the location of interest. ICRP has changed its definition to be the product of the absorbed dose and the radiation weighting factor. (The unit of dose equivalent is the rem. In SI units, the dose equivalent is the sievert, which equals 100 rem.)

Dose, Fractionation—A method of administering therapeutic radiation in which relatively small doses are given daily or at longer intervals.

Dose, Protraction—A method of administering therapeutic radiation by delivering it continuously over a relatively long period at a low dose rate.

Dose, Radiation—The amount of energy imparted to matter by ionizing radiation per unit mass of the matter, usually expressed as the unit rad, or in SI units, the gray. 100 rad=1 gray (Gy) (see Absorbed Dose).

Committed Dose Equivalent ($H_{T,50}$)—The dose equivalent to organs or tissues of reference (T) that will be received from an intake of radioactive material by an individual during the 50 years following the intake.

Committed Effective Dose Equivalent ($H_{E,50}$)—The sum of the products of the weighting factors applicable to each of the body organs or tissues that are irradiated and the committed dose equivalent to those organs or tissues.

Effective Dose—A dose value that attempts to normalize the detriment to the body (for cancer mortality and morbidity, hereditary effects, and years of life lost) from a non-uniform exposure to that of a uniform whole body exposure. Effective dose is calculated as the sum of products of the equivalent dose and the tissue weighting factor (w_T) for each tissue exposed. ($E = \sum D_{T,R}\ w_R\ w_{T)}$).

Effective Dose Equivalent (H_E)—This dose type is limited to internal exposures and is the sum of the products of the dose equivalent to the organ or tissue (H_T) and the weighting factors (w_T) applicable to each of the body organs or tissues that are irradiated. ($H_E = \sum w_T\ H_T$).

Equivalent Dose—A dose quantity that places the biological effect of all radiation types on a common scale for calculating tissue damage. Alpha particles, for example, are considered to cause 20 times more damage than gamma rays. Equivalent dose is calculated as the sum of products of the average absorbed dose (in gray) in an organ or tissue ($_{DT,R}$) from each type of radiation and the radiation weighting factor (w_R) for that radiation ($\sum D_{T,R}\ w_R$).

External Dose—That portion of the dose equivalent received from radiation sources outside the body.

Internal Dose—That portion of the dose equivalent received from radioactive material taken into the body.

Limit—A permissible upper bound on the radiation dose.

Maximum Permissible Dose (MPD)—The greatest dose equivalent that a person or specified part thereof shall be allowed to receive in a given period of time.

Median Lethal Dose (MLD)—Dose of radiation required to kill, within a specified period (usually 30 days), 50% of the individuals in a large group of animals or organisms. Also called the LD_{50}, or $LD_{50/30}$ if for 30 days..

Threshold Dose—The minimum absorbed dose that will produce a detectable degree of any given effect.

Tissue Dose—Absorbed dose received by tissue in the region of interest, expressed in rad (see Dose, Gray, and Rad).

Dose Rate—The amount of radiation dose delivered per unit time. Generically, the rate at which radiation dose is delivered to any material or tissue.

Dose-Response Relationship—The quantitative relationship between the amount of exposure to a toxicant and the incidence of the adverse effects.

Dosimetry—Quantification of radiation doses to cells, tissues, organs, individuals or populations resulting from radiation exposures.

Early Effects (of radiation exposure)—Effects that appear within 60 days of an acute exposure.

Electron—A stable elementary particle having an electric charge equal to $\pm 1.60210 \times 10^{-19}$ C (Coulombs) and a rest mass equal to 9.1091×10^{-31} kg. A positron is a positively charged "electron" (see Positron).

Electron Volt—A unit of energy equivalent to the energy gained by an electron in passing through a potential difference of one volt. Larger multiple units of the electron volt are frequently used: keV for thousand or kilo electron volts; MeV for million or mega electron volts (eV). 1 eV=1.6×10^{-12} erg.

Embryotoxicity and Fetotoxicity—Any toxic effect on the conceptus as a result of prenatal exposure to a chemical; the distinguishing feature between the two terms is the stage of development during which the insult occurred. The terms, as used here, include malformations and variations, altered growth, and *in utero* death.

Energy—Capacity for doing work. Gravitationally, "potential energy" is the energy inherent in a mass because of its spatial relation to other masses. Chemically or radiologically, "potential energy" is the energy released when a chemical reaction or radiological transformation goes to completion. "Kinetic energy" is the energy possessed by a mass because of its motion (SI unit: joules):

Binding Energy (Electron)—The amount of energy that must be expended to remove an electron from an atom.

Binding Energy (Nuclear)—The energy represented by the difference in mass between the sum of the component parts and the actual mass of the nucleus. It represents the amount of energy that must be expended to break a nucleus into its component neutrons and protons.

Excitation Energy—The energy required to change a system from its ground state to an excited state. Each different excited state has a different excitation energy.

Ionizing Energy—The energy required to knock an electron out of an atom. The average energy lost by electrons or beta particles in producing an ion pair in air or in soft tissue is about 34 eV.

Radiant Energy—The energy of electromagnetic radiation, such as radio waves, visible light, x and gamma rays.

Enrichment, Isotopic—An isotopic separation process by which the relative abundances of the isotopes of a given element are altered, thus producing a form of the element that has been enriched in one or more isotopes and depleted in others. In uranium enrichment, the percentage of uranium-235 in natural uranium can be increased from 0.7% to >90% in a gaseous diffusion process based on the different thermal velocities of the constituents of natural uranium (^{234}U, ^{235}U, ^{238}U) in the molecular form UF_6.

EPA Health Advisory—An estimate of acceptable drinking water levels for a chemical substance based on health effects information. A health advisory is not a legally enforceable federal standard, but serves as technical guidance to assist federal, state, and local officials.

Epidemiology—Refers to the investigation of factors that determine the frequency and distribution of disease or other health-related conditions within a defined human population during a specified period.

Equilibrium, Radioactive—In a radioactive series, the state which prevails when the ratios between the activities of two or more successive members of the series remains constant.

Secular Equilibrium—If a parent element has a very much longer half-life than the daughters (so there is not appreciable change in its amount in the time interval required for later products to attain equilibrium) then, after equilibrium is reached, equal numbers of atoms of all members of the series disintegrate in unit time. This condition is never exactly attained, but is essentially established in such a case as ^{226}Ra and its transformation series to stable ^{206}Pb. The half-life of ^{226}Ra is about 1,600 years; of ^{222}Rn, approximately 3.82 days, and of each of the subsequent members, a few minutes. After about a month, essentially the equilibrium amount of radon is present; then (and for a long time) all members of the series disintegrate the same number of atoms per unit time. At this time, the activity of the daughter is equal to the activity of the parent.

Transient Equilibrium—If the half-life of the parent is short enough so the quantity present decreases appreciably during the period under consideration, but is still longer than that of successive members of the series, a stage of equilibrium will be reached after which all members of the series decrease in activity exponentially with the period of the parent. At this time, the ratio of the parent activity to the daughter activity is constant.

Equilibrium, Electron—The condition in a radiation field where the energy of the electrons entering a volume equals the energy of the electrons leaving that volume.

Excitation—The addition of energy to a system, thereby transferring it from its ground state to an excited state. Excitation of a nucleus, an atom, or a molecule can result from absorption of photons or from inelastic collisions with other particles. The excited state of an atom is an unstable or metastable state and will return to ground state by radiation of the excess energy.

Exposure (Chemical)—Contact of an organism with a chemical or physical agent. Exposure is quantified as the amount of the agent available at the exchange boundaries of the organism (e.g., skin, lungs, gut) and available for absorption.

Exposure (Radiation)—Subjection to ionizing radiation or to a radioactive material. For example, exposure in air is a measure of the ionization produced in air by x or gamma radiation; the sum of the electric charges on all ions of one sign produced in air when all electrons liberated by photons in a volume of air are completely stopped in air (dQ), divided by the mass of the air in the volume (dm). The unit of exposure in air is the roentgen, or coulomb per kilogram (SI units). One roentgen is equal to 2.58×10^{-4} coulomb per kilogram (C/kg).

Fission, Nuclear—A nuclear transformation characterized by the splitting of a nucleus into at least two other nuclei with emission of several neutrons, accompanied by the release of a relatively large amount of energy.

Gamma Ray, Penetrating—Short wavelength electromagnetic radiation of nuclear origin.

Genetic Effect of Radiation—Inheritable change, chiefly mutations, produced by the absorption of ionizing radiation by germ cells. Genetic effects have not been observed in any human population exposed at any dose level.

Genotoxicity—A specific adverse effect on the genome of living cells that, upon the duplication of affected cells, can be expressed as a mutagenic, clastogenic or carcinogenic event because of specific alteration of the molecular structure of the genome.

Gray (Gy)—SI unit of absorbed dose, 1 J/kg. One gray equals 100 rad (see Units).

Half-life, Effective—See Half-Time, Effective.

Half-life, Radioactive—Time required for a radioactive substance to lose 50% of its activity by decay. Each radio-nuclide has a unique physical half-life. Known also as physical half-time and symbolized as T_r or T_{rad}.

Half-time, Biological—Time required for an organ, tissue, or the whole body to eliminate one-half of any absorbed substance by regular processes of elimination. This is the same for both stable and radioactive isotopes of a particular element, and is sometimes referred to as half-time, symbolized as t_{biol} or T_b.

Half-time, Effective—Time required for a radioactive element in an organ, tissue, or the whole body to be diminished 50% as a result of the combined action of radioactive decay and biological elimination, symbolized as T_e or T_{eff}.

$$\text{Effective half-time} = \frac{\text{Biological half-time} \times \text{Radioactive half-life}}{\text{Biological half-time} + \text{Radioactive half-life}}$$

Immediately Dangerous to Life or Health (IDLH)—The maximum environmental concentration of a contaminant from which one could escape within 30 minutes without any escape-impairing symptoms or irreversible health effects.

Immunologic Toxicity—The occurrence of adverse effects on the immune system that may result from exposure to environmental agents such as chemicals.

Immunological Effects—Functional changes in the immune response.

In Vitro—Isolated from the living organism and artificially maintained, as in a test tube. Literally, "in glass."

In Vivo—Occurring within the living organism. Literally, "in life."

Intensity—Amount of energy per unit time passing through a unit area perpendicular to the line of propagation at the point in question.

Intermediate Exposure—Exposure to a chemical for a duration of 15–364 days, as specified in the Toxicological Profiles.

Internal Conversion—Process in which a gamma ray knocks an electron out of the same atom from which the gamma ray was emitted. The ratio of the number of internal conversion electrons to the number of gamma quanta emitted in the de-excitation of the nucleus is called the "conversion ratio."

Ion—Atomic particle, atom or chemical radical bearing a net electrical charge, either negative or positive.

Ion Pair—Two particles of opposite charge, usually referring to the electron and positive atomic or molecular residue resulting after the interaction of ionizing radiation with the orbital electrons of atoms.

Ionization—The process by which a neutral atom or molecule acquires a positive or negative charge.

> **Primary Ionization**—(1) In collision theory: the ionization produced by the primary particles as contrasted to the "total ionization" which includes the "secondary ionization" produced by delta rays. (2) In counter tubes: the total ionization produced by incident radiation without gas amplification.
>
> **Specific Ionization**—Number of ion pairs per unit length of path of ionizing radiation in a medium; e.g., per centimeter of air or per micrometer of tissue.
>
> **Total Ionization**—The total electric charge of one sign on the ions produced by radiation in the process of losing its kinetic energy. For a given gas, the total ionization is closely proportional to the initial ionization and is nearly independent of the nature of the ionizing radiation. It is frequently used as a measure of absorption of radiation energy.

Ionization Density—Number of ion pairs per unit volume.

Ionization Path (Track)—The trail of ion pairs produced by an ionizing particle in its passage through matter.

Ionizing Radiation—Any radiation capable of knocking electrons out of atoms and producing ions. Examples: alpha, beta, gamma and x rays, and neutrons.

Isobars—Nuclides having the same mass number but different atomic numbers.

Isomers—Nuclides having the same number of neutrons and protons but capable of existing, for a measurable time, in different quantum states with different energies and radioactive properties. Commonly the isomer of higher energy decays to one with lower energy by the process of isomeric transition.

Isotopes—Nuclides having the same number of protons in their nuclei, and hence the same atomic number, but differing in the number of neutrons, and therefore in the mass number. Identical chemical properties exist in isotopes of a particular element. The term should not be used as a synonym for nuclide because isotopes refer specifically to different nuclei of the same element.

Stable Isotope—A nonradioactive isotope of an element.

Joule—The S.I. unit for work and energy. It is equal to the work done by raising a mass of one newton through a distance of one meter (J = Nm), which corresponds to about 0.7 ft-pound.

Kerma (k)—A measure of the kinetic energy transferred from gamma rays or neutrons to a unit mass of absorbing medium in the initial collision between the radiation and the absorber atoms. The SI unit is J/kg. The special name of this unit is the rad (traditional system of units) or Gray (SI).

Labeled Compound—A compound containing one or more radioactive atoms intentionally added to its structure. By observations of radioactivity or isotopic composition, this compound or its fragments may be followed through physical, chemical, or biological processes.

Late Effects (of radiation exposure)—Effects which appear 60 days or more following an acute exposure.

$LD_{50/30}$—The dose of a chemical or radiation expected to cause 50% mortality in those exposed within 30 days. For radiation, this is about 350 rad (3.5 gray) received by humans over a short period of time.

Lethal Concentration$_{(Lo)}$ (LC_{Lo})—The lowest concentration of a chemical in air that has been reported to have caused death in humans or animals.

Lethal Concentration$_{(50)}$ (LC_{50})—A calculated concentration of a chemical in air to which exposure for a specific length of time is expected to cause death in 50% of a defined experimental animal population within a specified time, usually 30 days.

Lethal Dose$_{(Lo)}$ (LD_{Lo})—The lowest dose of a chemical introduced by a route other than inhalation that is expected to have caused death in humans or animals within a specified time, usually 30 days.

Lethal Dose$_{(50)}$ (LD_{50})—The dose of a chemical which has been calculated to cause death in 50% of a defined experimental animal population.

Lethal Time$_{(50)}$ (LT_{50})—A calculated period of time within which a specific concentration of a chemical is expected to cause death in 50% of a defined experimental animal population.

Linear Energy Transfer (LET)—A measure of the energy that a charged particle transfers to a material per unit path length.

Average LET—The energy of a charged particle divided by the length of the path over which it deposits all its energy in a material. This is averaged over a number of particles.

High-LET—Energy transfer characteristic of heavy charged particles such as protons and alpha particles where the distance between ionizing events is small on the scale of a cellular nucleus.

Low-LET—Energy transfer characteristic of light charged particles such as electrons produced by x and gamma rays where the distance between ionizing events is large on the scale of a cellular nucleus.

Lowest-Observed-Adverse-Effect Level (LOAEL)—The lowest dose of chemical in a study, or group of studies, that produces statistically or biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control.

Lung Clearance Class (fast, F; medium, M; slow, S)—A classification scheme for inhaled material according to its rate of clearance from the pulmonary region of the lungs to the blood and the gastrointestinal tract.

Lymphoreticular Effects—Represent morphological effects involving lymphatic tissues such as the lymph nodes, spleen, and thymus.

Malformations—Permanent structural changes that may adversely affect survival, development, or function.

Mass Numbers (A)—The number of nucleons (protons and neutrons) in the nucleus of an atom.

Minimal Risk Level—An estimate of daily human exposure to a substance that is likely to be without an appreciable risk of adverse noncancerous effects over a specified duration of exposure.

Morbidity—State of being diseased; morbidity rate is the incidence or prevalence of disease in a specific population.

Mutagen—A substance that causes changes (mutations) in the genetic material in a cell. Mutations can lead to birth defects, miscarriages, or cancer.

Necropsy—The gross examination of the organs and tissues of a dead body to determine the cause of death or pathological conditions.

Neurotoxicity—The occurrence of adverse effects on the nervous system following exposure to a substance.

Neutrino (v)—A neutral particle of infinitesimally small rest mass emitted during beta plus or beta minus decay. This particle accounts for conservation of energy in beta plus and beta minus decays. It plays no role in damage from radiation.

No-Observed-Adverse-Effect Level (NOAEL)—The dose of a substance at which there were no statistically or biologically significant increases in frequency or severity of adverse effects seen between the exposed population and its appropriate control. Effects may be produced at this dose, but they are not considered to be adverse.

Nuclear Reactor—A power plant that heats the medium (typically water) by using the energy released from the nuclear fission of uranium or plutonium isotopes instead of burning coal, oil, or natural gas. All of these sources of energy simply heat water and use the steam which is produced to turn turbines that make electricity or propel a ship.

Nucleon—Common name for a constituent particle of the nucleus. Applied to a proton or neutron.

Nuclide—A species of atom characterized by the constitution of its nucleus. The nuclear constitution is specified by the number of protons (Z), number of neutrons (N), and energy content; or, alternatively, by the atomic number (Z), mass number A(N+Z), and atomic mass. To be regarded as a distinct nuclide, the atom must be capable of existing for a measurable time. Thus, nuclear isomers are separate nuclides, whereas promptly decaying excited nuclear states and unstable intermediates in nuclear reactions are not so considered.

Octanol-Water Partition Coefficient (K_{ow})—The equilibrium ratio of the concentrations of a chemical in n-octanol and water, in dilute solution.

Odds Ratio (OR)—A means of measuring the association between an exposure (such as toxic substances and a disease or condition) which represents the best estimate of relative risk (risk as a ratio of the incidence among subjects exposed to a particular risk factor divided by the incidence among subjects who were not exposed to the risk factor). An odds ratio of greater than 1 is considered to indicate greater risk of disease in the exposed group compared to the unexposed.

Pair Production—An absorption process for x- and gamma radiation in which the incident photon is absorbed in the vicinity of the nucleus of the absorbing atom, with subsequent production of an electron and positron pair (see annihilation). This reaction can only occur for incident photon energies exceeding 1.02 MeV.

Parent—Any radionuclide nuclide which, upon disintegration, yields a new nuclide (termed the progeny or daughter), either directly or as a later member of a radioactive series.

Permissible Exposure Limit (PEL)—A maximum allowable atmospheric level of a substance in workplace air averaged over an 8-hour shift.

Pharmacokinetic Model—A set of equations that can be used to describe the time course of a parent chemical or metabolite in an animal system. There are two types of pharmacokinetic models: data-based and physiologically-based. A data-based model divides the animal system into a series of compartments which, in general, do not represent real, identifiable anatomic regions of the body whereas the physiologically-based model compartments represent real anatomic regions of the body.

Pharmacokinetics—The dynamic behavior of a material in the body, used to predict the fate (disposition) of an exogenous substance in an organism. Utilizing computational techniques, it provides the means of studying the absorption, distribution, metabolism and excretion of chemicals by the body.

Physiologically Based Pharmacodynamic (PBPD) Model—A type of physiologically-based dose-response model which quantitatively describes the relationship between target tissue dose and toxic end points. These models advance the importance of physiologically based models in that they clearly describe the biological effect (response) produced by the system following exposure to an exogenous substance.

Physiologically Based Pharmacokinetic (PBPK) Model—A model comprising a series of compartments representing organs or tissue groups with realistic weights and blood flows. These models require a variety of physiological information: tissue volumes, blood flow rates to tissues, cardiac output, alveolar ventilation rates and, possibly membrane permeabilities. The models also utilize biochemical information such as air/blood partition coefficients, and metabolic parameters. PBPK models are also called biologically based tissue dosimetry models.

Photoelectric Effect—An attenuation process observed for x and gamma radiation in which an incident photon interacts with a tightly bound inner orbital electron of an atom delivering all of its energy to knock the electron out of the atom. The incident photon disappears in the process.

Photon—A quantum of electromagnetic energy (E) whose value is the product of its frequency (v) in hertz and Planck's constant (h). The equation is: $E = hv$.

Population dose—See Collective dose.

Positron—A positively charged electron.

Potential, Ionization—The energy expressed as electron volts (eV) necessary to separate one electron from an atom, resulting in the formation of an ion pair.

Power, Stopping—A measure of the ability of a material to absorb energy from an ionizing particle passing through it; the greater the stopping power, the greater the energy absorbing ability (see Linear Energy Transfer).

Progeny—The decay product or daughter products resulting after a radioactive decay or a series of radioactive decays. The progeny can also be radioactive, and the chain continues until a stable nuclide is formed.

Proton—Elementary nuclear particle with a positive electric charge equal numerically to the charge of the electron and a rest mass of 1.007 mass units.

Quality—A term describing the distribution of the energy deposited by a particle along its track; radiations that produce different densities of ionization per unit intensity are said to have different "qualities."

Quality Factor (Q)—The linear-energy-transfer-dependent factor by which absorbed doses are multiplied to obtain (for radiation protection purposes) a quantity that expresses - on a common scale for all ionizing radiation - the approximate biological effectiveness of the absorbed dose.

Type of radiation	Quality Factor
X, gamma, or beta	1
Alpha particles	20
Neutrons of unknown energy	10
High energy protons	10

Rad—The traditional unit of absorbed dose equal to 100 ergs per gram, or 0.01 joule per kilogram (0.01 Gy) in any medium (see Absorbed Dose).

Radiation—The emission and propagation of energy through space or through a material medium in the form of waves (e.g., the emission and propagation of electromagnetic waves, or of sound and elastic waves). The term radiation or radiant energy, when unqualified, usually refers to electromagnetic radiation. Such radiation commonly is classified according to frequency, as microwaves, infrared, visible (light), ultraviolet, and x and gamma rays (see Photon.) and, by extension, corpuscular emission, such as alpha and beta radiation, neutrons, or rays of mixed or unknown type, as cosmic radiation.

> **Radiation, Annihilation**—Photons produced when an electron and a positron unite and cease to exist. The annihilation of a positron-electron pair results in the production of two photons, each of 0.51 MeV energy.

Radiation, Background—See Background Radiation.

Radiation, Characteristic (Discrete)—Radiation originating from an excited atom after removal of an electron from an atom. The wavelength of the emitted radiation is specific, depending only on the element and particular energy levels involved.

Radiation, External—Radiation from a source outside the body.

Radiation, Internal—Radiation from a source within the body (as a result of deposition of radionuclides in body tissues).

Radiation, Ionizing—Any electromagnetic or particulate radiation capable of producing ions, directly or indirectly, in its passage through matter (see Radiation).

Radiation, Monoenergetic—Radiation of a given type in which all particles or photons originate with and have the same energy.

Radiation, Scattered—Radiation which during its passage through a substance, has been deviated in direction. It may also have been modified by a decrease in energy.

Radiation, Secondary—A particle or ray that is produced when the primary radiation interacts with a material, and which has sufficient energy to produce its own ionization, such as bremsstrahlung or electrons knocked from atomic orbitals with enough energy to then produce ionization (see Delta Rays).

Radiation Weighting Factor (also called Quality Factor)—In radiation protection, a factor (1 for x-rays, gamma rays, beta particles; 20 for alpha particles) weighting the absorbed dose of radiation of a specific type and energy for its effect on tissue.

Radioactive Material—Material containing radioactive atoms.

Radioactivity—Spontaneous nuclear transformations that result in the formation of new elements. These transformations are accomplished by emission of alpha or beta particles from the nucleus or by the capture of an orbital electron. Each of these reactions may or may not be accompanied by a gamma photon.

Radioactivity, Artificial—Man-made radioactivity produced by particle bombardment or nuclear fission, as opposed to naturally occurring radioactivity.

Radioactivity, Induced—Radioactivity produced in a substance after bombardment with neutrons or other particles. The resulting activity is "natural radioactivity" if formed by nuclear reactions occurring in nature and "artificial radioactivity" if the reactions are caused by man.

Radioactivity, Natural—The property of radioactivity exhibited by more than 50 naturally occurring radionuclides.

Radioisotope—An unstable or radioactive isotope of an element that decays or disintegrates spontaneously, emitting radiation.

Radionuclide—Any radioactive isotope of any element. Approximately 5,000 natural and artificial radioisotopes have been identified.

Radiosensitivity—Relative susceptibility of cells, tissues, organs, organisms, or any living substance to the injurious action of radiation. Radiosensitivity and its antonym, radioresistance, are used comparatively, rather than absolutely.

Reference Dose (RfD)—An estimate of the daily exposure of the human population to a potential hazard that is likely to be without risk of deleterious effects during a lifetime. The RfD is operationally derived from the NOAEL (from animal and human studies) by a consistent application of uncertainty factors that reflect various types of data used to estimate RfDs and an additional modifying factor, which is based on a professional judgment of the entire database on the chemical. The RfDs are not applicable to non-threshold effects such as cancer.

Relative Biological Effectiveness (RBE)—The RBE is a factor used to compare the biological effectiveness of absorbed radiation doses (i.e., rad) due to different types of ionizing radiation. More specifically, it is the experimentally determined ratio of an absorbed dose of a radiation in question to the absorbed dose of a reference radiation (typically ^{60}Co gamma rays or 200 kVp x rays) required to produce an identical biological effect in a particular experimental organism or tissue (see Quality Factor).

Rem—The traditional unit of dose equivalent that is used in the regulatory, administrative, and engineering design aspects of radiation safety practice. The dose equivalent in rem is numerically equal to the absorbed dose in rad multiplied by the quality factor (1 rem is equal to 0.01 sievert).

Reportable Quantity (RQ)—The quantity of a hazardous substance that is considered reportable under CERCLA. Reportable quantities are (1) 1 pound or greater or (2) for selected substances, an amount established by regulation either under CERCLA or under Sect. 311 of the Clean Water Act. Quantities are measured over a 24-hour period.

Reproductive Toxicity—The occurrence of adverse effects on the reproductive system that may result from exposure to a chemical. The toxicity may be directed to the reproductive organs and/or the related endocrine system. The manifestation of such toxicity may be noted as alterations in sexual behavior, fertility, pregnancy outcomes, or modifications in other functions that are dependent on the integrity of this system.

Roentgen (R)—A unit of exposure (in air) to ionizing radiation. It is the amount of x or gamma rays required to produce ions carrying 1 electrostatic unit of electrical charge in 1 cubic centimeter of dry air under standard conditions. Named after William Roentgen, a German scientist who discovered x rays in 1895.

Retrospective Study—A type of cohort study based on a group of persons known to have been exposed at some time in the past. Data are collected from routinely recorded events, up to the time the study is undertaken. Retrospective studies are limited to causal factors that can be ascertained from existing records and/or examining survivors of the cohort.

Self-Absorption—Absorption of radiation (emitted by radioactive atoms) by the material in which the atoms are located; in particular, the absorption of radiation within a sample being assayed.

Short-Term Exposure Limit (STEL)—The maximum concentration to which workers can be exposed for up to 15 minutes continually. No more than four excursions are allowed per day, and there must be at least 60 minutes between exposure periods. The daily TLV-TWA may not be exceeded.

SI Units—The International System of Units as defined by the General Conference of Weights and Measures in 1960. These units are generally based on the meter/kilogram/second units, with special quantities for radiation including the becquerel, gray, and sievert.

Sickness, Acute Radiation (Syndrome)—The complex symptoms and signs characterizing the condition resulting from excessive exposure of the whole body (or large part) to ionizing radiation. The earliest of these symptoms are nausea, fatigue, vomiting, and diarrhea, and may be followed by loss of hair (epilation), hemorrhage, inflammation of the mouth and throat, and general loss of energy. In severe cases, where the radiation dose is relatively high (over several hundred rad or several gray), death may occur within two to four weeks. Those who survive six weeks after exposure of a single high dose of radiation may generally be expected to recover.

Sievert (Sv)—The SI unit of any of the quantities expressed as dose equivalent. The dose equivalent in sieverts is equal to the absorbed dose, in gray, multiplied by the quality factor (1 sievert equals 100 rem). The sievert is also the SI unit for effective dose equivalent, which is the sum of the products of the dose equivalent to each organ or tissue and its corresponding tissue weighting factor.

Specific-Activity—Radioactivity per unit mass of a radionuclide, expressed, for example, as Ci/gram or Bq/kilogram.

Specific Energy—The actual energy per unit mass deposited per unit volume in a small target, such as the cell or cell nucleus, as the result of one or more energy-depositing events. This is a stochastic quantity as opposed to the average value over a large number of instance (i.e., the absorbed dose).

Standardized Mortality Ratio (SMR)—A ratio of the observed number of deaths and the expected number of deaths in a specific standard population.

Stochastic Effect—A health effect that occurs randomly and for which the probability of the effect occurring, rather than its severity, is assumed to be a linear function of dose without a threshold (also called a nondeterministic effect).

Stopping Power—The average rate of energy loss of a charged particle per unit thickness of a material or per unit mass of material traversed.

Surface-seeking Radionuclide—A bone-seeking internal emitter that deposits and remains on the bone surface for a long period of time, although it may eventually diffuse into the bone mineral. This contrasts with a volume seeker, which deposits more uniformly throughout the bone volume.

Target Organ Toxicity—This term covers a broad range of adverse effects on target organs or physiological systems (e.g., renal, cardiovascular) extending from those arising through a single limited exposure to those assumed over a lifetime of exposure to a chemical.

Target Theory (Hit Theory)—A theory explaining some biological effects of radiation on the basis that ionization, occurring in a discrete volume (the target) within the cell, directly causes a lesion which subsequently results in a physiological response to the damage at that location. One, two, or more "hits" (ionizing events within the target) may be necessary to elicit the response.

Teratogen—A chemical that causes birth defects.

Threshold Limit Value (TLV)—The maximum concentration of a substance to which most workers can be exposed without adverse effect. TLV is a term used exclusively by the ACGIH. Other terms used to express similar concepts are the MAC (Maximum Allowable Concentration) and PEL (Permissible Exposure Limits).

Time-Weighted Average (TWA)—An allowable exposure concentration averaged over a normal 8-hour workday or 40-hour workweek.

Tissue Weighting Factor (W_t)—Organ- or tissue-specific factor by which the equivalent dose is multiplied to give the portion of the effective dose for that organ or tissue. Recommended values of tissue weighting factors are:

Tissue/Organ	Tissue Weighting Factor
Gonads	0.70
Bone marrow (red)	0.12
Colon	0.12
Lung	0.12
Stomach	0.12
Bladder	0.05
Breast	0.05
Liver	0.05
Esophagus	0.05
Thyroid	0.05
Skin	0.01
Bone surface	0.01
Remainder (adrenals, brain, upper large intestine, small intestine, pancreas, spleen, thymus, and uterus)	0.05

Toxic Dose (TD_{50})—A calculated dose of a chemical, introduced by a route other than inhalation, which is expected to cause a specific toxic effect in 50% of a defined experimental animal population.

Toxicokinetic—The absorption, distribution and elimination of toxic compounds in the living organism.

Toxicosis —A diseased condition resulting from poisoning.

Transformation, Nuclear—The process of radioactive decay by which a nuclide is transformed into a different nuclide by absorbing or emitting particulate or electromagnetic radiation.

Transition, Isomeric—The process by which a nuclide decays to an isomeric nuclide (i.e., one of the same mass number and atomic number) of lower quantum energy. Isomeric transitions (often abbreviated I.T.) proceed by gamma ray and internal conversion electron emission.

Tritium—The hydrogen isotope with one proton and two neutrons in the nucleus (Symbol: ^{3}H). It is radioactive and has a physical half-life of 12.3 years.

Unattached Fraction—That fraction of the radon daughters, usually ^{218}Po and ^{214}Po, which has not yet attached to a dust particle or to water vapor. As a free atom, it has a high probability of being exhaled and not retained within the lung. It is the attached fraction which is primarily retained.

Uncertainty Factor (UF)—A factor used in operationally deriving the RfD from experimental data. UFs are intended to account for (1) the variation in sensitivity among the members of the human population, (2) the uncertainty in extrapolating animal data to the case of human, (3) the uncertainty in extrapolating from data obtained in a study that is of less than lifetime exposure, and (4) the uncertainty in using LOAEL data rather than NOAEL data. Usually each of these factors is set equal to 10.

Units, Prefixes—Many units of measure are expressed as submultiples or multiples of the primary unit (e.g., 10^{-3} curie is 1 mCi and 10^{3} becquerel is 1 kBq).

Factor	Prefix	Symbol	Factor	Prefix	Symbol
10^{-18}	atto	A	10^{3}	kilo	k
10^{-15}	femto	F	10^{6}	mega	M
10^{-12}	pico	p	10^{9}	giga	G
10^{-9}	nano	N	10^{12}	tera	T
10^{-6}	micro	M	10^{15}	peta	P
10^{-3}	milli	M	10^{18}	exa	E
10^{-2}	centi	C			

Units, Radiological—

Units	Equivalents
Becquerel* (Bq)	1 disintegration per second = 2.7×10^{-11} Ci
Curie (Ci)	3.7×10^{10} disintegrations per second = 3.7×10^{10} Bq
Gray* (Gy)	1 J/kg = 100 rad
Rad (rad)	100 erg/g = 0.01 Gy
Rem (rem)	0.01 sievert
Sievert* (Sv)	100 rem

*International Units, designated (SI)

Working Level (WL)—Any combination of short-lived radon daughters in 1 liter of air that will result in the ultimate emission of 1.3×10^{5} MeV of potential alpha energy.

Working Level Month (WLM)—A unit of exposure to radon daughters corresponding to the product of the radon daughter concentration in Working Level (WL) and the exposure time in nominal months (1 nominal month = 170 hours). Inhalation of air with a concentration of 1 WL of radon daughters for 170 working hours results in an exposure of 1 WLM.

X rays—Penetrating electromagnetic radiations whose wave lengths are very much shorter than those of visible light. They are usually produced by bombarding a metallic target with fast electrons in a high vacuum. X rays (called characteristic x rays) are also produced when an orbital electron falls from a high energy level to a low energy level.

Zero-Threshold Linear Hypothesis (or No-Threshold Linear Hypothesis)—The assumption that a dose-response curve derived from data in the high dose and high dose-rate ranges may be extrapolated through the low dose and low dose range to zero, implying that, theoretically, any amount of radiation will cause some damage.

APPENDIX A. ATSDR MINIMAL RISK LEVELS AND WORKSHEETS

The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) [42 U.S.C. 9601 et seq.], as amended by the Superfund Amendments and Reauthorization Act (SARA) [Pub. L. 99–499], requires that the Agency for Toxic Substances and Disease Registry (ATSDR) develop jointly with the U.S. Environmental Protection Agency (EPA), in order of priority, a list of hazardous substances most commonly found at facilities on the CERCLA National Priorities List (NPL); prepare toxicological profiles for each substance included on the priority list of hazardous substances; and assure the initiation of a research program to fill identified data needs associated with the substances.

The toxicological profiles include an examination, summary, and interpretation of available toxicological information and epidemiologic evaluations of a hazardous substance. During the development of toxicological profiles, Minimal Risk Levels (MRLs) are derived when reliable and sufficient data exist to identify the target organ(s) of effect or the most sensitive health effect(s) for a specific duration for a given route of exposure. An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified duration of exposure. MRLs are based on noncancer health effects only and are not based on a consideration of cancer effects. These substance-specific estimates, which are intended to serve as screening levels, are used by ATSDR health assessors to identify contaminants and potential health effects that may be of concern at hazardous waste sites. It is important to note that MRLs are not intended to define clean-up or action levels.

MRLs are derived for hazardous substances using the no-observed-adverse-effect level/uncertainty factor approach. They are below levels that might cause adverse health effects in the people most sensitive to such chemical-induced effects. MRLs are derived for acute (1–14 days), intermediate (15–364 days), and chronic (365 days and longer) durations and for the oral and inhalation routes of exposure. Currently, MRLs for the dermal route of exposure are not derived because ATSDR has not yet identified a method suitable for this route of exposure. MRLs are generally based on the most sensitive chemical-induced end point considered to be of relevance to humans. Serious health effects (such as irreparable damage to the liver or kidneys, or birth defects) are not used as a basis for establishing MRLs. Exposure to a level above the MRL does not mean that adverse health effects will occur.

MRLs are intended only to serve as a screening tool to help public health professionals decide where to look more closely. They may also be viewed as a mechanism to identify those hazardous waste sites that

are not expected to cause adverse health effects. Most MRLs contain a degree of uncertainty because of the lack of precise toxicological information on the people who might be most sensitive (e.g., infants, elderly, nutritionally or immunologically compromised) to the effects of hazardous substances. ATSDR uses a conservative (i.e., protective) approach to address this uncertainty consistent with the public health principle of prevention. Although human data are preferred, MRLs often must be based on animal studies because relevant human studies are lacking. In the absence of evidence to the contrary, ATSDR assumes that humans are more sensitive to the effects of hazardous substance than animals and that certain persons may be particularly sensitive. Thus, the resulting MRL may be as much as a hundredfold below levels that have been shown to be nontoxic in laboratory animals.

Proposed MRLs undergo a rigorous review process: Health Effects/MRL Workgroup reviews within the Division of Toxicology, expert panel peer reviews, and agency wide MRL Workgroup reviews, with participation from other federal agencies and comments from the public. They are subject to change as new information becomes available concomitant with updating the toxicological profiles. Thus, MRLs in the most recent toxicological profiles supersede previously published levels. For additional information regarding MRLs, please contact the Division of Toxicology, Agency for Toxic Substances and Disease Registry, 1600 Clifton Road, Mailstop E-32, Atlanta, Georgia 30333.

APPENDIX A

MINIMAL RISK LEVEL (MRL) WORKSHEET

Chemical Name: Radioactive Cesium
CAS Number: Multiple
Date: March 11, 2004
Profile Status: Draft 3, Post-Public
Route: [ ] Inhalation [ ] Oral [X] External
Duration: [X] Acute [ ] Intermediate [ ] Chronic
Species: Human

Minimal Risk Level: 4 [ ] mg/kg/day [ ] ppm [ ] mg/m^3 [X] mSv (400 mrem)

Reference:

Schull WJ, Otake M, Yoshimaru H. 1988. Effect on intelligence test score of prenatal exposure to ionizing radiation in Hiroshima and Nagasaki: A comparison of the T65DR and DS86 dosimetry systems. Radiation Effects Research Foundation. RERF TR 3-88. Research project 24-62.

Burt C. 1966. The genetic determination of differences in intelligence: A study of monozygotic twins reared together and apart. Brit J Psychol 57(1 & 2):137-153.

Experimental design:

Schull et al. (1988) study: Schull et al. (1988) evaluated the quantitative effect of exposure to ionizing radiation on the developing fetal and embryonic human brain. The end point measured was changes in intelligence test scores. The effects on individuals exposed *in utero* to the atomic bombing of Hiroshima and Nagasaki were based on the original PE86 samples (n=1,759; data on available intelligence testing) and a clinical sample (n=1,598). The original PE86 sample included virtually all prenatally exposed individuals who received tissue-absorbed doses of 0.50 Gy or more. There were many more individuals in the dose range of 0–0.49 Gy in the PE86 sample than in the clinical sample. The clinical sample does not include children prenatally exposed at distances between 2,000 and 2,999 m in Hiroshima and Nagasaki. Children exposed at greater distances or not present in the city were selected as controls. In 1955–1956, Tanaka-B (emphasis on word-sense, arithmetic abilities, and the like, which were associated with the more subtle processing of visual clues than their simple recognition and depended more on connectedness) and the Koga (emphasis on perception of spatial relationships) intelligence tests were conducted in Nagasaki and the Koga test in Hiroshima.

Burt (1966) study: This study determined differences in intelligence in monozygotic twins reared together (n=95) and apart (n=53). All tests conducted in school consisted of (1) a group test of intelligence containing both nonverbal and verbal items, (2) an individual test (the London Revision of the Terman-Binet Scale) used primarily for standardization and for doubtful cases, and (3) a set of performance tests, based on the Pitner-Paterson tests and standardization. The methods and standard remained much the same throughout the study. Some of the reasons for separation of the twins were given as follows: death of the mother (n=9), unable to bring them up properly, mother's poor health (n=12), unmarried (n=6), and economic difficulties. The children were brought up by parents or foster parents (occupation ranged from unskilled to professional). IQ scores in the study group ranged from 66 to 137. The standard deviation of the group of separated monozygotic twins was reported at 15.3 as compared to 15.0 of ordinary siblings. Twins brought up in different environments were compared with those brought up in similar circumstances.

Effects noted in study and corresponding doses:

Schull et al. (1986) study: No evidence of radiation-related effect on intelligence was observed among individuals exposed within 0–7 weeks after fertilization or in the 26th or subsequent weeks. The highest risk of radiation damage to the embryonic and fetal brain occurs 8–15 weeks after fertilization under both dosimetric systems. The regression of intelligence score on estimated DS86 uterine absorbed dose is linear with dose, and the diminution in intelligence score is 21–29 points per Gy for the 8–15-week group and 10–26 points per Gy for the 16–25-week group. The results for 8–15 weeks applies regardless whether or not the mentally retarded individuals were included. The cumulative distribution of test scores suggested a progressive shift downwards in individual scores with increasing exposure. The mean IQ scores decrease significantly and systematically with uterine or fetal tissue dose within the 8–15- and 16–25-week groups.

In summary, analysis of intelligence test scores at 10–11 years of age of individuals exposed prenatally showed that:

There is no evidence of a radiation-related effect on intelligence scores among those individuals exposed within 0–7 weeks of fertilization or in the 26th week of gestation and beyond.

The cumulative distribution of test scores suggests a progressive shift downwards in intelligence scores with increasing exposure to ionizing radiation (dose-response relationship).

The most sensitive group was the 8–15 weeks exposure group. The regression in intelligence scores was found to be linear, with 1 Gy dose resulting in a 21–29 point decline in intelligence scores.

There was no indication of groups of individuals with differing sensitivities to radiation.

Burt (1966) study: The average intelligence of the twins measured on a conventional IQ scale (SD=15) was 97.8 for the separated monozygotes, 98.1 for monozygotes brought up together, 99.3 for the dizygotes as compared with 100.2 for the siblings, and 100.0 for the population as a whole. The difference of 0.3 IQ point between the separated and unseparated identical twins is considered a NOAEL for this study.

Dose and end point used for MRL derivation:

[X] NOAEL [ ] LOAEL 0.3 IQ point reduction in twins, between those raised together and those raised apart.

Uncertainty Factors used in MRL derivation:

[X] 1 [ ] 3 [ ] 10 (for use of a NOAEL)
[X] 1 [ ] 3 [ ] 10 (for extrapolation from animals to humans)
[ ] 1 [X] 3 [ ] 10 (for human variability/sensitive population)

Was a conversion used from ppm in food or water to a mg/body weight dose? No.

If an inhalation study in animals, list the conversion factors used in determining human equivalent dose: Not applicable

Was a conversion used from intermittent to continuous exposure? No.

Other additional studies or pertinent information which lend support to this MRL: Husen (1959) reported a study involving 269 pairs of Swedish monozygotic (identical) twins where the intrapair IQ difference was 4 IQ points for a combination of twins raised together and apart. This is somewhat lower than the value of 7 IQ points for identical twins raised apart, and just larger than the range of IQ scores for Washington, DC children repetitively tested (Jacobi and Glauberman 1995).

Supporting evidence for the acute MRL is provided by Jacobi and Glauberman (1995). Children in the 1st, 3rd, and 5th grades born in Washington, DC were tested, and average IQ levels of 94.2, 97.6, and 94.6 were reported. The range of 3.4 IQ points is considered to be a LOAEL for this study, which, if used for MRL derivation, would yield an MRL of 0.004 Sv (3.4 IQ points x 1 Sv/25 IQ points ÷ 30 [10 for use of a LOAEL and 3 for a sensitive population]).

Additional supporting evidence for the acute MRL is provided by Berger et al. 1997, in a case study of accidental radiation injury to the hand. A Mexican engineer suffered an accidental injury to the hand while repairing an x-ray spectrometer. The day after the accident, his symptoms included a tingling sensation and itching in the index and middle fingers. On days 4 and 7, a "pinching" sensation, swelling, and slight erythema were observed. By day 7, the tip of his index fingers was erythematous and a large blister developed with swelling on other fingers. On day 10, examination by a physician showed that the lesions had worsened and the fingers and palms were discolored. On day 10, he was admitted to the hospital where hyperbaric oxygen therapy was administered without success. One month after the accident, the patient entered the hospital again with pain, discoloration, and desquamation of his hand. Clinical examination showed decreased circulation in the entire hand, most notably in the index and middle finger. Total white blood count decreased to 3,000/μL (normal range 4,300–10,800/μL). Cytogenic studies of peripheral blood lymphocytes revealed four dicentrics, two rings, and eight chromosomal fragments in the 300 metaphases studied. The estimated whole body dose was reported to be 0.382 Gy (38.2 rad). This dose is a potential LOAEL for acute ionizing radiation and would yield an MRL of 0.004 Sv (0.38 Sv ÷100 [10 for use of LOAEL and 10 for sensitive human population]).

The NRC set a radiation exposure limit of 0.5 rem (50 mSv) for pregnant working women over the full gestational period (USNRC 1991). For the critical gestational period of 8–15 weeks ATSDR believes that the conservative acute MRL of 4 mSv is consistent with the NRC limit and could be applied to either acute (0–14-day) or intermediate (15–365-day) exposure periods.

Calculations:

Given: 0.3 IQ point is a NOAEL. A 1 Sv dose results in a 25 IQ point reduction (range=21–29 points; mean=25) and provides a conversion factor from IQ prediction to radiation dose. Assume that the radiation dose and the subsequent reduction in IQ is a linear relationship.

MRL = NOAEL x CF ÷ UF
MRL = 0.3 x 1/25 ÷ 3
MRL = 0.004 Sv =4 mSv (400 mrem)

Agency Contact (Chemical Manager): Malcolm Williams, Ph.D.

APPENDIX A

MINIMAL RISK LEVEL (MRL) WORKSHEET

Chemical Name: Radioactive Cesium
CAS Number: Multiple
Date: March 11, 2004
Profile Status: Draft 3, Post-Public
Route: [ ] Inhalation [ ] Oral [X] External
Duration: [ ] Acute [ ] Intermediate [X] Chronic
Species: Human

Minimal Risk Level: 1 [ ] mg/kg/day [ ] ppm [ ] mg/m^3 [X] mSv/year (100 mrem/year)

Reference: BEIR V. 1990. Health effects of exposure to low levels of ionizing radiation. Committee on the Biological Effects of Ionizing Radiations, National Research Council. National Academy Press. Washington, DC.

Experimental design: Not applicable

Effects noted in study and corresponding doses: No individual studies were identified that could be used to base a chronic-duration external exposure MRL that did not result in a cancer-producing end point. However, two sources of information were identified that did provide doses of ionizing radiation that have not been reported to be associated with detrimental effects (NOAELs). These sources provide estimates of background levels of primarily natural sources of ionizing radiation that have not been implicated in producing cancerous or noncancerous toxicological endpoints. BEIR V states that the average annual effective dose to the U.S. population is 3.6 mSv/year. A total annual effective dose equivalent of 3.6 mSv (360 mrem)/year to members of the U.S. population is obtained mainly by naturally occurring radiation from external sources, medical uses of radiation, and radiation from consumer products. The largest contribution (82%) is from natural sources, two-thirds of which is from naturally occurring radon and its decay products. Specific sources of this radiation are demonstrated in Table A-1.

The annual dose of 3.6 mSv per year has not been associated with adverse health effects or increases in the incidences of any type of cancers in humans or other animals.

Dose and end point used for MRL derivation: 3.6 mSv/year

[X] NOAEL [ ] LOAEL 3.6 mSv/year

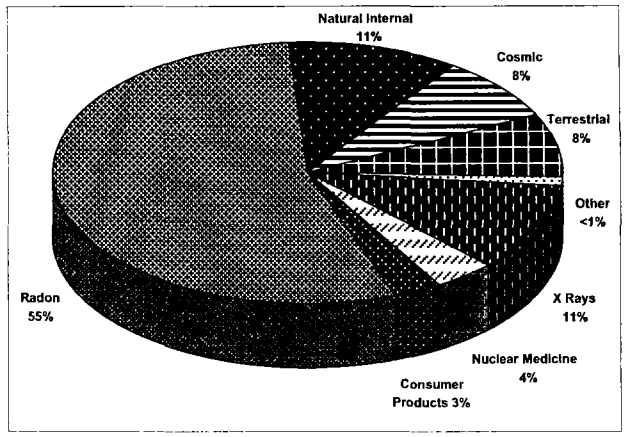
Uncertainty Factors used in MRL derivation:

[X] 1 [ ] 3 [ ] 10 (for use of a NOAEL)
[X] 1 [ ] 3 [ ] 10 (for extrapolation from animals to humans)
[ ] 1 [X] 3 [ ] 10 (for human variability)

Was a conversion used from ppm in food or water to a mg/body weight dose? No.

Table A-1. Average Annual Effective Dose Equivalent from Ionizing Radiation to a Member of the U.S. Population[a]

	Effective dose equivalent	
Source	mSv	Percent of total dose
Natural		
Radon[b]	2.0	55
Cosmic	0.27	8.0
Terrestrial	0.28	8.0
Internal	0.39	11
Total natural	3.0	82
Artificial		
Medical		
X-ray	0.39	11
Nuclear	0.14	4.0
Consumer products	0.10	3.0
Other		
Occupational	<0.01	<0.3
Nuclear fuel cycle	<0.01	<0.03
Fallout	<0.01	<0.03
Miscellaneous[c]	<0.01	<0.03
Total artificial	0.63	18
Total natural and artificial	3.6	100



[a]Adapted from BEIR V, Table 1-3 , page 18.
[b]Dose equivalent to bronchi from radon daughter products
[c]DOE facilities, smelter, transportation, etc.

If an inhalation study in animals, list the conversion factors used in determining human equivalent dose: Not applicable.

Was a conversion used from intermittent to continuous exposure? No.

Other additional studies or pertinent information which lend support to this MRL: ICRP has developed recommended dose limits for occupational and public exposure to ionizing radiation sources. The ICRP recommends limiting public exposure to 1 mSv/year (100 mrem/year), but does note that values at high altitudes above sea level and in some geological areas can sometimes be twice that value (≥2 mSv). In Annex C of ICRP 60, the commission provides data that suggests increasing the dose from 1 mSv to 5 mSv results in a very small, but detectable, increase in age-specific human mortality rate. ICRP states that the value of 1 mSv/year was chosen over the 5 mSv value because 5 mSv/year (500 mrem/year) causes this increase in age specific mortality rate, and 1 mSv/year (100 mrem/year) is typical of the annual effective dose from background, less radon (ICRP 1991). The 1 mSv estimate may underestimate the annual exposure to external sources of ionizing radiation to the U.S. population, as it does not include radiation from radon. Conversely, the 5 mSv estimate may be high, in that increases in mortality rate been reported. The most useful estimate appears to be the BEIR V estimate of 3.6 mSv, in that it accounts for an annual exposure to radon, is specific to the U.S. population, has not been associated with increases mortality, and it falls short of the 5 mSv value associated with small increases in human mortality.

Calculations:

$MRL = NOAEL_{(ADJ)} \div UF$
$MRL = 3.6 \text{ mSv/year} \div 3$
$MRL = 1.20 \text{ mSv/year}$
$MRL = 1.0 \text{ mSv/year} = 100 \text{ mrem/year above background}$

Agency Contact (Chemical Manager): Malcolm Williams, Ph.D.

APPENDIX B. USER'S GUIDE

Chapter 1

Public Health Statement

This chapter of the profile is a health effects summary written in nontechnical language. Its intended audience is the general public, especially people living in the vicinity of a hazardous waste site or chemical release. If the Public Health Statement were removed from the rest of the document, it would still communicate to the lay public essential information about the chemical.

The major headings in the Public Health Statement are useful to find specific topics of concern. The topics are written in a question and answer format. The answer to each question includes a sentence that will direct the reader to chapters in the profile that will provide more information on the given topic.

Chapter 2

Relevance to Public Health

This chapter provides a health effects summary based on evaluations of existing toxicologic, epidemiologic, and toxicokinetic information. This summary is designed to present interpretive, weight-of-evidence discussions for human health end points by addressing the following questions.

1. What effects are known to occur in humans?

2. What effects observed in animals are likely to be of concern to humans?

3. What exposure conditions are likely to be of concern to humans, especially around hazardous waste sites?

The chapter covers end points in the same order that they appear within the Discussion of Health Effects by Route of Exposure section, by route (inhalation, oral, and dermal) and within route by effect. Human data are presented first, then animal data. Both are organized by duration (acute, intermediate, chronic). *In vitro* data and data from parenteral routes (intramuscular, intravenous, subcutaneous, etc.) are also considered in this chapter.

The carcinogenic potential of the profiled substance is qualitatively evaluated, when appropriate, using existing toxicokinetic, genotoxic, and carcinogenic data. ATSDR does not currently assess cancer potency or perform cancer risk assessments. Minimal Risk Levels (MRLs) for noncancer end points (if derived) and the end points from which they were derived are indicated and discussed.

Limitations to existing scientific literature that prevent a satisfactory evaluation of the relevance to public health are identified in the Chapter 3 Data Needs section.

Interpretation of Minimal Risk Levels

Where sufficient toxicologic information is available, ATSDR has derived MRLs for inhalation and oral routes of entry at each duration of exposure (acute, intermediate, and chronic). These MRLs are not

meant to support regulatory action, but to acquaint health professionals with exposure levels at which adverse health effects are not expected to occur in humans.

MRLs should help physicians and public health officials determine the safety of a community living near a chemical emission, given the concentration of a contaminant in air or the estimated daily dose in water. MRLs are based largely on toxicological studies in animals and on reports of human occupational exposure.

MRL users should be familiar with the toxicologic information on which the number is based. Chapter 2, "Relevance to Public Health," contains basic information known about the substance. Other sections such as Chapter 3 Section 3.10, Interactions with Other Substances, and Section 3.11, Populations that are Unusually Susceptible provide important supplemental information.

MRL users should also understand the MRL derivation methodology. MRLs are derived using a modified version of the risk assessment methodology that the Environmental Protection Agency (EPA) provides (Barnes and Dourson 1988) to determine reference doses (RfDs) for lifetime exposure.

To derive an MRL, ATSDR generally selects the most sensitive end point which, in its best judgment, represents the most sensitive human health effect for a given exposure route and duration. ATSDR cannot make this judgment or derive an MRL unless information (quantitative or qualitative) is available for all potential systemic, neurological, and developmental effects. If this information and reliable quantitative data on the chosen end point are available, ATSDR derives an MRL using the most sensitive species (when information from multiple species is available) with the highest no-observed-adverse-effect level (NOAEL) that does not exceed any adverse effect levels. When a NOAEL is not available, a lowest-observed-adverse-effect level (LOAEL) can be used to derive an MRL, and an uncertainty factor (UF) of 10 must be employed. Additional uncertainty factors of 10 must be used both for human variability to protect sensitive subpopulations (people who are most susceptible to the health effects caused by the substance) and for interspecies variability (extrapolation from animals to humans). In deriving an MRL, these individual uncertainty factors are multiplied together. The product is then divided into the inhalation concentration or oral dosage selected from the study. Uncertainty factors used in developing a substance-specific MRL are provided in the footnotes of the levels of significant exposure (LSE) Tables.

Chapter 3

Health Effects

Tables and Figures for Levels of Significant Exposure (LSE)

Tables and figures are used to summarize health effects and illustrate graphically levels of exposure associated with those effects. These levels cover health effects observed at increasing dose concentrations and durations, differences in response by species, MRLs to humans for noncancer end points, and EPA's estimated range associated with an upper- bound individual lifetime cancer risk of 1 in 10,000 to 1 in 10,000,000. Use the LSE tables and figures for a quick review of the health effects and to locate data for a specific exposure scenario. The LSE tables and figures should always be used in conjunction with the text. All entries in these tables and figures represent studies that provide reliable, quantitative estimates of NOAELs, LOAELs, or Cancer Effect Levels (CELs).

The legends presented below demonstrate the application of these tables and figures. Representative examples of LSE Table 3-1 and Figure 3-1 are shown. The numbers in the left column of the legends correspond to the numbers in the example table and figure.

LEGEND

See Sample LSE Table 3-1 (page B-6)

(1) Route of Exposure. One of the first considerations when reviewing the toxicity of a substance using these tables and figures should be the relevant and appropriate route of exposure. Typically when sufficient data exist, three LSE tables and two LSE figures are presented in the document. The three LSE tables present data on the three principal routes of exposure, i.e., inhalation, oral, and dermal (LSE Table 3-1, 3-2, and 3-3, respectively). LSE figures are limited to the inhalation (LSE Figure 3-1) and oral (LSE Figure 3-2) routes. Not all substances will have data on each route of exposure and will not, therefore, have all five of the tables and figures.

(2) Exposure Period. Three exposure periods—acute (<15 days), intermediate (15–364 days), and chronic (365 days or more)—are presented within each relevant route of exposure. In this example, an inhalation study of intermediate exposure duration is reported. For quick reference to health effects occurring from a known length of exposure, locate the applicable exposure period within the LSE table and figure.

(3) Health Effect. The major categories of health effects included in LSE tables and figures are death, systemic, immunological, neurological, developmental, reproductive, and cancer. NOAELs and LOAELs can be reported in the tables and figures for all effects but cancer. Systemic effects are further defined in the "System" column of the LSE table (see key number 18).

(4) Key to Figure. Each key number in the LSE table links study information to one or more data points using the same key number in the corresponding LSE figure. In this example, the study represented by key number 18 has been used to derive a NOAEL and a Less Serious LOAEL (also see the two "18r" data points in sample Figure 3-1).

(5) Species. The test species, whether animal or human, are identified in this column. Chapter 2, "Relevance to Public Health," covers the relevance of animal data to human toxicity and Section 3.5, Toxicokinetics, contains any available information on comparative toxicokinetics. Although NOAELs and LOAELs are species specific, the levels are extrapolated to equivalent human doses to derive an MRL.

(6) Exposure Frequency/Duration. The duration of the study and the weekly and daily exposure regimen are provided in this column. This permits comparison of NOAELs and LOAELs from different studies. In this case (key number 18), rats were exposed to "Chemical X" via inhalation for 6 hours per day, 5 days per week, for 13 weeks. For a more complete review of the dosing regimen refer to the appropriate sections of the text or the original reference paper, i.e., Nitschke et al. 1981.

(7) System. This column further defines the systemic effects. These systems include respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, hepatic, renal, and dermal/ocular. "Other" refers to any systemic effect (e.g., a decrease in body weight) not covered in these systems. In the example of key number 18, one systemic effect (respiratory) was investigated.

(8) NOAEL. A NOAEL is the highest exposure level at which no harmful effects were seen in the organ system studied. Key number 18 reports a NOAEL of 3 ppm for the respiratory system,

which was used to derive an intermediate exposure, inhalation MRL of 0.005 ppm (see footnote "b").

(9) LOAEL. A LOAEL is the lowest dose used in the study that caused a harmful health effect. LOAELs have been classified into "Less Serious" and "Serious" effects. These distinctions help readers identify the levels of exposure at which adverse health effects first appear and the gradation of effects with increasing dose. A brief description of the specific end point used to quantify the adverse effect accompanies the LOAEL. The respiratory effect reported in key number 18 (hyperplasia) is a Less Serious LOAEL of 10 ppm. MRLs are not derived from Serious LOAELs.

(10) Reference. The complete reference citation is given in Chapter 9 of the profile.

(11) CEL. A CEL is the lowest exposure level associated with the onset of carcinogenesis in experimental or epidemiologic studies. CELs are always considered serious effects. The LSE tables and figures do not contain NOAELs for cancer, but the text may report doses not causing measurable cancer increases.

(12) Footnotes. Explanations of abbreviations or reference notes for data in the LSE tables are found in the footnotes. Footnote "b" indicates that the NOAEL of 3 ppm in key number 18 was used to derive an MRL of 0.005 ppm.

LEGEND

See Sample Figure 3-1 (page B-7)

LSE figures graphically illustrate the data presented in the corresponding LSE tables. Figures help the reader quickly compare health effects according to exposure concentrations for particular exposure periods.

(13) Exposure Period. The same exposure periods appear as in the LSE table. In this example, health effects observed within the acute and intermediate exposure periods are illustrated.

(14) Health Effect. These are the categories of health effects for which reliable quantitative data exists. The same health effects appear in the LSE table.

(15) Levels of Exposure. Concentrations or doses for each health effect in the LSE tables are graphically displayed in the LSE figures. Exposure concentration or dose is measured on the log scale "y" axis. Inhalation exposure is reported in mg/m^3 or ppm and oral exposure is reported in mg/kg/day.

(16) NOAEL. In this example, the open circle designated 18r identifies a NOAEL critical end point in the rat upon which an intermediate inhalation exposure MRL is based. The key number 18 corresponds to the entry in the LSE table. The dashed descending arrow indicates the extrapolation from the exposure level of 3 ppm (see entry 18 in the Table) to the MRL of 0.005 ppm (see footnote "b" in the LSE table).

(17) CEL. Key number 38r is one of three studies for which CELs were derived. The diamond symbol refers to a CEL for the test species-mouse. The number 38 corresponds to the entry in the LSE table.

(18) Estimated Upper-Bound Human Cancer Risk Levels. This is the range associated with the upper-bound for lifetime cancer risk of 1 in 10,000 to 1 in 10,000,000. These risk levels are derived from the EPA's Human Health Assessment Group's upper-bound estimates of the slope of the cancer dose response curve at low dose levels (q_1*).

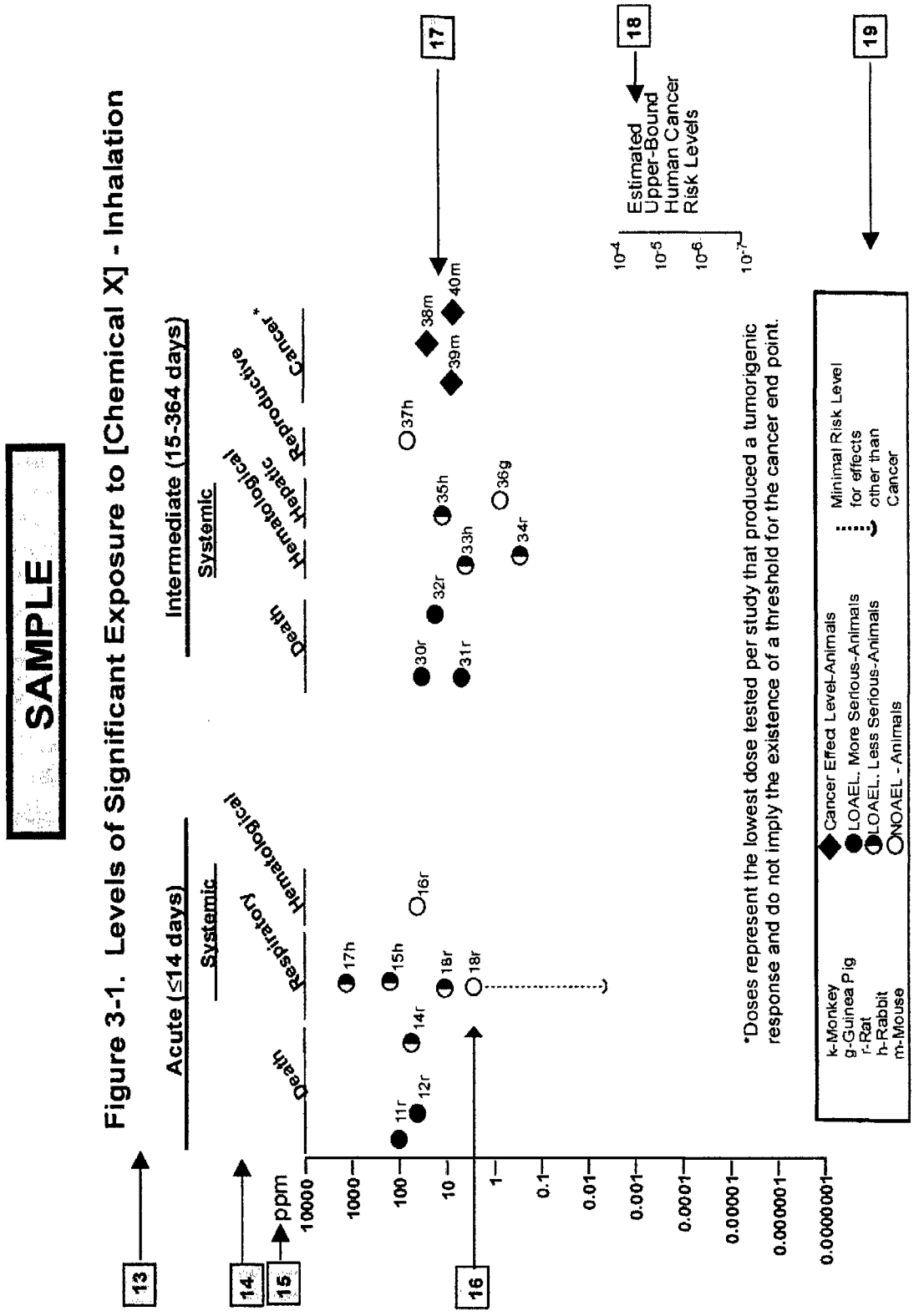
(19) Key to LSE Figure. The Key explains the abbreviations and symbols used in the figure.

SAMPLE

1 → **TABLE 3-1. Levels of Significant Exposure to [Chemical x] – Inhalation**

	Key to figure[a]	Species	Exposure frequency/ duration	System	NOAEL (ppm)	LOAEL (effect) Less serious (ppm)	LOAEL (effect) Serious (ppm)	Reference
2 →	INTERMEDIATE EXPOSURE							
		5	6	7	8	9		10
3 →	Systemic	↓	↓	↓	↓	↓		↓
4 →	18	Rat	13 wk 5 d/wk 6 hr/d	Resp	3[b]	10 (hyperplasia)		Nitschke et al. 1981
	CHRONIC EXPOSURE							
	Cancer					11		
						↓		
	38	Rat	18 mo 5 d/wk 7 hr/d			20	(CEL, multiple organs)	Wong et al. 1982
	39	Rat	89-104 wk 5 d/wk 6 hr/d			10	(CEL, lung tumors, nasal tumors)	NTP 1982
	40	Mouse	79-103 wk 5 d/wk 6 hr/d			10	(CEL, lung tumors, hemangiosarcomas)	NTP 1982

12 → a The number corresponds to entries in Figure 3-1.
b Used to derive an intermediate inhalation Minimal Risk Level (MRL) of 5x10-3 ppm; dose adjusted for intermittent exposure and divided by an uncertainty factor of 100 (10 for extrapolation from animal to humans, 10 for human variability).


SAMPLE
Figure 3-1. Levels of Significant Exposure to [Chemical X] - Inhalation
Acute (≤14 days)
Systemic
Intermediate (15-364 days)
Systemic
Death
Respiratory
Hematological
Death
Hematological
Hepatic
Reproductive
Cancer *
ppm
10000
1000
100
10
1
0.1
0.01
0.001
0.0001
0.00001
0.000001
0.0000001
11r
12r
14r
17h
15h
16r
18r
18r
30r
31r
32r
33h
35h
34r
36g
37h
38m
39m
40m
Estimated Upper-Bound Human Cancer Risk Levels
10^{-4}
10^{-5}
10^{-6}
10^{-7}
*Doses represent the lowest dose tested per study that produced a tumorigenic response and do not imply the existence of a threshold for the cancer end point.
k-Monkey
g-Guinea Pig
r-Rat
h-Rabbit
m-Mouse
Cancer Effect Level-Animals
LOAEL, More Serious-Animals
LOAEL, Less Serious-Animals
NOAEL - Animals
Minimal Risk Level for effects other than Cancer
13
14
15
16
17
18
19

APPENDIX C. ACRONYMS, ABBREVIATIONS, AND SYMBOLS

Some terms are generic and may not be used in this profile.

ACGIH	American Conference of Governmental Industrial Hygienists
ACOEM	American College of Occupational and Environmental Medicine
ADI	acceptable daily intake
ADME	absorption, distribution, metabolism, and excretion
AED	atomic emission detection
AFID	alkali flame ionization detector
AFOSH	Air Force Office of Safety and Health
ALI	annual limit on intake
ALT	alanine aminotransferase
AML	acute myeloid leukemia
AOAC	Association of Official Analytical Chemists
AOEC	Association of Occupational and Environmental Clinics
AP	alkaline phosphatase
APHA	American Public Health Association
AST	aspartate aminotransferase
atm	atmosphere
ATSDR	Agency for Toxic Substances and Disease Registry
AWQC	Ambient Water Quality Criteria
BAT	best available technology
BCF	bioconcentration factor
BEI	Biological Exposure Index
BMD	benchmark dose
BMR	benchmark response
BSC	Board of Scientific Counselors
C	centigrade
CAA	Clean Air Act
CAG	Cancer Assessment Group of the U.S. Environmental Protection Agency
CAS	Chemical Abstract Services
CDC	Centers for Disease Control and Prevention
CEL	cancer effect level
CELDS	Computer-Environmental Legislative Data System
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CFR	Code of Federal Regulations
Ci	curie
CI	confidence interval
CL	ceiling limit value
CLP	Contract Laboratory Program
cm	centimeter
CML	chronic myeloid leukemia
CPSC	Consumer Products Safety Commission
CWA	Clean Water Act
DAC	derived air concentration
DHEW	Department of Health, Education, and Welfare
DHHS	Department of Health and Human Services
DNA	deoxyribonucleic acid
DOD	Department of Defense

DOE	Department of Energy
DOL	Department of Labor
DOT	Department of Transportation
DOT/UN/NA/IMCO	Department of Transportation/United Nations/North America/International Maritime Dangerous Goods Code
DWEL	drinking water exposure level
ECD	electron capture detection
ECG/EKG	electrocardiogram
EEG	electroencephalogram
EEGL	Emergency Exposure Guidance Level
EPA	Environmental Protection Agency
F	Fahrenheit
F_1	first-filial generation
FAO	Food and Agricultural Organization of the United Nations
FDA	Food and Drug Administration
FEMA	Federal Emergency Management Agency
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
FPD	flame photometric detection
fpm	feet per minute
FR	Federal Register
FSH	follicle stimulating hormone
g	gram
GC	gas chromatography
gd	gestational day
GLC	gas liquid chromatography
GPC	gel permeation chromatography
HPLC	high-performance liquid chromatography
HRGC	high resolution gas chromatography
HSDB	Hazardous Substance Data Bank
IARC	International Agency for Research on Cancer
IDLH	immediately dangerous to life and health
ILO	International Labor Organization
IRIS	Integrated Risk Information System
Kd	adsorption ratio
kg	kilogram
kkg	metric ton
K_{oc}	organic carbon partition coefficient
K_{ow}	octanol-water partition coefficient
L	liter
LC	liquid chromatography
LC_{50}	lethal concentration, 50% kill
LC_{Lo}	lethal concentration, low
LD_{50}	lethal dose, 50% kill
LD_{Lo}	lethal dose, low
LDH	lactic dehydrogenase
LH	luteinizing hormone
LOAEL	lowest-observed-adverse-effect level
LSE	Levels of Significant Exposure
LT_{50}	lethal time, 50% kill
m	meter
MA	*trans,trans*-muconic acid

MAL	maximum allowable level
mCi	millicurie
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
MFO	mixed function oxidase
mg	milligram
mL	milliliter
mm	millimeter
mmHg	millimeters of mercury
mmol	millimole
mppcf	millions of particles per cubic foot
MRL	Minimal Risk Level
MS	mass spectrometry
NAAQS	National Ambient Air Quality Standard
NAS	National Academy of Science
NATICH	National Air Toxics Information Clearinghouse
NATO	North Atlantic Treaty Organization
NCE	normochromatic erythrocytes
NCEH	National Center for Environmental Health
NCI	National Cancer Institute
ND	not detected
NFPA	National Fire Protection Association
ng	nanogram
NHANES	National Health and Nutrition Examination Survey
NIEHS	National Institute of Environmental Health Sciences
NIOSH	National Institute for Occupational Safety and Health
NIOSHTIC	NIOSH's Computerized Information Retrieval System
NLM	National Library of Medicine
nm	nanometer
nmol	nanomole
NOAEL	no-observed-adverse-effect level
NOES	National Occupational Exposure Survey
NOHS	National Occupational Hazard Survey
NPD	nitrogen phosphorus detection
NPDES	National Pollutant Discharge Elimination System
NPL	National Priorities List
NR	not reported
NRC	National Research Council
NS	not specified
NSPS	New Source Performance Standards
NTIS	National Technical Information Service
NTP	National Toxicology Program
ODW	Office of Drinking Water, EPA
OERR	Office of Emergency and Remedial Response, EPA
OHM/TADS	Oil and Hazardous Materials/Technical Assistance Data System
OPP	Office of Pesticide Programs, EPA
OPPT	Office of Pollution Prevention and Toxics, EPA
OPPTS	Office of Prevention, Pesticides and Toxic Substances, EPA
OR	odds ratio
OSHA	Occupational Safety and Health Administration

OSW	Office of Solid Waste, EPA
OTS	Office of Toxic Substances
OW	Office of Water
OWRS	Office of Water Regulations and Standards, EPA
PAH	polycyclic aromatic hydrocarbon
PBPD	physiologically based pharmacodynamic
PBPK	physiologically based pharmacokinetic
PCE	polychromatic erythrocytes
PEL	permissible exposure limit
pg	picogram
PHS	Public Health Service
PID	photo ionization detector
pmol	picomole
PMR	proportionate mortality ratio
ppb	parts per billion
ppm	parts per million
ppt	parts per trillion
PSNS	pretreatment standards for new sources
RBC	red blood cell
REL	recommended exposure level/limit
RfC	reference concentration
RfD	reference dose
RNA	ribonucleic acid
RQ	reportable quantity
RTECS	Registry of Toxic Effects of Chemical Substances
SARA	Superfund Amendments and Reauthorization Act
SCE	sister chromatid exchange
SGOT	serum glutamic oxaloacetic transaminase
SGPT	serum glutamic pyruvic transaminase
SIC	standard industrial classification
SIM	selected ion monitoring
SMCL	secondary maximum contaminant level
SMR	standardized mortality ratio
SNARL	suggested no adverse response level
SPEGL	Short-Term Public Emergency Guidance Level
STEL	short term exposure limit
STORET	Storage and Retrieval
TD_{50}	toxic dose, 50% specific toxic effect
TLV	threshold limit value
TOC	total organic carbon
TPQ	threshold planning quantity
TRI	Toxics Release Inventory
TSCA	Toxic Substances Control Act
TWA	time-weighted average
UF	uncertainty factor
U.S.	United States
USDA	United States Department of Agriculture
USGS	United States Geological Survey
USNRC	United States Nuclear Regulatory Commission
VOC	volatile organic compound
WBC	white blood cell

WHO World Health Organization

>	greater than
≥	greater than or equal to
=	equal to
<	less than
≤	less than or equal to
%	percent
α	alpha
β	beta
γ	gamma
δ	delta
μm	micrometer
μg	microgram
q_1^*	cancer slope factor
–	negative
+	positive
(+)	weakly positive result
(–)	weakly negative result

APPENDIX D. OVERVIEW OF BASIC RADIATION PHYSICS, CHEMISTRY, AND BIOLOGY

Understanding the basic concepts in radiation physics, chemistry, and biology is important to the evaluation and interpretation of radiation-induced adverse health effects and to the derivation of radiation protection principles. This appendix presents a brief overview of the areas of radiation physics, chemistry, and biology and is based to a large extent on the reviews of Mettler and Moseley (1985), Hobbs and McClellan (1986), Eichholz (1982), Hendee (1973), Cember (1996), and Early et al. (1979).

D.1 RADIONUCLIDES AND RADIOACTIVITY

The substances we call elements are composed of atoms. Atoms in turn are made up of neutrons, protons and electrons: neutrons and protons in the nucleus and electrons in a cloud of orbits around the nucleus. Nuclide is the general term referring to any nucleus along with its orbital electrons. The nuclide is characterized by the composition of its nucleus and hence by the number of protons and neutrons in the nucleus. All atoms of an element have the same number of protons (this is given by the atomic number) but may have different numbers of neutrons (this is reflected by the atomic mass numbers or atomic weight of the element). Atoms with different atomic mass but the same atomic numbers are referred to as isotopes of an element.

The numerical combination of protons and neutrons in most nuclides is such that the nucleus is quantum mechanically stable and the atom is said to be stable, i.e., not radioactive; however, if there are too few or too many neutrons, the nucleus is unstable and the atom is said to be radioactive. Unstable nuclides undergo radioactive transformation, a process in which a neutron or proton converts into the other and a beta particle is emitted, or else an alpha particle is emitted. Each type of decay is typically accompanied by the emission of gamma rays. These unstable atoms are called radionuclides; their emissions are called ionizing radiation; and the whole property is called radioactivity. Transformation or decay results in the formation of new nuclides some of which may themselves be radionuclides, while others are stable nuclides. This series of transformations is called the decay chain of the radionuclide. The first radionuclide in the chain is called the parent; the subsequent products of the transformation are called progeny, daughters, or decay products.

In general there are two classifications of radioactivity and radionuclides: natural and artificial (man-made). Naturally-occurring radioactive materials (NORMs) exist in nature and no additional energy is necessary to place them in an unstable state. Natural radioactivity is the property of some naturally occurring, usually heavy elements, that are heavier than lead. Radionuclides, such as radium and uranium, primarily emit alpha particles. Some lighter elements such as carbon-14 and tritium (hydrogen-3) primarily emit beta particles as they transform to a more stable atom. Natural radioactive atoms heavier than lead cannot attain a stable nucleus heavier than lead. Everyone is exposed to background radiation from naturally-occurring radionuclides throughout life. This background radiation is the major source of radiation exposure to man and arises from several sources. The natural background exposures are frequently used as a standard of comparison for exposures to various artificial sources of ionizing radiation.

Artificial radioactive atoms are produced either as a by-product of fission of uranium or plutonium atoms in a nuclear reactor or by bombarding stable atoms with particles, such as neutrons or protons, directed at the stable atoms with high velocity. These artificially produced radioactive elements usually decay by emission of particles, such as positive or negative beta particles and one or more high energy photons (gamma rays). Unstable (radioactive) atoms of any element can be produced.

Both naturally occurring and artificial radioisotopes find application in medicine, industrial products, and consumer products. Some specific radioisotopes, called fall-out, are still found in the environment as a result of nuclear weapons use or testing.

D.2 RADIOACTIVE DECAY

D.2.1 Principles of Radioactive Decay

The stability of an atom is the result of the balance of the forces of the various components of the nucleus. An atom that is unstable (radionuclide) will release energy (decay) in various ways and transform to stable atoms or to other radioactive species called daughters, often with the release of ionizing radiation. If there are either too many or too few neutrons for a given number of protons, the resulting nucleus may undergo transformation. For some elements, a chain of daughter decay products may be produced until stable atoms are formed. Radionuclides can be characterized by the type and energy of the radiation emitted, the rate of decay, and the mode of decay. The mode of decay indicates how a parent compound undergoes transformation. Radiations considered here are primarily of nuclear origin, i.e., they arise from nuclear excitation, usually caused by the capture of charged or uncharged nucleons by a nucleus, or by the radioactive decay or transformation of an unstable nuclide. The type of radiation may be categorized as charged or uncharged particles, protons, and fission products) or electromagnetic radiation (gamma rays and x rays). Table D-1 summarizes the basic characteristics of the more common types of radiation encountered.

D.2.2 Half-Life and Activity

For any given radionuclide, the rate of decay is a first-order process that is constant, regardless of the radioactive atoms present and is characteristic for each radionuclide. The process of decay is a series of random events; temperature, pressure, or chemical combinations do not effect the rate of decay. While it may not be possible to predict exactly which atom is going to undergo transformation at any given time, it is possible to predict, on average, the fraction of the radioactive atoms that will transform during any interval of time.

The *activity* is a measure of the quantity of radioactive material. For these radioactive materials it is customary to describe the activity as the number of disintegrations (transformations) per unit time. The unit of activity is the curie (Ci), which was originally related to the activity of one gram of radium, but is now defined as that quantity of radioactive material in which there are:

1 curie (Ci) = 3.7×10^{10} disintegrations (transformations)/second (dps) or 2.22×10^{12} disintegrations (transformations)/minute (dpm).

The SI unit of activity is the becquerel (Bq); 1 Bq = that quantity of radioactive material in which there is 1 transformation/second. Since activity is proportional to the number of atoms of the radioactive material, the quantity of any radioactive material is usually expressed in curies, regardless of its purity or concentration. The transformation of radioactive nuclei is a random process, and the number of transformations is directly proportional to the number of radioactive atoms present. For any pure radioactive substance, the rate of decay is usually described by its radiological half-life, T_R, i.e., the time it takes for a specified source material to decay to half its initial activity. The specific activity is the activity of a radionuclide per mass of that radionuclide. If properly qualified, it can refer to activity per unit mass of related materials, such as the element itself or a chemical compound labeled with the radionuclide. The higher the specific activity of a radioisotope, the faster it is decaying.

The activity of a radionuclide at time t may be calculated by:

$$A = A_o e^{-0.693t/Trad}$$

where A is the activity in dps or curies or becquerels, A_o is the activity at time zero, t is the time at which measured, and T_{rad} is the radiological half-life of the radionuclide (T_{rad} and t must be in the same units of time). The time when the activity of a sample of radioactivity becomes one-half its original value is the radioactive half-life and is expressed in any suitable unit of time.

Table D-1. Characteristics of Nuclear Radiations

Radiation	Rest mass[a]	Charge	Typical energy range	Path length[b] Air	Path length[b] Solid	Comments
Alpha (α)	4.00 amu	+2	4–10 MeV	5–10 cm	25–80 μm	Identical to ionized He nucleus
Negatron (β^-)	5.48×10^{-4} amu; 0.51 MeV	–1	0–4 MeV	0–10 m	0–1 cm	Identical to electron
Positron (β^+)	5.48×10^{-4} amu; 0.51 MeV	+1	0-4 MeV	0–10 m	0–1 cm	Identical to electron except for sign of charge
Neutron	1.0086 amu; 939.55 MeV	0	0–15 MeV	b	b	Free half-life: 16 min
X ray (e.m. photon)	–	0	5 keV–100 keV	b	b	Photon from transition of an electron between atomic orbits
Gamma (γ) (e.m. photon)	–	0	10 keV–3 MeV	b	b	Photon from nuclear transformation

[a] The rest mass (in amu) has an energy equivalent in MeV that is obtained using the equation $E=mc^2$, where 1 amu = 932 MeV.
[b] Path lengths are not applicable to x- and gamma rays since their intensities decrease exponentially; path lengths in solid tissue are variable, depending on particle energy, electron density of material, and other factors.

amu = atomic mass unit; e.m. = electromagnetic; MeV = Megaelectron Volts

The specific activity is a measure of activity, and is defined as the activity of a radionuclide per mass of that radionuclide. This activity is usually expressed in curies per gram and may be calculated by

$$\text{curies/gram} = 1.3 \times 10^8 / (T_{rad})\ (\text{atomic weight}) \quad \text{or}$$

$$[3.577 \times 10^5 \times \text{mass(g)}] / [T_{rad} \times \text{atomic weight}]$$

where T_{rad} is the radiological half-life in days.

In the case of radioactive materials contained in living organisms, an additional consideration is made for the reduction in observed activity due to regular processes of elimination of the respective chemical or biochemical substance from the organism. This introduces a rate constant called the biological half-life (T_{biol}) which is the time required for biological processes to eliminate one-half of the activity. This time is virtually the same for both stable and radioactive isotopes of any given element.

Under such conditions the time required for a radioactive element to be halved as a result of the combined action of radioactive decay and biological elimination is the effective clearance half-time:

$$T_{eff} = (T_{biol} \times T_{rad}) / (T_{biol} + T_{rad}).$$

Table D-2 presents representative effective half-lives of particular interest.

Table D-2. Half-Lives of Some Radionuclides in Adult Body Organs

		Half-life[a]		
Radionuclide	Critical organ	Physical	Biological	Effective
Uranium 238	Kidney	4,460,000,000 y	4 d	4 d
Hydrogen 3[b] (Tritium)	Whole body	12.3 y	10 d	10 d
Iodine 131	Thyroid	8 d	80 d	7.3 d
Strontium 90	Bone	28 y	50 y	18 y
Plutonium 239	Bone surface	24,400 y	50 y	50 y
	Lung	24,400 y	500 d	474 d
Cobalt 60	Whole body	5.3 y	99.5 d	95 d
Iron 55	Spleen	2.7 y	600 d	388 d
Iron 59	Spleen	45.1 d	600 d	42 d
Manganese 54	Liver	303 d	25 d	23 d
Cesium 137	Whole body	30 y	70 d	70 d

[a]d = days, y = years
[b]Mixed in body water as tritiated water

D.2.3 Interaction of Radiation with Matter

Both ionizing and nonionizing radiation will interact with materials; that is, radiation will lose kinetic energy to any solid, liquid or gas through which it passes by a variety of mechanisms. The transfer of energy to a medium by either electromagnetic or particulate radiation may be sufficient to cause formation of ions. This process is called ionization. Compared to other types of radiation that may be absorbed, such as ultraviolet radiation, ionizing radiation deposits a relatively large amount of energy into a small volume.

The method by which incident radiation interacts with the medium to cause ionization may be direct or indirect. Electromagnetic radiations (x rays and gamma photons) are indirectly ionizing; that is, they give up their energy in various interactions with cellular molecules, and the energy is then utilized to produce a fast-moving charged particle such as an electron. It is the electron that then may react with a target molecule. This particle is called a "primary ionizing particle. Charged particles, in contrast, strike the tissue or medium and directly react with target molecules, such as oxygen or water. These particulate radiations are directly ionizing radiations. Examples of directly ionizing particles include alpha and beta particles. Indirectly ionizing radiations are always more penetrating than directly ionizing particulate radiations.

Mass, charge, and velocity of a particle, as well as the electron density of the material with which it interacts, all affect the rate at which ionization occurs. The higher the charge of the particle and the lower the velocity, the greater the propensity to cause ionization. Heavy, highly charged particles, such as alpha particles, lose energy rapidly with distance and, therefore, do not penetrate deeply. The result of these

interaction processes is a gradual slowing down of any incident particle until it is brought to rest or "stopped" at the end of its range.

D.2.4 Characteristics of Emitted Radiation

D.2.4.1 Alpha Emission. In alpha emission, an alpha particle consisting of two protons and two neutrons is emitted with a resulting decrease in the atomic mass number by four and reduction of the atomic number of two, thereby changing the parent to a different element. The alpha particle is identical to a helium nucleus consisting of two neutrons and two protons. It results from the radioactive decay of some heavy elements such as uranium, plutonium, radium, thorium, and radon. The alpha particles emitted by a given radionuclide have the same energy and intensity combination. Most of the alpha particles that are likely to be found have energies in the range of about 4 to 8 MeV, depending on the isotope from which they came.

The alpha particle has an electrical charge of +2. Because of this double positive charge and their size, alpha particles have great ionizing power and, thus, lose their kinetic energy quickly. This results in very little penetrating power. In fact, an alpha particle cannot penetrate a sheet of paper. The range of an alpha particle (the distance the charged particle travels from the point of origin to its resting point) is about 4 cm in air, which decreases considerably to a few micrometers in tissue. These properties cause alpha emitters to be hazardous only if there is internal contamination (i.e., if the radionuclide is inside the body).

D.2.4.2 Beta Emission. A beta particle (β) is a high-velocity electron ejected from a disintegrating nucleus. The particle may be either a negatively charged electron, termed a negatron (β-) or a positively charged electron, termed a positron (β+). Although the precise definition of "beta emission" refers to both β- and β+, common usage of the term generally applies only to the negative particle, as distinguished from the positron emission, which refers to the β+ particle.

D.2.4.2.1 Beta Negative Emission. Beta particle (β-) emission is another process by which a radionuclide, with a neutron excess achieves stability. Beta particle emission decreases the number of neutrons by one and increases the number of protons by one, while the atomic mass number remains unchanged.[1] This transformation results in the formation of a different element. The energy spectrum of beta particle emission ranges from a certain maximum down to zero with the mean energy of the spectrum being about one-third of the maximum. The range of betas is much less in tissue than in air. Beta negative emitting radionuclides can cause injury to the skin and superficial body tissues, but mostly present an internal contamination hazard.

D.2.4.2.2 Positron Emission. In cases in which there are too many protons in the nucleus, positron emission may occur. In this case a proton may be thought of as being converted into a neutron, and a positron (β+) is emitted.1 This increases the number of neutrons by one, decreases the number of protons by one, and again leaves the atomic mass number unchanged. The gamma radiation resulting from the annihilation (see glossary) of the positron makes all positron emitting isotopes more of an external radiation hazard than pure β emitters of equal energy.

D.2.4.2.3 Gamma Emission. Radioactive decay by alpha, beta, or positron emission, or electron capture often leaves some of the energy resulting from these changes in the nucleus. As a result, the nucleus is raised to an excited level. None of these excited nuclei can remain in this high-energy state. Nuclei release this energy returning to ground state or to the lowest possible stable energy level. The energy released is in the form of gamma radiation (high energy photons) and has an energy equal to the

[1] Neutrinos also accompany negative beta particles and positron emissions

change in the energy state of the nucleus. Gamma and x rays behave similarly but differ in their origin; gamma emissions originate in the nucleus while x rays originate in the orbital electron structure or from rapidly changing the velocity of an electron (e.g., as occurs when shielding high energy beta particles or stopping the electron beam in an x ray tube).

D.3 ESTIMATION OF ENERGY DEPOSITION IN HUMAN TISSUES

Two forms of potential radiation exposures can result: internal and external. The term exposure denotes physical interaction of the radiation emitted from the radioactive material with cells and tissues of the human body. An exposure can be "acute" or "chronic" depending on how long an individual or organ is exposed to the radiation. Internal exposures occur when radionuclides, which have entered the body (e.g., through the inhalation, ingestion, or dermal pathways), undergo radioactive decay resulting in the deposition of energy to internal organs. External exposures occur when radiation enters the body directly from sources located outside the body, such as radiation emitters from radionuclides on ground surfaces, dissolved in water, or dispersed in the air. In general, external exposures are from material emitting gamma radiation, which readily penetrate the skin and internal organs. Beta and alpha radiation from external sources are far less penetrating and deposit their energy primarily on the skin's outer layer. Consequently, their contribution to the absorbed dose of the total body dose, compared to that deposited by gamma rays, may be negligible.

Characterizing the radiation dose to persons as a result of exposure to radiation is a complex issue. It is difficult to: (1) measure internally the amount of energy actually transferred to an organic material and to correlate any observed effects with this energy deposition; and (2) account for and predict secondary processes, such as collision effects or biologically triggered effects, that are an indirect consequence of the primary interaction event.

D.3.1 Dose/Exposure Units

D.3.1.1 Roentgen. The roentgen (R) is a unit of x or gamma-ray exposure and is a measured by the amount of ionization caused in air by gamma or x radiation. One roentgen produces 2.58×10^{-4} coulomb per kilogram of air. In the case of gamma radiation, over the commonly encountered range of photon energy, the energy deposition in tissue for a dose of 1 R is about 0.0096 joules (J) /kg of tissue.

D.3.1.2 Absorbed Dose and Absorbed Dose Rate. The absorbed dose is defined as the energy imparted by radiation to a unit mass of the tissue or organ. The unit of absorbed dose is the rad; 1 rad = 100 erg/gram = 0.01 J/kg in any medium. An exposure of 1 R results in a dose to soft tissue of approximately 0.01 J/kg. The SI unit is the gray which is equivalent to 100 rad or 1 J/kg. Internal and external exposures from radiation sources are not usually instantaneous but are distributed over extended periods of time. The resulting rate of change of the absorbed dose to a small volume of mass is referred to as the absorbed dose rate in units of rad/unit time.

D.3.1.3 Working Levels and Working Level Months. Working level (WL) is a measure of the atmospheric concentration of radon and its short-lived progeny. One WL is defined as any combination of short-lived radon daughters (through polonium-214), per liter of air, that will result in the emission of 1.3×10^5 MeV of alpha energy. An activity concentration of 100 pCi radon-222/L of air, in equilibrium with its daughters, corresponds approximately to a potential alpha-energy concentration of 1 WL. The WL unit can also be used for thoron daughters. In this case, 1.3×10^5 MeV of alpha energy (1 WL) is released by the thoron daughters in equilibrium with 7.5 pCi thoron/L. The potential alpha energy exposure of miners is commonly expressed in the unit Working Level Month (WLM). One WLM corresponds to exposure to a concentration of 1 WL for the reference period of 170 hours, or more generally

WLM = concentration (WL) x exposure time (months) (one "month" = 170 working hours).

D.3.2 Dosimetry Models

Dosimetry models are used to estimate the dose from internally deposited to radioactive substances. The models for internal dosimetry consider the amount of radionuclides entering the body, the factors affecting their movement or transport through the body, distribution and retention of radionuclides in the body, and the energy deposited in organs and tissues from the radiation that is emitted during spontaneous decay processes. The dose pattern for radioactive materials in the body may be strongly influenced by the route of entry of the material. For industrial workers, inhalation of radioactive particles with pulmonary deposition and puncture wounds with subcutaneous deposition have been the most frequent. The general population has been exposed via ingestion and inhalation of low levels of naturally occurring radionuclides as well as radionuclides from nuclear weapons testing.

The models for external dosimetry consider only the photon doses (and neutron doses, where applicable) to organs of individuals who are immersed in air or are exposed to a contaminated object.

D.3.2.1 Ingestion. Ingestion of radioactive materials is most likely to occur from contaminated foodstuffs or water or eventual ingestion of inhaled compounds initially deposited in the lung. Ingestion of radioactive material may result in toxic effects as a result of either absorption of the radionuclide or irradiation of the gastrointestinal tract during passage through the tract, or a combination of both. The fraction of a radioactive material absorbed from the gastrointestinal tract is variable, depending on the specific element, the physical and chemical form of the material ingested, and the diet, as well as some other metabolic and physiological factors. The absorption of some elements is influenced by age, usually with higher absorption in the very young.

D.3.2.2 Inhalation. The inhalation route of exposure has long been recognized as being a major portal of entry for both nonradioactive and radioactive materials. The deposition of particles within the lung is largely dependent upon the size of the particles being inhaled. After the particle is deposited, the retention will depend upon the physical and chemical properties of the dust and the physiological status of the lung. The retention of the particle in the lung depends on the location of deposition, in addition to the physical and chemical properties of the particles. The converse of pulmonary retention is pulmonary clearance. There are three distinct mechanisms of clearance which operate simultaneously. Ciliary clearance acts only in the upper respiratory tract. The second and third mechanisms act mainly in the deep respiratory tract. These are phagocytosis and absorption. Phagocytosis is the engulfing of foreign bodies by alveolar macrophages and their subsequent removal either up the ciliary "escalator" or by entrance into the lymphatic system. Some inhaled soluble particles are absorbed into the blood and translocated to other organs and tissues.

D.3.3 Internal Emitters

An internal emitter is a radionuclide that is inside the body. The absorbed dose from internally deposited radionuclide depends on the energy absorbed per unit mass by the irradiated tissue. For a radionuclide distributed uniformly throughout an infinitely large medium, the concentration of absorbed energy must be equal to the concentration of energy emitted by the radionuclide. An infinitely large medium may be approximated by a tissue mass whose dimensions exceed the range of the particle. All alpha and most beta radiation will be absorbed in the organ (or tissue) of reference. Gamma-emitting radionuclide emissions are penetrating radiation, and a substantial fraction of gamma energy may be absorbed in tissue. The dose to an organ or tissue is a function of the effective retention half-time, the energy released in the tissue, the amount of radioactivity initially introduced, and the mass of the organ or tissue.

D.4 BIOLOGICAL EFFECTS OF RADIATION

When biological material is exposed to ionizing radiation, a chain of cellular events occurs as the ionizing particle passes through the biological material. A number of theories have been proposed to describe the interaction of radiation with biologically important molecules in cells and to explain the resulting damage to biological systems from those interactions. Many factors may modify the response of a living organism to a given dose of radiation. Factors related to the exposure include the dose rate, the energy of the radiation, and the temporal pattern of the exposure. Biological considerations include factors such as species, age, sex, and the portion of the body exposed. Several excellent reviews of the biological effects of radiation have been published, and the reader is referred to these for a more in-depth discussion (Brodsky 1996; Hobbs and McClellan 1986; ICRP 1984; Mettler and Moseley 1985; Rubin and Casarett 1968).

D.4.1 Radiation Effects at the Cellular Level

According to Mettler and Moseley (1985), at acute doses up to 10 rad (100 mGy), single strand breaks in DNA may be produced. These single strand breaks may be repaired rapidly. With doses in the range of 50–500 rad (0.5–5 Gy), irreparable double-stranded DNA breaks are likely, resulting in cellular reproductive death after one or more divisions of the irradiated parent cell. At large doses of radiation, usually greater than 500 rad (5 Gy), direct cell death before division (interphase death) may occur from the direct interaction of free-radicals with essential cellular macromolecules. Morphological changes at the cellular level, the severity of which are dose-dependent, may also be observed.

The sensitivity of various cell types varies. According to the Bergonie-Tribondeau law, the sensitivity of cell lines is directly proportional to their mitotic rate and inversely proportional to the degree of differentiation (Mettler and Moseley 1985). Rubin and Casarett (1968) devised a classification system that categorized cells according to type, function, and mitotic activity. The categories range from the most sensitive type, "vegetative intermitotic cells", found in the stem cells of the bone marrow and the gastrointestinal tract, to the least sensitive cell type, "fixed postmitotic cells," found in striated muscles or long-lived neural tissues.

Cellular changes may result in cell death, which if extensive, may produce irreversible damage to an organ or tissue or may result in the death of the individual. If the cell recovers, altered metabolism and function may still occur, which may be repaired or may result in the manifestation of clinical symptoms. These changes may also be expressed at a later time as tumors or cellular mutations, which may result in abnormal tissue.

D.4.2 Radiation Effects at the Organ Level

In most organs and tissues the injury and the underlying mechanism for that injury are complex and may involve a combination of events. The extent and severity of this tissue injury are dependent upon the radiosensitivity of the various cell types in that organ system. Rubin and Casarett (1968) describe and schematically display the events following radiation in several organ system types. These include: a rapid renewal system, such as the gastrointestinal mucosa; a slow renewal system, such as the pulmonary epithelium; and a nonrenewal system, such as neural or muscle tissue. In the rapid renewal system, organ injury results from the direct destruction of highly radiosensitive cells, such as the stem cells in the bone marrow. Injury may also result from constriction of the microcirculation and from edema and inflammation of the basement membrane, designated as the histohematic barrier, which may progress to fibrosis. In slow renewal and nonrenewal systems, the radiation may have little effect on the parenchymal

cells, but ultimate parenchymal atrophy and death over several months result from fibrosis and occlusion of the microcirculation.

D.4.3 Low Level Radiation Effects

Cancer is the major latent harmful effect produced by ionizing radiation and the one that most people exposed to radiation are concerned about. The ability of alpha, beta, and gamma radiation to produce cancer in virtually every tissue and organ in laboratory animals has been well-demonstrated. The development of cancer is not an immediate effect. Radiation-induced leukemia has the shortest latent period at about 2 years, while other radiation induced cancers, such as osteosarcoma, have latent periods greater than 20 years. The mechanism by which cancer is induced in living cells is complex and is a topic of intense study. Exposure to ionizing radiation can produce cancer at any site within the body; however, some sites appear to be more common than others, such as the breast, lung, stomach, and thyroid.

DNA is the major target molecule during exposure to ionizing radiation. Other macromolecules, such as lipids and proteins, are also at risk of damage when exposed to ionizing radiation. The genotoxicity of ionizing radiation is an area of intense study, as damage to the DNA is ultimately responsible for many of the adverse toxicological effects ascribed to ionizing radiation, including cancer. Damage to genetic material is basic to developmental or teratogenic effects, as well. However, for effects other than cancer, there is little evidence of human effects at low levels of exposure.

D.5 UNITS IN RADIATION PROTECTION AND REGULATION

D.5.1 Dose Equivalent (or Equivalent Dose)

Dose equivalent (as measured in rem or sievert) is a special radiation protection quantity that is used for administrative and radiation safety purposes to express the absorbed dose in a manner which considers the difference in biological effectiveness of various kinds of ionizing radiation. ICRP (1990) changed this term to equivalent dose, but it has not yet been adopted by the USNRC or DOE.

The USNRC defines the dose equivalent, H, as the product of the absorbed dose, D, and the quality factor, Q, at the point of interest in biological tissue. This relationship is expressed as $H = D \times Q$. The dose equivalent concept is applicable only to doses that are not great enough to produce biomedical effects.

The quality factor or radiation weighting factor is a dimensionless quantity that depends in part on the stopping power for charged particles, and it accounts for the differences in biological effectiveness found among the types of radiation. Originally relative biological effectiveness (RBE) was used rather than Q to define the quantity, rem, which was of use in risk assessment. The generally accepted values for quality factors and radiation weighting factors for various radiation types are provided in Table D-3. The dose equivalent rate is the time rate of change of the dose equivalent to organs and tissues and is expressed as rem/unit time or sievert/unit time.

Table D-3. Quality Factors (Q) and Absorbed Dose Equivalencies

Type of radiation	Quality factor (Q)	Radiation weighting factor (w_r)*
X, gamma, or beta radiation	1	1
Alpha particles, multiple-charged particles, fission fragments and heavy particles of unknown charge	20	0.05
Neutrons (other than thermal >> 100 keV to 2 MeV), protons, alpha particles, charged particles of unknown energy	10	20
Neutrons of unknown energy	10	
High-energy protons	10	0.1
Thermal neutrons		5

*Absorbed dose in rad equal to 1 rem or the absorbed dose in gray equal to 1 sievert.

Source: USNRC. 2004. Standards for the protection against radiation, table 1004(b).1. 10 CFR 20.1004. U.S. Nuclear Regulatory Commission, Washington, D.C. NCRP 1993

D.5.2 Relative Biological Effectiveness

RBE is used to denote the experimentally determined ratio of the absorbed dose from one radiation type to the absorbed dose of a reference radiation required to produce an identical biologic effect under the same conditions. Gamma rays from cobalt-60 and 200–250 kVp x-rays have been used as reference standards. The term RBE has been widely used in experimental radiobiology, and the term quality factor (or radiation weighting factor) used in calculations of dose equivalents for radiation safety purposes (ICRP 1977; NCRP 1971; UNSCEAR 1982). Any RBE value applies only to a specific biological end point, in a specific exposure, under specific conditions to a specific species. There are no generally applicable values of RBE since RBEs are specific to a given exposure scenario.

D.5.3 Effective Dose Equivalent (or Effective Dose)

The absorbed dose is usually defined as the mean energy imparted per unit mass to an organ or tissue. This represents a simplification of the actual problem. Normally when an individual ingests or inhales a radionuclide or is exposed to external radiation that enters the body (gamma), the dose is not uniform throughout the whole body. The simplifying assumption is that the detriment will be the same whether the body is uniformly or non-uniformly irradiated. In an attempt to compare detriment from absorbed dose of a limited portion of the body with the detriment from total body dose, the ICRP (1977) has derived a concept of effective dose equivalent. ICRP (1990) changed this term to effective dose, but it has not yet been adopted by the USNRC or DOE.

The effective dose equivalent, H_E, is

$$H_E = \text{(the sum of) } W_t H_t$$

where H_t is the dose equivalent (or equivalent dose) in the tissue t, W_t is the tissue weighting factor in that tissue, which represents the estimated proportion of the stochastic risk resulting from tissue, t, to the stochastic risk when the whole body is uniformly irradiated for occupational exposures under certain conditions (ICRP 1977). Tissue weighting factors for selected tissues are listed in Table D-4.

D.5.4 SI Units

The ICRU (1980), ICRP (1984), and NCRP (1985) now recommend that the rad, roentgen, curie, and rem be replaced by the SI units: gray (Gy), Coulomb per kilogram (C/kg), Becquerel (Bq), and sievert (Sv), respectively. The relationship between the customary units and the international system of units (SI) for radiological quantities is shown in Table D-5.

Table D-4. Tissue Weighting Factors for Calculating Effective Dose Equivalent and Effective Dose for Selected Tissues

	Tissue weighting factor	
Tissue	NCRP115/ ICRP60	USNRC/ICRP26
Bladder	0.05	–
Bone marrow	0.12	0.12
Bone surface	0.01	0.03
Breast	0.05	0.15
Colon	0.12	–
Esophagus	0.05	–
Gonads	0.20	0.25
Liver	0.05	–
Lung	0.12	0.12
Skin	0.01	–
Stomach	0.12	–
Thyroid	0.05	0.03
Remainder	0.05	0.30
Total	1.00	1.00

ICRP60 = International Commission on Radiological Protection, 1990 Recommendations of the ICRP
NCRP115 = National Council on Radiation Protection and Measurements. 1993. Risk Estimates for Radiation Protection, Report 115. Bethesda, Maryland
USNRC = Nuclear Regulatory Commission, Title 10, Code of Federal Regulations, Part 20

Table D-5. Comparison of Common and SI Units for Radiation Quantities

Quantity	Customary units	Definition	SI units	Definition
Activity (A)	curie (Ci)	3.7×10^{10} transformations s^{-1}	becquerel (Bq)	s^{-1}
Absorbed dose (D)	rad	10^{-2} Jkg^{-1}	gray (Gy)	Jkg^{-1}
Absorbed dose rate ($\check{D}$)	rad per second (rad s^{-1})	10^{-2} $Jkg^{-1}s^{-1}$	gray per second (Gy s^{-1})	Jkg^{-1} s^{-1}
Dose equivalent (H)	rem	10^{-2} Jkg^{-1}	sievert (Sv)	Jkg^{-1}
Dose equivalent rate ($\hat{H}$)	rem per second (rem s^{-1})	10^{-2} $Jkg^{-1}s^{-1}$	sievert per second (Sv s^{-1})	Jkg^{-1} s^{-1}
Effective dose	rem	10^{-2} Jkg^{-1}	Sievert (Sv)	Jkg^{-1}
Equivalent dose (H)	rem	10^{-2} Jkg^{-1}	Sievert (Sv)	Jkg^{-1}
Linear energy transfer (LET)	kiloelectron volts per micrometer (keV μm^{-1})	1.602×10^{-10} Jm^{-1}	kiloelectron volts per micrometer (keV μm^{-1})	1.602×10^{-10} Jm^{-1}

Jkg^{-1} = Joules per kilogram; $Jkg^{-1}s^{-1}$ = Joules per kilogram per second; Jm^{-1} = Joules per meter; s^{-1} = per second

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TOXICOLOGICAL PROFILE FOR COBALT

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry

April 2004

DISCLAIMER

The use of company or product name(s) is for identification only and does not imply endorsement by the Agency for Toxic Substances and Disease Registry.

UPDATE STATEMENT

A Toxicological Profile for cobalt, Draft for Public Comment was released in July 2001. This edition supersedes any previously released draft or final profile.

Toxicological profiles are revised and republished as necessary. For information regarding the update status of previously released profiles, contact ATSDR at:

Agency for Toxic Substances and Disease Registry
Division of Toxicology/Toxicology Information Branch
1600 Clifton Road NE,
Mailstop F-32
Atlanta, Georgia 30333

FOREWORD

This toxicological profile is prepared in accordance with guidelines* developed by the Agency for Toxic Substances and Disease Registry (ATSDR) and the Environmental Protection Agency (EPA). The original guidelines were published in the *Federal Register* on April 17, 1987. Each profile will be revised and republished as necessary.

The ATSDR toxicological profile succinctly characterizes the toxicologic and adverse health effects information for the hazardous substance described therein. Each peer-reviewed profile identifies and reviews the key literature that describes a hazardous substance's toxicologic properties. Other pertinent literature is also presented, but is described in less detail than the key studies. The profile is not intended to be an exhaustive document; however, more comprehensive sources of specialty information are referenced.

The focus of the profiles is on health and toxicologic information; therefore, each toxicological profile begins with a public health statement that describes, in nontechnical language, a substance's relevant toxicological properties. Following the public health statement is information concerning levels of significant human exposure and, where known, significant health effects. The adequacy of information to determine a substance's health effects is described in a health effects summary. Data needs that are of significance to protection of public health are identified by ATSDR and EPA.

Each profile includes the following:

(A) The examination, summary, and interpretation of available toxicologic information and epidemiologic evaluations on a hazardous substance to ascertain the levels of significant human exposure for the substance and the associated acute, subacute, and chronic health effects;

(B) A determination of whether adequate information on the health effects of each substance is available or in the process of development to determine levels of exposure that present a significant risk to human health of acute, subacute, and chronic health effects; and

(C) Where appropriate, identification of toxicologic testing needed to identify the types or levels of exposure that may present significant risk of adverse health effects in humans.

The principal audiences for the toxicological profiles are health professionals at the Federal, State, and local levels; interested private sector organizations and groups; and members of the public.

This profile reflects ATSDR's assessment of all relevant toxicologic testing and information that has been peer-reviewed. Staff of the Centers for Disease Control and Prevention and other Federal scientists have also reviewed the profile. In addition, this profile has been peer-reviewed by a nongovernmental panel and was made available for public review. Final responsibility for the contents and views expressed in this toxicological profile resides with ATSDR.

Julie Louise Gerberding, M.D., M.P.H.
Administrator
Agency for Toxic Substances and
Disease Registry

Background Information

The toxicological profiles are developed by ATSDR pursuant to Section 104(i) (3) and (5) of the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA or Superfund) for hazardous substances found at Department of Energy (DOE) waste sites. CERCLA directs ATSDR to prepare toxicological profiles for hazardous substances most commonly found at facilities on the CERCLA National Priorities List (NPL) and that pose the most significant potential threat to human health, as determined by ATSDR and the EPA. ATSDR and DOE entered into a Memorandum of Understanding on November 4, 1992 which provided that ATSDR would prepare toxicological profiles for hazardous substances based upon ATSDR's or DOE's identification of need. The current ATSDR priority list of hazardous substances at DOE NPL sites was announced in the Federal Register on July 24, 1996 (61 FR 38451).

QUICK REFERENCE FOR HEALTH CARE PROVIDERS

Toxicological Profiles are a unique compilation of toxicological information on a given hazardous substance. Each profile reflects a comprehensive and extensive evaluation, summary, and interpretation of available toxicologic and epidemiologic information on a substance. Health care providers treating patients potentially exposed to hazardous substances will find the following information helpful for fast answers to often-asked questions.

Primary Chapters/Sections of Interest

Chapter 1: Public Health Statement: The Public Health Statement can be a useful tool for educating patients about possible exposure to a hazardous substance. It explains a substance's relevant toxicologic properties in a nontechnical, question-and-answer format, and it includes a review of the general health effects observed following exposure.

Chapter 2: Relevance to Public Health: The Relevance to Public Health Section evaluates, interprets, and assesses the significance of toxicity data to human health.

Chapter 3: Health Effects: Specific health effects of a given hazardous compound are reported by type of health effect (death, systemic, immunologic, reproductive), by route of exposure, and by length of exposure (acute, intermediate, and chronic). In addition, both human and animal studies are reported in this section.

NOTE: Not all health effects reported in this section are necessarily observed in the clinical setting. Please refer to the Public Health Statement to identify general health effects observed following exposure.

Pediatrics: Four new sections have been added to each Toxicological Profile to address child health issues:

Section 1.6 How Can (Chemical X) Affect Children?
Section 1.7 How Can Families Reduce the Risk of Exposure to (Chemical X)?
Section 3.8 Children's Susceptibility
Section 6.6 Exposures of Children

Other Sections of Interest:

Section 3.9 Biomarkers of Exposure and Effect
Section 3.12 Methods for Reducing Toxic Effects

ATSDR Information Center

Phone: 1-888-42-ATSDR or (404) 498-0110 ***Fax:*** (770) 488-4178
E-mail: atsdric@cdc.gov ***Internet***: http://www.atsdr.cdc.gov

The following additional material can be ordered through the ATSDR Information Center:

Case Studies in Environmental Medicine: Taking an Exposure History—The importance of taking an exposure history and how to conduct one are described, and an example of a thorough exposure history is provided. Other case studies of interest include *Reproductive and Developmental*

Hazards; Skin Lesions and Environmental Exposures; Cholinesterase-Inhibiting Pesticide Toxicity; and numerous chemical-specific case studies.

Managing Hazardous Materials Incidents is a three-volume set of recommendations for on-scene (prehospital) and hospital medical management of patients exposed during a hazardous materials incident. Volumes I and II are planning guides to assist first responders and hospital emergency department personnel in planning for incidents that involve hazardous materials. Volume III—*Medical Management Guidelines for Acute Chemical Exposures*—is a guide for health care professionals treating patients exposed to hazardous materials.

Fact Sheets (ToxFAQs) provide answers to frequently asked questions about toxic substances.

Other Agencies and Organizations

The National Center for Environmental Health (NCEH) focuses on preventing or controlling disease, injury, and disability related to the interactions between people and their environment outside the workplace. Contact: NCEH, Mailstop F-29, 4770 Buford Highway, NE, Atlanta, GA 30341-3724 • Phone: 770-488-7000 • FAX: 770-488-7015.

The National Institute for Occupational Safety and Health (NIOSH) conducts research on occupational diseases and injuries, responds to requests for assistance by investigating problems of health and safety in the workplace, recommends standards to the Occupational Safety and Health Administration (OSHA) and the Mine Safety and Health Administration (MSHA), and trains professionals in occupational safety and health. Contact: NIOSH, 200 Independence Avenue, SW, Washington, DC 20201 • Phone: 800-356-4674 or NIOSH Technical Information Branch, Robert A. Taft Laboratory, Mailstop C-19, 4676 Columbia Parkway, Cincinnati, OH 45226-1998 • Phone: 800-35-NIOSH.

The National Institute of Environmental Health Sciences (NIEHS) is the principal federal agency for biomedical research on the effects of chemical, physical, and biologic environmental agents on human health and well-being. Contact: NIEHS, PO Box 12233, 104 T.W. Alexander Drive, Research Triangle Park, NC 27709 • Phone: 919-541-3212.

Radiation Emergency Assistance Center/Training Site (REAC/TS) provides support to the U.S. Department of Energy, the World Health Organization, and the International Atomic Energy Agency in the medical management of radiation accidents. A 24-hour emergency response program at the Oak Ridge Institute for Science and Education (ORISE), REAC/TS trains, consults, or assists in the response to all kinds of radiation accidents. Contact: Oak Ridge Institute for Science and Education, REAC/TS, PO Box 117, MS 39, Oak Ridge, TN 37831-0117 • Phone 865-576-3131 • FAX 865-576-9522 • 24-Hour Emergency Phone 865-576-1005 (ask for REAC/TS) • e-mail: cooleyp@orau.gov • website (including emergency medical guidance): http://www.orau.gov/reacts/default.htm

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Referrals

The Association of Occupational and Environmental Clinics (AOEC) has developed a network of clinics in the United States to provide expertise in occupational and environmental issues. Contact:

AOEC, 1010 Vermont Avenue, NW, #513, Washington, DC 20005 • Phone: 202-347-4976 • FAX: 202-347-4950 • e-mail: AOEC@AOEC.ORG • Web Page: http://www.aoec.org/.

The American College of Occupational and Environmental Medicine (ACOEM) is an association of physicians and other health care providers specializing in the field of occupational and environmental medicine. Contact: ACOEM, 55 West Seegers Road, Arlington Heights, IL 60005 • Phone: 847-818-1800 • FAX: 847-818-9266.

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THE PROFILE HAS UNDERGONE THE FOLLOWING ATSDR INTERNAL REVIEWS:

1. Health Effects Review. The Health Effects Review Committee examines the health effects chapter of each profile for consistency and accuracy in interpreting health effects and classifying end points.

2. Minimal Risk Level Review. The Minimal Risk Level Workgroup considers issues relevant to substance-specific Minimal Risk Levels (MRLs), reviews the health effects database of each profile, and makes recommendations for derivation of MRLs.

3. Data Needs Review. The Research Implementation Branch reviews data needs sections to assure consistency across profiles and adherence to instructions in the Guidance.

PEER REVIEW

A peer review panel was assembled for cobalt. The panel consisted of the following members:

1. Dr. Herman Cember, C.H.P., Ph.D., PE., Adjunct Professor, School of Health Sciences, Purdue University, Lafayette, Indiana;

2. Dr. James Hansen, Ph.D., Environmental Contaminant Specialist, U.S. Fish and Wildlife Service, Spokane, WA;

3. Dr. Dominique Lison, M.D., Ph.D., Vice-Chairman of the Doctoral School in Genetics and Immunology, Catholic University of Louvain, Brussels, Belgium, and

4. Dr. Nancy Pedigo, Ph.D., Research Assistant Professor, Department of Pharmacology, University of Kentucky Medical Center, Lexington, KY.

These experts collectively have knowledge of cobalt's physical and chemical properties, toxicokinetics, key health end points, mechanisms of action, human and animal exposure, and quantification of risk to humans. All reviewers were selected in conformity with the conditions for peer review specified in Section 104(I)(13) of the Comprehensive Environmental Response, Compensation, and Liability Act, as amended.

Scientists from the Agency for Toxic Substances and Disease Registry (ATSDR) have reviewed the peer reviewers' comments and determined which comments will be included in the profile. A listing of the peer reviewers' comments not incorporated in the profile, with a brief explanation of the rationale for their exclusion, exists as part of the administrative record for this compound. A list of databases reviewed and a list of unpublished documents cited are also included in the administrative record.

The citation of the peer review panel should not be understood to imply its approval of the profile's final content. The responsibility for the content of this profile lies with the ATSDR.

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1. PUBLIC HEALTH STATEMENT

This public health statement tells you about cobalt and the effects of exposure.

The Environmental Protection Agency (EPA) identifies the most serious hazardous waste sites in the nation. These sites make up the National Priorities List (NPL) and are the sites targeted for long-term federal cleanup activities. Stable cobalt has been found in at least 426 of the 1,636 current or former NPL sites. Radioactive cobalt, as ^{60}Co, has been found in at least 13 of the 1,636 current or former NPL sites. However, the total number of NPL sites evaluated for this substance is not known. As more sites are evaluated, the sites at which cobalt is found may increase. This information is important because exposure to this substance may harm you and because these sites may be sources of exposure.

When a substance is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment. This release does not always lead to exposure. You are exposed to a substance only when you come in contact with it. You may be exposed by breathing, eating, or drinking the substance, or by skin contact. External exposure to radiation may occur from natural or man-made sources. Naturally occurring sources of radiation are cosmic radiation from space or radioactive materials in soil or building materials. Man-made sources of radioactive materials are found in consumer products, industrial equipment, atom bomb fallout, and to a smaller extent from hospital waste and nuclear reactors.

If you are exposed to cobalt, many factors determine whether you'll be harmed. These factors include the dose (how much), the duration (how long), and how you come in contact with it. You must also consider the other chemicals you're exposed to and your age, sex, diet, family traits, lifestyle, and state of health.

1.1 WHAT IS COBALT?

Cobalt is a naturally-occurring element that has properties similar to those of iron and nickel. It has an atomic number of 27. There is only one stable isotope of cobalt, which has an atomic mass number of 59. (An element may have several different forms, called isotopes, with different weights depending on the number of neutrons that it contains. The isotopes of an element, therefore, have different atomic mass numbers [number of protons and neutrons], although the atomic number [number of protons] remains the same.) However, there are many unstable or radioactive isotopes, two of which are commercially important, cobalt-60 and cobalt-57, also written as Co-60 or ^{60}Co and Co-57 or ^{57}Co, and read as cobalt sixty and cobalt fifty-seven. All isotopes of cobalt behave the same chemically and will therefore have the same chemical behavior in the environment and the same chemical effects on your body. However, isotopes have different mass numbers and the radioactive isotopes have different radioactive properties, such as their half-life and the nature of the radiation they give off. The half-life of a cobalt isotope is the time that it takes for half of that isotope to give off its radiation and change into a different isotope. After one half-life, one-half of the radioactivity is gone. After a second half-life, one-fourth of the original radioactivity is left, and so on. Radioactive isotopes are constantly changing into different isotopes by giving off radiation, a process referred to as radioactive decay. The new isotope may be a different element or the same element with a different mass.

Small amounts of cobalt are naturally found in most rocks, soil, water, plants, and animals, typically in small amounts. Cobalt is also found in meteorites. Elemental cobalt is a hard, silvery grey metal. However, cobalt is usually found in the environment combined with other elements such as oxygen, sulfur, and arsenic. Small amounts of these chemical compounds can be found in rocks, soil, plants, and animals. Cobalt is even found in water in dissolved or ionic form, typically in small amounts. (Ions are atoms, collections of atoms, or molecules containing a positive or negative electric charge.) A biochemically important cobalt compound is vitamin B_{12} or cyanocobalamin. Vitamin B_{12} is essential for good health in animals and humans. Cobalt is not currently mined in the United States, but has been mined in the past. Therefore, we

obtain cobalt and its other chemical forms from imported materials and by recycling scrap metal that contains cobalt.

Cobalt metal is usually mixed with other metals to form alloys, which are harder or more resistant to wear and corrosion. These alloys are used in a number of military and industrial applications such as aircraft engines, magnets, and grinding and cutting tools. They are also used in artificial hip and knee joints. Cobalt compounds are used as colorants in glass, ceramics, and paints, as catalysts, and as paint driers. Cobalt colorants have a characteristic blue color; however, not all cobalt compounds are blue. Cobalt compounds are also used as trace element additives in agriculture and medicine.

Cobalt can also exist in radioactive forms. A radioactive isotope of an element constantly gives off radiation, which can change it into an isotope of a different element or a different isotope of the same element. This newly formed nuclide may be stable or radioactive. This process is called radioactive decay. ^{60}Co is the most important radioisotope of cobalt. It is produced by bombarding natural cobalt, ^{59}Co, with neutrons in a nuclear reactor. ^{60}Co decays by giving off a beta ray (or electron), and is changed into a stable nuclide of nickel (atomic number 28). The half-life of ^{60}Co is 5.27 years. The decay is accompanied by the emission of high energy radiation called gamma rays. ^{60}Co is used as a source of gamma rays for sterilizing medical equipment and consumer products, radiation therapy for treating cancer patients, and for manufacturing plastics. ^{60}Co has also been used for food irradiation; depending on the radiation dose, this process may be used to sterilize food, destroy pathogens, extend the shelf-life of food, disinfest fruits and grain, delay ripening, and retard sprouting (e.g., potatoes and onions). ^{57}Co is used in medical and scientific research and has a half-life of 272 days. ^{57}Co undergoes a decay process called electron capture to form a stable isotope of iron (^{57}Fe). Another important cobalt isotope, ^{58}Co, is produced when nickel is exposed to a source of neutrons. Since nickel is used in nuclear reactors, ^{58}Co may be unintentionally produced and appear as a contaminant in cooling water released by nuclear reactors. ^{58}Co also decays by electron capture, forming another stable isotope of iron (^{58}Fe). ^{60}Co may be similarly produced from cobalt alloys in nuclear reactors and

released as a contaminant in cooling water. ^{58}Co has a half-life of 71 days and gives off beta and gamma radiation in the decay process.

Quantities of radioactive cobalt are normally measured in units of radioactivity (curies or becquerels) rather than in units of mass (grams). The becquerel (Bq) is a new international unit, and the curie (Ci) is the traditional unit; both are currently used. A becquerel is the amount of radioactive material in which 1 atom transforms every second, and a curie is the amount of radioactive material in which 37 billion atoms transform every second. For an overview of basic radiation physics, chemistry, and biology see Appendix D of this profile. For more information on radiation, see the *ATSDR Toxicological Profile for Ionizing Radiation.*

To learn more about the properties and uses of cobalt, see Chapters 4 and 5.

1.2 WHAT HAPPENS TO COBALT WHEN IT ENTERS THE ENVIRONMENT?

Cobalt may enter the environment from both natural sources and human activities. Cobalt occurs naturally in soil, rock, air, water, plants, and animals. It may enter air and water, and settle on land from windblown dust, seawater spray, volcanic eruptions, and forest fires and may additionally get into surface water from runoff and leaching when rainwater washes through soil and rock containing cobalt. Soils near ore deposits, phosphate rocks, or ore smelting facilities, and soils contaminated by airport traffic, highway traffic, or other industrial pollution may contain high concentrations of cobalt. Small amounts of cobalt may be released into the atmosphere from coal-fired power plants and incinerators, vehicular exhaust, industrial activities relating to the mining and processing of cobalt-containing ores, and the production and use of cobalt alloys and chemicals. ^{58}Co and ^{60}Co may be released to the environment as a result of nuclear accidents (i.e, Chernobyl), radioactive waste dumping in the sea or from radioactive waste landfills, and nuclear power plant operations.

Cobalt cannot be destroyed in the environment. It can only change its form or become attached or separated from particles. Cobalt released from power plants and other combustion processes

is usually attached to very small particles. Cobalt contained in windborne soil is generally found in larger particles than those released from power plants. These large particles settle to the ground or are washed out of the air by rain. Cobalt that is attached to very small particles may stay in the air for many days. Cobalt released into water may stick to particles in the water column or to the sediment at the bottom of the body of water into which it was released, or remain in the water column in ionic form. The specific fate of cobalt will depend on many factors such as the chemistry of the water and sediment at a site as well as the cobalt concentration and water flow. Cobalt deposited on soil is often strongly attached to soil particles and therefore would not travel very far into the ground. However, the form of the cobalt and the nature of the soil at a particular site will affect how far cobalt will penetrate into the soil. Both in soil and sediment, the amount of cobalt that is mobile will increase under more acidic conditions. Ultimately, most cobalt ends up in the soil or sediment.

Plants can accumulate very small amounts of cobalt from the soil, especially in the parts of the plant that you eat most often, such as the fruit, grain, and seeds. While animals that eat these plants will accumulate cobalt, cobalt is not known to biomagnify (produce increasingly higher concentrations) up the food chain. Therefore, vegetables, fruits, fish, and meat that you consume will generally not contain high amounts of cobalt. Cobalt is an essential element, required for good health in animals and humans, and therefore, it is important that foodstuffs contain adequate quantities of cobalt.

^{60}Co and ^{58}Co are moderately short-lived, manufactured radioactive isotopes that are produced in nuclear reactors. Although these isotopes are not produced by nuclear fission, small amounts of these radioisotopes are also produced by the neutron interaction with the structural materials found in the reactor of nuclear plants, and are produced during the routine operation of nuclear plants. Small amounts may be released to the environment as contaminants in cooling water or in radioactive waste. Since these isotopes are not fission products, they are not produced in nuclear weapons testing and are not associated with nuclear fallout. In the environment, radioactive isotopes of cobalt will behave chemically like stable cobalt. However, ^{60}Co and ^{58}Co

will also undergo radioactive decay according to their respective half-lives, 5.27 years and 71 days.

For more information about what happens to cobalt in the environment, see Chapter 6.

1.3 HOW MIGHT I BE EXPOSED TO COBALT?

Cobalt is widely dispersed in the environment in low concentrations. You may be exposed to small amounts of cobalt by breathing air, drinking water, and eating food containing it. Children may also be exposed to cobalt by eating dirt. You may also be exposed by skin contact with soil, water, cobalt alloys, or other substances that contain cobalt. Analytical methods used by scientists to determine the levels of cobalt in the environment generally do not determine the specific chemical form of cobalt present. Therefore, we do not always know the chemical form of cobalt to which a person may be exposed. Similarly, we do not know what forms of cobalt are present at hazardous waste sites. Some forms of cobalt may be insoluble or so tightly attached to particles or embedded in minerals that they are not taken up by plants and animals. Other forms of cobalt that are weakly attached to particles may be taken up by plants and animals.

The concentration of cobalt in soil varies widely, generally ranging from about 1 to 40 ppm (1 ppm=1 part of cobalt in a million parts of soil by weight), with an average level of 7 ppm. Soils containing less than about 3 ppm of cobalt are considered cobalt-deficient because plants growing in them do not have sufficient cobalt to meet the dietary requirements of cattle and sheep. Such cobalt-deficient soils are found in some areas in the southeast and northeast parts of the United States. On the other hand, soils near cobalt-containing mineral deposits, mining and smelting facilities, or industries manufacturing or using cobalt alloys or chemicals may contain much higher levels of cobalt.

Usually, the air contains very small amounts of cobalt, less than 2 nanograms (1 nanogram=one-billionth part of a gram) per cubic meter (ng/m^3). The amount of cobalt that you breathe in a day

is much less than what you consume in food and water. You may breathe in higher levels of cobalt in dust in areas near cobalt-related industries or near certain hazardous waste sites.

The concentration of cobalt in surface and groundwater in the United States is generally low—between 1 and 10 parts of cobalt in 1 billion parts of water (ppb) in populated areas; concentration may be hundreds or thousands times higher in areas that are rich in cobalt-containing minerals or in areas near mining or smelting operations. In most drinking water, cobalt levels are less than 1–2 ppb.

For most people, food is the largest source of cobalt intake. The average person consumes about 11 micrograms of cobalt a day in their diet. Included in this food is vitamin B_{12}, which is found in meat and diary products. The recommended daily intake of vitamin B_{12} is 6 micrograms (1 microgram=one-millionth part of a gram).

You may also be exposed to higher levels of cobalt if you work in metal mining, smelting, and refining, in industries that make or use cutting or grinding tools, or in other industries that produce or use cobalt metal and cobalt compounds. If good industrial hygiene is practiced, such as the use of exhaust systems in the workplace, exposure can be reduced to safe levels. Industrial exposure results mainly from breathing cobalt-containing dust.

When we speak of exposure to ^{60}Co, we are interested in exposure to the radiation given off by this isotope, primarily the gamma rays. The general population is rarely exposed to this radiation unless a person is undergoing radiation therapy. However, workers at nuclear facilities, irradiation facilities, or nuclear waste storage sites may be exposed to ^{60}Co or ^{58}Co. Exposures to radiation at these facilities are regulated and carefully monitored and controlled.

You can find more information on how you may be exposed to cobalt in Chapter 6.

1.4 HOW CAN COBALT ENTER AND LEAVE MY BODY?

Cobalt can enter your body when you breathe in air containing cobalt dust, when you drink water that contains cobalt, when you eat food that contains cobalt, or when your skin touches materials that contain cobalt. If you breathe in air that contains cobalt dust, the amount of inhaled cobalt that stays in your lungs depends on the size of the dust particles. The amount that is then absorbed into your blood depends on how well the particles dissolve. If the particles dissolve easily, then it is easier for the cobalt to pass into your blood from the particles in your lungs. If the particles dissolve slowly, then they will remain in your lungs longer. Some of the particles will leave your lungs as they normally clean themselves out. Some of the particles will be swallowed into your stomach. The most likely way you will be exposed to excess cobalt is by eating contaminated food or drinking contaminated water. Levels of cobalt normally found in the environment, however, are not high enough to result in excess amounts of cobalt in food or water. The amount of cobalt that is absorbed into your body from food or water depends on many things including your state of health, the amount you eat or drink, and the number of days, weeks, or years you eat foods or drink fluids containing cobalt. If you do not have enough iron in your body, the body may absorb more cobalt from the foods you eat. Once cobalt enters your body, it is distributed into all tissues, but mainly into the liver, kidney, and bones. After cobalt is breathed in or eaten, some of it leaves the body quickly in the feces. The rest is absorbed into the blood and then into the tissues throughout the body. The absorbed cobalt leaves the body slowly, mainly in the urine. Studies have shown that cobalt does not readily enter the body through normal skin, but it can if the skin has been cut.

Further information on how cobalt can enter or leave your body can be found in Chapter 3.

1.5 HOW CAN COBALT AFFECT MY HEALTH?

To protect the public from the harmful effects of toxic chemicals and to find ways to treat people who have been harmed, scientists use many tests.

One way to see if a chemical will hurt people is to learn how the chemical is absorbed, used, and released by the body. In the case of a radioactive chemical, it is also important to gather information concerning the radiation dose and dose rate to the body. For some chemicals, animal testing may be necessary. Animal testing may also be used to identify health effects such as cancer or birth defects. Without laboratory animals, scientists would lose a basic method to get information needed to make wise decisions to protect public health. Scientists have the responsibility to treat research animals with care and compassion. Laws today protect the welfare of research animals, and scientists must comply with strict animal care guidelines.

Cobalt has both beneficial and harmful effects on human health. Cobalt is beneficial for humans because it is part of vitamin B_{12}, which is essential to maintain human health. Cobalt (0.16–1.0 mg cobalt/kg of body weight) has also been used as a treatment for anemia (less than normal number of red blood cells), including in pregnant women, because it causes red blood cells to be produced. Cobalt also increases red blood cell production in healthy people, but only at very high exposure levels. Cobalt is also essential for the health of various animals, such as cattle and sheep. Exposure of humans and animals to levels of cobalt normally found in the environment is not harmful.

When too much cobalt is taken into your body, however, harmful health effects can occur. Workers who breathed air containing 0.038 mg cobalt/m^3 (about 100,000 times the concentration normally found in ambient air) for 6 hours had trouble breathing. Serious effects on the lungs, including asthma, pneumonia, and wheezing, have been found in people exposed to 0.005 mg cobalt/m^3 while working with hard metal, a cobalt-tungsten carbide alloy. People exposed to 0.007 mg cobalt/m^3 at work have also developed allergies to cobalt that resulted in asthma and skin rashes. The general public, however, is not likely to be exposed to the same type or amount of cobalt dust that caused these effects in workers.

In the 1960s, some breweries added cobalt salts to beer to stabilize the foam (resulting in exposures of 0.04–0.14 mg cobalt/kg). Some people who drank excessive amounts of beer (8–25 pints/day) experienced serious effects on the heart. In some cases, these effects resulted in

death. Nausea and vomiting were usually reported before the effects on the heart were noticed. Cobalt is no longer added to beer so you will not be exposed from this source. The effects on the heart, however, may have also been due to the fact that the beer-drinkers had protein-poor diets and may have already had heart damage from alcohol abuse. Effects on the heart were not seen, however, in people with anemia treated with up to 1 mg cobalt/kg, or in pregnant women with anemia treated with 0.6 mg cobalt/kg. Effects on the thyroid were found in people exposed to 0.5 mg cobalt/kg for a few weeks. Vision problems were found in one man following treatment with 1.3 mg cobalt/kg for 6 weeks, but this effect has not been seen in other human or animal studies.

Being exposed to radioactive cobalt may be very dangerous to your health. If you come near radioactive cobalt, cells in your body can become damaged from gamma rays that can penetrate your entire body, even if you do not touch the radioactive cobalt. Radiation from radioactive cobalt can also damage cells in your body if you eat, drink, breathe, or touch anything that contains radioactive cobalt. The amount of damage depends on the amount of radiation to which you are exposed, which is related to the amount of activity in the radioactive material and the length of time that you are exposed. Most of the information regarding health effects from exposure to radiation comes from exposures for only short time periods. The risk of damage from exposure to very low levels of radiation for long time periods is not known. If you are exposed to enough radiation, you might experience a reduction in white blood cell number, which could lower your resistance to infections. Your skin might blister or burn, and you may lose hair from the exposed areas. This happens to cancer patients treated with large amounts of radiation to kill cancer. Cells in your reproductive system could become damaged and cause temporary sterility. Exposure to lower levels of radiation might cause nausea, and higher levels can cause vomiting, diarrhea, bleeding, coma, and even death. Exposure to radiation can also cause changes in the genetic materials within cells and may result in the development of some types of cancer.

Studies in animals suggest that exposure to high amounts of nonradioactive cobalt during pregnancy might affect the health of the developing fetus. Birth defects, however, have not been

found in children born to mothers who were treated with cobalt for anemia during pregnancy. The doses of cobalt used in the animal studies were much higher than the amounts of cobalt to which humans would normally be exposed.

Nonradioactive cobalt has not been found to cause cancer in humans or in animals following exposure in the food or water. Cancer has been shown, however, in animals who breathed cobalt or when cobalt was placed directly into the muscle or under the skin. Based on the animal data, the International Agency for Research on Cancer (IARC) has determined that cobalt is possibly carcinogenic to humans.

Much of our knowledge of cobalt toxicity is based on animal studies. Cobalt is essential for the growth and development of certain animals, such as cows and sheep. Short-term exposure of rats to high levels of cobalt in the air results in death and lung damage. Longer-term exposure of rats, guinea pigs, hamsters, and pigs to lower levels of cobalt in the air results in lung damage and an increase in red blood cells. Short-term exposure of rats to high levels of cobalt in the food or drinking water results in effects on the blood, liver, kidneys, and heart. Longer-term exposure of rats, mice, and guinea pigs to lower levels of cobalt in the food or drinking water results in effects on the same tissues (heart, liver, kidneys, and blood) as well as the testes, and also causes effects on behavior. Sores were seen on the skin of guinea pigs following skin contact with cobalt for 18 days. Generally, cobalt compounds that dissolve easily in water are more harmful than those that are hard to dissolve in water.

Much of what we know about the effects of radioactive cobalt comes from studies in animals. The greatest danger of radiation seen in animals is the risk to the developing animal, with even moderate amounts of radiation causing changes in the fetus. High radiation doses in animals have also been shown to cause temporary or permanent sterility and changes in the lungs, which affected the animals' breathing. The blood of exposed animals has lower numbers of white blood cells, the cells that aid in resistance to infections, and red blood cells, which carry oxygen in the blood. Radioactive cobalt exposures in animals have also caused genetic damage to cells, cancer, and even death.

More information on how cobalt can affect your health can be found in Chapter 3.

1.6 HOW CAN COBALT AFFECT CHILDREN?

This section discusses potential health effects from exposures during the period from conception to maturity at 18 years of age in humans.

Children can be exposed to cobalt in the same ways as adults. In addition, cobalt may be transferred from the pregnant mother to the fetus or from the mother to the infant in the breast milk. Children may be affected by cobalt the same ways as adults. Studies in animals have suggested that children may absorb more cobalt from foods and liquids containing cobalt than adults. Babies exposed to radiation while in their mother's womb are believed to be much more sensitive to the effects of radiation than adults.

1.7 HOW CAN FAMILIES REDUCE THE RISK OF EXPOSURE TO COBALT

If your doctor finds that you have been exposed to significant amounts of cobalt, ask whether your children might also be exposed. Your doctor might need to ask your state health department to investigate.

Since cobalt is naturally found in the environment, people cannot avoid being exposed to it. However, the relatively low concentrations present do not warrant any immediate steps to reduce exposure. If you are accidentally exposed to large amounts of cobalt, consult a physician immediately.

Children living near waste sites containing cobalt are likely to be exposed to higher environmental levels of cobalt through breathing, touching soil, and eating contaminated soil. Some children eat a lot of dirt. You should discourage your children from eating dirt. Make sure

they wash their hands frequently and before eating. Discourage your children from putting their hands in their mouths or hand-to-mouth activity.

You are unlikely to be exposed to high levels of radioactive cobalt unless you are exposed as part of a radiotherapy treatment, there is an accident involving a cobalt sterilization or radiotherapy unit, or there is an accidental release from a nuclear power plant. In such cases, follow the advice of public health officials who will publish guidelines for reducing exposure to radioactive material when necessary. Workers who work near or with radioactive cobalt should follow the workplace safety guidelines of their institution carefully to reduce the risk of accidental irradiation.

1.8 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO COBALT?

We have reliable tests that can measure cobalt in the urine and the blood for periods up to a few days after exposure. The amount of cobalt in your blood or urine can be used to estimate how much cobalt you had taken into your body. The tests are not able to accurately predict potential health effects following exposure to cobalt.

It is difficult to determine whether a person has been exposed only to external radiation from radioactive cobalt unless the radiation dose was rather large. Health professionals examining people who have health problems similar to those resulting from radiation exposure would need to rely on additional information in order to establish if such people had been near a source of radioactivity. It is relatively easy to determine whether a person has been internally exposed to radioactive cobalt, as discussed in Chapter 7. More information on medical tests can be found in Chapters 3 and 7.

1.9 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

The federal government develops regulations and recommendations to protect public health. Regulations can be enforced by law. Federal agencies that develop regulations for toxic substances include the Environmental Protection Agency (EPA), the Occupational Safety and Health Administration (OSHA), the Food and Drug Administration (FDA), and the U.S. Nuclear Regulatory Commission (USNRC).

Recommendations provide valuable guidelines to protect public health but cannot be enforced by law. Federal organizations that develop recommendations for toxic substances include the Agency for Toxic Substances and Disease Registry (ATSDR), the National Institute for Occupational Safety and Health (NIOSH), and the FDA.

Regulations and recommendations can be expressed in not-to-exceed levels in air, water, soil, or food that are usually based on levels that affect animals; they are then adjusted to help protect people. Sometimes these not-to-exceed levels differ among federal organizations because of different exposure times (an 8-hour workday or a 24-hour day), the use of different animal studies, or other factors.

Recommendations and regulations are also periodically updated as more information becomes available. For the most current information, check with the federal agency or organization that provides it. Some regulations and recommendations for cobalt include the following:

EPA requires that the federal government be notified if more than 1,000 pounds of cobalt (as the bromide, formate, and sulfamate compounds) are released into the environment in a 24-hour period. OSHA regulates levels of nonradioactive cobalt in workplace air. The limit for an 8-hour workday, 40-hour workweek is an average of 0.1 mg/m^3. The USNRC and the Department of Energy (DOE) regulate occupational exposures as well as exposures of the general public to radioactive cobalt.

1.10 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns, please contact your community or state health or environmental quality department, your regional Nuclear Regulatory Commission office, or contact ATSDR at the address and phone number below.

ATSDR can also tell you the location of occupational and environmental health clinics. These clinics specialize in recognizing, evaluating, and treating illnesses resulting from exposure to hazardous substances.

Toxicological profiles are also available on-line at www.atsdr.cdc.gov and on CD-ROM. You may request a copy of the ATSDR ToxProfiles CD-ROM by calling the information and technical assistance toll-free number at 1-888-42ATSDR (1-888-422-8737), by email at atsdric@cdc.gov, or by writing to:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE
Mailstop F-32
Atlanta, GA 30333
Fax: 1-770-488-4178

For-profit organizations may request a copy of final profiles from the following:

National Technical Information Service (NTIS)
5285 Port Royal Road
Springfield, VA 22161
Phone: 1-800-553-6847 or 1-703-605-6000
Web site: http://www.ntis.gov/

2. RELEVANCE TO PUBLIC HEALTH

2.1 BACKGROUND AND ENVIRONMENTAL EXPOSURES TO COBALT IN THE UNITED STATES

Cobalt is a naturally-occurring element that has properties similar to those of iron and nickel. The largest use of metallic cobalt is in superalloys that are used in gas turbine aircraft engines. Cobalt compounds are used as pigments in glass, ceramics, and paints; as catalysts in the petroleum industry; as paint driers; and as trace element additives in agriculture and medicine.

Cobalt may be released to the environment by human activities, as well by weathering of rocks and soil. The primary anthropogenic sources of cobalt in the environment are from the burning of fossil fuels, application of cobalt-containing sludge or phosphate fertilizers, mining and smelting of cobalt-containing ores, processing of cobalt-containing alloys, and industries that use or process cobalt compounds. Cobalt released to the atmosphere is deposited onto soil or water surfaces by wet and dry deposition. In soils, cobalt generally has low mobility and strong adsorption. However its mobility increases in moist, acidic soils. In water, cobalt largely partitions to sediment and to suspended solids in the water column; however, the amount that is adsorbed to suspended solids is highly variable.

Exposure of the general population to cobalt occurs through inhalation of ambient air and ingestion of food and drinking water. In general, intake from food sources is much greater than from drinking water and air. The cobalt intake in food has been estimated to be 5.0–40.0 μg/day. Occupational exposure to cobalt occurs for workers in the hard metal industry (tool production, grinding, etc.) and in industries such as coal mining, metal mining, smelting and refining, cobalt dye painters, and the cobalt chemical production industry. The concentrations of cobalt in the air of hard metal manufacturing, welding, and grinding factories may range from 1 to 300 $\mu g/m^3$, compared to normal atmospheric levels of 0.4–2.0 ng/m^3.

While there is only one stable isotope of cobalt, ^{59}Co, there are many radioactive isotopes of cobalt. Of these radioactive isotopes, two are commercially important, ^{60}Co and ^{57}Co. ^{60}Co is produced by irradiating ^{59}Co with thermal neutrons in a nuclear reactor, and is used as a source of gamma rays for sterilizing medical equipment or consumer products, food irradiation, radiation therapy for treating cancer

patients, and for manufacturing plastics. The general population is not significantly exposed to radioactive forms of cobalt. Cancer patients being treated with radiation therapy may be exposed to gamma rays from a ^{60}Co source; however, the effects of external exposure to gamma radiation is not unique to ^{60}Co, but is similar for all gamma-emitting radionuclides. Workers at nuclear facilities and nuclear waste storage sites may be exposed to potentially high levels of radioactive cobalt.

2.2 SUMMARY OF HEALTH EFFECTS

As a component of cyanocobalmin (vitamin B_{12}), cobalt is essential in the body; the Recommended Dietary Allowance of vitamin B_{12} is 2.4 μg/day, which contains 0.1 μg of cobalt. Cobalt has been identified in most tissues of the body, with the highest concentrations found in the liver.

Following inhalation exposure to cobalt-containing particles, the primary target of exposure is the respiratory tract. Occupational exposure of humans to cobalt metal or cobalt-containing hard metal have reported primarily respiratory effects, including decreased pulmonary function, asthma, interstitial lung disease, wheezing, and dyspnea; these effects were reported at occupational exposure levels ranging from 0.015–0.13 mg Co/m^3. Animal studies have further identified respiratory tract hyperplasia, pulmonary fibrosis, and emphysema as sensitive effects of inhaled cobalt on respiratory tissues. Many of the respiratory tract effects are believed to be the result of the generation of oxidants and free radicals by the cobalt ion. In particular, hard metal (a tungsten carbide/cobalt alloy) is a potent generator of free electrons, resulting in the generation of active oxygen species. However, some of the respiratory effects, such as cobalt-induced asthma, are likely the result of immunosensitization to cobalt.

Other sensitive targets of cobalt inhalation in humans include effects on the thyroid and allergic dermatitis, manifesting as eczema and erythema; it is believed that the allergic dermatitis is due, at least in part, to concurrent dermal exposure and the development of immunosensitization to cobalt.

Adequate chronic studies of the oral toxicity of cobalt or cobalt compounds in humans and animals are not presently available. The most sensitive endpoint following oral exposure to cobalt in humans appears to be an increase in erythrocyte numbers (polycythemia). This effect has been observed in both normal subjects and in patients who were anemic as a result of being anephric. However, treatment of pregnant women with cobalt did not prevent the reduction in hematocrit and hemoglobin levels often found during

pregnancy. Exposure of humans to beer containing cobalt as a foam stabilizer resulted in severe effects on the cardiovascular system, including cardiomyopathy and death, as well as gastrointestinal effects (nausea, vomiting) and hepatic necrosis. However, the subjects in these studies were alcoholics, and it is not known what effect excessive alcohol consumption may have played in the development of the observed effects.

Following dermal exposure, the most commonly observed effect is dermatitis, as demonstrated by a large number of human studies. Using patch tests and intradermal injections, it has been demonstrated that the dermatitis is probably caused by an allergic reaction to cobalt, with the cobalt ion functioning as a hapten.

Available studies of the carcinogenic effects of cobalt in occupationally-exposed humans have reported mixed results, with both positive and negative results. Lifetime inhalation of cobalt sulfate resulted in increased tumor incidences in both rats and mice; NTP reported that there was some evidence of carcinogenicity in male Fischer 344 (F344) strain rats, and clear evidence of carcinogenicity in female F344 strain rats and male and female B6C3F1 strain mice following inhalation exposure. Oral data on the carcinogenic effects of cobalt and cobalt compounds are not available. IRIS does not report a cancer classification for cobalt or cobalt compounds. IARC has classified cobalt and cobalt compounds as *possibly carcinogenic to humans (Group 2B)*.

A more detailed discussion of the health effects of cobalt and cobalt compounds is presented in Chapter 3. An enhanced discussion of sensitive end points of stable cobalt toxicity is presented below.

Respiratory Effects. The primary effects of cobalt on respiratory tissues are seen following inhalation exposure, and include diminished pulmonary function, increased frequency of cough, respiratory inflammation, and fibrosis; reported effect levels in occupationally-exposed humans have ranged from 0.015–0.13 mg Co/m^3. Animal studies have further identified respiratory tract hyperplasia, pulmonary fibrosis, and emphysema as sensitive effects of cobalt on respiratory tissues. A number of these effects are believed to be the result of the generation of oxidants and free radicals by the cobalt ion. *In vitro* exposure to soluble cobalt increases indices of oxidative stress, including diminished levels of reduced glutathione, increased levels of oxidized glutathione, activation of the hexose monophosphate shunt, and free-radical-induced DNA damage. Cobalt exposure also results in sensitization of the immune system, which may result in asthmatic attacks following inhalation of cobalt in sensitized individuals.

Hard metal is a metal alloy with a tungsten carbide and cobalt matrix. It is used to make cutting tools because of its hardness and resistance to high temperature. Exposure to hard metal has been shown in a number of studies to cause respiratory effects, including respiratory irritation, diminished pulmonary function, asthma, and fibrosis, at exposure levels lower than those that would produce similar effects following exposure to cobalt metal alone (0.007–0.14 mg Co/m^3). Studies suggest that cobalt and not tungsten carbide is the probable causative agent for the respiratory effects observed in hard metal workers (see Section 3.5). A mechanism by which hard metal may exert its effects has been proposed by a group of Belgian researchers. In this proposed mechanism, tungsten carbide, which is a very good conductor of electrons, facilitates the oxidation of cobalt metal to ionic cobalt (presumably Co^{2+}) by transferring electrons from the cobalt atom to molecular oxygen adjacent to the tungsten carbide molecule. The result is an increased solubility of cobalt, relative to cobalt metal alone, and the generation of active oxygen species. *In vitro* evidence for this mechanism includes the ability of hard metal particles, but neither cobalt nor tungsten carbide alone at the same concentrations, to generate oxidant species and cause lipid peroxidation. Hard metal particles have also been shown to increase the levels of inducible nitric oxide synthase (iNOS), a gene responsive to oxidant stress.

Hematological Effects. Exposure to cobalt and cobalt compounds has been demonstrated to increase levels of erythrocytes and hemoglobin in both humans and animals. Davis and Fields reported increased (~16–20%) erythrocyte levels in six of six healthy men exposed orally to cobalt chloride (~1 mg Co/kg-day); erythrocyte counts returned to normal 9–15 days after cessation of cobalt administration. Increased levels of erythrocytes were also found following oral treatment of anephric patients (with resulting anemia) with cobalt chloride. The increase in hemoglobin resulted in a decreased need for blood transfusions. Treatment of pregnant women for 90 days with cobalt chloride, however, did not prevent the reduction in hematocrit and hemoglobin levels often found during pregnancy.

Increased levels of hemoglobin were observed in rats and guinea pigs, but not in dogs, exposed to cobalt hydrocarbonyl by inhalation. Polycythemia was reported in rats, but not mice, exposed to airborne cobalt sulfate. Significantly increased erythrocyte (polycythemia), hematocrit, and hemoglobin levels were found in animals treated orally with cobalt as either a single dose or with longer-term exposure. Of particular note is an 8-week study in rats, which reported dose- and time-related increases in erythrocyte number following oral administration of cobalt chloride.

The mechanisms regarding cobalt-induced polycythemia are not well understood. Cobalt is thought to inhibit heme synthesis *in vivo* by acting upon at least two different sites in the biosynthetic pathway. This inhibitory activity might result in the formation of cobalt protoporphyrin rather than heme. Cobalt treatment also stimulates heme oxidation in many organs, due to the induction of heme oxygenase. Conversely, cobalt acts, through a mechanism believed to involve a heme-containing protein, to increase erythropoietin, which stimulates the production of red blood cells. The regulatory mechanisms behind this apparent dichotomy have not been fully elucidated.

Cardiac Effects. Cardiomyopathy has been reported in both humans and animals following exposure to cobalt. Occupational exposure of humans to cobalt-containing dust, either as cobalt metal or as hard metal, is believed to result in cardiomyopathy characterized by functional effects on the ventricles and enlargement of the heart, but the exposure levels associated with cardiac effects of inhaled cobalt in humans have not been determined. Rats exposed to 11.4 mg Co/m^3 for 13 weeks developed a mild cardiomyopathy; however, rats and mice exposed to 1.14 mg Co/m^3 for 2 years showed no signs of cardiomyopathy.

Beer-cobalt cardiomyopathy was observed in people who heavily consumed beer that contained cobalt sulfate as a foam stabilizer. The beer drinkers ingested an average of 0.04 mg Co/kg/day to 0.14 mg Co/kg/day for a period of years. The cardiomyopathy was characterized by sinus tachycardia, left ventricular failure, cardiogenic shock, diminished myocardial compliance, absence of a myocardial response to exercise or catecholamine, enlarged heart, pericardial effusion, and extensive intracellular changes (changes in the myofibers, mitochondria, glycogen, and lipids). The beer-cobalt cardiomyopathy appeared to be similar to alcoholic cardiomyopathy and beriberi, but the onset of beer-cobalt cardiomyopathy was very abrupt. It should be noted, however, that the cardiomyopathy may have also been due to the fact that the beer-drinkers had protein-poor diets and may have had prior cardiac damage from alcohol abuse. Studies in animals, and limited human data, have supported this possibility, as much greater oral exposure levels (on the order of 8-30 mg Co/kg-day) are necessary to induce cardiac effects.

The mechanism for cobalt-induced cardiomyopathy is not presently understood. Exposure to cobalt may result in accumulation in cardiac tissues, and is thought to stimulate carotid-body chemoreceptors, mimicking the action of hypoxia. Microscopic analysis of the hearts of those with beer-cobalt cardiomyopathy revealed fragmentation and degeneration of myofibers and aggregates of abnormal mitochondria. These mitochondrial changes are indicative of disturbances in energy production or

utilization may possibly be related to cobalt effects on lipoic acid. Cobalt irreversibly chelates lipoic acids under aerobic conditions. Lipoic acid is a required cofactor for oxidative decarboxylation of pyruvate to acetyl CoA and of α-ketoglutarate to succinate. In the myocardium of rats treated with cobalt, oxidation of pyruvate or fatty acids is impaired. However, the relative contribution of these mechanisms to the cardiac effects of cobalt has not been determined.

Dermal Effects. Dermatitis is a common result of dermal exposure to cobalt in humans. Using patch tests and intradermal injections, it has been demonstrated that the dermatitis is probably caused by an allergic reaction to cobalt. Exposure levels associated with the development of dermatitis have not been identified. It appears that cobalt metal may be a more potent allergen than some cobalt salts, as Nielsen et al. demonstrated that daily repeated exposure to aqueous cobalt salts did not result in hand eczema in patients known to have cobalt allergy. In animals, scabs and denuded areas were found after six doses of 51.75 mg Co/kg (5 days/week) as dicobalt octacarbonyl were applied to the shaved abdomens (uncovered area of approximately 50 cm^2) of guinea pigs. By the 11th dose, the lesions disappeared. No adverse effects were observed in vehicle controls (methyl ethyl ketone). It is not known whether or not a similar reaction would result from metallic or inorganic forms of cobalt.

Immunological Effects. Exposure of humans to cobalt by the inhalation and dermal routes has resulted in sensitization to cobalt. Exposure to inhaled cobalt chloride aerosols can precipitate an asthmatic attack in sensitized individuals, believed to be the result of an allergic reaction within the lungs. Similarly, the dermatitis seen in dermally-exposed subjects is likely the result of an allergic reaction, with cobalt functioning as a hapten. IgE and IgA antibodies specific to cobalt have been reported in humans. There is evidence that cobalt sensitivity in humans may also be regulated by T-lymphocytes; a human helper T-lymphocyte cell line specific for cobalt ($CoCl_2$) has been established. Cobalt may also interact directly with immunologic proteins, such as antibodies or Fc receptors, to result in immunosensitization. *In vitro*, cobalt(III) has been shown to reduce the proliferation of both B and T lymphocytes, as well as the release of the cytokines IL-2, IL-6, and IFN-Gamma. Interrelationships exist between nickel and cobalt sensitization, with cross-reactivity between the two having been reported in several studies.

Radioactive Cobalt. Exposure to radioisotopes of cobalt is also a human health concern. Energy released by radioactive isotopes can result in significant damage to living cells. Both ^{60}Co and ^{57}Co emit beta particles and gamma rays, which may ionize molecules within cells penetrated by these emissions and result in tissue damage and disruption of cellular function. The most important exposure route for

radioisotopes of cobalt is external exposure to the radiation released by the radioisotopes. It should be noted that there is nothing unique about the effects of external exposure to ^{60}Co and ^{57}Co when compared to other gamma- and beta-emitting radionuclides.

Generally, acute radiation doses below 15 rad (0.15 Gy) do not result in observable adverse health effects. At doses in the range of 15–50 rad (0.15–0.5 Gy), subclinical responses such as chromosomal breaks and transient changes in formed elements of the blood may be seen in sensitive individuals. Symptoms of acute radiation syndrome begin to be observed at radiation doses above 50 rad, characterized by transient hematopoietic manifestations, nausea and vomiting, and moderate leukopenia at doses near 100 rad (1 Gy), progressing through more serious hematopoietic symptoms, clinical signs, and gastrointestinal symptoms with increasing dose (100–800 rad or 1–8 Gy), and usually death in persons receiving total doses ≥1,000 rad (10 Gy). Other health effects from acute or continued high-level exposure to ionizing radiation may include reproductive, developmental, and latent cancer effects.

Signs and symptoms of acute toxicity from external and internal exposure to high levels of radiation from ^{60}Co and ^{57}Co are typical of those observed in cases of high exposure to ionizing radiation in general. Depending on the radiation dose, symptoms may include those typical of acute radiation syndrome (vomiting, nausea, and diarrhea), skin and ocular lesions, neurological signs, chromosomal abnormalities, compromised immune function, and death.

Acute or repeated exposure of humans or animals to ionizing radiation (from radioisotopes of cobalt or other radioactive elements) may result in reduced male fertility, abnormal neurological development following exposure during critical stages of fetal development, and genotoxic effects such as increased frequencies of chromosomal aberrations, sister-chromatid exchanges, and micronucleus formation.

Due to the ionizing properties of radionuclides such as ^{60}Co and ^{57}Co, increased cancer risk would be expected among exposed individuals. However, studies of increased cancer risk specifically associated with exposure of humans to radioactive cobalt isotopes were not located. Similarly, studies of the carcinogenic effects of radioactive cobalt isotopes in animals were not located.

2.3 MINIMAL RISK LEVELS (MRLs)

Inhalation MRLs

- An MRL of 0.0001 mg cobalt/m^3 has been derived for chronic-duration inhalation exposure (>365 days) to cobalt.

An MRL for inhalation exposure to cobalt was derived for chronic duration only. The chronic inhalation MRL of 0.0001 mg cobalt/m^3 was based on a no-observed-adverse-effect-level (NOAEL) of 0.0053 mg cobalt/m^3 and a LOAEL of 0.0151 mg cobalt/m^3 (both NOAEL and LOAEL values were adjusted for continuous exposure prior to MRL derivation) for decreases in forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), forced expiratory flow between 25 and 75% of the FVC (MMEF), and mean peak expiratory flow rate (PEF) in diamond polishers (Nemery et al. 1992); a further discussion of the results and limitations of this study is presented in Appendix A.

The National Toxicology Program (NTP) has conducted a chronic-duration carcinogenicity study in rats and mice. Exposure of rats and mice to aerosols of cobalt (as cobalt sulfate) at concentrations ranging from 0.11 to 1.14 mg cobalt/m^3 for 2 years resulted in a spectrum of inflammatory, fibrotic, and proliferative lesions in the respiratory tract of male and female rats and mice (NTP 1998). Squamous metaplasia of the larynx occurred in rats and mice at exposure concentrations of 0.11 mg cobalt/m^3, with severity of the lesion increasing with increased exposure concentration. Hyperplastic lesions of the nasal epithelium occurred in rats at concentrations of 0.11 mg cobalt/m^3, and in mice at concentrations of 0.38 mg cobalt/m^3. Both sexes of rats had greatly increased incidences (>90% incidence) of alveolar lesions at all exposure levels, including inflammatory changes, fibrosis, and metaplasia. Similar changes were seen in mice at all exposure levels, though the changes in mice were less severe. The study in diamond polishers, being a well-conducted study in humans, was selected as the critical study for the derivation of a MRL because it examined a human population and identified a NOAEL, neither of which occurred in the NTP study. The chronic inhalation MRL was derived by adjusting the NOAEL of 0.0053 mg Co/m^3 for intermittent exposure (adjusted to 0.0013 mg/m^3 to simulate continuous exposure), and applying an uncertainty factor of 10 (for human variability). It should be noted that this MRL may not be protective for individuals already sensitive to cobalt.

An acute inhalation MRL was not derived because the threshold was not defined for human effects and animal studies reported effects that were serious and occurred at levels above those reported in the few

human studies. An acute-duration study of hard metal exposure in humans (Kusaka et al. 1986b) was not utilized for MRL derivation because the toxicity of hard metal is not directly due to cobalt metal, but rather to an interaction between cobalt metal and tungsten carbide. An intermediate-duration MRL was not derived because available studies did not examine the dose-response relationship at low doses; the chronic inhalation MRL should be protective for intermediate exposures (see Appendix A).

Oral MRLs

- An MRL of 0.01 mg Co/kg-day has been derived for intermediate-duration oral exposure (<365 days) to cobalt.

An intermediate-duration MRL of 0.01 mg Co/kg/day was derived based on a LOAEL of 1 mg cobalt/kg-day for polycythemia as reported in a study by Davis and Fields (1958). The authors exposed six men to 120 or 150 mg/day of cobalt chloride (~1 mg Co/kg/day) for up to 22 days. Exposure to cobalt resulted in the development of polycythemia in all six patients, with increases in red blood cell numbers ranging from 0.5 to 1.19 million (~16–20% increase above pre-treatment levels). Polycythemic erythrocyte counts returned to normal 9–15 days after cessation of cobalt administration. An 8–week study in rats (Stanley et al. 1947) also reported increases in erythrocyte number, with a no-observed-effect-level (NOEL) of 0.6 mg/kg-day and a lowest-observed-effect-level (LOEL) of 1 mg/kg/day. The intermediate oral MRL was derived by dividing the LOAEL of 1 mg Co/kg-day by an uncertainty factor of 100 (10 for use of a LOAEL and 10 for human variability).

Oral MRL values were not derived for acute or chronic exposure to cobalt. An acute MRL was not derived because the reported effects in animals were serious and occurred at levels above those reported in the few human oral studies. No chronic oral studies were available in animals; the chronic studies of beer-cobalt cardiomyopathy (Alexander 1969, 1972; Bonenfant et al. 1969; Morin et al. 1967, 1971; Sullivan et al. 1969) were not used because the effects were serious (death) and because the effects of concurrent alcoholism were not controlled for. Therefore, a chronic oral MRL was not derived for cobalt.

MRLs for External Exposure to Cobalt Isotopes

Two MRLs have been derived for ionizing radiation (Agency for Toxic Substances and Disease Registry 1999) and are applicable to external exposure to radioisotopes of cobalt:

- An MRL of 400 mrem (4.0 mSv) has been derived for acute-duration external exposure to ionizing radiation (14 days or less).

The acute MRL is based on results of a study by Schull et al. (1988) in which neurological effects of radiation, measured by intelligence test scores, were evaluated in children 10–11 years of age who had been exposed at critical stages of fetal development (gestation weeks 8–15) during the atomic bombing of Hiroshima and Nagasaki. When IQ scores were regressed on radiation dose estimates, IQ diminished linearly with increasing dose, resulting in an estimated decrease in IQ score of approximately 25 points per 100 rad (or 100 rem in dose equivalent) or 0.25 points/rem (25 points/Sv). To derive the MRL of 400 mrem (4.0 mSv), Agency for Toxic Substances and Disease Registry (1999) divided the dose associated with a predicted change of 0.25 IQ points/rem by an uncertainty factor of 3 (for human variability and/or the potential existence of sensitive populations). Agency for Toxic Substances and Disease Registry (1999) noted that a change in IQ points of 0.25 is less than the reported difference of 0.3 IQ points between separated and unseparated identical twins (Burt 1966).

The USNRC set a radiation exposure limit of 500 mrem (5 mSv) for pregnant working women over the full gestational period (USNRC 1991). For the critical gestational period of 8–15 weeks, Agency for Toxic Substances and Disease Registry believes that the acute MRL of 400 mrem (4 mSv) is consistent with the USNRC limit and could be applied to either acute (0–14-day) or intermediate (15–365-day) exposure periods.

- An MRL of 100 mrem/year (1.0 mSv/year) above background has been derived for chronic-duration external ionizing radiation (365 days or more).

The MRL is based on the BEIR V (1990) report that the average annual effective dose of ionizing radiation to the U.S. population is 360 mrem/year (3.6 mSv/year), a dose not expected to produce adverse noncancerous health effects. This dose is obtained mainly by naturally-occurring radiation from external sources, medical uses of radiation, and radiation from consumer products. An uncertainty factor of 3 (for human variability) was applied to the NOAEL of 360 mrem/year to derive the MRL of 100 mrem/year.

3. HEALTH EFFECTS

3.1 INTRODUCTION

The primary purpose of this chapter is to provide public health officials, physicians, toxicologists, and other interested individuals and groups with an overall perspective on the toxicology of cobalt. It contains descriptions and evaluations of toxicological studies and epidemiological investigations and provides conclusions, where possible, on the relevance of toxicity and toxicokinetic data to public health. Section 3.2 contains a discussion of the chemical toxicity of stable cobalt; radiation toxicity associated with exposure to radioactive cobalt (primarily ^{60}Co) is discussed in Section 3.3. The chemical properties of stable and radioactive cobalt isotopes are identical and are described in Chapter 4.

A glossary and list of acronyms, abbreviations, and symbols can be found at the end of this profile.

3.2 DISCUSSION OF HEALTH EFFECTS BY ROUTE OF EXPOSURE

Section 3.2 discusses the chemical toxicity of stable cobalt. Radiation toxicity resulting from exposure to radioactive cobalt is discussed in Section 3.3.

To help public health professionals and others address the needs of persons living or working near hazardous waste sites, the information in this section is organized first by route of exposure (inhalation, oral, and dermal) and then by health effect (death, systemic, immunological, neurological, reproductive, developmental, genotoxic, and carcinogenic effects). These data are discussed in terms of three exposure periods: acute (14 days or less), intermediate (15–364 days), and chronic (365 days or more).

Levels of significant exposure for each route and duration are presented in tables and illustrated in figures. The points in the figures showing no-observed-adverse-effect levels (NOAELs) or lowest-observed-adverse-effect levels (LOAELs) reflect the actual doses (levels of exposure) used in the studies. LOAELs have been classified into "less serious" or "serious" effects. "Serious" effects are those that evoke failure in a biological system and can lead to morbidity or mortality (e.g., acute respiratory distress or death). "Less serious" effects are those that are not expected to cause significant dysfunction or death,

or those whose significance to the organism is not entirely clear. ATSDR acknowledges that a considerable amount of judgment may be required in establishing whether an end point should be classified as a NOAEL, "less serious" LOAEL, or "serious" LOAEL, and that in some cases, there will be insufficient data to decide whether the effect is indicative of significant dysfunction. However, the Agency has established guidelines and policies that are used to classify these end points. ATSDR believes that there is sufficient merit in this approach to warrant an attempt at distinguishing between "less serious" and "serious" effects. The distinction between "less serious" effects and "serious" effects is considered to be important because it helps the users of the profiles to identify levels of exposure at which major health effects start to appear. LOAELs or NOAELs should also help in determining whether or not the effects vary with dose and/or duration, and place into perspective the possible significance of these effects to human health.

The significance of the exposure levels shown in the Levels of Significant Exposure (LSE) tables and figures may differ depending on the user's perspective. Public health officials and others concerned with appropriate actions to take at hazardous waste sites may want information on levels of exposure associated with more subtle effects in humans or animals (LOAELs) or exposure levels below which no adverse effects (NOAELs) have been observed. Estimates of levels posing minimal risk to humans (Minimal Risk Levels or MRLs) may be of interest to health professionals and citizens alike.

Estimates of exposure levels posing minimal risk to humans (Minimal Risk Levels or MRLs) have been made for cobalt. An MRL is defined as an estimate of daily human exposure to a substance that is likely to be without an appreciable risk of adverse effects (noncarcinogenic) over a specified duration of exposure. MRLs are derived when reliable and sufficient data exist to identify the target organ(s) of effect or the most sensitive health effect(s) for a specific duration within a given route of exposure. MRLs are based on noncancerous health effects only and do not consider carcinogenic effects. MRLs can be derived for acute, intermediate, and chronic duration exposures for inhalation and oral routes. Appropriate methodology does not exist to develop MRLs for dermal exposure.

Although methods have been established to derive these levels (Barnes and Dourson 1988; EPA 1990), uncertainties are associated with these techniques. Furthermore, ATSDR acknowledges additional uncertainties inherent in the application of the procedures to derive less than lifetime MRLs. As an example, acute inhalation MRLs may not be protective for health effects that are delayed in development or are acquired following repeated acute insults, such as hypersensitivity reactions, asthma, or chronic

bronchitis. As these kinds of health effects data become available and methods to assess levels of significant human exposure improve, these MRLs will be revised.

A User's Guide has been provided at the end of this profile (see Appendix B). This guide should aid in the interpretation of the tables and figures for Levels of Significant Exposure and the MRLs.

Studies have shown that soluble cobalt compounds are generally more acutely toxic than insoluble cobalt compounds. When expressed in terms of the cobalt ion for the sake of comparison, however, the differences in lethality values from the available studies are within an order of magnitude and therefore do not warrant presentation in separate LSE tables and figures. Therefore, data regarding both soluble and insoluble cobalt compounds are presented in Tables 3-1, 3-2, and 3-3.

3.2.1 Inhalation Exposure

3.2.1.1 Death

Conclusive evidence for human deaths related to inhalation exposure to cobalt has not been reported; however, results of several studies and case reports suggest a possible relationship between exposure and deaths from lung cancer and cardiomyopathy, respectively.

In general, available cohort studies in humans have not reported a significant increase in total mortality as a result of cobalt exposure. Several studies have noted increased mortality rates resulting from lung cancer following occupational exposure to cobalt, either as a mixture of cobalt compounds (Mur et al. 1987) or as hard metal, a metal alloy with a tungsten carbide and cobalt matrix (Lasfargues et al. 1994; Moulin et al. 1998). Fatal cases of hard metal disease (Figueroa et al. 1992; Ruokonen et al. 1996) and cardiomyopathy (Barborik and Dusek 1972) believed to have resulted from occupational cobalt exposure have also been reported. However, in the majority of these and other reported occupational studies, co-exposure to other substances was common, and was unable to be corrected for in the analysis.

Cobalt inhalation can be lethal in animals if exposure is sufficiently high or prolonged. The acute LC^{50} for a 30-minute inhalation exposure in rats was 165 mg cobalt/m^3 as cobalt hydrocarbonyl (Palmes et al. 1959). Exposure to 9 mg cobalt/m^3 as cobalt hydrocarbonyl for 6 hours/day, 5 days/week for

3 months resulted in 16 deaths out of 75 rats (Palmes et al. 1959). Death was reported in rats and mice exposed to 19 mg cobalt/m^3 (but not 1.9 mg cobalt/m^3) as cobalt sulfate over 16 days, but exposure to 11.4 mg cobalt/m^3 over 13 weeks was lethal only to mice and not to rats (Bucher et al. 1990; NTP 1991). Exposure to 1.14 mg cobalt/m^3 as cobalt sulfate for 104 weeks resulted in no increase in mortality in rats and mice of either sex (Bucher et al. 1999; NTP 1998). Lethal levels for each species and duration category are recorded in Table 3-1 and plotted in Figure 3-1.

3.2.1.2 Systemic Effects

No data were located regarding dermal effects in humans or animals after inhalation exposure to stable cobalt. Inhalation of stable cobalt by humans and/or animals resulted in respiratory, cardiovascular, hematological, hepatic, renal, endocrine, ocular, and body weight effects. For each effect, the highest NOAEL values and all reliable LOAEL values for each species and duration category are reported in Table 3-1 and plotted in Figure 3-1.

Respiratory Effects. Hard metal is a metal alloy with a tungsten carbide and cobalt matrix. It is used to make cutting tools because of its hardness and resistance to high temperature. Studies (Davison et al. 1983; Harding 1950) suggest that cobalt (and not tungsten carbide) is the probable causative agent for the respiratory effects observed in hard metal workers (see Section 3.6).

The effects of chronic occupational exposure to cobalt and cobalt compounds on the respiratory system in humans are well-documented. These effects include respiratory irritation, diminished pulmonary function, wheezing, asthma, pneumonia, and fibrosis and occurred at exposure levels ranging from 0.007 to 0.893 mg cobalt/m^3 (exposure from 2 to 17 years) (Anttila et al. 1986; Davison et al. 1983; Demedts et al. 1984a, 1984b; Deng et al. 1991; Gennart and Lauwerys 1990; Gheysens et al. 1985; Hahtola et al. 2000; Hartung et al. 1982; Kusaka et al. 1986a, 1986b, 1996a, 1996b; Nemery et al. 1992; Raffn et al. 1988; Rastogi et al. 1991; Ruokonen et al. 1996; Shirakawa et al. 1988, 1989; Sprince et al. 1988; Sundaram et al. 2001; Swennen et al. 1993; Tabatowski et al. 1988; Van Cutsem et al. 1987; Zanelli et al. 1994). These effects have been observed in workers employed in cobalt refineries, as well as hard metal workers, diamond polishers, and ceramic dish painters (painting with cobalt blue dye).

Table 3-1 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Inhalation

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/m³)	LOAEL Less Serious (mg/m³)	LOAEL Serious (mg/m³)	Reference Chemical Form
	ACUTE EXPOSURE						
	Systemic						
1	Human	6 hr	Resp		0.038 (bronchial irritation, reduced FVC)		Kusaka et al. 1986b Hard Metal
2	Rat SD-Jcl	5 hr	Resp	2.72			Kyono et al. 1992 Metal
3	Rat SD-Jcl	4 d	Resp		2.12 M (Slight damage to respiratory tissues, assessed by electron microscopy)		Kyono et al. 1992 Metal
4	Rat	30 min	Resp	7	26 (edema)	83 (severe edema)	Palmes et al. 1959 Hydrocarbonyl
	INTERMEDIATE EXPOSURE						
	Death						
5	Rat	16 d 5 d/wk 6 hr/d		1.9		19 (2/5 males died)	NTP 1991 Sulfate

Table 3-1 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Inhalation (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/m³)	LOAEL Less Serious (mg/m³)		LOAEL Serious (mg/m³)		Reference Chemical Form
6	Mouse	13 wk 5 d/wk 6 hr/d		3.8			11.4	(2 males died)	NTP 1991 Sulfate
	Systemic								
7	Rat	13 wk 5 d/wk 6 hr/d	Resp		0.11	(laryngial squamous metaplasia and polyps)	0.38	(chronic inflammation of larynx)	NTP 1991 Sulfate
			Cardio		11.4	(increase in severity of cardiomyopathy)			
			Hemato		1.14[b] M	(polycythemia)			
			Renal	11.4					
			Bd Wt		11.4	(15% lower body weight in males)			
8	Rat	3 mo 5 d/wk 7 h/d	Resp		9	(lung inflamm)			Palmes et al. 1959 Hydrocarbonyl
			Hemato		9[b]	(10% increase in hemoglobin)			
			Bd Wt	9					

Table 3-1 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Inhalation (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/m³)	LOAEL Less Serious (mg/m³)	LOAEL Serious (mg/m³)	Reference Chemical Form
9	Mouse	16d 5 d/wk 6 hr/d	Resp	0.2	1.9 (respiratory tract inflammation)	19 (necrosis)	NTP 1991 Sulfate
			Cardio	76			
			Gastro	76			
			Musc/skel	76			
			Hepatic			19 (necrosis)	
			Renal	76			
			Dermal	76			

Table 3-1 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Inhalation (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/m³)	LOAEL Less Serious (mg/m³)	LOAEL Serious (mg/m³)	Reference Chemical Form
10	Mouse	13 wk 5 d/wk 6 hr/d	Resp		0.11 (larynx metaplasia)	3.8 M (acute inflam of nose) 1.14[c] F (acute inflam of nose)	NTP 1991 Sulfate
			Gastro	11.4			
			Hemato	11.4			
			Musc/skel	11.4			
			Hepatic	11.4			
			Renal	11.4			
			Dermal	11.4			
			Bd Wt		11.4 (13-20% decrease in body weight)		
11	Gn Pig (Hartley)	66 d	Resp			2.4 F (Increased lung weight, increased retention of lavage fluid)	Camner et al. 1993 Chloride

Table 3-1 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Inhalation (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/m³)	LOAEL Less Serious (mg/m³)	LOAEL Serious (mg/m³)	Reference Chemical Form
12	Gn Pig	3 mo 5 d/wk 7 h/d	Hemato		9[b] (5% increase in hemoglobin)		Palmes et al. 1959 Hydrocarbonyl
13	Dog	3 mo 3d/wk 7h/d	Hemato	9			Palmes et al. 1959 Hydrocarbonyl
			Bd Wt		9 (wt loss)		
14	Rabbit	4 mo 5 d/wk 6 h/d	Resp		0.4 (moderate lung inflammation)	2 (severe lung inflammation)	Johansson et al. 1987
15	Rabbit	4 mo	Resp	0.5 M			Johansson et al. 1991 Chloride
16	Rabbit	4 mo	Resp		0.6 M (Histologic alterations in pulmonary tissue; altered BAL parameters)		Johansson et al. 1992 Chloride

Table 3-1 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Inhalation (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/m³)	LOAEL Less Serious (mg/m³)	LOAEL Serious (mg/m³)	Reference Chemical Form
17	Pig	3 mo 5d/wk 6hr/d	Resp		0.1	(decr compliance)	Kerfoot 1975 Metal
			Cardio		0.1	(EKG changes)	
			Hepatic	1			
			Renal	1			
			Bd Wt		0.1	(decr wt gain)	
	Immuno/ Lymphoret						
18	Rat	16 d 5 d/wk 6 hr/d			19	(necrosis of thymus)	NTP 1991 Sulfate
19	Mouse	13 wk 5 d/wk 6 hr/d			11.4	(lymph node hyperplasia)	NTP 1991 Sulfate
	Neurological						
20	Rat	16 d 5 d/wk 6 hr/d			19	(congestion of vessels in brain)	NTP 1991 Sulfate
21	Mouse	16 d 5 d/wk 6 hr/d			19	(congestion of vessels in brain)	NTP 1991 Sulfate

Table 3-1 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Inhalation (continued)

Key to[a] figure	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/m³)	LOAEL Less Serious (mg/m³)	LOAEL Serious (mg/m³)	Reference Chemical Form
	Reproductive						
22	Rat	16 d 5 d/wk 6 hr/d				19 M (testes atrophy)	NTP 1991 Sulfate
23	Mouse	13 wk 5 d/wk 6 hr/d			1.14 M (decreased sperm motility)	11.4 (testes atrophy- increased length estrous cycle)	NTP 1991 Sulfate
	CHRONIC EXPOSURE						
	Systemic						
24	Human	occup (occup)	Resp	0.0175			Deng et al. 1991 Metal
25	Human	occup (occup)	Resp		0.1355 (Decreased FEV1 and FVC ~10%; increased cough, sputum, dyspnea)		Gennart and Lauwerys 1990 Hard-Metal
26	Human	occup (occup)	Resp	0.0053[d]	0.0151 (Decreased FEV1, FVC increased cough and upper airway irritation)		Nemery et al. 1992 Metal
27	Human	occup (occup)	Endocr		0.05 F (Decreased thyroid volume; increases in T4 and FT4I levels)		Prescott et al. 1992 Zinc-Silicate Dye

Table 3-1 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Inhalation (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/m³)	LOAEL Less Serious (mg/m³)		LOAEL Serious (mg/m³)		Reference Chemical Form
28	Human	occup	Resp				0.007	(asthma)	Shirakawa et al. 1988 Hard Metal
29	Human	occup	Resp				0.051	(interst lung dis)	Sprince et al. 1988 Hard Metal
30	Human	8 yr (occup)	Resp		0.125	(Dyspnoea and wheezing)			Swennen et al. 1993 Metal
			Hemato		0.125	(Decreased red cell counts ~5%; decreased total hemoglobin ~4%)			
			Endocr		0.125	(Slight (~7%) decrease in T3 levels)			
			Dermal		0.125	(Eczema and erythema)			
31	Rat (Fischer- 344)	104 wk	Resp				0.11	(Hyper- and metaplasia of respiratory tract tissues; pulmonary fibrosis)	NTP 1998 Sulfate
32	Mouse (B6C3F1)	104 wk	Resp		0.11	(Laryngial metaplasia)			NTP 1998 Sulfate

Table 3-1 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Inhalation (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/m³)	LOAEL Less Serious (mg/m³)	LOAEL Serious (mg/m³)	Reference Chemical Form
33	Hamster	life 5d/wk 7h/d	Resp			7.9 (emphysema)	Wehner et al. 1977 Oxide
			Bd Wt	7.9			
	Immuno/ Lymphoret						
34	Human	occup			0.007 (sensitization)		Shirakawa et al. 1986a Hard Metal
35	Human	8 yr (occup)			0.125 (Increased white cell count by 19%)		Swennen et al. 1993 Metal
	Cancer						
36	Rat (Fischer- 344)	104 wk				1.14 M (alveoloar/bronchiolar neoplasms) 1.14 F (pheochromocytoma) 0.38 F (alveoloar/bronchiolar neoplasms)	NTP 1998 Sulfate

Table 3-1 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Inhalation (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/m³)	LOAEL Less Serious (mg/m³)	LOAEL Serious (mg/m³)	Reference Chemical Form
37	Mouse (B6C3F1)	104 wk				1.14 M (Combined alveolar/bronchiolar adenoma/carcinoma) 0.38 F (Combined alveolar/bronchiolar adenoma/carcinoma)	NTP 1998 Sulfate

[a] The number corresponds to entries in Figure 3-1.

[b] An increase in hemoglobin or red blood cells (polycythemia) is not necessarily considered an adverse effect.

[c] Differences in levels of health effects and cancer effect between males and females are not indicated in Figure 3-1. Where such differences exist, only the levels of effect for the most sensitive gender are presented.

[d] Used to derive a chronic inhalation Minimal Risk level (MRL) of 0.0001 mg Co/m3., dose adjusted for intermittent exposure, and divided by an uncertainty factor of 10 (for human variability).

Bd = body weight; Cardio = cardiovascular, d = day(s); Derm = dermal; Endocr = endocrine; F = female; Gastro = gastrointestinal; Gn Pig = guinea pig; Hemato = hematological; hr = hour(s); LOAEL = lowest-observed-adverse-effect level; M = male; mo = month(s); Musc/skel = muscular/skeletal; NOAEL = no-observed-adverse-effect level; (occup) = occupational; Resp = respiratory; wk = week(s); yr = year(s).

Figure 3-1. Levels of Significant Exposure to Cobalt - Chemical Toxicity - *Inhalation*

Acute (≤14 days)

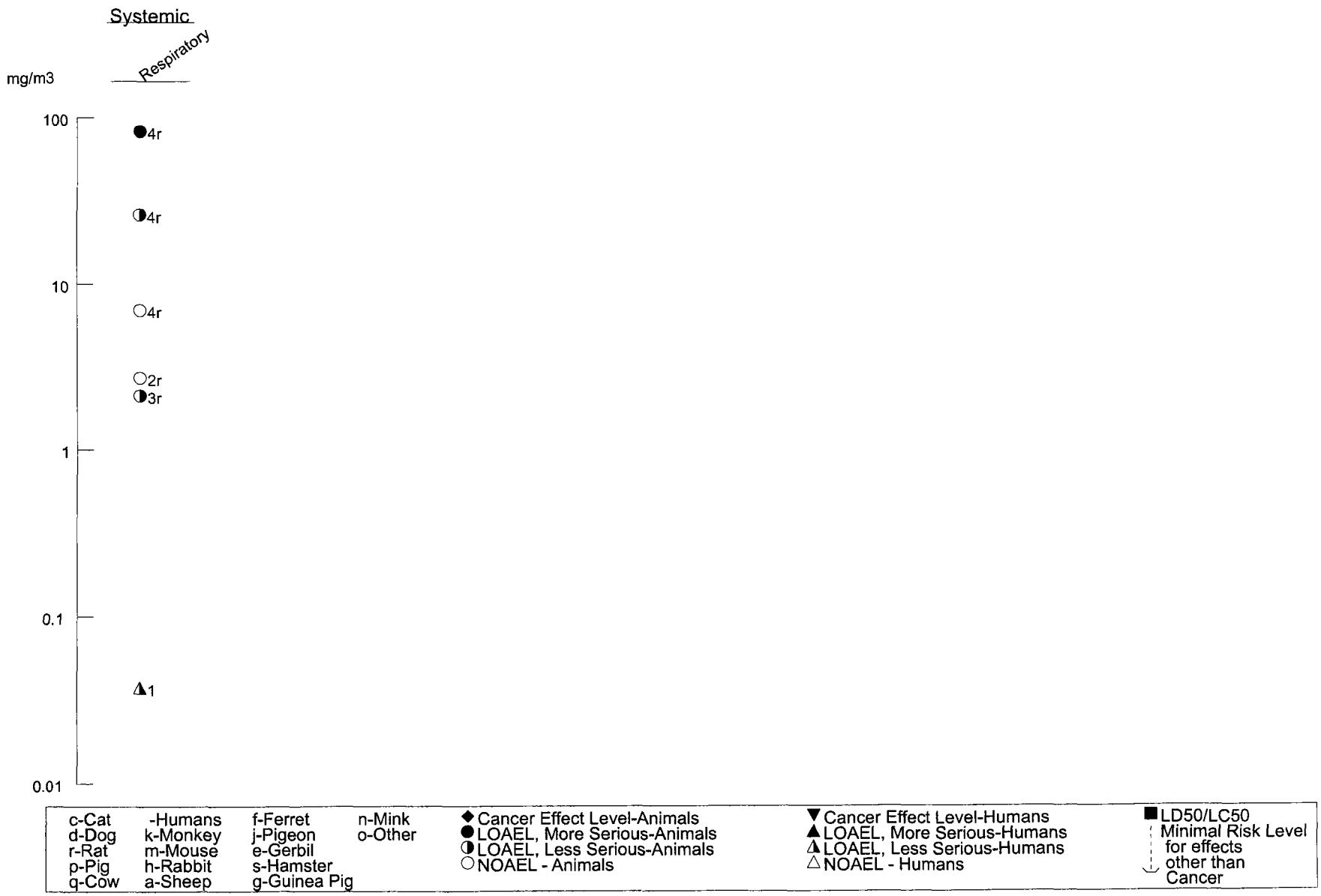


Figure 3-1. Levels of Significant Exposure to Cobalt - Chemical Toxicity - Inhalation (*Continued*)

Intermediate (15-364 days)

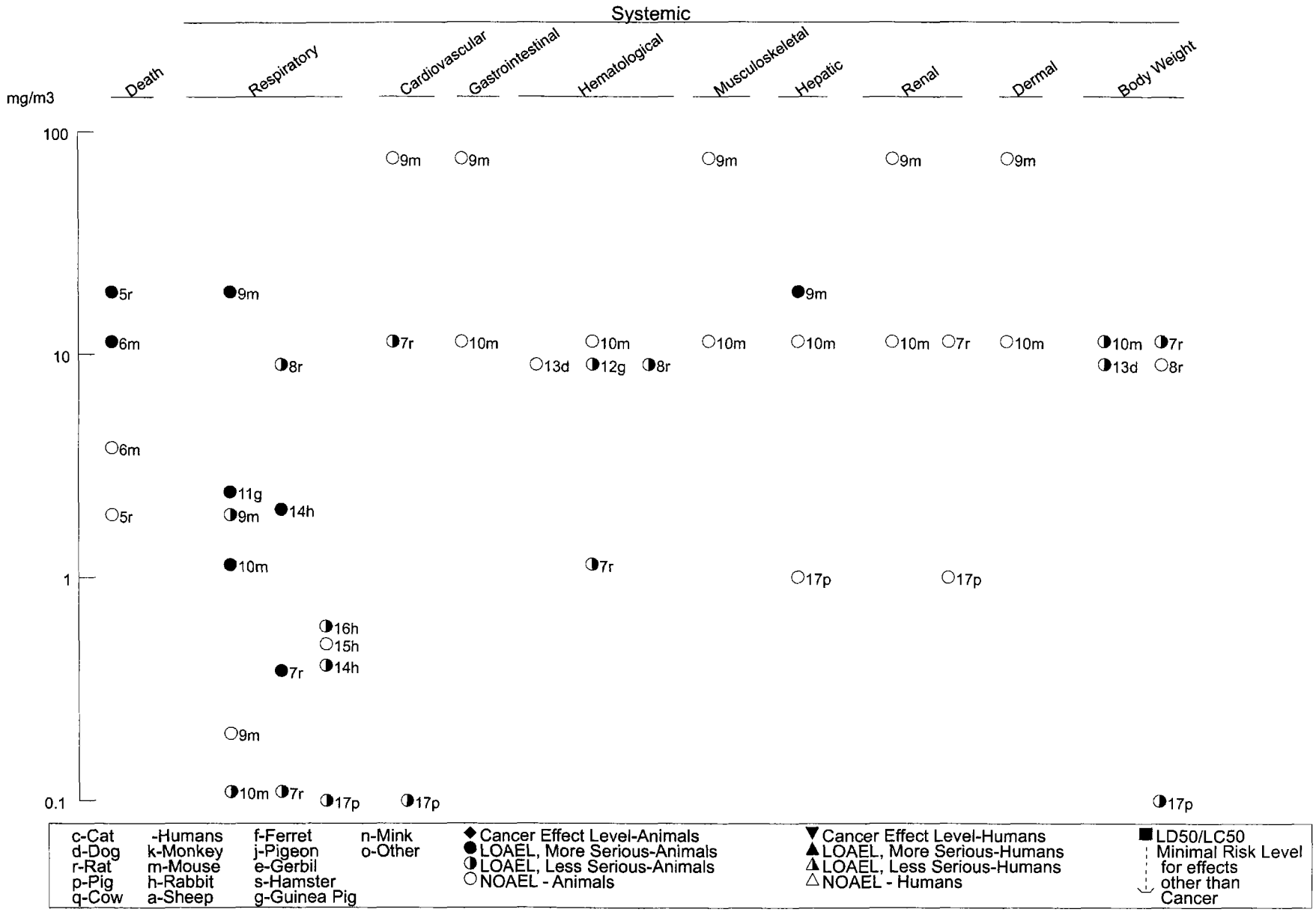


Figure 3-1. Levels of Significant Exposure to Cobalt - Chemical Toxicity - Inhalation (*Continued*)

Intermediate (15-364 days)

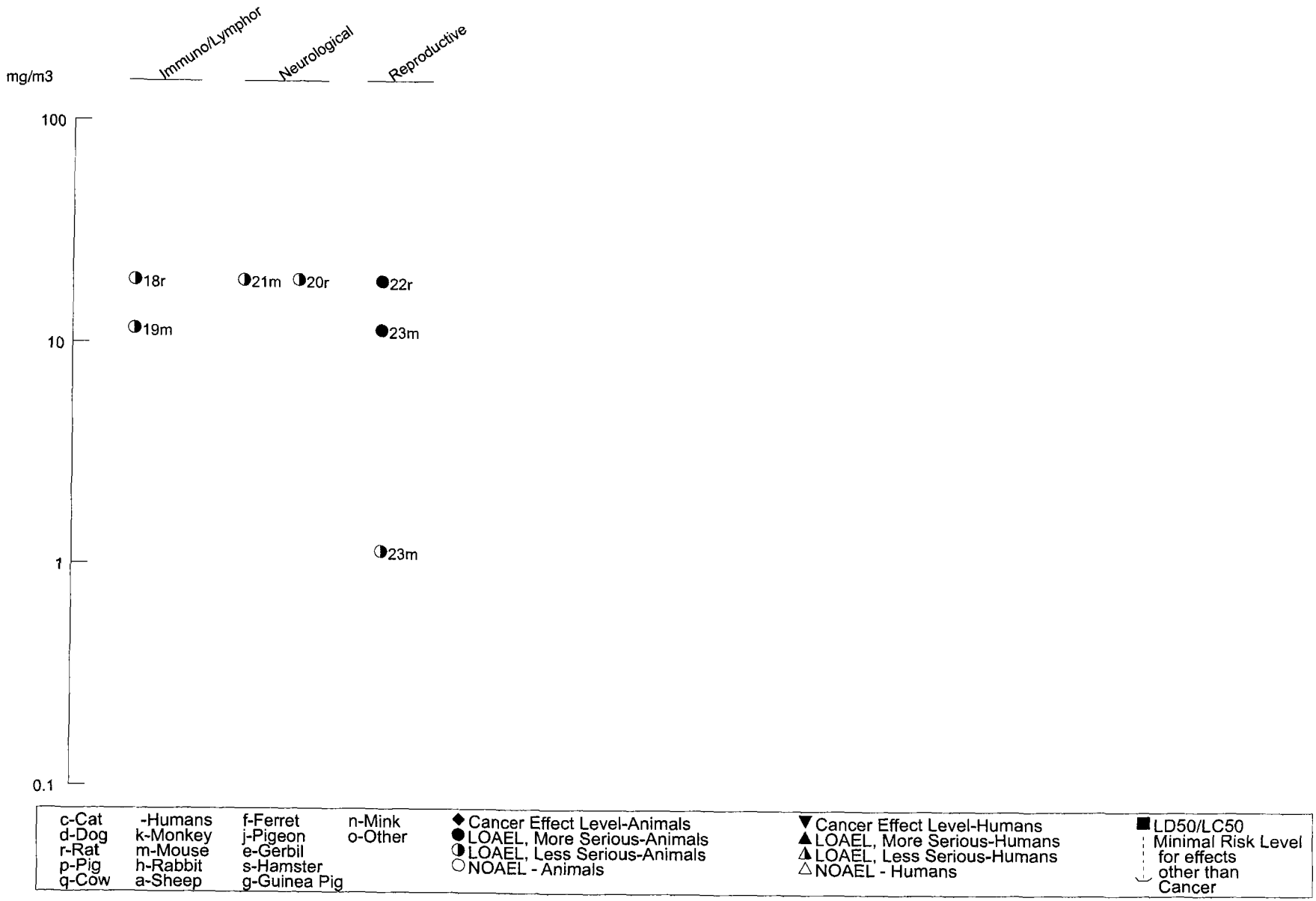
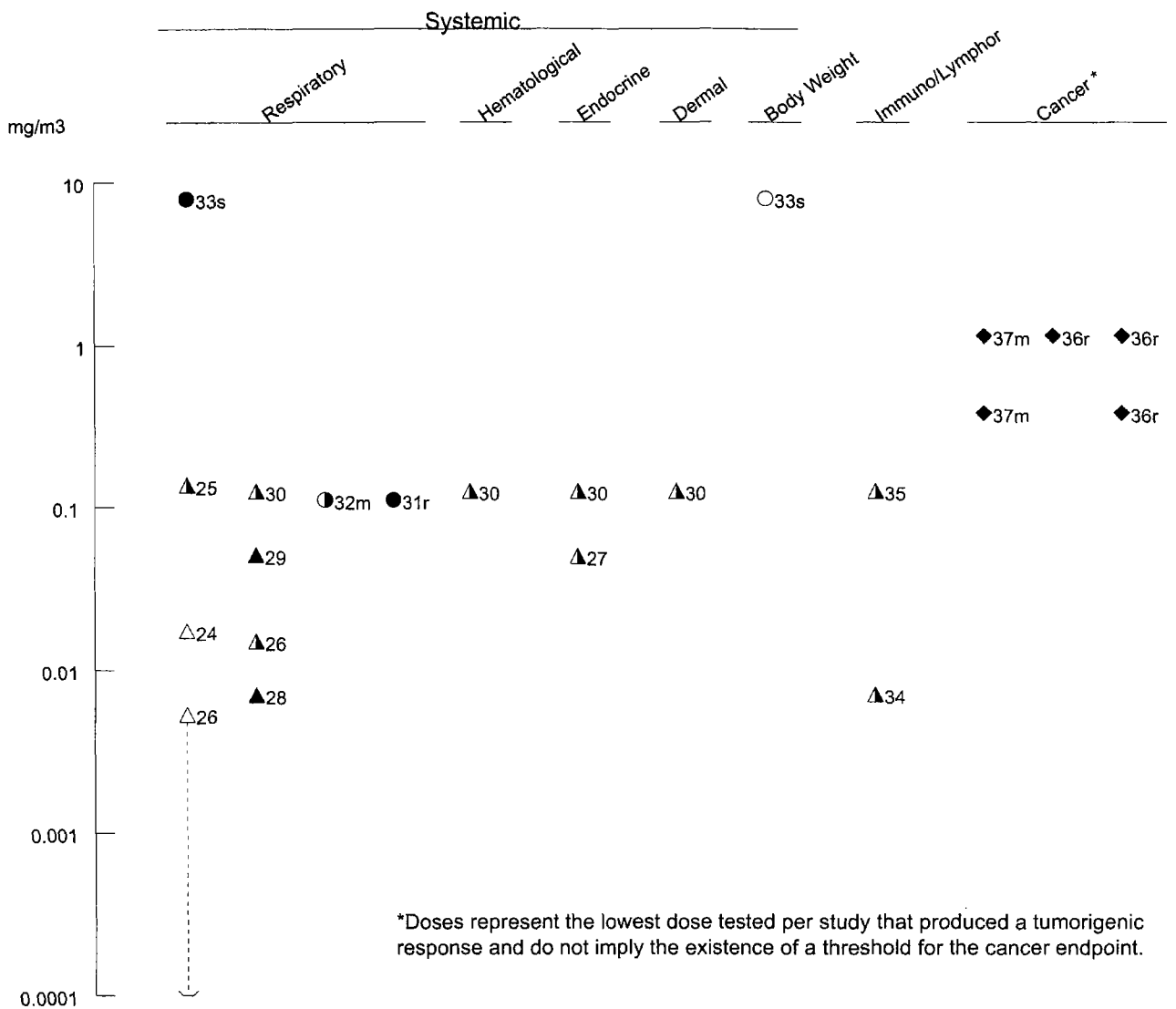


Figure 3-1. Levels of Significant Exposure to Cobalt - Chemical Toxicity - Inhalation (*Continued*)

Chronic (≥365 days)



*Doses represent the lowest dose tested per study that produced a tumorigenic response and do not imply the existence of a threshold for the cancer endpoint.

c-Cat	-Humans	f-Ferret	n-Mink	◆ Cancer Effect Level-Animals	▼ Cancer Effect Level-Humans	■ LD50/LC50
d-Dog	k-Monkey	j-Pigeon	o-Other	● LOAEL, More Serious-Animals	▲ LOAEL, More Serious-Humans	Minimal Risk Level for effects other than Cancer
r-Rat	m-Mouse	e-Gerbil		◑ LOAEL, Less Serious-Animals	◮ LOAEL, Less Serious-Humans	
p-Pig	h-Rabbit	s-Hamster		○ NOAEL - Animals	△ NOAEL - Humans	
q-Cow	a-Sheep	g-Guinea Pig				

Kusaka et al. (1986b) described an acute exposure of 15 healthy young men to atmospheres of hard metal dust containing 0.038 mg cobalt/m^3 for 6 hours. Forced vital capacity (FVC) was reduced, but no dose-response relation could be discerned. By contrast, 42 workers occupationally exposed to hard metal showed no decrease in ventilatory function at 0.085 mg cobalt/m^3, but significant changes in FEV_1 (forced expiratory volume in 1 second) at 0.126 mg cobalt/m^3 (Kusaka et al. 1986b). Several other studies of hard metal workers have shown respiratory effects, including decreased ventilatory function, wheezing, asthma, and fibrosis (Kusaka et al. 1996a, 1996b; Ruokonen et al. 1996; Zanelli et al. 1994), but have had less complete reports of exposure.

Swennen et al. (1993) performed a cross-sectional study on 82 workers in a cobalt refinery. Workers were examined for cobalt in blood and urine, a number of erythropoietic variables, thyroid metabolism, pulmonary function, skin lesions, and several serum enzymes. The concentrations of cobalt in blood and in urine after the shift were significantly correlated with those in air. Workers exposed to airborne cobalt metal, salts, or oxides (mean concentration 0.125 mg/m^3, range 0.001–7.7 mg/m^3) showed an increased ($p<0.05$) prevalence of dyspnea and wheezing and had significantly more skin lesions (eczema, erythema) than control workers. A dose-effect relation was found between the reduction of the FEV_1 and the intensity of the current exposure to cobalt, as assessed by measurement of cobalt in blood, air, or urine.

Gennart and Lauwerys (1990) examined the ventilatory functions of 48 diamond polishing workers, relative to 23 control workers. Exposure occurred mainly in one of two rooms, with mean airborne concentrations of 0.0152 and 0.1355 mg cobalt/m^3; control subjects worked in other areas of the facilities, where no exposure to cobalt occurred. Significant decreases in ventilatory function were found in the exposed workers relative to the control workers. Duration of exposure played a significant factor, with no significant differences in workers who had been exposed for ≤5 years; reported decreases in ventilatory function were noted in workers exposed for > 5 years. Inhalation exposure to cobalt salts (exposure levels not reported) among glass bangle workers resulted in decreases in decreased ventilatory function, generally restrictive in nature, relative to controls (Rastogi et al. 1991).

Nemery et al. (1992) conducted a cross-sectional study of cobalt exposure and respiratory effects in diamond polishers. Exposure occurred mainly from the generation of airborne cobalt resulting from the use of cobalt-containing polishing discs. The study groups were composed of 194 polishers working in 10 different workshops, and were divided into control, low-, and high-exposure groups. The low-exposure group (n=102) was exposed to an average of 0.0053 mg cobalt/m^3, based on personal sampling

measurements, while the exposure level for the high dose group (n=92) was 0.0151 mg cobalt/m^3; there was considerable overlap in the total range of concentrations for the low- and high-exposure groups. Workers in the high-exposure group were more likely than those in the other groups to complain about respiratory symptoms; the prevalence of eye, nose, and throat irritation and cough, as well as the fraction of these symptoms related to work, were significantly increased in the high-exposure group. Workers in the high-exposure group also had significantly reduced lung function compared to controls and low-exposure group workers, as assessed by FVC, FEV_1, MMEF (forced expiratory flow between 25 and 75% of the FVC) and mean PEF (peak expiratory flow rate). Results in the low-exposure group did not differ from controls. Based on the NOAEL of 0.0053 mg cobalt/m^3 for decreased ventilatory function in exposed workers, a chronic inhalation MRL of 1x10-4 mg cobalt/m^3 was calculated as described in footnote (d) in Table 3-1. It should be noted that this MRL value may not be protective for some hypersensitive individuals.

As with exposures in humans, exposures of animals to cobalt-containing aerosols have resulted in pronounced respiratory effects. Animals exposed to aerosols of cobalt oxides and cobalt sulfate developed respiratory effects that varied in severity with exposure level and duration. A single 30-minute exposure of rats to relatively high levels (26–236 mg cobalt/m^3 as cobalt hydrocarbonyl) resulted in congestion, edema, and hemorrhage of the lung (Palmes et al. 1959). Prolonged exposure (3–4 months) of rats and rabbits to mixed cobalt oxides (0.4–9 mg cobalt/m^3) resulted in lesions in the alveolar region of the respiratory tract characterized histologically by nodular accumulation of Type II epithelial cells, accumulations of enlarged highly vacuolated macrophages, interstitial inflammation, and fibrosis (Johansson et al. 1984, 1987, 1991, 1992; Kyono et al. 1992; Palmes et al. 1959). In at least one instance, the lesions appeared to regress when exposure was terminated (Palmes et al. 1959). Guinea pigs sensitized to cobalt by repeated dermal application and then exposed to 2.4 mg cobalt/m^3 as cobalt chloride showed pulmonary inflammatory changes (altered BAL fluid recovery, increased neutrophils and eosinophils in the recovered BAL fluid) that were different than those in exposed animals not sensitized to cobalt (Camner et al. 1993). Decreased lung compliance was found in pigs exposed to 0.1 mg cobalt/m^3 as cobalt dust for 3 months (Kerfoot 1975). Lifetime exposure of hamsters to 7.9 mg cobalt/m^3 as cobalt oxide resulted in emphysema (Wehner et al. 1977).

Necrosis and inflammation of the respiratory tract epithelium (nasal turbinates, larynx, trachea, bronchioles) were reported in rats exposed to 19 mg cobalt/m^3 and mice exposed to 1.9 mg cobalt/m^3 or greater as cobalt sulfate over 16 days (Bucher et al. 1990; NTP 1991). Exposure of rats and mice to

cobalt as cobalt sulfate for 13 weeks resulted in adverse effects on all parts of the respiratory tract, with the larynx being the most sensitive part (Bucher et al. 1990; NTP 1991). At concentrations of ≥0.11 mg cobalt/m^3, rats and mice developed squamous metaplasia of the larynx. Histiocytic infiltrates in the lung were also reported at similar levels in both the rats and mice. In rats, chronic inflammation of the larynx was found at ≥0.38 mg cobalt/m^3, and more severe effects on the nose, larynx, and lung were reported at higher exposures. In mice, acute inflammation of the nose was found at ≥1.14 mg cobalt/m^3, and more severe effects on the nose, larynx, and lung were reported at higher exposures. Exposure of rats and mice to aerosols of cobalt (as cobalt sulfate) at concentrations from 0.11 to 1.14 mg cobalt/m^3 for 2 years resulted in a spectrum of inflammatory, fibrotic, and proliferative lesions in the respiratory tract of male and female rats and mice (Bucher et al. 1999; NTP 1998). Squamous metaplasia of the larynx occurred in rats and mice at exposure concentrations of ≥0.11 mg cobalt/m^3, with severity of the lesion increasing with increased cobalt concentration. Hyperplastic lesions of the nasal epithelium occurred in rats at concentrations of ≥0.11 mg cobalt/m^3, and in mice at concentrations of ≥0.38 mg cobalt/m^3. Both sexes of rats had greatly increased incidences (>90% incidence) of alveolar lesions at all exposure levels, including inflammatory changes, fibrosis, and metaplasia. Similar changes were seen in mice at all exposure levels, though the changes in mice were less severe.

Cardiovascular Effects. Occupational exposure of humans to cobalt-containing dust, either as cobalt metal or as hard metal, has been shown to result in cardiomyopathy, characterized by functional effects on the ventricles (Horowitz et al. 1988) and/or enlargement of the heart (Barborik and Dusek 1972; Jarvis et al. 1992), but the exposure levels associated with cardiac effects of inhaled cobalt in humans have not been determined. Jarvis et al. (1992) reported on two patients (exposure histories not specified) who had been admitted to the emergency room for cardiac failures; these failures were believed to be associated with cobalt exposure. Barborik and Dusek (1972) reported a case of a 41-year-old man who was admitted to the hospital with cardiac failure following occupational exposure to cobalt; cobalt concentrations in heart, liver, lung, spleen, and kidney were elevated over two control patients. Horowitz et al. (1988) reported that in a cohort of 30 hard metal workers (exposure histories not specified), significant decreases in exercise right ventricular ejection fraction (EF) were seen in workers with abnormal chest x-rays relative to those with normal chest x-rays. It is possible that these effects were secondary to the respiratory effects of inhaled cobalt. It was concluded that cobalt is a weak cardiomyopathic agent following occupational exposure (Horowitz et al. 1988). Cardiomyopathy is a characteristic toxic effect of cobalt following oral exposure in both humans and animals (Section 3.2.2.2).

In rats, exposure to 11.4 mg cobalt/m^3 as cobalt sulfate over 13 weeks resulted in a marginal increase in the severity of cardiomyopathy as compared to controls (minimal-mild in treated animals versus minimal in controls; 3/10 animals affected in either group) (Bucher et al. 1990; NTP 1991). Cardiomyopathy was not observed in mice exposed to ≤76 mg cobalt/m^3 as cobalt sulfate over 16 days (Bucher et al. 1990; NTP 1991), nor in mice or rats exposed to up to 1.14 mg cobalt/m^3 for 2 years (Bucher et al. 1999; NTP 1998). Electrocardiogram abnormalities that may reflect ventricular impairment have been observed in miniature swine (n=5) exposed to 0.1 mg cobalt dust/m^3 for 6 hours/day, 5 days/week for 3 months (Kerfoot 1975).

Gastrointestinal Effects. No studies were located regarding gastrointestinal effects in humans after inhalation exposure to stable cobalt.

No histological lesions were reported in the esophagus, stomach, duodenum, ileum, jejunum, cecum, colon, or rectum of rats or mice of either sex exposed to 76 mg cobalt/m^3 or less as cobalt sulfate for 16 days, up to 11.4 mg cobalt/m^3 for 13 weeks, or up to 1.14 mg cobalt/m^3 for 104 weeks (Bucher et al. 1990, 1999; NTP 1991, 1998).

Hematological Effects. Swennen et al. (1993) reported slightly, but statistically significantly, decreased levels of red cells and total hemoglobin (~4–5% decreases) in a group of 82 workers occupationally exposed to a mean concentration of 0.125 mg cobalt/m^3 as cobalt metal dust. No other studies were located regarding hematological effects in humans after inhalation exposure to cobalt.

Increased levels of hemoglobin and increased numbers of basophils and monocytes have been observed in rats and guinea pigs, but not in dogs, exposed to 9 mg cobalt/m^3 as cobalt hydrocarbonyl for 3 months (Palmes et al. 1959). Polycythemia was reported in rats, but not mice, exposed to 1.14 mg cobalt/m^3 as cobalt sulfate for 13 weeks (Bucher et al. 1990; NTP 1991).

Musculoskeletal Effects. No studies were located regarding musculoskeletal effects in humans after inhalation exposure to cobalt.

No histological lesions were reported in the sternebrae (segments of the sternum), including the bone marrow, of rats or mice exposed to ≤76 mg cobalt/m^3 as cobalt sulfate for 16 days, up to 11.4 mg

cobalt/m^3 for 13 weeks, or up to 1.14 mg cobalt/m^3 for 104 weeks (Bucher et al. 1990, 1999; NTP 1991, 1998) (see the above section on respiratory effects for detailed descriptions of exposure conditions).

Hepatic Effects. Congestion of the liver was observed upon autopsy of a metal worker (exposure history not reported) who had been occupationally exposed to an unknown level of cobalt for 4 years (Barborik and Dusek 1972). The cause of death was determined to be cardiomyopathy.

Necrosis and congestion of the liver were observed in both rats and mice that died following exposure to 19 mg cobalt/m^3 as cobalt sulfate over 16 days (Bucher et al. 1990; NTP 1991). No histological effects on the liver were found in pigs exposed to up to 1.0 mg cobalt/m^3 as cobalt metal dust for 3 months (Kerfoot 1975).

Renal Effects. Congestion of the kidneys was observed upon autopsy of a metal worker who had been occupationally exposed to an unknown level of cobalt for 4 years (Barborik and Dusek 1972). The cause of death was determined to be cardiomyopathy.

A significant increase in the relative weight of the kidneys was reported in male rats exposed to 0.11 mg cobalt/m^3 or greater as cobalt sulfate for 13 weeks (Bucher et al. 1990; NTP 1991). No effects were observed upon histological examination of the kidneys in rats or mice following exposure to $\leq$76 mg cobalt/m^3 as cobalt sulfate for 16 days, up to 11.4 mg cobalt/m^3 for 13 weeks, or up to 1.14 mg cobalt/m^3 for 104 weeks (Bucher et al. 1990, 1999; NTP 1991, 1998). No histological effects on the kidneys were found in pigs exposed to up to 1.0 mg cobalt/m^3 as cobalt metal for 3 months (Kerfoot 1975).

Dermal Effects. No studies were located regarding dermal effects in humans or animals after inhalation exposure to stable cobalt.

Endocrine Effects. A group of female workers occupationally exposed to a semisoluble cobalt glaze (cobalt-zinc silicate, estimated concentrations of 0.05 mg Co/m^3) showed significantly elevated levels of serum thyroxine (T4) and free thyroxine, but no change in T3 levels (Prescott et al. 1992). In contrast to this, Swennen et al. (1993) reported no significant change in serum T4 levels, but a significant reduction in serum T3 in workers occupationally exposed to cobalt oxides, cobalt salts, and cobalt metal.

Ocular Effects. Congestion of the conjunctiva was observed in a metal worker after occupational exposure to an unknown level of cobalt for 4 years (Barborik and Dusek 1972); however, due to the nature of the exposure, this effect may also have been the result of direct dermal or ocular contact. Upon autopsy, the cause of death was determined to be cardiomyopathy.

No histological lesions were reported in the eyes or on the skin of rats or mice exposed to ≤76 mg cobalt/m^3 as cobalt sulfate for 16 days, up to 11.4 mg cobalt/m^3 for 13 weeks, or up to 1.14 mg cobalt/m^3 for 104 weeks (Bucher et al. 1990, 1999; NTP 1991, 1998).

Body Weight Effects. Weight loss, measured individually from time of initial examination throughout followup, was observed in a group of five diamond polishers suffering from cobalt-induced interstitial lung disease (Demedts et al. 1984b), but the exposure level of cobalt was not reported.

Decreased body weight, relative to controls at study termination, was reported in both rats and mice exposed to 19 mg cobalt/m^3 as cobalt sulfate over 16 days or to 11.4 mg cobalt/m^3 for 13 weeks (Bucher et al. 1990; NTP 1991). A 13-week exposure to 11.4 mg cobalt /m^3 resulted in ruffled fur in male rats, with no clinical signs reported in female rats or either sex of mice (Bucher et al. 1990; NTP 1991). Chronic exposure of rats and mice to up to 1.14 mg cobalt/m^3 did not result in decreased body weight (Bucher et al. 1999; NTP 1998).

Weight loss was found in dogs, but not rats or guinea pigs, exposed for 3 months to cobalt at a level of 9 mg cobalt/m^3 as cobalt hydrocarbonyl (Palmes et al. 1959). Lifetime exposure of hamsters to a similar concentration (7.9 mg cobalt/m^3 as cobalt oxide) did not result in decreased body weight gain (Wehner et al. 1977).

3.2.1.3 Immunological and Lymphoreticular Effects

Cobalt is known to function as a hapten, resulting in the generation of antibodies against cobalt-protein complexes. Although the minimum exposure level associated with cobalt sensitization has not been determined, sensitization has been demonstrated in hard metal workers with work-related asthma who have experienced prolonged occupational exposure (>3 years) to levels ranging from 0.007 to 0.893 mg cobalt/m^3 (Shirakawa et al. 1988, 1989). The lower end of this range, 0.007 mg/m^3, is reported in

Table 3-1 and plotted in Figure 3-1 as a LOAEL. The sensitization phenomenon includes the production of IgE and IgA antibodies to cobalt (Bencko et al. 1983; Shirakawa et al. 1988, 1989). Exposure to inhaled cobalt chloride aerosols can precipitate an asthmatic attack in sensitized individuals (Shirakawa et al. 1989), believed to be the result of an allergic reaction within the lungs.

Necrosis of the thymus was reported in rats exposed to 19 mg cobalt/m^3 as cobalt sulfate over 16 days, and hyperplasia of the mediastinal lymph nodes was found in mice exposed to 11.4 mg cobalt/m^3 for 13 weeks (Bucher et al. 1990; NTP 1991). Tests of immunological function, however, were not performed on the rats or mice.

3.2.1.4 Neurological Effects

Occupational exposure to cobalt in humans has been reported to cause several effects on the nervous system, including memory loss (Wechsler Memory Scale-Revised), nerve deafness, and a decreased visual acuity (Jordan et al. 1990; Meecham and Humphrey 1991). It should be noted, though, that both of these studies had small numbers of subjects (n=38 for Jordan et al. 1990, n=1 for Meecham and Humphrey 1991), and exposure characterization was not reported.

Congestion in the vessels of the brain/meninges was reported in rats and mice exposed to 19 mg cobalt/m^3 or greater as cobalt sulfate over 16 days (Bucher et al. 1990; NTP 1991).

3.2.1.5 Reproductive Effects

No studies were located regarding reproductive effects in humans after inhalation exposure to cobalt.

In animals, long-term exposure to cobalt-containing aerosols has resulted in effects on reproductive end points. Testicular atrophy was reported in rats, but not in mice, exposed to 19 mg cobalt/m^3 as cobalt sulfate over 16 days (Bucher et al. 1990; NTP 1991). Following exposure of mice to cobalt (as cobalt sulfate) for 13 weeks, a decrease in sperm motility was found at 1.14 mg cobalt/m^3, and testicular atrophy was found at 11.4 mg cobalt/m^3. A significant increase in the length of the estrous cycle was reported in female mice exposed to 11.4 mg cobalt/m^3 for 13 weeks (Bucher et al. 1990; NTP 1991). No effects on

the male or female reproductive systems were observed in rats similarly treated for 13 weeks (Bucher et al. 1990; NTP 1991), or in mice or rats exposed to up to 1.14 mg cobalt/m^3 for 104 weeks (Bucher et al. 1999; NTP 1998).

3.2.1.6 Developmental Effects

No studies were located regarding developmental effects in humans or animals after inhalation exposure to cobalt.

3.2.1.7 Cancer

Several studies have evaluated the effects of inhalation of cobalt-containing compounds on possible carcinogenicity in humans. The mortality of a cohort of 1,143 workers in a plant that refined and processed cobalt and sodium was analyzed (Mur et al. 1987); the French national population mortality data were used as a reference population. An increase in deaths due to lung cancer was found in workers exposed only to cobalt (standardized mortality ratio [SMR] of 4.66; four cases in the exposed group versus one case in the controls). In a study within the cohort that controlled for date of birth, age at death, and smoking habits, 44% (four workers) in the group exposed to cobalt and 17% (three workers) in the group not exposed to cobalt died of lung cancer. The authors, however, indicated that the difference was not statistically significant and that the workers were exposed to both arsenic and nickel as well as cobalt. The nonneoplastic lung diseases commonly found in cobalt-exposed workers (see Section 3.2.1.2) were not reported in this group. These lung diseases may have been present in these workers, but if they were not listed as the cause of death on the death certificate, they would not have been mentioned. Inhalation was probably a prominent route of exposure to cobalt; however, oral and dermal exposure probably occurred as well. No adjustments were made for smoking habits in the larger study, and the exposure levels of cobalt were not reported for either study. However, a followup study of this cohort (Moulin et al. 1993) did not report significant increases in mortality due to respiratory or circulatory diseases. Similarly, no increase in the SMR for lung cancer was noted in exposed workers, relative to controls. While an elevated SMR for lung cancer was seen in maintenance workers (SMR=1.80, 95% confidence interval [CI]=0.78–3.55), it was not statistically significant, since the 95% confidence interval included an SMR of 1.

Lasfargues et al. (1994) reported on the mortality of a cohort of 709 male workers in a French hard metal plant, using the national rates for French males for comparison. The overall mortality did not differ from expected, but there was a significant increase in mortality due to cancer of the trachea, bronchus, and lung (SMR=2.13, 95% CI=1.02–3.93). Smoking alone did not account for the lung cancer excesses, although the influence of smoking on the observed mortality could not be entirely ruled out.

A cohort of 5,777 males and 1,682 females who were exposed occupationally to cobalt (concentrations ranging from 1 to 515 $\mu g/m^3$, means of exposure levels ranging from 39.37 to 169.0 $\mu g/m^3$) and tungsten carbide (as hard metal dust) was examined by Moulin et al. (1998). A significantly increased mortality rate (SMR=1.30, 95% CI=1.00–1.66) was seen for lung cancer in exposed workers, when compared to the national average. Within this study group, 61 cases and 180 controls were selected for a case-control study of cancer risk. When exposures during the last 10 years were ignored, presumably because cancer is a late-developing disease, a significant increase in lung cancer mortality (OR=1.93, 95% CI=1.03–3.62) relative to controls was seen among workers simultaneously exposed to cobalt and tungsten carbide. Significant trends for increasing cancer risk with increasing cumulative exposure and exposure duration were noted. Adjustments for smoking and for coexposures to other carcinogens did not change the results, though occupational risk was greatest among smokers.

A later study by the same group (Moulin et al. 2000) examined the lung cancer mortality of 4,288 male and 609 female workers employed in the production of stainless and alloyed steel from 1968 to 1992. No significant changes in mortality rate from lung cancer were seen among exposed workers (SMR=1.19, 95% CI=0.88–1.55), and a concurrent case control study identified no correlation between lung cancer excess and for exposure to cobalt (OR=0.64, 95% CI=0.33–1.25).

Wild et al. (2000) reported on a cohort of 2,216 male hard metal workers who had been employed for at least 3 months; this cohort was the same as that in Moulin et al. (2000), with some modifications. The total mortality was not increased in workers, relative to local mortality rates. However, lung cancer mortality was significantly increased (SMR=1.70, 95% CI=1.24–2.26). The risks increased with increasing exposure scores, even after adjustment for smoking and coexposure to other known or suspected carcinogens.

Inhalation exposure to 7.9 mg cobalt/m^3 as cobalt oxide intermittently for a lifetime did not increase the incidence of malignant or benign tumors in hamsters (Wehner et al. 1977).

NTP (1998) exposed groups of rats and mice of both sexes to 0, 0.11, 0.38, or 1.14 mg cobalt/m^3 as cobalt sulfate for 2 years. Increased incidence of alveolar/bronchiolar neoplasms was noted following lifetime exposure of male rats to 1.14 mg cobalt/m^3 and in female rats exposed to 0.38 mg cobalt/m^3 (Bucher et al. 1999; NTP 1998). Statistical analysis revealed that tumors occurred with significantly positive trends in both sexes of rats. Similarly, mice of both sexes exposed to 1.14 mg cobalt/m^3 showed an increase in alveolar/bronchiolar neoplasms, again with lung tumors occurring with significantly positive trends.

3.2.2 Oral Exposure

3.2.2.1 Death

In several studies, lethal cardiomyopathy was reported in people who consumed large quantities of beer containing cobalt sulfate (Alexander 1969, 1972; Bonenfant et al. 1969; Morin et al. 1967, 1971; Sullivan et al. 1969). The deaths occurred during the early to mid 1960s, at which time, breweries in Canada, the United States, and Europe were adding cobalt to beer as a foam stabilizer (Alexander 1969, 1972; Bonenfant et al. 1969; Morin et al. 1967, 1971; Sullivan et al. 1969); this practice has been discontinued. Deaths occurred following ingestion of beer containing 0.04–0.14 mg cobalt/kg/day for a period of years (approximately 8–30 pints of beer each day). "Acute mortality" (death within several days of admission) accounted for 18% of the deaths (Alexander 1972). Approximately 43% of the patients admitted to the hospital with cardiomyopathy died within several years of the initial hospital visit. It should be noted, however, that the cardiomyopathy may have also been due to the fact that the beer-drinkers had protein-poor diets and may have had prior cardiac damage from alcohol abuse.

Treatment of both pregnant and nonpregnant anemic patients with doses of cobalt (0.6–1 mg/kg/day) that were much higher than the doses in the beer did not result in mortality (Davis and Fields 1958; Holly 1955). A 19-month-old male child who swallowed an unknown amount of a cobalt chloride solution died approximately 6.5 hours after ingestion, despite repeated induced vomiting, gastric lavage, and supportive therapy (Jacobziner and Raybin 1961).

Oral LD_{50} values for several cobalt compounds have been determined in Wistar rats (FDRL 1984a, 1984b, 1984c; Singh and Junnarkar 1991; Speijers et al. 1982). The LD_{50} values ranged from 42.4 mg cobalt/kg as cobalt chloride to 317 mg cobalt/kg as cobalt carbonate. An LD_{50} of 3,672 mg cobalt/kg was also found for tricobalt tetraoxide, a highly insoluble cobalt compound (FDRL 1984c). The exact cause of death in rats is unknown, but effects on the heart, liver, gastrointestinal tract, and kidneys have been observed. In Sprague-Dawley rats, death has been reported to occur at 161 mg cobalt/kg given by gavage as cobalt chloride (Domingo and Llobet 1984). In male Swiss mice, the LD_{50} values for cobalt chloride and cobalt sulfate have been reported to be 89.3 and 123 mg cobalt/kg, respectively (Singh and Junnarkar 1991).

Following 5 weeks of exposure to 20 mg cobalt/kg/day as cobalt sulfate by gavage, 20–25% of the guinea pigs died (Mohiuddin et al. 1970). The animals were given cobalt sulfate alone or in combination with ethanol (as part of a liquid diet) to compare the effects seen in animals to those seen in humans suffering from beer-cobalt cardiomyopathy. Although effects on the heart were found in the treated animals, alcohol did not appear to intensify the toxic effect.

The LD_{50} and all reliable LOAEL values for each species and duration category are reported in Table 3-2 and plotted in Figure 3-2.

3.2.2.2 Systemic Effects

Oral cobalt exposure in humans and/or animals resulted in respiratory, cardiovascular, gastrointestinal, hematological, hepatic, renal, endocrine, dermal, ocular, hypothermic, and body weight effects. For each effect, the highest NOAEL values and all reliable LOAEL values for each species and duration category are reported in Table 3-2 and plotted in Figure 3-2.

Respiratory Effects. In 50 patients with beer-cobalt cardiomyopathy, pulmonary rales and pulmonary edema were observed and were attributed to cobalt-induced cardiac failure (Morin et al. 1971). These patients had ingested, over a period of years, an average of 0.04 mg cobalt/kg/day in beer containing cobalt sulfate that was added to stabilize the foam. It should be noted that these patients consumed significant quantities of alcohol, and the effect that this may have had on the symptoms seen is not known.

Table 3-2 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Oral

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/kg/day)	LOAEL Less Serious (mg/kg/day)	LOAEL Serious (mg/kg/day)	Reference Chemical Form
	ACUTE EXPOSURE						
	Death						
1	Rat (Sprague-Dawley)	1x (GW)				161.1 (LD50)	Domingo and Llobet 1984 Chloride
2	Rat (Wistar)	1x (GW)				42.4 (LD50)	Singh and Junnarkar 1991 Chloride
3	Rat (Wistar)	1x (GW)				194 (LD50)	Singh and Junnarkar 1991 Sulfate
4	Rat (Wistar)	1 x (GO)				91 (LD50)	Speijers et al. 1982 Fluoride
5	Rat (Wistar)	1 x (GO)				187 (LD50)	Speijers et al. 1982 Phosphate
6	Rat (Wistar)	1 x (GW)				109 (LD50)	Speijers et al. 1982 Bromide
7	Rat (Wistar)	1 x (GO)				159 (LD50)	Speijers et al. 1982 Oxide

Table 3-2 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Oral (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/kg/day)	LOAEL Less Serious (mg/kg/day)	LOAEL Serious (mg/kg/day)	Reference Chemical Form
8	Rat (Wistar)	1 x (GW)				168 (LD50)	Speijers et al. 1982 Acetate
9	Rat (Wistar)	1 x (GW)				190 (LD50)	Speijers et al. 1982 Chloride
10	Rat (Wistar)	1 x (GW)				140 (LD50)	Speijers et al. 1982 Bromide
11	Rat (Wistar)	1 x (GW)				161 (LD50)	Speijers et al. 1982 Sulfate
12	Mouse (Swiss-Webster)	1x (GW)				123 (LD50)	Singh and Junnarkar 1991 Sulfate
13	Mouse (Swiss-Webster)	1x (GW)				89.3 (LD50)	Singh and Junnarkar 1991 Chloride
	Systemic						
14	Human	2 wk (C)	Endocr		1 (decreased Iodine uptake in thyroid)		Roche and Layrisse 1958 Chloride

Table 3-2 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Oral (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/kg/day)	LOAEL Less Serious (mg/kg/day)		LOAEL Serious (mg/kg/day)		Reference Chemical Form
15	Rat	1x (GW)	Hemato		161.1[b]	(increased hematocrit 8%)			Domingo and Llobet 1984 Chloride
16	Rat	1 x (GW)	Other	110	209	(Clinical signs, including decreased activity, ataxia, diarrhea, salivation)			FDRL 1984a Sulfate
17	Rat (Sprague-Dawley)	1 x (GO)	Other		149	(Decreased activity, diarrhea)			FDRL 1984b Carbonate
18	Rat (Wistar)	1x (GW)	Renal		19.4	(Increased urinary output)			Singh and Junnarkar 1991 Sulfate
19	Rat (Wistar)	1 x (GO)	Cardio	109.6			176.6	(proliferative interstitial tissues, swollen muscle fibers, focal myocardial degeneration)	Speijers et al. 1982 Fluoride
			Hepatic	42.6			68.2	(hyperemia)	
			Renal		42.6	(swollen proximal tubules)	176.6	(degeneration of proximal tubules)	
			Other				109.6	(hypothermia)	

Table 3-2 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Oral (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/kg/day)	LOAEL Less Serious (mg/kg/day)	LOAEL Serious (mg/kg/day)	Reference Chemical Form
20	Rat (Wistar)	1 x (GO)	Cardio			794.5 (hemorrhage)	Speijers et al. 1982 Oxide
			Hepatic			157.3 (hyperemia)	
			Renal			157.3 (hyperemia)	
			Other			157.3 (hypothermia)	
21	Mouse (Swiss-Webster)	48 hr (W)	Hemato		76.4 M (Alteration in electrophoretic profile of serum proteins)		Bryan and Bright, 1973 Chloride
22	Mouse (Swiss-Webster)	3 mo (W)	Hemato	76.4 M			Bryan and Bright, 1973 Chloride
	Neurological						
23	Rat (Wistar)	1x (GW)			19.4 (Mild depression of spontaneous activity, muscle tone, and respiration)		Singh and Junnarkar 1991 Sulfate

Table 3-2 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Oral (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/kg/day)	LOAEL Less Serious (mg/kg/day)	LOAEL Serious (mg/kg/day)	Reference Chemical Form
24	Rat (Wistar)	1x (GW)			4.25 (Mild depression of spontaneous activity, muscle tone, and respiration)		Singh and Junnarkar 1991 Chloride
	Developmental						
25	Rat	Gd 6-15 (GW)		24.8			Paternian et al. 1988 Chloride
26	Mouse	Gd 8-12 (GW)		81.7			Seidenberg 1986 Chloride
	INTERMEDIATE EXPOSURE						
	Death						
27	Human	NR (W)				0.04 (death)	Morin et al. 1971 Sulfate
28	Gn Pig	5 wk (F)				20 (death)	Mohiuddin et al. 1970 Sulfate
	Systemic						
29	Human	NR (W)	Cardio			0.07 (beer-cobalt cardiomyopathy)	Alexander 1972 Sulfate

Table 3-2 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Oral (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/kg/day)	LOAEL Less Serious (mg/kg/day)	LOAEL Serious (mg/kg/day)	Reference Chemical Form
30	Human	1x/d 25 d (C)	Hemato		1[c] (polycythemia)		Davis and Fields 1958 Chloride
31	Human	12-32 wk (C)	Gastro		0.18 (nausea)		Duckham and Lee 1976b Chloride
			Hemato		0.18[b] (increased hemoglobin, 23-102% increase)		
32	Human	90 d (C)	Gastro		0.5 (gastric intolerance)		Holly 1955 Chloride
			Hemato	0.6			
			Hepatic	0.6			
33	Human	NR (W)	Resp		0.04 (edema)		Morin et al. 1971 Sulfate
			Cardio			0.04 (beer-cobalt cardiomyopathy)	
			Gastro		0.04 (vomiting, nausea)		
			Hepatic		0.04 (necrosis)		

Table 3-2 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Oral (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/kg/day)	LOAEL Less Serious (mg/kg/day)	LOAEL Serious (mg/kg/day)	Reference Chemical Form
34	Human	10-25 d 1x/d (C)	Other		0.54 (decreased iodine uptake)		Paley et al. 1958
35	Human	12-32 wk 7d/wk (C)	Hemato		0.16[b] (increased hemoglobin)		Taylor et al. 1977 Chloride
36	Rat (Sprague-Dawley)	4 wk (F)	Bd Wt		3.79 M (45-65% reduction in body weight gain)		Chetty et al. 1979 Chloride
37	Rat	8 wk 1x/d (F)	Bd Wt		4.2 (33% decrease in body weight gain)		Clyne et al. 1988

Table 3-2 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Oral (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/kg/day)	LOAEL Less Serious (mg/kg/day)	LOAEL Serious (mg/kg/day)	Reference Chemical Form
38	Rat	3 mo (W)	Resp		30.2 (increased lung weight 33%)		Domingo et al. 1984
			Cardio		30.2 (increased heart weight 9.4%)		
			Gastro	30.2			
			Hemato		30.2 (increased hematocrit 29%)c		
			Musc/skel	30.2			
			Hepatic	30.2			
			Renal	30.2			
39	Rat	8 wk (F)	Cardio			26 (degeneration)	Grice et al. 1969
40	Rat (Sprague-Dawley)	24 wk (F)	Cardio			8.4 M (Left ventricular hypertrophy and impaired ventricular function)	Haga et al. 1996 Sulfate

Table 3-2 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Oral (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/kg/day)	LOAEL Less Serious (mg/kg/day)	LOAEL Serious (mg/kg/day)	Reference Chemical Form
41	Rat	4 mo (G)	Resp	18			Holly 1955 Chloride
			Cardio	18			
			Gastro	18			
			Hemato		18[b] (erythrocytosis)		
			Hepatic	18			
			Renal			18 (tubular necrosis)	
42	Rat	7 mo 6 d/wk (GW)	Hemato	0.05	0.5[b] (increased RBC, hemoglobin)		Krasovskii and Fridlyand 1971
			Hepatic	2.5			
43	Rat CFY	3 wk (G)	Cardio			12.4 M (Incipient, multifocal myocytolysis, with degeneration of myofibrilles)	Morvai et al. 1993 Chloride
			Bd Wt		12.4 M (Decreased body weight 8%)		

Table 3-2 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Oral (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/kg/day)	LOAEL Less Serious (mg/kg/day)	LOAEL Serious (mg/kg/day)	Reference Chemical Form
44	Rat	150 d 5 d/wk (GW)	Hemato		10[b] (increased hemoglobin)		Murdock 1959 Chloride
			Hepatic		10 (increased weight 17%)		
			Renal			10 (necrosis of tubular lining cells)	
			Bd Wt	10			
45	Rat (Sprague-Dawley)	8 wk	Hemato	8.4 M			Pehrsson et al. 1991 Sulfate
			Bd Wt			8.4 M (>20% decrease from appropriate control)	
46	Rat (Sprague-Dawley)	12-16 d (W)	Bd Wt	10.6 M			Saker et al. 1998 Chloride
			Metab		10.6 M (Decreased serum glucose levels in diabetic rats, but not control rats)		

Table 3-2 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Oral (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/kg/day)	LOAEL Less Serious (mg/kg/day)	LOAEL Serious (mg/kg/day)	Reference Chemical Form
47	Rat	6 wk 7 d/wk (C)	Hemato	0.6	2.5[b] (polycythemia)		Stanley et al. 1947 Chloride
48	Rat (Long- Evans)	3 d (F)	Bd Wt	20 M	100 M (<20% reduction of body weights)		Wellman et al. 1984 Chloride
49	Mouse Parkes	45 d (W)	Endocr			26 F (Necrosis and inflammation of thyroid)	Shrivastava et al. 1996 Chloride
50	Gn Pig	5 wk (F)	Cardio			20 (cardiomyopathy)	Mohiuddin et al. 1970
			Bd Wt	20			
51	Dog	4 wk 7 d/wk (F)	Hemato		5[b] (polycythemia)		Brewer 1940
	Immuno/ Lymphoret						
52	Rat (Sprague-Dawley)	4 wk (F)				3.79 M (Atrophy of the thymus)	Chetty et al. 1979 Chloride

Table 3-2 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Oral (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/kg/day)	LOAEL Less Serious (mg/kg/day)	LOAEL Serious (mg/kg/day)	Reference Chemical Form
53	Rat	7 mo 6 d/wk (GW)		0.05	0.5 (decreased phagocytic ability)		Krasovskii and Fridlyand 1971 Chloride
	Neurological						
54	Rat (Sprague-Dawley)	57 d (W)			20 M (Increased latency during retention testing)		Bourg et al. 1985 Chloride
55	Rat	57 d (W)			20 (increased reactivity)		Bourg et al. 1985 Chloride
56	Rat	7 mo 6 d/wk (GW)		0.05	0.5 (mildly increased latent reflex)	2.5 (pronounced increase in latent reflex)	Krasovskii and Fridlyand 1971 Chloride
57	Rat (Wistar)	30 d (W)			4.96 M (Alterations in sympathetically-induced contractility of vas deferens)		Mutafova-Yambolieva et al. 1994 Chloride
58	Rat	69 d (F)		5	20 (changes in schedule training, conditioned suppression, and mixed schedule training tests)		Nation et al. 1983

Table 3-2 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Oral (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/kg/day)	LOAEL Less Serious (mg/kg/day)	LOAEL Serious (mg/kg/day)	Reference Chemical Form
59	Rat (Wistar)	30 d (W)			6.44 M (Alterations in cholinergic sensitivity)		Vassilev et al. 1993 Nitrate
60	Rat (Long- Evans)	3 d (F)		20 M	100 M (Saccharin and food aversion)		Wellman et al. 1984 Chloride
	Reproductive						
61	Rat (Sprague-Dawley)	98 days (F)				20 M Pronounced histologic alteration of seminiferous tubules	Corrier et al. 1985 Chloride
62	Rat (Sprague-Dawley)	90 d (W)			30.2 M 26% decrease in testicular weight		Domingo et al. 1984 Chloride
63	Rat	98 d 7 d/wk (F)				13.25 (testicular degeneration)	Mollenhauer et al. 1985
64	Rat	69 d (F)		5		20 M (testicular atrophy)	Nation et al. 1983 Chloride

Table 3-2 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Oral (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/kg/day)	LOAEL Less Serious (mg/kg/day)	LOAEL Serious (mg/kg/day)	Reference Chemical Form
65	Mouse (CD-1)	13 wk (W)				43.4 M (Irreversible testicular degeneration)	Anderson et al. 1992 Chloride
66	Mouse (CD-1)	13 wk (W)				43.4 M (Testicular degeneration)	Anderson et al. 1993 Chloride
67	Mouse	13 wk (W)			23 (reversible testicular degeneration)		Pedigo et al. 1988 Chloride
68	Mouse (B6C3F1)	10 wk (W)				58.9 M (Reduced pregnant females and pups per litter; reduced fertility)	Pedigo et al. 1993 Chloride
	Developmental						
69	Human	90 d (C)		0.6			Holly 1955 Chloride

Table 3-2 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Oral (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (mg/kg/day)	LOAEL Less Serious (mg/kg/day)	LOAEL Serious (mg/kg/day)	Reference Chemical Form
70	Rat	Gd 14- Ld 21 (G)				5.4 (stunted pup growth)	Domingo et al. 1985 Chloride

[a] The number corresponds to entries in Figure 3-2.

[b] An increase in hemoglobin or red blood cells is not necessarily considered an adverse effect.

[c] Used to derive an intermediate oral MRL; concentration was divided by an uncertainty factor of 100 (10 for use of a LOAEL and 10 for human variability), resulting in an MRL of 0.01 mg/kg/day.

Bd Wt = body weight; (C) = capsule; Cardio = cardiovascular; d = day(s); Endocr = endocrine; (F) = feed; F = female; (G) = gavage; Gd = gestation day; (GO) = gavage oil; (GW) = gavage-water, Gastro = gastrointestinal; Hemato = hematological; hr = hour(s); Ld = lactation day; LD50 = dose producing 50% death; LOAEL = lowest-observed-adverse-effect level; M = male; Metab = metabolism; mo = month(s); NOAEL = no observed-adverse-effect level; NS = not specified; (W) = drinking water; wk = week(s); x = times.

Figure 3-2. Levels of Significant Exposure to Cobalt - Chemical Toxicity - Oral

Acute (≤14 days)

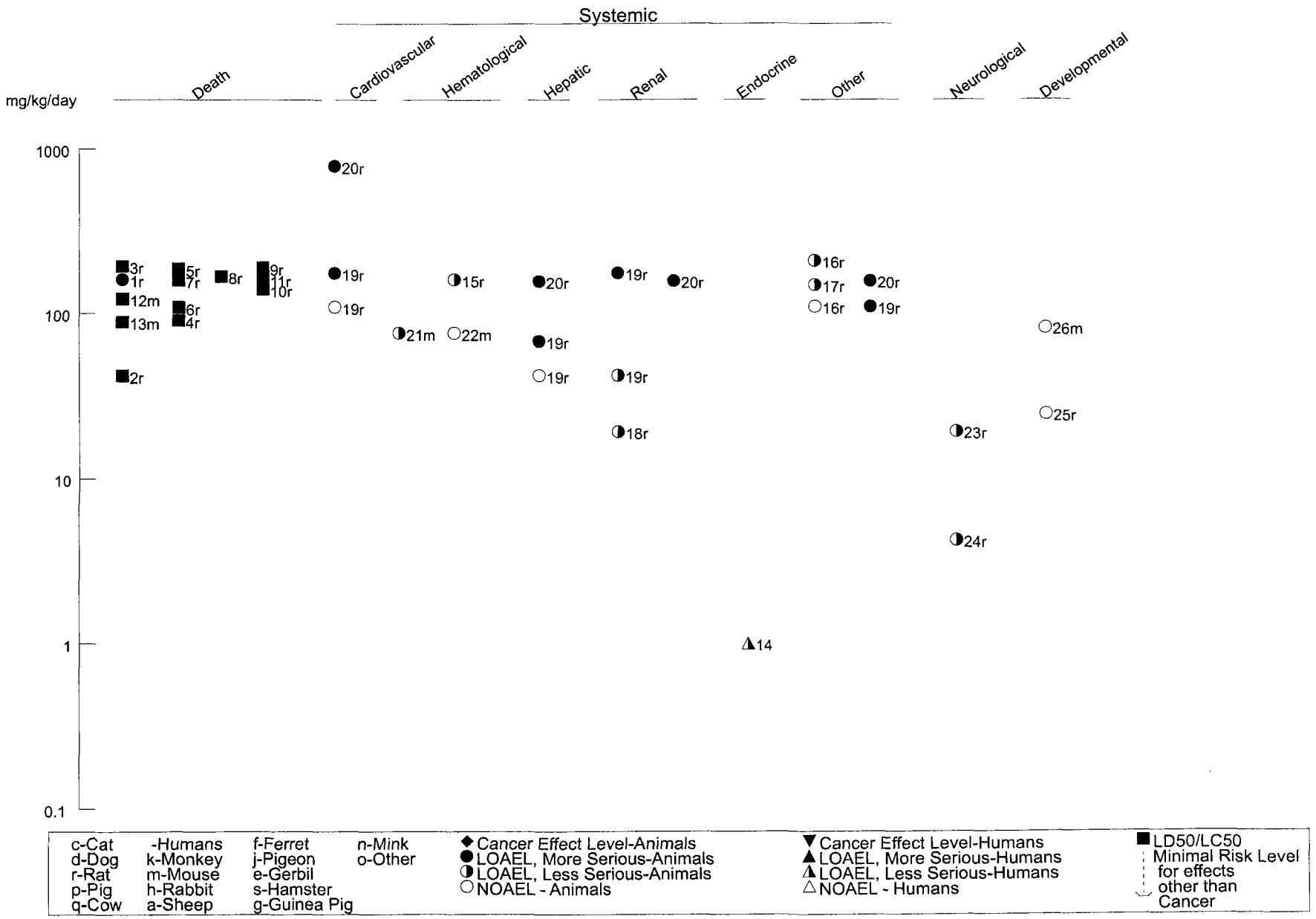
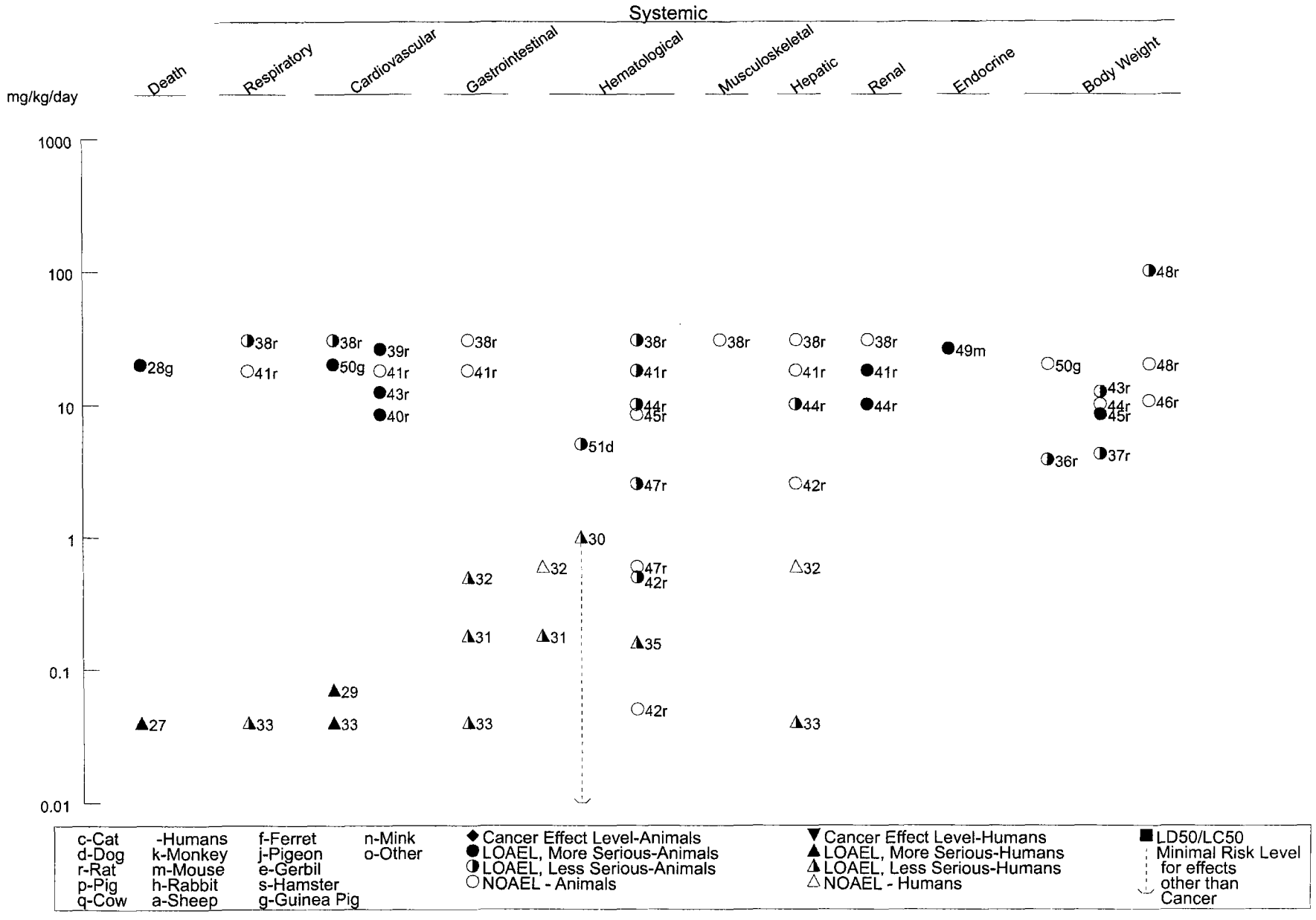


Figure 3-2 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Oral (*Continued*)

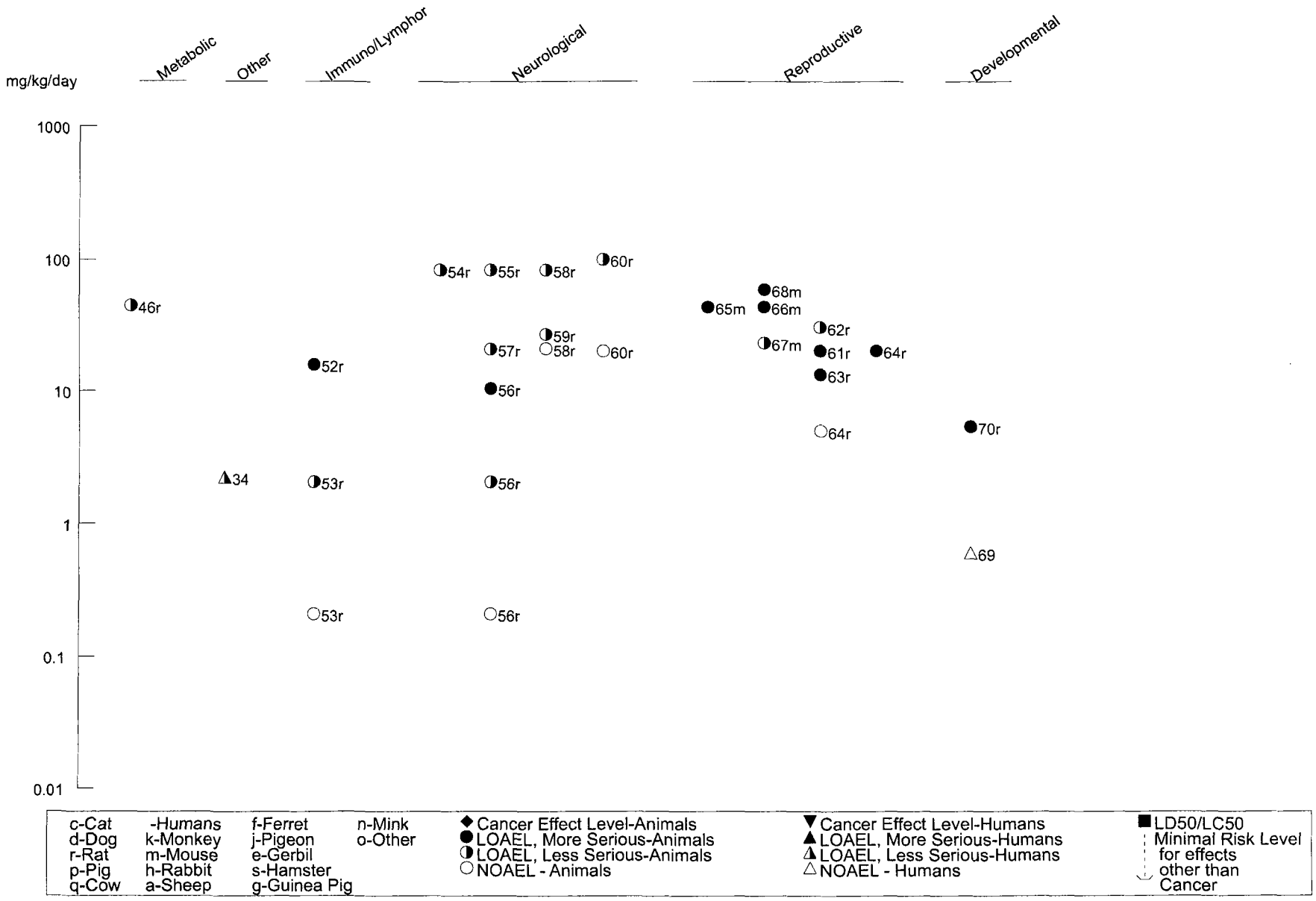
Intermediate (15-364 days)



c-Cat	-Humans	f-Ferret	n-Mink	◆ Cancer Effect Level-Animals	▼ Cancer Effect Level-Humans	■ LD50/LC50
d-Dog	k-Monkey	j-Pigeon	o-Other	● LOAEL, More Serious-Animals	▲ LOAEL, More Serious-Humans	Minimal Risk Level for effects other than Cancer
r-Rat	m-Mouse	e-Gerbil		◑ LOAEL, Less Serious-Animals	◮ LOAEL, Less Serious-Humans	
p-Pig	h-Rabbit	s-Hamster		○ NOAEL - Animals	△ NOAEL - Humans	
q-Cow	a-Sheep	g-Guinea Pig				

Figure 3-2 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Oral (*Continued*)

Intermediate (15-364 days)



A significant increase in the weight of the lungs, without morphological or histological changes, was found in rats that received 30.2 mg cobalt/kg/day as cobalt chloride in drinking water for 3 months, as compared with controls (Domingo et al. 1984). No morphological changes were seen in the lungs of rats treated with 18 mg cobalt/kg/day for 4 months (Holly 1955).

Cardiovascular Effects. Beer-cobalt cardiomyopathy was observed in people who heavily consumed beer containing cobalt sulfate as a foam stabilizer (Alexander 1969, 1972; Bonenfant et al. 1969; Kesteloot et al. 1968; Morin et al. 1967, 1971; Sullivan et al. 1969). The beer drinkers ingested an average of 0.04 mg cobalt/kg/day (Morin et al. 1971, n=50) to 0.14 mg cobalt/kg/day for a period of years (Alexander 1969, 1972, n=28). The cardiomyopathy was characterized by sinus tachycardia, left ventricular failure, cardiogenic shock, diminished myocardial compliance, absence of a myocardial response to exercise or catecholamine, enlarged heart, pericardial effusion, and extensive intracellular changes (changes in the myofibers, mitochondria, glycogen, and lipids). The beer-cobalt cardiomyopathy appeared to be similar to alcoholic cardiomyopathy and beriberi, but the onset of beer-cobalt cardiomyopathy was very abrupt. It should be noted, however, that the cardiomyopathy may have also been due to the fact that the beer-drinkers had protein-poor diets and may have had prior cardiac damage from alcohol abuse. Treatment of both pregnant and nonpregnant anemic patients for 90 days with doses of cobalt (0.6–1 mg/kg/day as cobalt chloride) that were much higher than the doses in the beer did not result in effects on the heart (Davis and Fields 1958; Holly 1955).

Approximately 40–50% of the patients admitted to the hospital with cardiomyopathy died within several years of diagnosis. In a followup study of four different sites, 0–43% of the survivors, depending on the site, showed a residual cardiac disability and 23–41% had abnormal electrocardiograms (Alexander 1972).

In an experiment designed to simulate conditions leading to beer-cobalt cardiomyopathy in humans, guinea pigs were given 20 mg cobalt/kg/day as cobalt sulfate by gavage either alone or in combination with ethanol (as part of a liquid diet) for 5 weeks (Mohiuddin et al. 1970). The experiment resulted in cardiomyopathy, which was characterized by abnormal EKGs; increased heart weights; lesions involving the pericardium, myocardium, and endocardium; and disfigured mitochondria. Alcohol did not intensify the cardiac effects. Myocardia changes (proliferative interstitial tissue, swollen muscle fibers, and focal degeneration) were also found in rats following a single dose of 176.6 mg cobalt/kg administered by

gavage as cobalt fluoride or a single dose of 795 mg cobalt/kg administered as cobalt oxide (Speijers et al. 1982).

Three weeks of exposure to 12.4 mg cobalt/kg/day as cobalt chloride in male rats resulted in cardiac damage, presenting as incipient, multifocal myocytolysis, with degeneration of myofibrilles (Morvai et al. 1993). After longer-term exposure (2–3 months) of rats to 26–30.2 mg cobalt/kg/day as cobalt sulfate in the diet or as cobalt chloride in the drinking water, degenerative heart lesions (Grice et al. 1969) and an increase in heart weight were found (Domingo et al. 1984). Exposure of rats to 8.4 mg cobalt/kg/day as cobalt sulfate resulted in left ventricular hypertrophy and impaired left ventricular systolic and diastolic functions in an isolated working rat heart model (Haga et al. 1996). Clyne et al. (2001) reported that exposure of rats to 8.4 mg cobalt/kg/day, as cobalt sulfate, in the diet for 24 weeks resulted in significant reductions in a number of enzymes in cardiac tissues, including manganese-superoxide dismutase, succinate-cytochrome c oxidase, NADH-cytochrome c reductase, and cytochrome c oxidase, as well as reducing the mitochondrial ATP production rate.

Gastrointestinal Effects. The first signs of the beer-cobalt cardiomyopathy syndrome were gastrointestinal effects and included nausea, vomiting, and diarrhea (Morin et al. 1971). Signs of heart failure subsequently appeared. These individuals had ingested an average of 0.04 mg cobalt/kg/day for a period of years during which cobalt sulfate was added to beer as a foam stabilizer; however, it is likely that alcohol consumption was also a factor.

In pregnant women given cobalt supplements (alone or combined with iron) to prevent the decrease in hematocrit and hemoglobin levels commonly found during pregnancy (n=78), a small percentage of those treated complained of gastric intolerance (Holly 1955). The women were treated with 0.5–0.6 mg cobalt/kg/day as cobalt chloride for 90 days. Nausea was reported in one anemic patient following treatment with 0.18 mg cobalt/kg/day as cobalt chloride (Duckham and Lee 1976b).

No morphological changes in the gastrointestinal system were observed following exposure of 20 male rats for 3 months to 30.2 mg cobalt/kg/day as cobalt chloride in the drinking water (Domingo et al. 1984) or exposure for 4 months to 18 mg cobalt/kg/day as cobalt chloride by gavage (Holly 1955).

Hematological Effects. Cobalt has been shown to stimulate the production of red blood cells in humans. Davis and Fields (1958) exposed six apparently normal men, ages 20–47, to a daily dose of

cobalt chloride, administered as a 2% solution diluted in either water or milk, for up to 22 days. Five of the six received 150 mg cobalt chloride per day for the entire exposure period, while the sixth was started on 120 mg/day and later increased to 150 mg/day. Blood samples were obtained daily from free-flowing punctures of fingertips at least 2 hours after eating, and at least 15 hours after the last dosage of cobalt. Blood was analyzed for red blood cell counts, hemoglobin percentage, leukocyte counts, reticulocyte percentages, and thrombocyte counts. Exposure to cobalt resulted in the development of polycythemia in all six subjects, with increases in red blood cell numbers ranging from 0.5 to 1.19 million (~16–20% increase above pretreatment levels). Polycythemic erythrocyte counts returned to normal 9–15 days after cessation of cobalt administration. Hemoglobin levels were also increased by cobalt treatment, though to a lesser extent than the erythrocyte values, with increases of 6–11% over pretreatment values. In five of the six subjects, reticulocyte levels were elevated, reaching at least twice the pre-experiment values. Thrombocyte and total leukocyte counts did not deviate significantly from pretreatment values. From the LOAEL of 1 mg/kg-day identified by this study, an intermediate-duration oral MRL of $1x10^{-2}$ mg/kg-day was derived (for derivation, see Section 2.3 and Appendix A).

Increased levels of erythrocytes were also found following oral treatment of anephric patients (with resulting anemia) with 0.16–1.0 mg cobalt/kg/day daily as cobalt chloride for 3–32 weeks (Duckham and Lee 1976b; Taylor et al. 1977). The increase in hemoglobin resulted in a decreased need for blood transfusions. Treatment of pregnant women for 90 days with 0.5–0.6 mg cobalt/kg/day as cobalt chloride, however, did not prevent the reduction in hematocrit and hemoglobin levels often found during pregnancy (Holly 1955).

Significantly increased erythrocyte (polycythemia), hematocrit, and hemoglobin levels were found in animals treated orally with cobalt chloride as a single dose of 161 mg cobalt/kg (Domingo and Llobet 1984) or with longer-term exposure (3 weeks to 2 months) to ≥0.5 mg/kg/day (Brewer 1940; Davis 1937; Domingo et al. 1984; Holly 1955; Krasovskii and Fridlyand 1971; Murdock 1959; Stanley et al. 1947). Of particular note is an 8-week study in rats (Stanley et al. 1947), which reported dose- and time-related increases in erythrocyte number following oral administration of cobalt chloride, with an apparent NOAEL of 0.6 mg cobalt/kg/day and a LOAEL of 2.5 mg cobalt/kg/day. Changes in the levels of other blood proteins (transferrin, several haptoglobulins, and ceruloplasmin) were noted in male Swiss mice following 4, 24, and 48 hours of treatment with 76.4 mg cobalt/kg as cobalt chloride in the drinking water (Bryan and Bright 1973). Exposure for 3 weeks or 3 months to 76.4 mg cobalt/kg as cobalt chloride in the drinking water resulted in no alterations in serum proteins examined.

Musculoskeletal Effects. No studies were located regarding musculoskeletal effects in humans after oral exposure to cobalt.

No morphological changes were found in the skeletal muscle of rats exposed to 30.2 mg cobalt/kg/day as cobalt chloride in the drinking water for 3 months (Domingo et al. 1984). This NOAEL in rats for intermediate-duration exposure is reported in Table 3-2 and plotted in Figure 3-2.

Hepatic Effects. Liver injury was evident in patients with beer-cobalt cardiomyopathy, characterized by central hepatic necrosis accompanied by increased levels of serum bilirubin and serum enzymes (serum glutamic oxaloacetic transaminase [SGOT], serum glutamic pyruvic transaminase [SGPT], lactate dehydrogenase [LDH]), creatine phosphokinase, ornithine carbamyl transferase, isocitric dehydrogenase, aldolase) (Alexander 1972; Morin et al. 1971). The hepatic injury may have resulted from ischemia, secondary to the cardiac effects of cobalt, and/or from excessive alcohol consumption. The cardiomyopathy resulted from the ingestion of beer containing 0.04 mg cobalt/kg/day as cobalt sulfate that had been added as a foam stabilizer (Morin et al. 1971). Liver function tests were found to be normal in pregnant women receiving up to 0.6 mg cobalt/kg/day as cobalt chloride for 90 days for treatment of the decreases in hematocrit and hemoglobin levels commonly found during pregnancy (Holly 1955).

Data from animals have also indicated that cobalt has hepatic effects. Hyperemia of the liver and cytoplasmic changes in hepatocytes (clumpy cytoplasm located along the cell membrane) were found in rats administered a single dose of 68.2 mg cobalt/kg as cobalt fluoride or a single dose of 157.3 mg cobalt/kg as cobalt oxide (Speijers et al. 1982).

Increased liver weight (17%) was found in rats exposed to 10 mg cobalt/kg/day (as cobalt chloride) for 5 months (Murdock 1959). No morphological or enzymatic changes were found in the livers of rats exposed to 2.5–30.2 mg cobalt/kg as cobalt chloride by gavage or as cobalt chloride in the drinking water for 3–7 months (Domingo et al. 1984; Holly 1955; Krasovskii and Fridlyand 1971).

Renal Effects. No studies were located regarding renal effects in humans after oral exposure to cobalt.

Acute and prolonged exposure to cobalt results in renal tubular degeneration in rats. Renal injury, evidenced by histologic alteration of the proximal tubules, was observed in rats after a single oral exposure to 42 mg cobalt/kg as cobalt fluoride (Speijers et al. 1982) and after exposure to 10–18 mg cobalt/kg/day as cobalt chloride for 4–5 months (Holly 1955; Murdock 1959). A slightly decreased urinary output was observed in rats exposed to 19.4 mg cobalt/kg as cobalt sulfate, but not in rats exposed to 4.25 mg cobalt/kg as cobalt chloride (Singh and Junnarker 1991).

Endocrine Effects. Roy et al. (1968) reported on 20 Québécois patients who died of beer drinkers' myocardosis. Of these, 14 thyroids were available for examination. Three of those were normal, and the other 11 formed the basis of the study. "Abnormal" thyroids did not show gross changes, but upon histologic examination, they showed irregular follicle morphology and decreased follicular size.

Kriss et al. (1955) reported on five patients who had been receiving cobalt therapy for sickle-cell anemia or renal amyloidosis. Three of five developed goiter, one severe, while four of five showed microscopic alterations of the thyroid gland. Two of the patients died from non-cobalt-related causes, while the other three recovered once cobalt treatment was removed. A similar study was reported by Gross et al. (1955) in which stable cobalt was used therapeutically in four cases of sickle-cell anemia. Treatment with cobalt resulted in an enlargement of the thyroid gland, which was reversible upon cessation of cobalt therapy. Similar effects on the thyroid, including enlargement, hyperplasia, and an increased firmness, have been reported in several other cases where cobalt therapy for anemia was used (Chamberlain 1961; Little and Sunico 1958; Soderholm et al. 1968; Washburn and Kaplan 1964). No other studies examining the endocrine effects of stable cobalt in humans were located.

NTP (1998; Bucher et al. 1999) reported increased incidence of pheochromocytoma, a tumor of the adrenal medulla, in female rats exposed to 1.14 mg cobalt/m^3 for 2 years, but did not measure any other endocrine effects. Female mice exposed to 26 mg cobalt/kg-day in the drinking water for up to 45 days showed histopathological changes to the thyroid gland (Shrivastava et al. 1996). Cobalt significantly stimulated serum testosterone in mice treated orally with 23 mg cobalt/kg as cobalt chloride, though no dose-response relationship was present (Pedigo et al. 1988).

Dermal Effects. Allergic dermatitis has been reported in some cobalt-sensitized people following oral challenge with cobalt. Several patients with eczema of the hands were challenged orally with 1 mg cobalt as cobalt sulfate given in tablet form once per week for 3 weeks (0.014 mg/kg/day). A flaring of the

eczema was considered to be a positive allergic response to cobalt (Veien et al. 1987). No other studies were located regarding dermal effects in humans or animals after oral exposure to cobalt.

Ocular Effects. Severe visual disturbances (optic atrophy, impaired choroidal perfusion) developed in a man who was treated with cobalt chloride for pancytopenia and hypercellular bone marrow (Licht et al. 1972). He received 1.3 mg cobalt/kg daily for four series of treatments with a total duration of 6 weeks. However, no other cases of visual disturbances due to therapeutic administration of cobalt have been reported, and no such effects have been observed in animals.

Body Weight Effects. No effects on body weight in animals were found following longer-term (1–5 months) exposure of rats to 10–30.2 mg cobalt/kg/day as cobalt chloride (Bourg et al. 1985; Domingo et al. 1984; Murdock 1959) or of guinea pigs to 20 mg cobalt/kg/day as cobalt sulfate (Mohiuddin et al. 1970). A significant decrease (33%) in body weight gain was observed following 8 weeks of exposure of rats to 4.2 mg cobalt/kg/day as cobalt sulfate (Clyne et al. 1988).

Metabolic Effects. Treatment of rats with 10.6 mg Co/kg/day as $CoCl_2$ in the drinking water for 12–16 days resulted in a significant decrease in serum glucose levels in diabetic rats, but not in control rats (Saker et al. 1998).

Other Systemic Effects. Hypothermia occurred in rats following a single oral dose of 157 mg cobalt/kg given as cobalt oxide or a single oral dose of 110 mg cobalt/kg given as cobalt fluoride (Speijers et al. 1982). The hypothermia was time- and dose-related. Hypothermia was reported as an effect during LD_{50} studies with other cobalt compounds, but the exact dose for the onset of hypothermia with these compounds was not reported (Speijers et al. 1982). Other physiological signs noted in LD_{50} studies include decreased activity, ataxia, diarrhea, and salivation (FDRL 1984a, 1984b).

3.2.2.3 Immunological and Lymphoreticular Effects

Cobalt is known to function as a hapten, resulting in the generation of antibodies against cobalt-protein complexes. Allergic dermatitis has been reported in some cobalt-sensitized people following oral challenge with cobalt. Several patients with eczema of the hands were challenged orally with 1 mg cobalt as cobalt sulfate given in tablet form once per week for 3 weeks (0.014 mg/kg/day). A flaring of the

eczema was considered to be a positive allergic response to cobalt (Veien et al. 1987). Using both the oral challenge test and dermal patch tests, it was determined that the cobalt allergy was systemically induced. The exposure level associated with sensitization to cobalt was not established. After sensitization, allergic reactivity may be independent of dose. Cobalt has been found to be a sensitizer following inhalation exposure (Section 3.2.1.3). This LOAEL value was not reported in Table 3-2 because sensitized individuals only represent a small percent of the population.

A case report of a 6-year-old boy who had ingested approximately 1.7 mg of cobalt chloride reported neutropenia by 7 hours post-exposure (Mucklow et al. 1990).

Thymic atrophy was reported in male Sprague-Dawley rats exposed to 3.79 mg cobalt/kg/day as cobalt chloride in the feed for 4 weeks (Chetty et al. 1979). A deterioration in immunological reactivity, manifested by a decline in phagocytic activity, was reported in rats following 6–7 months of treatment with 0.5 mg cobalt/kg or greater as cobalt chloride (Krasovskii and Fridlyand 1971). This value is presented in Table 3-2 and Figure 3-2.

3.2.2.4 Neurological Effects

No studies were located regarding neurological effects in humans after oral exposure to stable cobalt.

Several rodent studies have identified neurological effects following cobalt exposure. In Wistar rats, a single gavage dose of 4.25 mg cobalt/kg as cobalt chloride resulted in a moderate reduction in spontaneous activity, muscle tone, touch response, and respiration, while 19.4 mg cobalt/kg as cobalt sulfate caused a mild reduction the same parameters (Singh and Junnarkar 1991). In rats exposed to 4.96 mg cobalt/kg/day as cobalt chloride for 30 days in the drinking water, cobalt led to changes in sympathetically mediated contractile activity of isolated rat vas deferens (Mutafova-Yambolieva et al. 1994). Rats exposed to 6.44 mg cobalt/kg/day as cobalt nitrate in the drinking water showed an increased sensitivity and decreased maximal response to a cholinergic agonist (Vassilev et al. 1993). In rats exposed to 20 mg cobalt/kg/day as cobalt chloride for 57 days in the drinking water, cobalt enhanced behavioral reactivity to stress (the animals were less likely to descend from a safe platform to an electrified grid) (Bourg et al. 1985). Rats exposed to the same dose in the diet for 69 days showed a slower rate of lever pressing than controls, but no change in behavioral reactivity to stress (Nation et al.

1983). Longer-term exposure of rats to cobalt chloride (7 months) resulted in a significant increase in the latent reflex period at ≥0.5 mg cobalt/kg as cobalt chloride and a pronounced neurotropic effect (disturbed conditioned reflexes) at 2.5 mg cobalt/kg (Krasovskii and Fridlyand 1971).

The NOAEL value and the LOAEL value for rats for intermediate duration are reported in Table 3-2 and plotted in Figure 3-2.

3.2.2.5 Reproductive Effects

No studies were located regarding reproductive effects in humans after oral exposure to stable cobalt.

Testicular degeneration and atrophy have been reported in rats exposed to 13.3–58.9 mg cobalt/kg/day as cobalt chloride for 2–3 months in the diet or drinking water (Corrier et al. 1985; Domingo et al. 1984; Mollenhauer et al. 1985; Nation et al. 1983; Pedigo and Vernon 1993; Pedigo et al. 1988), or in mice exposed to 43.4 mg cobalt/kg/day as cobalt chloride for 13 weeks in the drinking water (Anderson et al. 1992, 1993).

The highest NOAEL and all reliable LOAEL values for rats in the intermediate-duration category are reported in Table 3-2 and plotted in Figure 3-2.

3.2.2.6 Developmental Effects

No developmental effects on human fetuses were observed following treatment of pregnant women with cobalt chloride to raise hematocrit and hemoglobin levels that are often depressed during pregnancy. Dosages up to 0.6 mg cobalt/kg/day for 90 days were given (Holly 1955). Examination of the fetuses, however, was limited to the reporting of obvious birth defects, and exposure only occurred in the final trimester.

Oral exposure of female rats to cobalt chloride at 5.4 or 21.8 mg cobalt/kg/day from gestation day 14 through lactation day 21 has been shown to result in stunted growth and decreased survival, respectively, of newborn pups (Domingo et al. 1985b). The effects on the offspring occurred at levels

that also caused maternal toxicity (reduced body weight and food consumption, and altered hematological measurements) and might therefore have been an indirect effect of maternal toxicity rather than a direct effect of cobalt on the fetus (Domingo et al. 1985b). Teratogenic effects were not observed.

Szakmary et al. (2001) reported that exposure of pregnant rats to 0–38 mg Co/kg-day as cobalt sulfate did not result in changes in fetal death rates, maternal body weigh gain, average litter size, or average fetal or placental weights; however, a dose-related trend was seen for the percent of fetuses with retarded body weights. In contrast, no effects on fetal growth or survival were found following exposure of rats to 24.8 mg cobalt/kg/day as cobalt chloride during gestation days 6–15 (Paternian et al. 1988). In mice, exposure to 81.7 mg cobalt/kg/day as cobalt chloride during gestation days 8–12 was reported to have no effect on fetal growth or mortality in mice (Seidenberg et al. 1986). In a later mouse study that exposed pregnant mice to 19 mg Co/kg-day as cobalt sulfate, no changes in litter size, postimplantation loss, or average fetal or placental weights were seen; the only difference seen was an increase in the percent of fetuses with retarded body weights (Szakmary et al. 2001). The same study reported that rabbits exposed to ≥ 38 mg Co/kg-day, as cobalt sulfate, showed nearly complete maternal lethality, and complete fetal loss. Rabbits exposed to 7.6 mg Co/kg, as cobalt sulfate, showed significant increases in mortality and fetal resorption, as well as an increase in fetuses with retarded body weight (Szakmary et al. 2001). The highest NOAEL and all reliable LOAEL values for each species and duration category are reported in Table 3-2 and plotted in Figure 3-2.

3.2.2.7 Cancer

In a survey assessing the correlation between cancer mortality and trace metals in water supplies (10 basins) throughout the United States, no correlation was found between cancer mortality and the level of cobalt in the water (Berg and Burbank 1972). Cobalt levels of 1–19 μg/L, with resulting human intakes ranging from 0.03 to 0.54 μg/kg/day, were reported.

No studies were located regarding carcinogenic effects in animals after oral exposure to stable cobalt.

3.2.3 Dermal Exposure

3.2.3.1 Death

No studies were located regarding lethal effects in humans after dermal exposure to cobalt.

No mortality was observed in guinea pigs dermally exposed to 51.75 mg cobalt/kg for 5 days/week as dicobalt octacarbonyl for a total of 18 applications (Kincaid et al. 1954).

3.2.3.2 Systemic Effects

No studies were located regarding respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, hepatic, renal, or ocular effects in humans or animals after dermal exposure to stable cobalt.

Dermal Effects. Dermatitis is a common result of dermal exposure to cobalt in humans that has been verified in a large number of studies (Alomar et al. 1985; Bedello et al. 1984; Dooms-Goossens et al. 1980; Fischer and Rystedt 1983; Goossens et al. 2001; Kanerva et al. 1988, 1998; Kiec-Swierczyńska and Krecisz 2002; Marcussen 1963; Minamoto et al. 2002; Pryce and King 1990; Swennen et al. 1993; Romaguera et al. 1982; Valer et al. 1967). Using patch tests and intradermal injections, it has been demonstrated that the dermatitis is probably caused by an allergic reaction to cobalt. Contact allergy was reported in 22 of 223 (9.9%) nurses who were tested with a patch test of 1.0% cobalt chloride (Kieć-Świerczyńska and Kręcisz 2000), as well as 16 of 79 (20.3%) of examined dentists (Kieć-Świerczyńska and Kręcisz 2002). Persons with body piercings showed an increased prevalence of allergy to cobalt, with the incidence of contact allergy being proportional to number of piercings (Ehrlich et al. 2001). The prevalence of sensitivity to cobalt following exposure to cobalt as a component of metal implants is low, with only 3.8% of patients developing a new sensitivity to cobalt following insertion of the implant (Swiontkowski et al. 2001). Exposure levels associated with the development of dermatitis have not been identified. It appears that the allergic properties of cobalt result mainly from exposure to the metal itself, rather than a salt, as Nielsen et al. (2000) demonstrated that daily repeated exposure to aqueous cobalt salts did not result in hand eczema in patients known to have cobalt allergy.

In animals, scabs and denuded areas were found after six doses of 51.75 mg cobalt/kg (5 days/week) as dicobalt octacarbonyl were applied to the shaved abdomens (uncovered area of approximately 50 cm^2) of guinea pigs (Kincaid et al. 1954). By the 11th dose, the lesions disappeared. No adverse effects were observed in vehicle controls (methyl ethyl ketone). It is not known whether or not a similar reaction would result from metallic or inorganic forms of cobalt. This LOAEL value is reported in Table 3-3.

3.2.3.3 Immunological and Lymphoreticular Effects

Cobalt-induced dermatitis is well documented in the literature, and the studies indicate that cobalt is a sensitizer (Alomar et al. 1985; Dooms-Goossens et al. 1980; Fischer and Rystedt 1983; Goh et al. 1986; Kanerva et al. 1988; Marcussen 1963; Valer et al. 1967). Patch testing and intradermal injections were performed, but exposure levels of cobalt were not reported. Interrelationships exist between nickel and cobalt sensitization (Bencko et al. 1983; Rystedt and Fisher 1983); however, the extent of any potential interactions between the two metals on immunologic end points is not well understood. In guinea pigs, nickel and cobalt sensitization appear to be interrelated and mutually enhancing (Lammintausta et al. 1985), though cross-reactivity was not reported to occur.

Single or multiple dermal exposures of BALB/c mice to $CoCl_2$ in dimethylsulfoxide or in ethanol resulted in an increased cellular proliferation in the local lymph node assay in a concentration-dependant manner (Ikarashi et al. 1992a). The effect of three consecutive exposures to varying concentrations of $CoCl_2$ in DMSO on lymph node proliferation was measured in rats, mice, and guinea pigs (Ikarashi et al. 1992b). Stimulation Indices of 3 or greater, indicated by the authors as a significant response, were reported for mice exposed to 1, 2.5, or 5% $CoCl_2$, rats exposed to 2.5 or 5% $CoCl_2$, and guinea pigs exposed to 5% $CoCl_2$; these treatments resulted in dose levels of 10.8, 27, or 54.1 mg cobalt/kg/day for mice, 9.60 or 19.2 mg cobalt/kg/day for rats, and 14.7 mg cobalt/kg/day for guinea pigs.

No studies were located regarding the following health effects in humans or animals after dermal exposure to stable cobalt:

Table 3-3 Levels of Significant Exposure to Cobalt - Chemical Toxicity - Dermal

Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL	LOAEL Less Serious	LOAEL Serious	Reference Chemical Form
ACUTE EXPOSURE						
Immuno/ Lymphoret						
Rat (Fischer- 344)	1x/d 3d		3.84 F mg/kg/day	9.6 F mg/kg/day (Increased proliferation of lymphatic cells)		Ikarashi et al. 1992b Chloride
Mouse (BALB/c)	1x or 1x/d for 3 d			10.8 F mg/kg/day (Increased proliferation of lymphatic cells)		Ikarashi et al. 1992a Chloride
Mouse CBA/N	1x/d 3 d		5.4 F mg/kg/day	10.8 F mg/kg/day (Increased proliferation of lymphatic cells)		Ikarashi et al. 1992b Chloride
Gn Pig (Hartley)	1x/d 3 d		7.39 F mg/kg/day	14.7 F mg/kg/day (Increased proliferation of lymphatic cells)		Ikarashi et al. 1992b Chloride
INTERMEDIATE EXPOSURE						
Systemic						
Gn Pig (NS)	18 d 5 d/wk	Dermal		51.75 mg/kg/day (skin lesions (scabs and denuded areas) at application site)		Kincaid et al. 1954

d =day(s); F = female; LOAEL = lowest-observed-adverse-effect level; NOAEL = no observed-adverse-effect level; wk = week(s); x = times.

3.2.3.4 Neurological Effects

3.2.3.5 Reproductive Effects

3.2.3.6 Developmental Effects

3.2.3.7 Cancer

No studies were located regarding carcinogenic effects in humans or animals after dermal exposure to cobalt.

3.2.4 Other Routes of Exposure

Endocrine Effects. Patients (n=12) injected with a single diagnostic dose of radioactive iodine, and then treated 48 hours later with 1 mg cobalt/kg/day as cobalt chloride for 2 weeks, resulted in a greatly reduced uptake of radioactive iodine by the thyroid in 1 week, with uptake nearing 0 by the second week (Roche and Layrisse 1956). When the cobalt treatment ended, the uptake values returned to normal. The decrease of radioactive iodine uptake found in patients administered 0.54 mg cobalt/kg/day for 10–25 days prior to iodine injection was found to result from cobalt blocking the organic binding of iodine (Paley et al. 1958).

In various species of animals, parenteral administration of cobalt resulted in cytotoxic effects on the alpha cells of the pancreas (Beskid 1963; Goldner et al. 1952; Hakanson et al. 1974; Lacy and Cardeza 1958; Lazarus et al. 1953; Van Campenhout 1955). Because this effect has never been reported in humans or animals following inhalation, oral, or dermal exposure to cobalt, the relevance of the effect to humans is not known.

Moger (1983) exposed primary cultures of mouse Leydig cells to 0–2.5 mM cobalt as cobalt for 3 hours, and measured the effects on androgen production. Cobalt exposure caused a dose-related decrease in both basal and LH-stimulated androgen production, with no effects on protein synthesis. The author suggested that these effects are the result of cobalt inhibition of calcium influx across the plasma membrane.

3.3 DISCUSSION OF HEALTH EFFECTS OF RADIOACTIVE COBALT BY ROUTE OF EXPOSURE

Section 3.3 discusses radiation toxicity associated with exposure to radionuclides of cobalt and is organized in the same manner as that of Section 3.2, first by route of exposure (inhalation, oral, and external) and then by health effect (death, systemic, immunological, neurological, reproductive, developmental, genotoxic, and carcinogenic effects). These data are discussed in terms of three exposure periods: acute (14 days or less), intermediate (15–364 days), and chronic (365 days or more).

Levels of significant exposure for each route and duration are presented in tables and illustrated in figures. The points in the figures showing NOAELs or LOAELs reflect the actual dose (levels of exposure) used in the studies. Refer to Section 3.2 for detailed discussion of the classification of endpoints as a NOAEL, less serious LOAEL, or serious LOAEL.

Refer to Appendix B for a User's Guide, which should aid in the interpretation of the tables and figures for Levels of Significant Exposure.

3.3.1 Inhalation Exposure

No studies were located regarding the following health effects in humans or animals after inhalation exposure to radioactive cobalt:

3.3.1.1 Death

3.3.1.2 Systemic Effects

3.3.1.3 Immunological and Lymphoreticular Effects

3.3.1.4 Neurological Effects

3.3.1.5 Reproductive Effects

3.3.1.6 Developmental Effects

3.3.1.7 Cancer

3.3.2 Oral Exposure

No studies were located regarding the following health effects in humans or animals after oral exposure to radioactive cobalt:

3.3.2.1 Death

3.3.2.2 Systemic Effects

3.3.2.3 Immunological and Lymphoreticular Effects

3.3.2.4 Neurological Effects

3.3.2.5 Reproductive Effects

3.3.2.6 Developmental Effects

3.3.2.7 Cancer

3.3.3 External Exposure

This section contains information regarding health effects related to external exposure to radioactive cobalt sources. Radionuclides of cobalt may emit beta particles and/or gamma rays, which may be a health hazard in living organisms because they ionize the atoms that they hit while passing through the tissues of the body (see Table 3-4 and Figure 3-3). Beta particles can travel appreciable distances in air, but travel only a few millimeters in solids. External exposure to beta particles may result in damage to skin and superficial body tissues at sufficiently high doses. Beta radiation is only a threat to internal organs if the radiation source is internalized. Gamma radiation, on the other hand, can easily pass completely through the human body and cause ionization of atoms in its path. For most radionuclides of public interest, the fraction of gamma rays that actually deposits energy and contributes to the radiation

Table 3-4 Levels of Significant Exposure to Cobalt - Radiation Toxicity - External Radiation

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (rad)	LOAEL Less Serious (rad)	LOAEL Serious (rad)	Reference Chemical Form
	ACUTE EXPOSURE						
	Death						
1	Human					2250 M (Death)	Stavem et al. 1985
2	Mouse (BALB/c)					627 (30-day LD50 value, single exposure)	Darwezah et al. 1988
3	Mouse CBA/Ca.Lac.C	1x				1420 M (Death)	Down et al. 1986
	Systemic						
4	Human	(occup)	Gastro	12.7			House et al. 1992
			Hemato	12.7			
5	Human		Dermal			159 M (Severe alterations to skin of left hand)	Klener et al. 1986
			Ocular			159 M (Progressive occlusion of vision of left eye)	

Table 3-4 Levels of Significant Exposure to Cobalt - Radiation Toxicity - External Radiation (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (rad)	LOAEL Less Serious (rad)	LOAEL Serious (rad)	Reference Chemical Form
6	Human		Cardio			2250 M (left ventricular hypertrophy)	Stavem et al. 1985
			Gastro			2250 M (Pronounced atrophy in intestines; less severe in stomach)	
			Hemato			2250 M (>35% decrease in hemogolbin and >90% decrease in thrombocytes)	
			Renal		2250 M (Enlarged kidneys)		
7	Monkey (Rhesus)	30 min	Cardio		1000 M (Minor changes: increased heart rate; decreased blood pressure; variable cardiac output and total peripheral resistance)		Bruner 1977
8	Monkey (Rhesus)	1 hr	Cardio			10000 M (Pronounced decreases in mean arterial blood pressure and blood flow to the brain)	Cockerham et al. 1986
9	Rat (Wistar)		Cardio		2500 M (Increased brain uptake index)		Bezek et al. 1990

Table 3-4 Levels of Significant Exposure to Cobalt - Radiation Toxicity - External Radiation (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (rad)	LOAEL Less Serious (rad)	LOAEL Serious (rad)	Reference Chemical Form
10	Rat (Sprague-Dawley)	1x	Resp			1500 M (Severe inflammation, pulmonary histopathology, fibrosis)	Lafuma et al. 1987
11	Mouse (Swiss-Webster)		Gastro		1000 M (Intestinal crypt cell damage, including necrosis and altered mitotic figures)		Devi et al. 1979
12	Mouse CBA/Ca.Lac.C	1x	Resp		1330 M (Increased breathing rate)		Down et al. 1986
			Dermal		1800 M (Mild epilation)		
13	Mouse (Swiss-Webster)	24 hr	Hepatic		1000 M (Transient decrease in total liver protein)		Mazur et al. 1991
14	Dog (Mongrel)	198 d	Cardio			4355 (Cardiac arrhythmia)	Dick et al. 1979
15	Dog (Beagle)	10.44 min	Gastro		800 (Repeated emesis)		Gomez-de-Segura et al. 1998

Table 3-4 Levels of Significant Exposure to Cobalt - Radiation Toxicity - External Radiation (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (rad)	LOAEL Less Serious (rad)	LOAEL Serious (rad)	Reference Chemical Form
16	Rabbit (New Zealand)	1x	Dermal		1730 M (Alopecia)		Cox et al. 1981
17	Pig Large White	1x	Resp		900 F (Reversible decrease in ventilation capacity)	1090 F (Irreversible decrease in ventilation capacity, histopathology, pulmonary atrophy)	Rezvani et al. 1989
18	Pig Large White	1x	Renal			874 F (50% loss in effective renal plasma flow)	Robbins et al. 1989a
19	Pig Large White	1x	Hemato		780 F (Slight decreases in erythrocytes, hemoglobin, and hematocrit)	1190 F (Severe decreases in erythrocytes, hemoglobin, and hematocrit)	Robbins et al. 1989b
			Renal		780 F (Reversible changes in effective renal plasma flow and glomerular filtration rate)	980 F (Persistent changes in effective renal plasma flow and glomerular filtration rate)	
20	Pig Large White	1x	Renal			557 F (50% loss in effective renal plasma flow)	Robbins et al. 1989c

Table 3-4 Levels of Significant Exposure to Cobalt - Radiation Toxicity - External Radiation (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (rad)	LOAEL Less Serious (rad)	LOAEL Serious (rad)	Reference Chemical Form
21	Pig Large White	1x	Renal			980 F (Progressive inflammatory and degenerative changes in the glomerulus)	Robbins et al. 1991
22	Baboon	3-4 wk, 1x/wk	Resp			3000 (Severe pulmonary fibrosis)	Collins et al. 1978
23	Ferret	2 hr	Gastro	49 M	77 M (Emesis with wretching)		King 1988
	Immuno/ Lymphoret						
24	Human	(occup)		12.7			House et al. 1992
25	Human				159 M (Minor reduction in white cell counts)		Klener et al. 1986
26	Human					2250 M (Pronounced decrease in lymphocytes and granulocytes)	Stavem et al. 1985

Table 3-4 Levels of Significant Exposure to Cobalt - Radiation Toxicity - External Radiation (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (rad)	LOAEL Less Serious (rad)	LOAEL Serious (rad)	Reference Chemical Form
27	Mouse (Swiss-Webster)	24 hr			1000 M (>50% decrease in spleen weight and protein; increased spleen acid phosphatase)		Mazur et al. 1991
	Neurological						
28	Rat (CD)	140 d		150 M	450 M (Reversible deficits in fixed-ratio behavior parameters)		Mele et al. 1988
29	Mouse (Swiss-Webster)	97 d		300 M	500 M (Reversible decreases in aggressive behavior)		Maier and Landauer 1989
30	Rabbit Burgundy fawn	12 hr			450 M (Altered firing rates and patterns of hippocampal neurons)		Bassant and Court 1978
	Reproductive						
31	Rat (Sprague-Dawley)	1x			330 M (Decreased testis weight and altered spermatogenesis, with some evidence of recovery)		Cunningham and Huckins 1978
32	Rat (Wistar)	1x			80 M (Reversible decrease in testicular weight)		Laporte et al. 1985

Table 3-4 Levels of Significant Exposure to Cobalt - Radiation Toxicity - External Radiation (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (rad)	LOAEL Less Serious (rad)	LOAEL Serious (rad)	Reference Chemical Form
	Developmental						
33	Monkey Squirrel	1x				100 (Developmental retardation, neurobehavioral deficits)	Brizzee et al. 1978
34	Rat (Sprague-Dawley)	1x				50 F (Defective eye development and spinal curvature)	Bruni et al. 1994
35	Rat	1x				260 (Testicular trophy; adrenal atrophy)	Inano et al. 1989
36	Rat (Wistar)	1x			260 M (Reduced NADPH cytochrome p450 reductase)		Inano et al. 1990
37	Rat (Wistar)	4d or 6d		11	16 (slightly decreased (2.6%) brain weight in offspring)	560 (decreased (13.1%) brain weight in offspring)	Reyners et al. 1992
38	Rat (Wistar)	1x				210 M (Testicular atrophy)	Suzuki et al. 1990

Table 3-4 Levels of Significant Exposure to Cobalt - Radiation Toxicity - External Radiation (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (rad)	LOAEL Less Serious (rad)	LOAEL Serious (rad)	Reference Chemical Form
39	Mouse (Swiss-Webster)	1x		5	10 (Decreased brain weight 3-4%, significantly increased microphthalmia)	50 (Increased fetal mortality and growth retardation)	Devi et al. 1994
40	Mouse (Swiss-Webster)	1x			25 (Decreased body weight 5%, liver weight 5%, and spleen weight 12%. Decreased spleen cellularity.)		Devi et al. 1998
41	Mouse (B6C3F1)	1x				100 M (Increased number of tumor-bearing animals after in utero exposure)	Nitta et al. 1992
42	Mouse (Swiss-Webster)	1x				200 (Atrophy or lack of development of corpus callosum)	Schmidt and Lent 1987
43	Mouse	6d				20 F (Altered neurobehavioral parameters, growth retardation)	Want et al. 1993

Table 3-4 Levels of Significant Exposure to Cobalt - Radiation Toxicity - External Radiation (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (rad)	LOAEL Less Serious (rad)	LOAEL Serious (rad)	Reference Chemical Form
44	Mouse LACA	1x				50 (Delayed development, altered hindlimb splay)	Zhong et al. 1996
45	Hamster (Golden Syrian)	1x				200 F (Severe developmental abnormalities of multiple organ systems, embryo death)	Harvey et al. 1962
46	Dog (Beagle)	1x				83 (Increased risk of thyroid neoplasia)	Benjamin et al. 1997
47	Dog (Beagle)	1x				15.6 (Increased cancer-related mortality - multiple tumor types)	Benjamin et al. 1998b
48	Dog (Beagle)	1x		16	83 (Hypodontia)		Lee et al. 1989
49	Dog (Beagle)	1x				96 (Optic atrophy/degeneration)	Schweitzer et al. 1987

Table 3-4 Levels of Significant Exposure to Cobalt - Radiation Toxicity - External Radiation (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (rad)	LOAEL Less Serious (rad)	LOAEL Serious (rad)	Reference Chemical Form
	Cancer						
50	Dog (Beagle)	1x				15.6 (Increased cancer-related mortality)	Benjamin et al. 1998b
	INTERMEDIATE EXPOSURE						
	Death						
51	Human					7500 F (Death)	Roscher and Woodard 1969
	Systemic						
52	Human		Ocular			4800 F (Progressive visual impairment and blindness)	Fishman et al. 1976
53	Human	22 - 35 d teletherapy	Cardio			4623 (Persistent pericarditis)	Martin et al. 1975
54	Human	18 d	Gastro		3600 (Loose bowel movements, impaired absorption of vitamin B12)		McBrien 1973
55	Human	17 d	Dermal		4056 F (Comedones, which were resolved with treatment)		Myskowski and Safai 1981

Table 3-4 Levels of Significant Exposure to Cobalt - Radiation Toxicity - External Radiation (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (rad)	LOAEL Less Serious (rad)	LOAEL Serious (rad)	Reference Chemical Form
56	Human		Gastro			7500 F (Severe gastrointestinal necrosis and fibrosis)	Roscher and Woodard 1969
57	Human	3 yr	Other	2400			Thibadoux et al. 1980
58	Human	7 wk	Dermal		4700 (Reversible changes in skin pigmentation)		van Oort et al. 1984
59	Rat (albino)	10 wk	Other	2400 M	4800 M (Transient alterations in incisor histopathology)	7200 M (Lasting alterations in incisor histopathology)	Sweeney et al. 1977
60	Dog (Beagle)	150-300 d	Hemato			1125 M (Aplastic anemia)	Seed et al. 1989
	Immuno/ Lymphoret						
61	Dog (Beagle)	150-300 d				1125 M (Dose- and time-related reduction in granulocytes, monocytes, and lymphocytes)	Seed et al. 1989

Table 3-4 Levels of Significant Exposure to Cobalt - Radiation Toxicity - External Radiation (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (rad)	LOAEL Less Serious (rad)	LOAEL Serious (rad)	Reference Chemical Form
	Neurological						
62	Human					4800 F (Optic nerve damage, resulting in visual impairment and blindness)	Fishman et al. 1976
63	Human	9 mo				13150 F (Neural necrosis and gliosis)	Llena et al. 1976
64	Human					5500 M (Partial paralysis secondary to radiation myelopathy) 5000[b] F (Partial paralysis secondary to radiation myelopathy)	Sanyal et al. 1979
	Reproductive						
65	Human	47 d				6600 M (Calcification of the prostate)	Keys and Reed 1980
66	Mouse	32 wk				1282 F (Decreased offspring per litter and sterility)	Searle et al. 1980
	Cancer						
67	Human	NS				1800 F (Basal cell carcinoma)	Garcia-Silva et al. 1996
68	Human	8 mo				25150 M (Multiple basal cell carcinomas)	Wollenberg et al. 1995

Table 3-4 Levels of Significant Exposure to Cobalt - Radiation Toxicity - External Radiation (continued)

Key to figure[a]	Species (Strain)	Exposure/ Duration/ Frequency (Specific Route)	System	NOAEL (rad)	LOAEL Less Serious (rad)	LOAEL Serious (rad)	Reference Chemical Form
	CHRONIC EXPOSURE						
	Systemic						
69	Human	3 yr	Cardio			13150 F (Endothelial hyperplasia, dysplasia, and fibrosis)	Llena et al. 1976

[a] The number corresponds to entries in Figure 3-3.

[b] Differences in levels of health and cancer effects between males and females are not indicated in Figure 3-3. Where such differences exits, only the levels of effect for the most sensitive gender are represented.

Bd Wt = body weight; Cardio = cardiovascular; d = day(s); Endocr = endocrine; F = female; Gastro = gastrointestinal; Hemato = hematological; hr = hour(s); LOAEL = lowest-observed-adverse-effect level; M = male; Metab = metabolism; min = minute(s); mo = month(s); NOAEL = no observed-adverse-effect level; NS = not specified; (occup) = occupational; Resp = respiratory; wk = week(s); yr = year(s).

Figure 3-3. Levels of Significant Exposure to Cobalt - Radiation Toxicity - External Radiation

Acute (≤14 days)

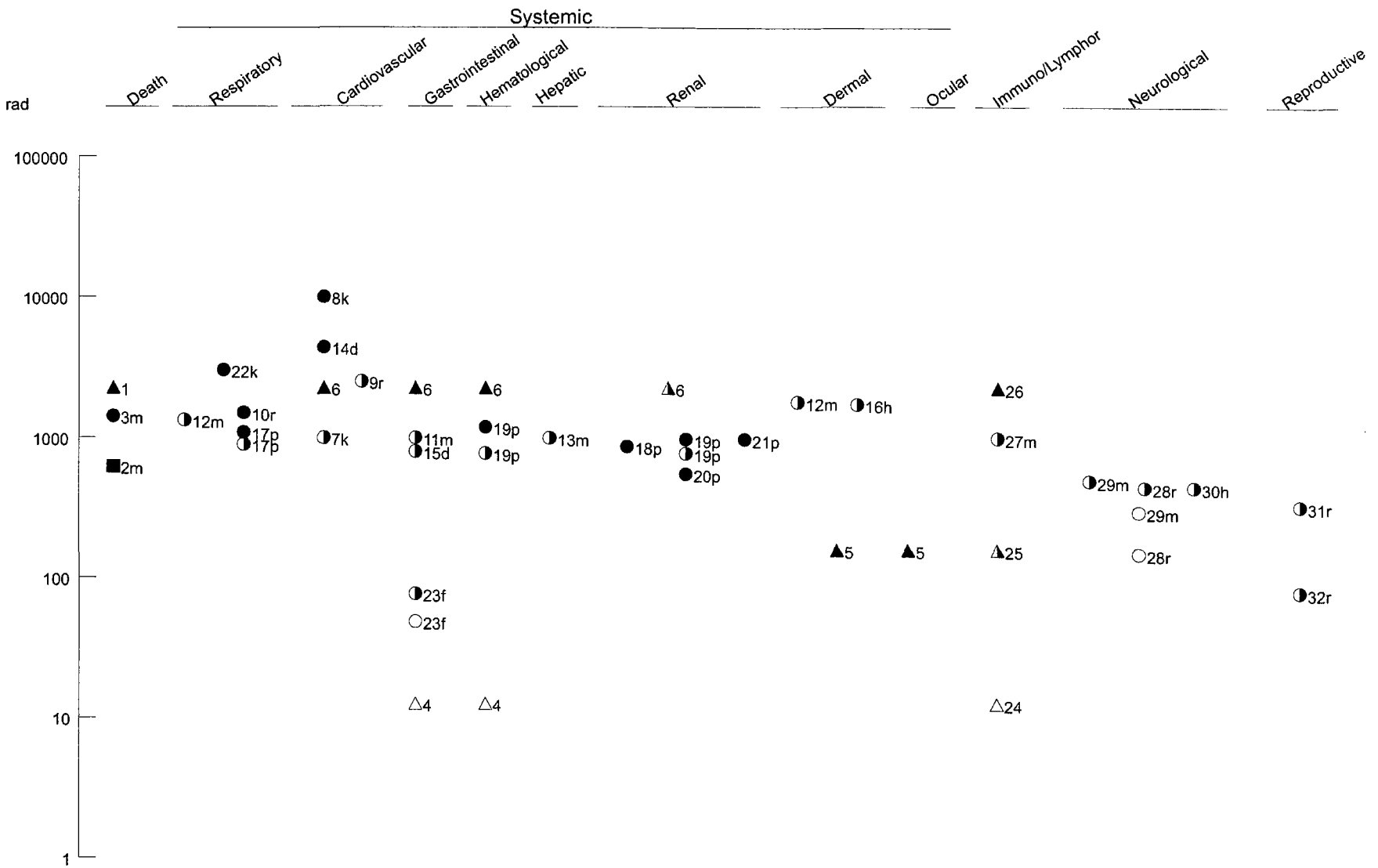
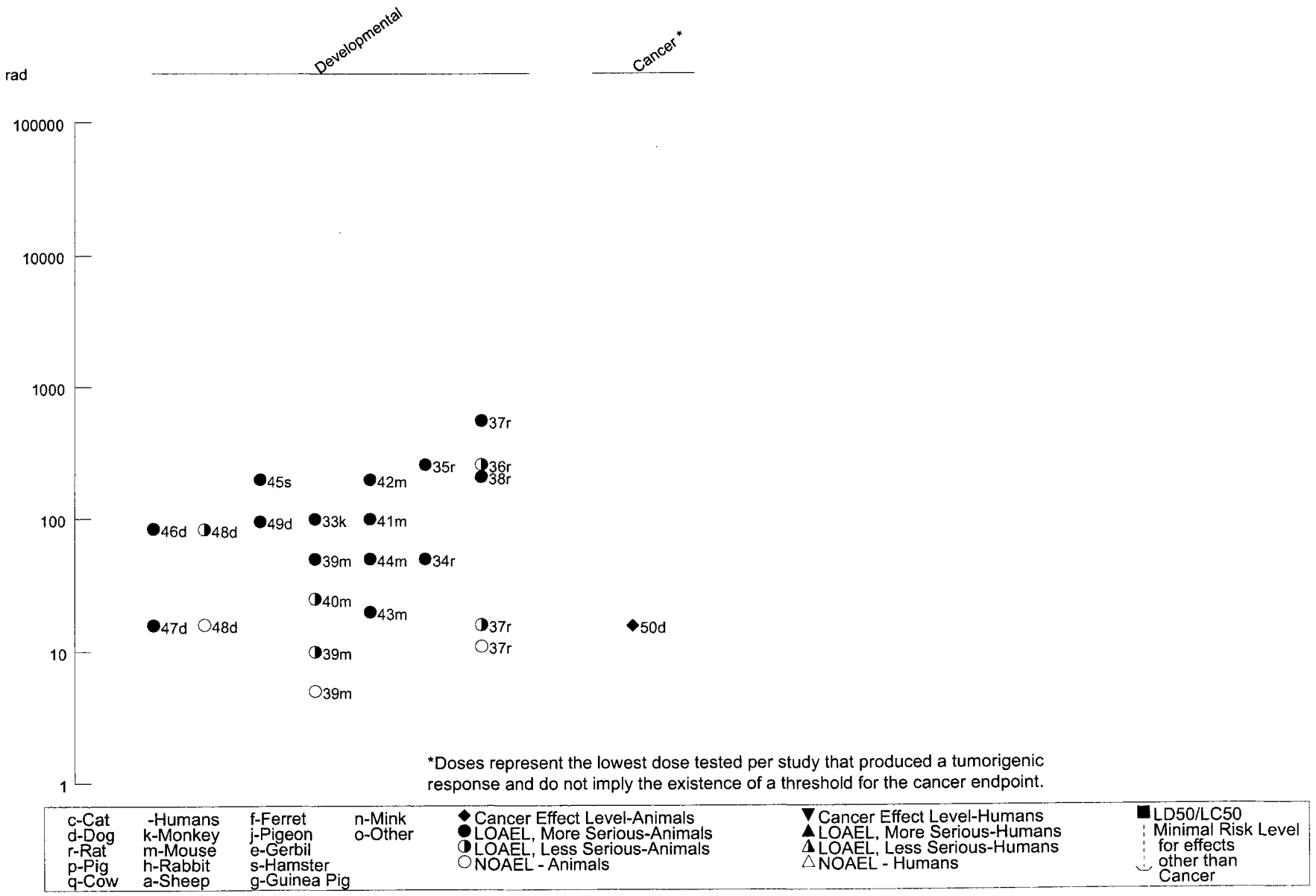


Figure 3-3. Levels of Significant Exposure to Cobalt - Radiation Toxicity - External Radiation (*Continued*)

Acute (≤14 days)



*Doses represent the lowest dose tested per study that produced a tumorigenic response and do not imply the existence of a threshold for the cancer endpoint.

c-Cat	-Humans	f-Ferret	n-Mink	◆ Cancer Effect Level-Animals	▼ Cancer Effect Level-Humans	■ LD50/LC50
d-Dog	k-Monkey	j-Pigeon	o-Other	● LOAEL, More Serious-Animals	▲ LOAEL, More Serious-Humans	Minimal Risk Level for effects other than Cancer
r-Rat	m-Mouse	e-Gerbil		◑ LOAEL, Less Serious-Animals	◭ LOAEL, Less Serious-Humans	
p-Pig	h-Rabbit	s-Hamster		○ NOAEL - Animals	△ NOAEL - Humans	
q-Cow	a-Sheep	g-Guinea Pig				

Figure 3-3. Levels of Significant Exposure to Cobalt - Radiation Toxicity - External Radiation (*Continued*)

Intermediate (15-364 days)

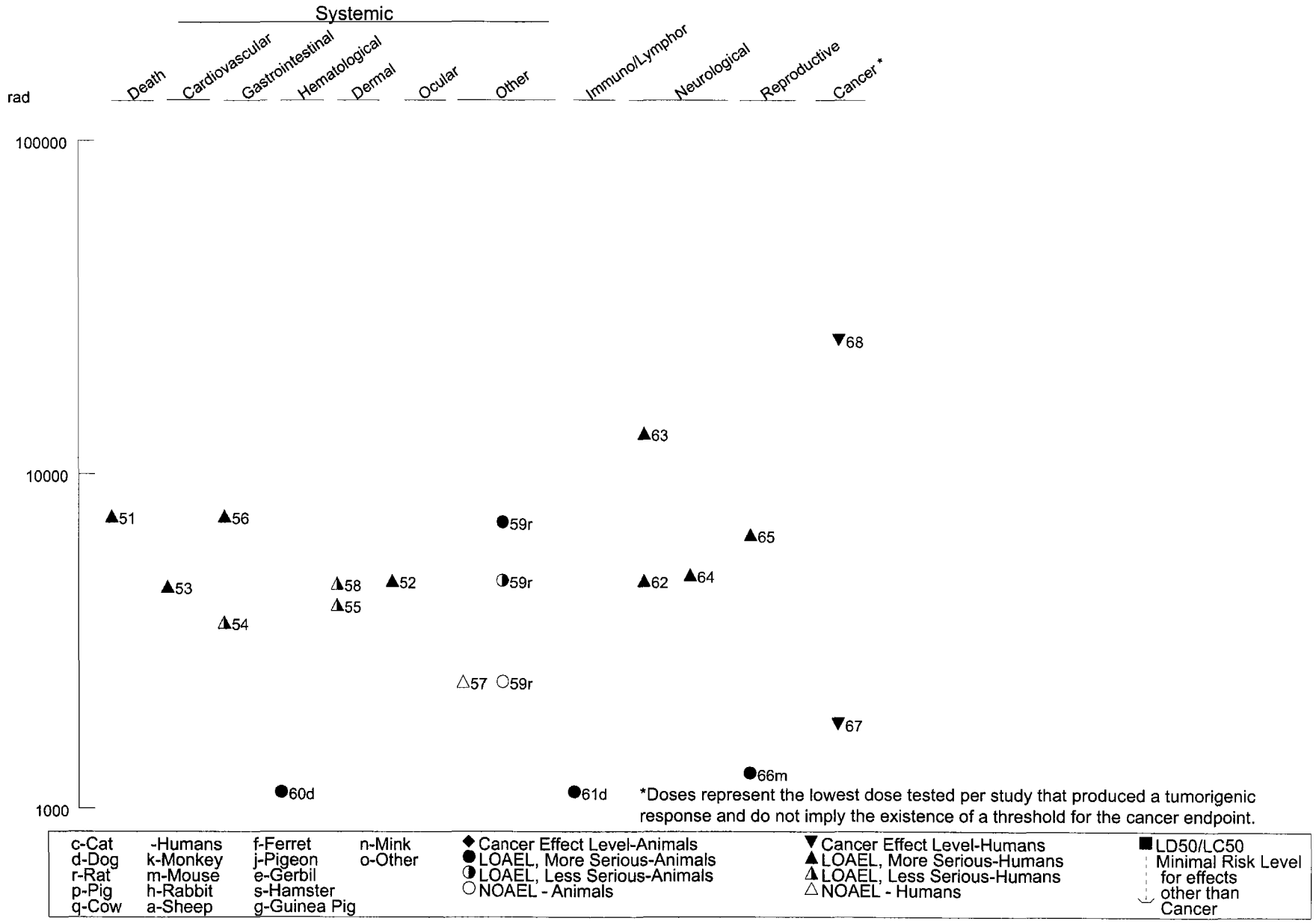
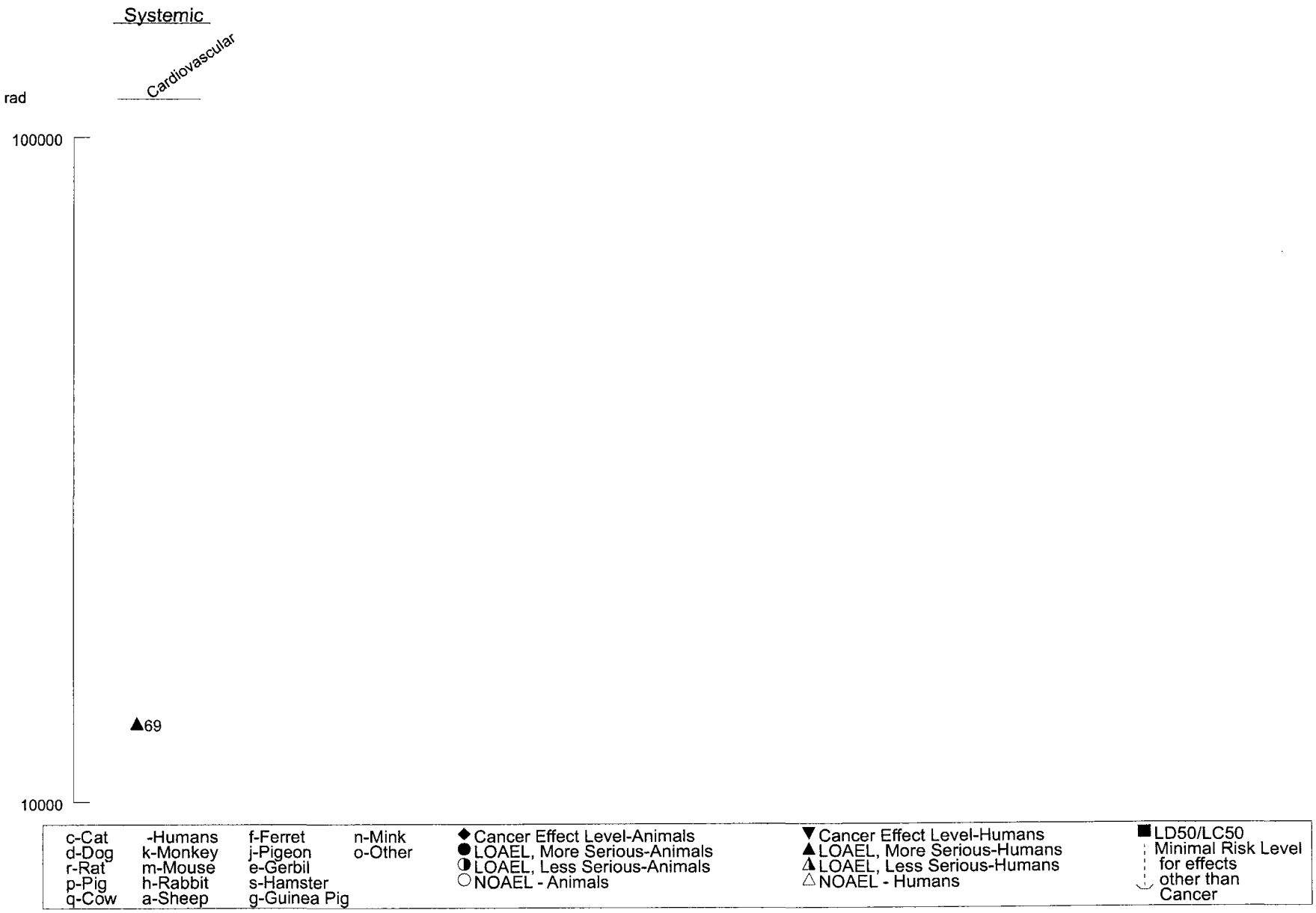


Figure 3-3. Levels of Significant Exposure to Cobalt - Radiation Toxicity - External Radiation (*Continued*)

Chronic (≥365 days)



dose increases with tissue density (resulting in a higher dose to bone than soft tissue) and decreases with energy. Several feet of concrete or a few inches of lead are typical shield thicknesses for protection from gamma rays. Because it is so highly penetrating, gamma radiation released by radionuclides such as cobalt may be a radiation hazard to internal organs (Agency for Toxic Substances and Disease Registry 1999; EPA 1997b). ^{60}Co gamma rays are commonly used for human radiotherapy. The purpose of this section is to provide information regarding health effects associated with external exposure to a radioactive cobalt source. These health effects are not specific to cobalt, but apply to any radionuclide delivering the same beta and gamma radiation dose at a comparable dose rate. Refer to Agency for Toxic Substances and Disease Registry (1999) for a detailed description of health effects from external exposure to ionizing radiation in general.

3.3.3.1 Death

Exposure to high levels of external radiation, including radiation from cobalt radionuclides, may result in mortality when the whole body dose exceeds 300 rads. Stavem et al. (1985) reported a case in which a worker was exposed to 2,250 rad (22.5 Gy) within a few minutes time, resulting in death due to acute radiation sickness (depressed leukocyte counts, vomiting, diarrhea, etc.). Complications resulting from cobalt radiotherapy resulted in the death of a patient from severe gastrointestinal complications (Roschler and Woodard 1969).

Norris and Poole (1969) reported on the mortality of dogs exposed to ^{60}Co gamma rays at a rate of 35 rad (0.35 Gy) per day for 40 days, resulting in a cumulative exposure of 1,400 rad (14 Gy). Twelve of 40 animals died prior to termination of the 40-day exposure period, 13 of 40 died within the 23-day post-exposure observation period, and 15 survived to the end of the study period, indicating an LD_{50} of <1,400 rad at 35 rad/day. Darwezah et al. (1988) reported single, whole-body exposure LD_{50} values in mice of 913 rad (9.13 Gy) and 627 rad (6.27 Gy) at 6 and 30 days post-irradiation, respectfully. Down et al. (1986) reported a slightly higher LD_{50} of 1,400–1,450 rad (14–14.5 Gy) for ^{60}Co thoracic irradiation in mice at 26 days postirradiation. Several studies have demonstrated that decreasing the dose rate or the portion of the body exposed will increase the LD_{50} for ^{60}Co gamma rays (Darwezah et al. 1988; Down et al. 1986; Hanks et al. 1966; Page et al. 1968).

3.3.3.2 Systemic Effects

Respiratory Effects. Ionizing radiation is known to exert dramatic effects on the tissue of the lung (Agency for Toxic Substances and Disease Registry 1999; Davis et al. 1992; Libshitz 1993; Roswit and White 1977), particularly at the high doses used in radiotherapy. The first phase of damage usually consists of radiation pneumonitis, which occurs between 3 and 13 weeks after irradiation and is characterized by low-grade fever, mild exertional dyspnea, congestion, and unproductive cough. The second phase is characterized by radiation-induced lung fibrosis, emphysema, and pleural thickening. Patients receiving radiotherapy treatment regimens of ≥4,000 rad (40 Gy) to the chest region almost always develop radiographic changes in the lung (Davis et al. 1992), whereas lower therapeutic doses (2,500–3,000 rad, 25–30 Gy) generally result in a lower risk of adverse pulmonary symptoms (Davis et al. 1992; Roswit and White 1977). Prophylactic protective measures may be taken, and these symptoms may be treated later if detected early enough in their progression (Roswit and White 1977).

At similar doses, studies in animals, including rats, mice, baboons, and pigs, using ^{60}Co radiation have also shown radiation pneumonitis and fibrosis, similar to effects seen in humans (Collins et al. 1978; Down et al. 1986; Lafuma et al. 1987; Rezvani et al. 1989). Other respiratory changes seen in animal experiments included an increased breathing rate, effects on the surfactant system, edema, increased pleural fluid content, pulmonary atrophy, and histologic alterations of the lung parenchyma (Bellet-Barthas et al. 1980; Collins et al. 1978; Down et al. 1986; Lafuma et al. 1987).

Cardiovascular Effects. Martin et al. (1975) reported that 24 of 81 patients who underwent ^{60}Co teletherapy for Hodgkin's disease, using an upper mantle treatment regimen of 4,000 rad (40 Gy) over 22–35 days, developed radiation-related pericarditis. In 14 of these patients, the condition was transient, while it persisted in the other 10 patients. Llena et al. (1976) presented a case wherein a 51-year-old woman who had received a localized dose of 13,150 rad (131.5 Gy) of ^{60}Co radiation between the nasopharynx and cervical lymph nodes as part of radiotherapy developed severe alterations in the endothelial cells of the brain, including proliferation, increased cytoplasmic organelles, and infoldings of the plasma membrane.

Whole-body exposure of Rhesus monkeys to 10,000 rad (100 Gy) over a 90-second period resulted in dramatic decreases in mean systemic arterial blood pressure, as well as in mean blood flow in the pons and pre-central gyrus, beginning at 10 minutes post-irradiation and persisting throughout the 60-minute

observation period (Cockerham et al. 1986). Bruner (1977) examined cardiovascular parameters in Rhesus monkeys exposed to 1,000 rad (10 Gy) at rates of 129–164 rad/minute (1.29–1.64 Gy/minute). Heart rate was elevated post-exposure, blood pressure was reduced near the end of exposure and thereafter, cardiac output increased at the end of exposure, but thereafter fell to below control levels, and total peripheral resistance to blood flow decreased at early times post-exposure, but thereafter rose to above control levels. Ten of 12 dogs irradiated with 4,355–5,655 rad (43.6–56.6 Gy), focused on the interatrial septum of the heart, developed cardiac arrhythmias (Dick et al. 1979). The permeability of the blood-brain barrier was significantly increased, particularly for hydrophillic compounds, in rats exposed to 2,500 rad (25 Gy) from a ^{60}Co source (Bezek et al. 1990).

Gastrointestinal Effects. A worker accidentally exposed to an acute whole-body dose of 2,250 rad (22.5 Gy) showed slight atrophy of the stomach glands, marked atrophy in the small intestine, and total atrophy of the glands in the large intestine (Stavem et al. 1985). Two years after a woman received ^{60}Co radiation therapy amounting to 4,000 rad (40 Gy) anteriorly and 3,500 rad (35 Gy) posteriorly over a 6-week period, she reported severe gastrointestinal difficulties, including epigastric pain, vomiting, bloody stools, and weight loss (Roschler and Woodard 1969), eventually resulting in death. Autopsy revealed dense fibrous layers around the sacrum, with severe fibrosis confirmed by microscopic examination. Cobalt radiotherapy for carcinoma of the bladder (~3,100–3,600 rad, 31–36 Gy, over 18 days) resulted in loose bowel movements and a decreased absorption of vitamin B12 following oral exposure in 8 of 14 patients (McBrien 1973). No gastrointestinal symptoms were reported in three workers who were accidentally exposed to much lower exposure levels, ranging from 2.24 to 12.7 rad (0.022–0.127 Gy) (House et al. 1992).

Exposure of male Sprague-Dawley rats to 850 rad (8.5 Gy) of ^{60}Co gamma radiation resulted in marked alterations in drug absorption, primarily due to a decrease in gastric emptying rate (Brady and Hayton 1977b). Exposure of young adult beagle dogs to 800 rad (8 Gy) of ^{60}Co radiation at a rate of 177.5 rad/minute (1.775 Gy/minute) resulted in a 100% emesis rate within 10 hours post-irradiation, with an average of 2.4 episodes per animal and an average time to emesis of 82 minutes (Gomez-d-Segura et al. 1998). King (1988a) reported a NOAEL of 49 rad (0.49 Gy) and an EC_{50} of 77 rad (0.77 Gy) for emesis and wretching following exposure of male ferrets to ^{60}Co gamma radiation. Exposure of male Swiss mice to 1,000 rad (10 Gy) of ^{60}Co radiation resulted in necrosis of the intestinal crypt cells (Devi et al. 1979).

Hematological Effects. No changes in hematologic parameters were reported in three workers who were accidentally exposed to levels ranging from 2.24 to 12.7 rad (0.022–0.127 Gy) (House et al. 1992). Hashimoto and Mitsuyasu (1967) reported that in 50 of 58 patients receiving local radiotherapy, irradiated bone marrow was more hypoplastic in the hematopoietic elements than in non-irradiated marrow in the same individual. A male worker exposed to 159 rad (1.59 Gy) showed minor reductions in leukocytes, neutrophils, and lymphocytes (Klener et al. 1986). Stavem et al. (1985) reported that a male worker exposed to 2,250 rad (22.5 Gy) showed a progressive decrease in hemoglobin and circulating thrombocytes prior to death. Autopsy showed a pronounced hypocellularity of the bone marrow.

Seed et al. (1989) exposed male Beagle dogs to 7.5 rad/day (0.075 Gy/day) gamma radiation for 150–700 days from a ^{60}Co source. The irradiated dogs initially showed a significant suppression, compared with levels from the control animals, of the five circulating types of cells studied (granulocytes, monocytes, platelets, erythrocytes, and lymphocytes), which lasted ~250 days; this was followed by a recovery phase for the remainder of the study period. Hashimoto and Mitsuyasu (1967) exposed guinea pigs to whole-body ^{60}Co radiation, and reported an initial hypoplasia of the bone marrow followed by recovery of hematopoietic activity by 3 weeks post-irradiation. Robbins et al. (1989b) reported significant reductions in erythrocyte count, hematocrit, and hemoglobin levels within 6–8 weeks of irradiation of the kidneys of female pigs with 980–1,400 rad (9.8–14 Gy) of ^{60}Co gamma rays.

Musculoskeletal Effects. No studies were located regarding musculoskeletal effects in humans or animals following external exposure to ^{60}Co radiation. These tissues are among the most radioresistant in both humans and animals.

Hepatic Effects. No studies were located regarding hepatic effects in humans following external exposure to ^{60}Co radiation.

No changes in liver weight were seen in male Swiss mice exposed to 1,000 rad (10 Gy) of ^{60}Co radiation and examined every 4 hours for 24 hours post-irradiation (Mazur et al. 1991). Andrzejewski et al. (1980) reported increased respiration rates in rat liver mitochondria after whole-body exposure to 1,000 or 3,000 rad (10 or 30 Gy) of ^{60}Co radiation; the increase was greater and more persistent at the higher dose level.

Renal Effects. Stavem et al. (1985) reported that a 64-year-old man who accidentally received a fatal dose (2,250 rad) of cobalt radiation developed enlarged kidneys. No other studies were located regarding renal effects in humans after external exposure to cobalt radiation.

Robbins et al. (1989a, 1989b, 1989c, 1991a, 1991b) performed a series of studies in female White pigs wherein the kidneys of the animals were exposed to single doses of 780–1,400 rad (7.8–14 Gy) of ^{60}Co radiation and examined for periods up to 24 weeks postirradiation. Irradiation resulted in an initial increase in glomerular filtration rate (GFR), followed by a dose-related decrease in the GFR, beginning at 4 weeks postexposure. Effective renal plasma flow (ERPF) was also decreased in a dose-related manner beginning at 4 weeks postexposure, but did not show the initial increase seen in GFR. Some recovery of GFR and ERPF occurred by 24 weeks postirradiation, though values were still significantly reduced below controls in all groups but the 780 rad (7.8 Gy) group. Histology was performed on animals exposed to 980 rad (9.8 Gy) and killed between 2 and 24 weeks after exposure. Beginning at 2 weeks postirradiation, increased numbers of inflammatory cells were present within the glomerulus, and there was an increase in mesangial matrix and number of mesangial cells. The glomerular changes continued to progress in severity throughout the observation period, with generalized thickening of the capillary walls, extensive duplication of the basement membrane, and progressive inflammation. Tubular changes appeared to be maximal at 6 weeks, including focal degeneration and necrosis, with partial recovery at later timepoints.

Endocrine Effects. Prager et al. (1972) reported that 5 of 23 patients receiving cobalt radiotherapy (3,900–4,600 rad, 39–46 Gy) for Hodgkin's disease developed hypothyroidism, with substantial decreases in levels of T4 relative to patients with normal thyroids. Chang et al. (2001) examined the residents of ^{60}Co-contaminated buildings for effects on the thyroid. There was an increased prevalence of goiter in males of all ages and females <15 years of age, as well as a dose-related increase in the prevalence of thyroid cysts in females of all ages, and elevated tri-iodothyronine levels in males <15 years of age. No other studies examining the endocrine effects of radioactive cobalt exposure, either internal or external, in humans were located.

Whole-body acute exposure of rats to 330 rad (3.3 Gy) did not affect FSH, LH, or testosterone levels (Cunningham and Huckins 1978). Similarly, male Wistar rats exposed to a single dose of 80 rad (0.8 Gy) of testicular radiation showed no changes in FSH, LH, prolactin, or testosterone (Laporte et al. 1985). No

other studies examining the endocrine effects of radioactive cobalt exposure, either internal or external, in animals were located.

Dermal Effects. Several studies in humans have demonstrated that high-dose exposure to cobalt radiation can result in damage to the skin. Klener et al. (1986) described the accidental irradiation of a worker who was attempting to bring under control a sealed ^{60}Co source. The patient's left palm (the patient was left-handed) developed an irregular oval defect 3x4 cm with whitish edges and bleeding, as well as superficial lesions on the third and fourth finger. Considerable spontaneous pain required the administration of analgesics. The lesions showed no tendency to heal, instead spreading to the adjacent digits. After several failed skin graft attempts, the condition worsened, necessitating the amputation of fingers five through two. Walter (1980) reported that a patient who had undergone ^{60}Co radiotherapy (dose not reported) of the forehead and scalp developed a pronounced acneform reaction, characterized primarily by alopecia with multiple open comedones on the scalp and forehead, and hair loss. With treatment, the comedones were 80% cleared at 9 months post-diagnosis (13 months post-treatment), but no hair regrowth was noted. Myskowski and Safai (1981) have likewise reported localized comedones in a patient following 4,056 rad (40.6 Gy) of ^{60}Co radiotherapy. Van Oort et al. (1984) reported that patients receiving 4,700–6,000 rad (47–60 Gy) of ^{60}Co radiotherapy over a 7-week period showed significant differences in baseline color of the skin, primarily erythema, and pigmentation, beginning the third week of exposure and persisting throughout the fifth week postirradiation (study week 12). Johansson et al. (2000) reported that 86% of women who had been treated with 5,400–5,700 rad (54–57 Gy) after a radical mastectomy developed fibrosis of the skin of the treated area.

Cox et al. (1981) reported a dose-related loss of hair in rabbits exposed to 1,730–3,210 rad (17.3–32.1 Gy) ^{60}Co gamma rays, targeted at the skin near the eyes or of the ears, with recovery initially noted in animals exposed to 2,140 rad (<21.4 Gy) by day 200 postirradiation. Beginning at day 500 postirradiation, a substantial loss of hair again was seen, persisting throughout the end of the study. Mice exposed to 1,800 rad (18 Gy) of ^{60}Co radiation showed a slight increase in epilation score (Down et al. 1986).

Ocular Effects. Exposure to high-dose radiation from cobalt sources has been shown to result in effects on the eye, in particular the development of cataracts. Augsburger and Shields (1985) described 13 patients who developed cataracts following ^{60}Co plaque radiotherapy; estimated doses to the eyes ranged from 2,000 to 10,000 rad (20–100 Gy). Fishman et al. (1976) reported on two patients who

received head-only 60Co radiotherapy, in combination with chemotherapy, for the treatment of acute lymphocytic leukemia. Both patients, who received 2,400 rad (24 Gy) over an initial 16-day course of treatment followed later by either 2,400 or 2,500 rad (24 or 25 Gy) in followup therapy, developed progressively severe vision disorders, resulting in partial or total blindness. Exposure of a male worker to a whole-body dose of 159 rad (1.59 Gy) of ^{60}Co radiation resulted in a progressive deterioration of visual acuity, due to cataract development, in the left eye (which was more exposed than the right) over time (Klener et al. 1986). Chen et al. (2001) evaluated subjects that had been exposed to 120–194 mSv (range: 1.11–1493.4 mSv) for an undisclosed period of time for lenticular opacities. Subjects <20 years old showed a dose-dependent increase in the numbers of focal lens defects, while for those aged 20–40 and >40, no such statistical correlation was seen.

Other Systemic Effects. Thibadoux et al. (1980) reported that of 61 children receiving a course of 2,400 rad (24 Gy) of cranial radiotherapy, none developed significant reductions in hearing levels by the end of the third year after irradiation.

Taiwanese children (48 boys, 37 girls) who were raised in apartments contaminated with ^{60}Co were compared to 21,898 age- and sex-matched nonexposed children from a nationwide surveillance program (Wang et al. 2001). After adjusting for effects from parental heights and body mass index, clear dose-related decreases in height percentile (HP) and age-specific relative height differences (RHD) were seen in exposed boys, but not in exposed girls. Average cumulative doses were 120.8 and 129.9 mSv for the boys and girls, respectively.

Sweeney et al. (1977) examined the effects of 60Co radiation on the teeth of rats exposed to 0, 2,400, 4,800, or 7,200 rad (0, 24, 48, or 72 Gy). Animals exposed to 4,800 rad (48 Gy) showed transient effects on the incisors only, while at 7,200 rad (72 Gy), the effects lasted throughout the 10-week study period.

3.3.3.3 Immunological and Lymphoreticular Effects

A worker accidentally exposed to an acute dose of 2,250 rad (22.5 Gy) showed a rapid fall in circulating lymphocytes and granulocytes prior to death (Stavem et al. 1985). Chronic exposure to low amounts of ^{60}Co radiation in people living in a contaminated building significantly reduced the numbers of circulating CD4+ lymphocytes in the blood (Chang et al. 1997, 1999b); mean total radiation dose was estimated to be

0.169 Gy (16.9 rad) over a 2–13-year period. Similarly, children chronically-exposed to low levels (estimated dose of 0.002–0.085 Gy [0.2–8.5 rad]) of ^{60}Co radiation in a contaminated kindergarten building showed significant decreases in total leucocytes and neutrophils, but an increase in eosinophils, 5–7 years after exposure had ceased (Chang et al. 1999a).

In male Swiss mice exposed to 1,000 rad (10 Gy) of ^{60}Co radiation, significant decreases in weight of the spleen were seen as early as 1 hour post-exposure and persisted throughout the following 24 hours (Mazur et al. 1991). Spleen acid phosphatase activity, expressed as activity per gram of protein, was significantly increased in irradiated animals beginning at 13 hours post-exposure.

3.3.3.4 Neurological Effects

Exposure of both humans and animals to high doses of cobalt radiation has been shown to result in damage to nervous tissue, particularly peripheral nerves. Llena et al. (1976) presented a case wherein a 51-year-old woman who had received 13,150 rad (131.5 Gy) of 60Co radiation between the nasopharynx and cervical lymph nodes as part of radiotherapy developed focal necrosis of the brain in the frontal lobe, as confirmed by gross and microscopic examination. Fishman et al. (1976) reported on two patients who received head-only ^{60}Co radiotherapy, in combination with chemotherapy, for the treatment of acute lymphocytic leukemia. Both patients, who received 2,400 rad (24 Gy) over an initial 16-day course of treatment followed later by either 2,400 or 2,500 rad (24 or 25 Gy) in followup therapy, developed progressively severe vision disorders, resulting in partial or total blindness. Histopathology from one patient demonstrated severe alterations in the optic nerve, including severe atrophy, terminal beading, lack of myelin, and calcification. Sanyal et al. (1979) reported on five patients who received doses of 4,500–6,000 rad (45–60 Gy) ^{60}Co radiation as radiotherapy, who developed varying degrees of myelopathy, resulting in minimal to mild paralysis. In patients that had been treated with ^{60}Co radiation (total dose of 54–57 Gy, or 5,400–5,700 rad) following mastectomy, 63% developed brachial plexus neuropathy and 5% developed vocal chord paresis over the 30-year period reported by the study (Johansson et al. 2000).

Mele et al. (1988) exposed male rats to 50, 150, or 450 rad (0.5, 1.5, or 4.5 Gy) 3 times, at 43-day intervals, and examined them for changes in behavior daily for 30 days following each exposure. Rats exposed to 450 rad (4.5 Gy), but not those exposed to 150 rad (1.5 Gy) or 50 rad (0.5 Gy), showed

significant deficits in fixed-ratio response rates and running rates after each exposure, beginning the day after exposure and persisting for 4–5 days, after which both rates returned to normal. After the third exposure, all rats were exposed to 650 rad (6.5 Gy), which resulted in similar performance decrements as were seen in the 450 rad (4.5 Gy) animals, again beginning 24 hours after exposure, with previous exposure resulting in no differences in behavioral parameters. Maier and Landauer (1989) reported significant decreases in offensive behavior in mice acutely exposed to whole-body doses of 500 or 700 rad (5 or 7 Gy), but not those exposed to 300 rad (3 Gy), with changes occurring in the second week postirradiation and responses returning to normal by day 19 postirradiation. Rabin et al. (1998) reported that exposure of rats to ^{60}Co radiation (up to 30 Gy or 3,000 rad) showed a dose-related decrease in the acquisition of controlled taste aversion behavior. Bassant and Court (1978) reported that rabbits exposed to 450 rad (4.5 Gy) of ^{60}Co radiation whole-body showed an altered activity of hippocampal cells, with a slowed mean discharge rate and increased interspike variability persisting for at least 12 hours postirradiation.

3.3.3.5 Reproductive Effects

Ionizing radiation in general, and gamma-emitting isotopes in particular, is known to have profound effects on reproductive tissues, with effects seen primarily in rapidly-dividing germ cells resulting in temporary or permanent sterility in both sexes, as well as other effects (Agency for Toxic Substances and Disease Registry 1999). These effects are usually observed only at high radiation doses. Keys and Reed (1980) reported a case of a man who, as treatment for a prostate tumor, received an estimated dose of 6,600 rad (66 Gy) to the prostate over a 47-day period, and who later developed a severe prostatic calcification necessitating surgical correction.

^{60}Co radiation at high doses has been shown to elicit profound decrements in reproductive ability in animal species. Whole-body acute exposure of rats to 330 rad (3.3 Gy) decreased testicular weights beginning at 22 days postirradiation, with recovery of testicular weight beginning about day 65 (Cunningham and Huckins 1978). Histologic examination of the testes revealed destruction of the spermatogonial population, with a slow recovery as the spermatogonial population was rebuilt from the surviving stem cells. Searl et al. (1976) reported that exposure of male mice to 1,128 rad (11.3 Gy) over a 28-week period resulted in significant reductions of testis mass and epididymal sperm count. Male Wistar rats exposed to a single dose of 80 rad (0.8 Gy) to the testes showed increased tubular fluid production

and decreased testicular weight at 30 and 45 days postirradiation, but not at later time points (Laporte et al. 1985). Single doses of >100 rad (1 Gy) of ^{60}Co radiation caused decreased fertility in exposed female mice (Philippe 1975). Continuous exposure of female mice to an average daily dose of 8 or 16 rad/day (0.08 or 0.16 Gy/day) caused a decreased number of offspring per litter and decreased reproductive performance, with 100% sterility occurring at 32 weeks of exposure at 8 rad/day (0.08 Gy/day) or 20 weeks of exposure at 16 rad/day (0.16 Gy/day) (Searl et al. 1980). Female rabbits exposed to 400 rad (4 Gy) prior to implantation showed dramatic decreases in implantation (Chang et al. 1963).

3.3.3.6 Developmental Effects

No studies were located regarding developmental effects in humans after external exposure to cobalt radiation.

In utero exposure to cobalt radiation has been extensively studied in animal species, and may elicit substantial effects across many organ systems of the developing organism. Effects have been noted following single-dose exposures as low as 10 rad (0.10 Gy) in mice (Devi et al. 1994; Wang et al. 1993), 50 rad (0.5 Gy) in rats (Bruni et al. 1994), 200 rad (2 Gy) in hamsters (Harvey and Chang 1962), 250 rad in rabbits (Chang et al. 1963), 15.6 rad (0.16 Gy) in dogs (Benjamin et al. 1998a, 1998b), and 100 rad (1 Gy) in monkeys (Brizzee et al. 1978). Organs known to be affected include the brain (Brizzee et al. 1978; Bruni et al. 1994; Devi et al. 1994; Hamilton et al. 1989; Reyners et al. 1992; Schmidt and Lent 1987), eyes (Brizzee et al. 1978; Bruni et al. 1994; Schweitzer et al. 1987), hair (Hirobe 1994; Hirobe and Zhou 1990), kidney (Benjamin et al. 1998a; Brizzee et al. 1978), liver (Devi et al. 1998), ovaries (Inano et al. 1989), pituitary (Brizzee et al. 1978), skeleton (including cleft palate, shortened digits, fused digits, and other gross abnormalities) (Bruni et al. 1994; Chang et al. 1963; Harvey and Chang 1962), spleen (Devi et al. 1998), teeth (Lee et al. 1989), testes (Inano et al. 1989; Suzuki et al. 1990), and thyroid (Benjamin et al. 1997). ^{60}Co radiation *in utero* has also shown to cause functional alterations, including postnatal growth retardation (Wang et al. 1993; Zhong et al. 1996), neurobehavioral changes (Brizzee et al. 1978; Wang et al. 1993), hormonal production (Brizzee et al. 1978; Inano et al. 1989; Suzuki et al. 1990), alterations in hepatic enzymes (Inano et al. 1990), and diabetes mellitus (Benjamin et al. 1998a). *In utero* irradiation with cobalt also leads to increased tumor incidence later in life (Benjamin et al. 1991, 1997, 1998b; Nitta et al. 1992).

Devi et al. (1994) exposed pregnant mice to a single dose of 0–50 rad (0–0.50 Gy) of ^{60}Co radiation on day 11.5 of gestation. A significant decrease in pup brain weight and an increase in the incidence of microphthalmia was seen at 10 rad (0.10 Gy), with decreases in head width, head length, body length, and body weight occurring at higher doses. A later study (Devi et al. 1998) found decreases in body weight, liver weight, and spleen weight in pups 72 hours after irradiation with 25 rad (0.25 Gy) of ^{60}Co radiation on day 17 of gestation. Male offspring, but not female offspring, of mice exposed to 50 rad (0.5 Gy) on gestation day 9 showed decreased body weights on postnatal days 0, 3, and 7, while offspring of both sexes showed delays in pinna detachment, incisor eruption, eye opening, and testes descent (Zhong et al. 1996). Wang et al. (1993) reported that mice exposed to a cumulative *in utero* dose of 10 rad (0.10 Gy) showed alterations in visual placing reflex tests, while those exposed to 20 or 40 rad (0.20 or 0.40 Gy) showed decreased mean body weight, delayed eye opening, and alterations in the air righting reflex.

Rats exposed to 50 rad (0.50 Gy) of ^{60}Co radiation on gestational day 9.5 showed histologic damage to the neuro-epithelium 4 hours post-exposure, with abnormal flexion of the embryo and abnormal flexion of the head at 48 hours post-exposure (Bruni et al. 1994). At birth, rats showed increased incidence of defective eye development, spinal curvature, and visceral anomalies. Reyners et al. (1992) reported decreased brain weight in 3-month-old rats that had been exposed to cumulative doses of 160 rad (1.6 Gy) over gestation days 12–16 or 170 rad (1.7 Gy) over gestation days 14–20. Male rats exposed to 210 rad (2.1 Gy) on day 20 of gestation showed atrophy of the testes, prostates, and seminal vesicles, as well as a complete disappearance of germinal cells within the testes, on postnatal day 70 (Suzuki et al. 1990). Inano et al. (1989) exposed rats on gestation day 20 to 260 rad (2.6 Gy) of ^{60}Co radiation. Seminiferous tubules of male offspring and ovaries of female offspring showed pronounced atrophy, and steroid hormone production was significantly altered.

Benjamin et al. (1997, 1998a, 1998b) exposed groups of pregnant Beagle dogs to 15.6–17.5 or 80.8–88.3 rad (0.15–0.175 or 0.8–0.88 Gy) of ^{60}Co radiation on day 8, 28, or 55 post-breeding. Animals were allowed to live their full life span and were observed for radiation-related illnesses and cause of death. No change in the mean age at death was seen as a result of exposure. Males exposed to either exposure level at day 55 post-breeding, but not females at any time or males exposed at days 8 or 28, showed an increase in deaths due to renal disease. High-dose females exposed on days 28 or 55 showed an increase in the frequency of diabetes mellitus. Both sexes showed an increase in malignant neoplasias in general when exposed to radiation at 8 or 55 days postcoitus, but not at 28 days, while females exposed on day 55 also showed an increase in lymphoid neoplasia. A similar exposure on day 28 or 55 postcoitus

also resulted in a dose-dependent decrease in brain weight (Hamilton et al. 1989). *In utero* radiation of dogs to higher doses (100–380 rad [1–3.8 Gy]) resulted in retinal dysplasia and atrophy (Schweitzer et al. 1987).

3.3.3.7 Cancer

The carcinogenic effects of high doses of ionizing radiation have been well documented (Agency for Toxic Substances and Disease Registry 1999), though the effects of lower doses are less clearly defined. Duncan et al. (1977) reported on a cohort of patients who had received radiotherapy for carcinoma of the cervix. Eight of 2,674 patients developed bladder tumors within 6 months to 20 years following irradiation; the incidence rate was over 57 times greater than the general female population. All eight patients had received high (therapeutic) doses of ^{60}Co irradiation, though five of the eight also received radium therapy in conjunction with ^{60}Co irradiation. Wollenberg et al. (1995) presented a case of a 55-year-old farmer who received a total of 25,150 rad (251.5 Gy) distributed over six areas of the body over an 8-month period as a ^{60}Co teletherapy treatment regimen. Twenty years after irradiation, the patient developed a total of 43 basal cell carcinomas of the skin over the treated areas, all of which were successfully removed with cryosurgery. A 2-year-old girl exposed to 1,800 rad (18 Gy) of ^{60}Co radiation as part of a treatment regimen for acute lymphoblastic leukemia L1 developed, at age 12, a basal cell carcinoma of the scalp (Garcia-Silva et al. 1996). Three patients receiving cobalt irradiation as part of a chemotherapy/radiation treatment developed basal cell carcinoma of the scalp 8–15 years after treatment in the area of radiation treatment (Dinehart et al. 1991).

3.3.4 Other Routes of Exposure

This section includes injection and *in vitro* studies that provide evidence for the biological basis of toxicity of stable and radioactive cobalt in humans and animals. Since these studies are not directly relevant to general population exposure conditions, no LSE tables have been created for this section.

3.4 GENOTOXICITY

Stable Cobalt. No studies were located regarding genotoxic effects in humans following oral or dermal exposure to cobalt. No studies were located regarding genotoxic effects in animals following inhalation exposure to cobalt.

Gennart et al. (1993) examined a cohort of 26 male workers who had been occupationally-exposed to cobalt, chromium, nickel, and iron. Analysis of variance on sister-chromatid exchange rank values revealed that exposure status (exposed vs. controls) and smoking habits had statistically significant effects. De Boeck et al. (2000) reported no significant change in the comet assay on lymphocytes from nonsmoking workers who had been occupationally exposed to cobalt or hard metal dusts; a positive association was found between hard metal exposure and increased micronucleus formation in smokers only.

Single oral exposure of male Swiss mice to 0, 4.96, 9.92, or 19.8 mg cobalt/kg as cobalt chloride resulted in significantly increased percentages of both chromosomal breaks and chromosomal aberrations in bone marrow cells, with significant linear trends toward increasing aberrations with increased exposure (Palit et al. 1991a, 1991b, 1991c, 1991d).

Results of genetic testing of cobalt are presented in Table 3-5. Several different forms of cobalt, including cobalt chloride and cobalt sulfide, were tested. No profound differences were found among the various forms.

Cobalt was found to be generally nonmutagenic in bacteria (*Salmonella typhimurium*, *Escherichia coli*) and yeast when compounds with a valence state of II were tested (Arlauskas et al. 1985; Fukunaga et al. 1982; Kanematsu et al. 1980; Kharab and Singh 1985; Ogawa et al. 1986; Singh 1983; Tso and Fung 1981). A very weak mutagenic response was found with *Bacillus subtilis* (Kanematsu et al. 1980). A mutagenic response to cobalt was found, however, when compounds with a valence state of III were tested in *S. typhimurium* and *E. coli* (Schultz et al. 1982). The authors suggested that this may be due to the formation of cobalt(III) complexes that are inert to ligand substitution, allowing optimal interaction of cobalt with genetic material (Schultz et al. 1982). Other studies have shown cobalt to be a comutagen in combination with 4-substituted pyridines in *S. typhimurium* (Ogawa et al. 1988). It has been reported that cobalt acts as an antimutagen in bacterial (*S. typhimurium*, *B. subtilis*, *E. coli*) and yeast test systems

Table 3-5. Genotoxicity of Cobalt *In Vitro*

Species (test system)	End point	Results: With activation	Results: Without activation	Reference	Valence state
Stable Cobalt					
Prokaryotic organisms:					
Salmonella typhimurium (plate incorporation)	Gene mutations	No data	–	Tso and Fung 1981	II
S. typhimurium (plate incorporation)	Gene mutations	No data	–	Arlauskas et al. 1985	II
S. typhimurium (plate incorporation)	Gene mutations	No data	–	Ogawa et al. 1986	II
S. typhimurium (plate incorporation)	Gene mutations	No data	+	Schultz et al. 1982	III
Bacillus subtilis (rec assay)	Gene mutations	No data	(+)	Kanematsu et al. 1980	II
Escherichia coli (reversion assay)	Gene mutations	No data	–	Kanematsu et al. 1980	II
E. coli (repair assay)	DNA damage	No data	+	Schultz et al. 1982	III
Eukaryotic organisms:					
Fungi:					
Saccharomyces cerevisiae (plate assay)	Reversion	No data	–	Kharab and Singh 1985	II
S. cerevisiae (plate assay)	Reversion	No data	–	Fukunaga et al. 1982	II
S. cerevisiae (plate assay)	Reversion	No data	–	Singh 1983	II
S. cerevisiae (plate assay)	Conversion	No data	+	Kharab and Singh 1985	II
S. cerevisiae (plate assay)	Conversion	No data	+	Fukunaga et al. 1982	II
S. cerevisiae (plate assay)	Conversion	No data	+	Singh 1983	II
Mammalian cells:					
Hamster ovary cells	Clastogenic effects	No data	+	Hamilton-Koch et al. 1986	II
Hamster embryo cells	Transformation	No data	+	Costa et al. 1982	II
Human lymphocytes	Sister chromatid exchange	No data	+	Andersen 1983	II
Human HeLa cells	Inhibition of DNA synthesis	No data	+	Painter and Howard 1982	II
Human diploid fibroblasts	DNA damage	No data	+	Hamilton-Koch et al. 1986	II

Table 3-5. Genotoxicity of Cobalt *In Vitro*

Species (test system)	End point	Results: With activation	Results: Without activation	Reference	Valence state
Radioactive Cobalt					
Mammalian cells:					
Chinese hamster ovary cells	DNA amplification	No data	+	Luecke-Huhle et al. 1986	N/A
Hamster embryo cells	DNA amplification	No data	+	Luecke-Huhle et al. 1990	N/A
Mouse lymphosarcoma cells	Chromosomal aberrations	No data	+	Juraskova and Drasil 1987	N/A
Mouse lymphosarcoma cells	Sister-chromatid exchanges	No data	+	Juraskova and Drasil 1987	N/A
Human lymphocytes	Chromosomal aberrations	No data	+	Koksal et al. 1995	N/A
Human lymphocytes	Micronucleus formation	No data	+	Koksal et al. 1996	N/A
Human leukocytes	DNA strand breaks	No data	+	Rueff et al. 1993	N/A
Human leukocytes	Chromosomal aberrations	No data	+	Rueff et al. 1993	N/A
Human leukocytes	Chromosome breaks	No data	+	Lindahl-Kiessling et al. 1970	N/A
Human fibroblasts	Transformation	No data	+	Namba et al. 1981	N/A
Human fibroblasts	Transformation	No data	+	Namba et al. 1985	N/A
Human fibroblasts	DNA strand breaks	No data	+	Coquerelle et al. 1987	N/A
Human fibroblasts	Transformation	No data	+	Namba et al. 1988	N/A
Human fibroblasts	Retinoblastoma gene alterations	No data	+	Endo et al. 1993	N/A
Human fibroblasts	DNA strand breaks	No data	+	Dolling et al. 1998	N/A
Human kidney cells	DNA strand breaks	No data	+	Feinendegen et al. 1977	N/A
Human kidney cells	DNA strand breaks	No data	+	Feinendegen et al. 1978	N/A

DNA = deoxyribonucleic acid; + = positive results; – = negative results; (+) = weakly positive results

(*Saccharomyces cerevisiae*) (Inoue et al. 1981; Kada et al. 1986; Kuroda and Inoue 1988). A possible explanation was that cobalt acts by correcting the error-proneness of deoxyribonucleic acid (DNA) replicating enzymes by improving their performance in DNA synthesis (Inoue et al. 1981; Kada et al. 1986; Kuroda and Inoue 1988). However, cobalt has also been shown to increase the frequency of genetic conversions in *S. cerevisiae* (Kharab and Singh 1985; Singh 1983). The reasons for this apparent dichotomy in yeast cells is not known.

In contrast to the results seen in bacteria, stable cobalt compounds were generally found to be genotoxic or mutagenic in mammalian assay systems. Exposure to cobalt compounds (metal, salts, or hard metal) has been shown to produce clastogenic effects in mammalian cells, including human lymphocytes (Anard et al. 1997; Hamilton-Koch et al. 1986; Painter and Howard 1982); transformation in hamster cells (Costa et al. 1982); sister chromatid exchanges in human lymphocytes (Andersen 1983); and micronucleus formation in mouse bone marrow cells (Suzuki et al. 1993) and human lymphocytes (Capomazza and Botta 1991; Olivero et al. 1995; Van Goethem et al. 1997). Hard metal is generally more genotoxic in *in vitro* tests than other cobalt compounds. Cobalt ions are also thought to inhibit DNA repair in mammalian cells by interaction with zinc-finger proteins involved in DNA excision repair (Asmuß et al. 2000; De Boeck et al. 1998; Hartwig et al. 1991; Kasten et al. 1997; Sarkar 1995).

Thirty hours following single intraperitoneal injection of cobalt(II) chloride in BALB/c mice, an increase in micronucleus formation was seen at 12.4 or 22.3 mg cobalt/kg (as cobalt chloride), but not at 6.19 mg/kg (Suzuki et al. 1993). Single injection of mg cobalt/kg (as cobalt chloride) resulted in significantly increased micronucleus formation at 24 hours post-injection, but not at 12, 48, 72, or 96 hours. Two or 10 days following intraperitoneal injection of male and female F344 rats with 3 or 6 mg cobalt/kg, increased levels of oxidatively-damaged DNA bases were noted in the liver, kidney, and to a lesser extent, the lung (Kasprzak et al. 1994).

Radioactive Cobalt. The ability of ionizing radiation to induce genotoxic damage is well-documented (Agency for Toxic Substances and Disease Registry 1999). Chang et al. (1999c) reported increased micronucleus frequency, both of single and multiple nucleates, in 48 people who had been exposed to 12–1,600 rad (0.12–16 Gy) over a 2–10-year period as a result of a building contaminated with ^{60}Co-containing steel. Subjects who had left the building showed a decrease in micronucleus formation that correlated with time since cessation of exposure. Three workers accidentally exposed to 2.2–12.7 rad (0.022–0.127 Gy) showed no elevation in frequency of chromosome alterations (House et al. 1992). Ten

children who received chemotherapy and 1,725–2,405 rad (17.25–24.05 Gy) as cobalt radiotherapy for acute lymphatic leukemia showed no clastogenic changes after chemotherapy but before irradiation. After radiotherapy, significant dose-related increases in chromosomal aberrations were seen (Rauscher and Bauchinger 1983).

Radiation from cobalt isotopes has been shown to induce numerous genetic changes, including translocations (Gilot-Delhalle et al. 1988; Grahn and Carnes 1988; Grahn et al. 1983; Searl et al. 1976), decreased DNA synthesis (Lohmann et al. 1966), dominant lethal mutations (Grahn et al. 1988; Searl et al. 1976; Zhou et al. 1986), chromosome deletions (Brooks et al. 1971b, 1974), polycentrics (Brooks et al. 1971a, 1974), and aberrations (Brooks et al. 1971a, 1971b) in exposed animals.

Radiation from cobalt isotopes was genotoxic in several assay systems in mammalian cells: DNA amplification in hamster cells (Lucke-Huhle et al. 1986, 1990); chromosomal aberrations and sister-chromatid exchanges in mouse lymphosarcoma cells (Juraskova and Drasil 1987); chromosomal aberrations and micronucleus formation in human lymphocytes (Koksal et al. 1995, 1996; Schmid et al. 2002); DNA breakage in human leukocytes (Lindahl-Kiessling et al. 1970; Reuff et al. 1993), kidney cells (Feinendegen et al. 1977), and fibroblasts (Coquerelle et al. 1987; Dolling et al. 1998); chromosomal aberrations in human leukocytes (Reuff et al. 1993); transformation of human fibroblasts (Namba et al. 1981, 1985, 1988); and retinoblastoma gene alterations in human fibroblasts (Endo et al. 1993).

3.5 TOXICOKINETICS

3.5.1 Absorption

3.5.1.1 Inhalation Exposure

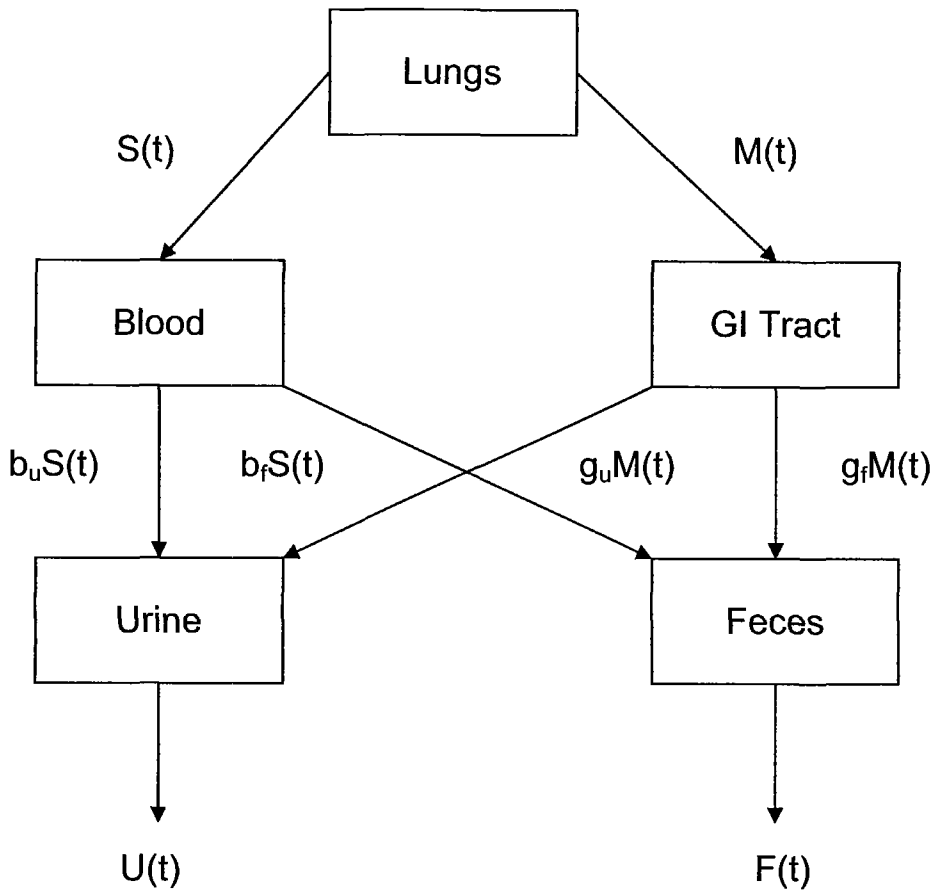
Inhaled cobalt particles are deposited in the upper and lower respiratory tract and cobalt is subsequently absorbed by several mechanisms (Casarett and Doull 1986); however, two of these mechanisms in particular appear to be most relevant. The deposition pattern in the respiratory tract is related to particle size, which determines the degree to which particles are affected by inertial impaction, sedimentation, diffusion, and electrostatic precipitation. Large particles (diameter >2 μm) tend to deposit in the upper respiratory tract where high airstream velocities and airway geometry promote inertial impaction of larger particles. Smaller particles escape inertial impaction and enter the lower respiratory tract where lower

airstream velocities and airway geometry favor the process of sedimentation, diffusion, and electrostatic precipitation of small particles. Fractional deposition can be expected to vary considerably with age, particle size, and breathing patterns (see Table 3-10). Fractional deposition of inhaled cobalt oxide particles in humans varied from approximately 50% of the inhaled dose for particles with a geometric mean diameter of 0.8 μm to approximately 75% of the inhaled dose for particles with a geometric mean diameter of 1.7 μm (Foster et al. 1989).

The transfer pathways of cobalt oxide (^{57}Co used as a tracer) from the lungs in humans and animals are shown in Figure 3-4. Particles of cobalt deposited in the respiratory tract can be absorbed into the blood after dissolution (S(t)) or mechanically transferred to the gastrointestinal tract by mucociliary action of the respiratory tract and swallowing action (M(t)). Only a portion (probably <50%) of the cobalt that enters the gastrointestinal tract will be absorbed into the body. The relative magnitude of the translocation and mechanical clearance pathways depends on the size and solubility of the cobalt particles that are inhaled. Large particles (>2 μm) will tend to deposit in the middle and upper airways where mechanical clearance mechanisms predominate over translocation. Smaller particles that enter the lower respiratory tract will tend to remain until dissolved or phagocytized by macrophages and translocation occurs. The sum of the activities of translocation and mechanical clearance determine the kinetics of absorption of inhaled cobalt. In humans, the ratio of translocation (S(t)) to mechanical clearance (M(t)) is approximately 5–1 for particle sizes ranging from 0.8 to 1.7 μm (mean geometric diameter) (Foster et al. 1989).

Data on retention of cobalt oxide (^{57}Co used as a tracer) in the respiratory tracts of humans and several animal species are summarized in Table 3-6. Considerable variability exists among species. In humans, almost one-half of the original lung burden persisted 6 months after exposure; in rats, clearance of cobalt from the lungs was nearly complete after 6 months. The elimination half-time for cobalt in the human lung increased with increasing time after exposure (Foster et al. 1989; Sedlet et al. 1958). This may reflect slower clearance of cobalt that is bound to cellular components in the lung (Kreyling et al. 1985, 1986).

Figure 3-4. Transfer Parameters for Cobalt Following Inhalation of Cobalt Oxide* (CO_3O_4) Particles, Showing the Fractions of the Lung Content, L(t), and Time, t, Cleared Per Day by Each Route**



GI tract = gastrointestinal tract;
$b_fS(t)$ = fraction of cobalt excreted in the feces after translocation;
$b_uS(t)$ = fraction of cobalt excreted in the urine after translocation;
F(t) = fecal excretion rate;
$g_fM(t)$ = fraction of cobalt excreted in the feces after mechanic clearance to the gastrointestinal tract;
$g_uM(t)$ = fraction of cobalt excreted in the urine after mechanic clearance to the gastrointestinal tract;
M(t) = rate of mechanical transport of cobalt particles from the lungs to the gastrointestinal tract;
S(t) = rate of translocation of cobalt from the lungs to the blood;
U(t) = urinary excretion rate

*Cobalt-57 tracer used
**Derived from Bailey et al. 1989

Table 3-6. Initial (Day 3) Lung Deposits of Cobalt Oxide and Summary of Lung Retention at 90 and 180 Days[a,b]

	Mean initial ^{57}Co activity in lung L(3) (kBq)		Lung retention L(90)/L(3) (%)		Lung retention L(180)/L(3) (%)	
Species (strain)	0.8 μm	1.7 μm	0.8 μm	1.7 μm	0.8 μm	1.7 μm
Human	53	42	64	75	45	56
Baboon	2,100	1,700	55	55	26	37
Beagle dog	1,150	1,450	27	45	5.5	12
Guinea pig (Harwell)	8.4	1.4	49	46	8.3	15
Rat (HMT, 1985)	10.8	4.7	5.2	20	1.3	8.0
Rat (HMT, 1986)	3.2	0.7	5.3	18	1.2	9.2
Rat (F344, SPF)	8.8	4.4	14	25	4.7	9.2
Rat (Sprague-Dawley)	0.9	0.10	8	39	1	15
Syrian hamster	4.0	1.2	21	35	3.4	12
Mouse (CBA/H)	1.8	No data	15	No data	2.8	No data

[a]Derived from Bailey et al. 1989
[b]Cobalt-57 used as tracer

3.5.1.2 Oral Exposure

Gastrointestinal absorption of cobalt in humans varies considerably (18–97% of the given dose) based on the type and dose of cobalt compound given and the nutritional status of the subjects (Harp and Scoular 1952; Smith et al. 1972; Sorbie et al. 1971; Valberg et al. 1969). More cobalt was absorbed through the gastrointestinal tract of humans when the body was deficient in iron (31–71% in iron deficiency; 18–44% in controls) (Sorbie et al. 1971; Valberg et al. 1969). One study in humans has shown that oral exposure to cobalt chloride resulted in significantly higher urinary excretion in females relative to males (Christensen et al. 1993).

In animal studies, many factors have been shown to influence the absorption of cobalt compounds following oral exposure. In several studies in rats (Ayala-Fierro et al. 1999; Barnaby et al. 1968; Hollins and McCullough 1971; Kirchgessner et al. 1994; Schade et al. 1970; Taylor 1962), soluble cobalt chloride was absorbed in the range of 13–34%, whereas physiologically insoluble cobalt oxide particles have been shown to be poorly absorbed, in the range of 1–3% (Bailey et al. 1989; Collier et al. 1989; Patrick et al. 1989). The particle size of the given dose of cobalt oxide had no significant effect on gastrointestinal absorption (Table 3-7). Administration of cobalt chloride labeled with radioactive 58Co and complexed with histidine, lysine, glycylglycine, ethylenediaminetetraacetic acid (EDTA), casein, or glycine resulted in decreased gastrointestinal absorption of cobalt; administration of cobalt chloride (with 58Co tracer) in cows' milk permitted a significantly greater (about 40%) absorption through the gastrointestinal tract (Taylor 1962). The same study found that while there was no difference in the chlorides of cobalt(II) and cobalt(III), a cobalt(II) glycine complex was absorbed in greater quantities than a cobalt(III) glycine complex. Other studies have also demonstrated that the chemical form of the cobalt compound can affect the absorption of cobalt following oral exposure (Deka et al. 1981; Firriolo et al. 1999; Inaba et al. 1980; Kinoshita and Fujita 1972), with more water-soluble compounds generally showing greater absorption.

Iron deficiency led to increased absorption of cobalt from the gastrointestinal tract, and simultaneous administration of cobalt and iron reduced the amount of cobalt absorbed (Reuber et al. 1994; Schade et al. 1970). Increasing oral doses of cobalt resulted in decreased fractional absorption (Houk et al. 1946; Kirchgessner et al. 1994; Taylor 1962), and more soluble forms of cobalt were better absorbed than less soluble compounds (Kreyling et al. 1986). Absorption is 3- to 15-fold greater in younger animals (rats and guinea pigs examined from days 1–60 of life) than in adult (200 days of age) animals (Naylor and

Table 3-7. Summary of Measurements of Retention and Excretion After Intragastric Administration of Cobalt Oxide (Co_3O_4) Particles (Mean Percentage of Recovered Activity at 7 Days After Administration)[a,b]

Species (strain)	Cumulative fecal excretion		Whole body retention		Cumulative urinary excretion		Absorption	
	0.8 μm	1.7 μm	0.8 μm	1.7 μm	0.8 μm	1.7 μm	0.8 μm	1.7 μm
Baboon	97.8	98.4	0.12	0.20	2.0	1.4	2.6	1.9
Guinea pig	98.7	97.6	0.16	0.66	1.1	1.9	1.3	2.3
Rat (HMT)	96.3	99.4	0.09	0.02	2.8	0.6	3.9	1.0
Rat (F-344)	99.6	99.7	0.04	0.03	0.4	0.3	0.4	0.3
Hamster	96.0	96.3	0.50	0.18	3.5	3.5	5.1	5.1
Mouse (CBA/H)	99.1	No data	0.3	No data	0.6	No data	0.8	No data

[a]Derived from Bailey et al. 1989
[b]Cobalt-57 used as tracer

Harrison 1995). Species differences in absorption of cobalt oxide do not appear to exist (Bailey et al. 1989), but absorption of soluble cobalt compounds is greater in rats (13–34%) than in dairy cows (1–2%) and guinea pigs (4–5%) following oral exposure (Ayala-Fierro et al. 1999; Barnaby et al. 1968; Hollins and McCullough 1971; Kirchgessner et al. 1994; Naylor and Harrison 1995; Schade et al. 1970; Taylor 1962; van Bruwaene et al. 1984).

3.5.1.3 Dermal Exposure

Four humans who placed their right hands into a box filled with hard metal dust (~5–15% cobalt metal, 95–85% tungsten carbide) for 90 minutes showed an increase in urinary cobalt levels by an order of magnitude in the post-exposure samples, remaining elevated for as long as 48–60 hours (Scansetti et al. 1994). Similarly, cobalt was detected in the fingernails of three volunteers who placed their fingers in cobalt solution 10 minutes/day for 7 days (Nielsen et al. 2000), even after the cessation of exposure. These findings demonstrate that cobalt from these metal dusts can be absorbed through the skin. The absorption of 2.2×10^{-5} mg ^{60}Co/kg as cobalt chloride in 1.4N HCl through 1 cm^2 of intact or abraded skin of guinea pigs was examined by Inaba and Suzuki-Yasumoto (1979). Absorption through intact skin was very small (<1%), while absorption through abraded skin was almost 80% 3 hours after exposure. A study in hamsters (Lacy et al. 1996) also reported a low amount of absorption of cobalt through unabraded skin.

3.5.1.4 Other Routes of Exposure

No studies were located regarding absorption of cobalt in humans or animals after other routes of exposure.

3.5.2 Distribution

As a component of vitamin B_{12}, cobalt is an essential element and, therefore, is found in most body tissues. It has been identified in liver, muscle, lung, lymph nodes, heart, skin, bone, hair, stomach, brain, pancreatic juice, kidneys, plasma, and urinary bladder of nonexposed subjects, with the highest cobalt concentration found in the liver (Collecchi et al. 1986; Forbes et al. 1954; Hewitt 1988; Ishihara et al.

1987; Muramatsu and Parr 1988; Teraoka 1981; Yamagata et al. 1962; Yukawa et al. 1980) (see Chapter 6 for more information). Tissue levels reflected exposure from all routes. The total body content of cobalt has been estimated at 1.1–1.5 mg (ICRP 1979; Yamagata et al. 1962); about 0.11 mg was found in the liver (ICRP 1979).

In patients with laryngeal carcinoma, levels of cobalt in the tumor were significantly higher ($p<0.001$) than levels in the nonmalignant tissues around the tumor (68.7 ng/g tissue versus 39.6 ng/g) (Collecchi et al. 1986). The mean cobalt concentrations in plasma (18.3 ng/mL) were also significantly higher in these patients than in the comparison population (0.73 ng/mL). The clinical significance of these findings is not known.

3.5.2.1 Inhalation Exposure

In workers occupationally exposed to airborne cobalt, increased cobalt levels were found in tissues at death. Significant increases in cobalt in the lung have been found in copper smelter and metal workers and coal miners occupationally exposed to cobalt (Gerhardsson et al. 1984; Hewitt 1988; Hillerdal and Hartung 1983; Teraoka 1981). No increase in liver or kidney cobalt levels were found in the copper smelter workers as compared to controls (Gerhardsson et al. 1984). In metal workers, increased cobalt levels were also found in the lymph nodes, liver, spleen, and kidneys (Hillerdal and Hartung 1983; Teraoka 1981).

The tissue distribution of cobalt in animals is similar to that in humans, with marked increases in the concentration of cobalt in the lungs following inhalation exposure (Barnes et al. 1976; Brune et al. 1980; Collier et al. 1991; Kreyling et al. 1986; Kyono et al. 1992; Patrick et al. 1989; Talbot and Morgan 1989). Histologically, the particles of cobalt in the lung are found in macrophages within the bronchial wall or in the interstitium close to the terminal bronchioli (Brune et al. 1980). Significant concentrations of cobalt have been found in the liver, kidney, trachea, spleen, bones, and heart (Barnes et al. 1976; Brune et al. 1980; Kerfoot 1975; Kreyling et al. 1986; Wehner and Craig 1972), with the greatest concentrations in the liver and the kidney (Kerfoot 1975; Wehner and Craig 1972).

3.5.2.2 Oral Exposure

No studies were located regarding distribution in humans after oral exposure to cobalt.

In animals, the cobalt absorbed through the gastrointestinal tract was primarily retained in the liver (Ayala-Fierro et al. 1999; Greenberg et al. 1943; Simesen 1939). Appreciable levels were also found in the kidneys, heart, stomach, and intestines (Ayala-Fierro et al. 1999; Persson et al. 1992; Simesen 1939). Following a single oral dose of cobalt napthenate, appreciable levels of cobalt were found in the heart, liver, and kidney, but not in the spleen or testes (Firriolo et al. 1999). Following oral exposure to pregnant rats, a dose-dependent increase in cobalt levels in fetal blood and amniotic fluid was seen (Szakmary et al. 2001).

Following longer-term exposure (8 weeks) to cobalt sulfate in the diet, exposed rats showed a 30-fold increase in the cobalt concentration in the myocardium, a 26-fold increase in the concentration in the soleus muscle, and a 100-fold increase in the concentration in serum compared with nonexposed controls (Clyne et al. 1988; Pehrsson et al. 1991). Long-term oral exposure of rats to cobalt chloride resulted in significantly increased levels of cobalt in the liver, kidney, muscle, brain, and testes of treated rats (Barnaby et al. 1968; Bourg et al. 1985; Thomas et al. 1976).

3.5.2.3 Dermal Exposure

No studies were located regarding distribution in humans or animals after dermal exposure to cobalt.

3.5.2.4 Other Routes of Exposure

Following intravenous injection of cobalt chloride (as a combination of radioactive $^{55}CoCl_2$ and $^{56}CoCl_2$) in two humans, the liver and bladder contained the highest portions of cobalt (Jansen et al. 1996).

Distribution in animals after an intravenous dose appears to be similar to what we know of cobalt distribution in humans following injection of cobalt compounds. Two hours after intravenous injection of cobalt chloride (with a radioactive ^{57}Co tracer) in rats, accumulation was found in the liver (22.8% of the dose), kidneys (10.2%), and intestines (3.16%) (Gregus and Klaassen 1986). Similar results (29% liver,

10% kidneys, 4.6% intestines) were found following intracardiac injection of cobalt nitrate in rats (Patrick et al. 1989) or intravenous injection of a combination of radioactive $^{55}CoCl_2$ and $^{56}CoCl_2$ in rats (exact percentages were not provided) (Jansen et al. 1996). One hundred days after intravenous injection of $^{60}CoCl_2$ in rats, the greatest concentrations were found in spleen>heart>bone, while liver and kidney, initially the highest in cobalt, contained comparatively low amounts of cobalt (Thomas et al. 1976). Similar results were seen 132 days following an intraperitoneal injection of $^{60}CoCl_2$ in rats (Barnaby et al. 1968). Intramuscular injection of cobalt mesoporphyrin in rats yielded the greatest levels of cobalt in liver and blood, followed by kidney, lung, spleen, adrenal glands, and heart at 7 days post-injection and later (Feng et al. 1998). Four weeks after subcutaneous administration of cobalt protoporphyrin, the greatest tissue levels of cobalt occurred in the kidney, followed by spleen, liver, lung, thymus, and gonads (Rosenberg 1993). When cobalt (with a ^{57}Co tracer) encapsulated in liposomes was intravenously injected into rats, decreased distribution to the heart (40% less than animals receiving cobalt chloride), kidneys, and carcass, and increased distribution to the spleen and bones were found (Szebeni et al. 1989).

3.5.3 Metabolism

Cobalt is essential in the body because it is a component of cyanocobalamin (vitamin B_{12}) (Vouk 1986). Vitamin B_{12} acts as coenzyme in many enzymatic reactions, most notably a methyl transfer reaction that converts homocysteine to methionine and for a separate reaction that converts L-methylmalonylcoenzyme A (CoA) to succinyl-CoA (Institute of Medicine 2000). Vitamin B_{12} is also a part of some enzymes involved in hematopoiesis; deficiency can lead to pernicious anemia (Domingo 1989). No other essential function of cobalt has been reported. The Recommended Dietary Allowance (RDA) for vitamin B_{12} for adults is 2.4 μg/day, which contains 0.1 μg of cobalt (Institute of Medicine 2000).

3.5.4 Elimination and Excretion

3.5.4.1 Inhalation Exposure

No data are available on the clearance of soluble cobalt particles in humans. Following exposure of humans to physiologically insoluble cobalt compounds (cobalt metal, cobalt oxides), clearance from the body, assessed by both urinary/fecal clearance and a reduction in whole-body retention, appears to follow

three-phase kinetics. The first phase, likely representing mucociliary clearance of particles deposited in the tracheobronchial region, has a half-time on the order of 2–44 hours (Apostoli et al. 1994; Mosconi et al. 1994b). The second phase, with a half-time on the order of 10–78 days, may represent macrophage-mediated clearance of cobalt particles from the lung (Beleznay and Osvay 1994; Mosconi et al. 1994b). The third clearance phase, representing long-term clearance from the lungs, has a half-time on the order of years (Bailey et al. 1989; Beleznay and Osvay 1994; Mosconi et al. 1994b; Newton and Rundo 1971). Following a controlled aerosol exposure in humans, about 40% of the initial lung burden of inhaled cobalt oxide (with a 57Co tracer) was retained for a period of 6 months after exposure (Foster et al. 1989). Within the first week, about 17% of the initial lung burden was eliminated, with the majority (about 90%) mechanically cleared to the gastrointestinal tract and excreted in the feces (Foster et al. 1989). Six months after exposure, a cumulative elimination of 33% of the initial lung burden was found in the urine and 28% was found in the feces (Foster et al. 1989). The ratio of peak absorption rate to average mechanical clearance rate (Figure 3-4 and Table 3-8) was about 5 to 1. The elimination of cobalt following inhalation exposure was affected by the time after exposure (urinary excretion increases as time increases) and particle size (more cobalt is initially mechanically cleared to the gastrointestinal tract when the aerosol consists of bigger particles) (Bailey et al. 1989; Foster et al. 1989).

In animals, the solubility of the cobalt compound appears to greatly affect its long-term clearance. Studies with cobalt oxides have shown that the more soluble CoO is cleared from the lungs at a greater rate than the less soluble Co_3O_4 (Barnes et al. 1976; Kreyling 1984a). More soluble cobalt compounds are absorbed into the blood at a greater rate, and excreted in the urine and, to a lesser extent, the feces (Barnes et al. 1976). The rate of urinary excretion appears to correlate with the rate of translocation of cobalt from the lungs to the blood, and the rate of fecal clearance with the rate of mechanical clearance of cobalt from the lungs to the gastrointestinal tract (Andre et al. 1989; Bailey et al. 1989; Collier et al. 1989; Kreyling et al. 1986, 1989; Patrick et al. 1989; Talbot and Morgan 1989). Following an initial high rate of fecal clearance, urinary excretion was the primary route of cobalt elimination after a single inhalation exposure (2 weeks of observation) (Palmes et al. 1959) or 3 months of exposure (Kerfoot 1975; Palmes et al. 1959). In several species of animals, most of the inhaled Co_3O_4 (with a ^{57}Co tracer) following a single exposure was cleared from the lungs by 6 months after exposure (Table 3-6) (Andre et al. 1989; Bailey et al. 1989; Collier et al. 1989; Kreyling et al. 1989; Patrick et al. 1989; Talbot and Morgan 1989). The peak translocation and average mechanical clearance of cobalt from the lungs for different species are reported in Table 3-8, with the rate (high to low) following as mouse > rat > hamster > guinea pig > baboon, human > beagle dog.

Table 3-8. Peak Translocation and Average Mechanical Clearance Rates After Inhalation of Cobalt Oxide[a,b]

	Percent of lung content cleared per day				
	Translocation at peak				Average mechanical clearance[c]
Species (strain)	0.8 μm	Peak day	1.7 μm	Peak day	
Human	0.45	180	0.5	180	0.1
Baboon	0.6	180	0.2	d	0.1
Beagle dog	2.1	85	1.7	180	0.03
Guinea pig	2.1	180	1.0	75	0.3
Rat HMT	2.4	40	0.6	d	0.9
Rat (F-344)	1.1	10	0.4	d	1.0
Hamster	1.8	180	0.7	180	0.8
Mouse	1.7	180	No data	No data	1.05

[a]Derived from Bailey et al. 1989
[b]Cobalt-57 used as tracer
[c]Clearance rates were virtually identical in both particle size groups
[d]Constant value over 180 days

3.5.4.2 Oral Exposure

In humans orally exposed to cobalt, fecal elimination, which is the primary route of elimination, varies considerably (3–99% of the dose) and depends on the amount and type of cobalt given and on the nutritional status of the subjects (Section 3.5.1.2) (Harp and Scoular 1952; Paley et al. 1958; Smith et al. 1972; Sorbie et al. 1971; Valberg et al. 1969). Within days after oral exposure, 10 times more cobalt was excreted in feces than in the urine (Paley et al. 1958). Less cobalt was eliminated in the feces (more was absorbed) in subjects with an iron deficiency (Sorbie et al. 1971; Valberg et al. 1969).

Fecal elimination of cobalt is the primary route of elimination in animals following oral exposure and depends mainly upon the particle solubility (decreasing fecal clearance with increasing solubility) of the cobalt compound. The cumulative urinary and fecal elimination in several species following oral administration of Co_3O_4 (with a ^{57}Co tracer) is reported in Table 3-7 (Bailey et al. 1989). Following oral administration in several species, very little Co_3O_4 was absorbed through the gastrointestinal tract and most (>96%) was quickly eliminated in the feces. No significant differences in elimination of Co_3O_4 were found among species of animals (Andre et al. 1989; Bailey et al. 1989; Collier et al. 1989; Patrick et al. 1989; Talbot and Morgan 1989). For the more soluble cobalt(II) chloride, reported fecal elimination levels have ranged from 70 to 83% of the administered dose for rats, with urinary excretion accounting for the majority of the remainder of the dose (Ayala-Fierro et al. 1999; Barnaby et al. 1968; Hollins and McCullough 1971). In lactating dairy cows, about 97% of an oral dose of cobalt chloride was recovered in the feces by day 70 post-exposure, while the urine and milk contained 0.26 and 0.012% of the dose, respectively (van Bruwaene et al. 1984). Following a single exposure in beagle dogs, more Co_3O_4 (physiologically insoluble) was eliminated in the feces (90% in the feces and 5% in the urine) than following an exposure to cobalt nitrate (soluble) (70% in the feces and 25% in the urine) (Kreyling et al. 1986).

As is the case for absorption of cobalt compounds, the iron status of the animal also appears to affect the elimination of cobalt compounds. Following oral exposure, iron-deficient rats eliminated less of a given dose in the feces than normal rats, while co-administration of iron compounds resulted in an increased fecal excretion of cobalt compounds (Reuber et al. 1994; Schade et al. 1970).

3.5.4.3 Dermal Exposure

No studies were located regarding excretion in humans after dermal exposure to cobalt.

Lacy et al. (1996) reported that the majority of the absorbed dose of $CoCl_2$ was excreted in the urine 48 hours after a single dermal exposure in Syrian hamsters. No other studies were located regarding excretion in animals after dermal exposure to cobalt.

3.5.4.4 Other Routes of Exposure

Following intravenous injection of cobalt chloride in humans, about 30% of the dose was excreted in the urine within 24 hours (Smith et al. 1972), 56–73% was excreted within 48 hours (Paley et al. 1958), and 57% was excreted within 2 weeks (Kent and McCance 1941).

Following intravenous injection of cobalt nitrate (with a 57Co tracer) in various species of animals, most of the injected dose was excreted in the urine; about 80% of the given dose was excreted in the urine within 21 days (Table 3-9) (Andre et al. 1989; Bailey et al. 1989; Collier et al. 1989; Patrick et al. 1989; Talbot and Morgan 1989). Other investigators have also found that the urine is the primary route of cobalt excretion following intravenous administration (Ayala-Fierro et al. 1999; Barnaby et al. 1968; Gregus and Klaassen 1986; Kreyling et al. 1986; Onkelinx 1976; Thomas et al. 1976). Most of the remaining cobalt (5–30% of the total dose) after intravenous exposure was excreted in the feces, with the majority of studies reporting very little long-term retention. Excretion of cobalt (about 2–7% of the injected dose) in the bile was also reported (Cikrt and Tich 1981; Gregus and Klaasen 1986; Sheline et al. 1945). Elimination following intraperitoneal injection is similar to that seen following intravenous exposure, with urinary excretion being the major route of elimination, and fecal excretion accounting for the majority of the remainder of the dose (Barnaby et al. 1968; Hollins and McCullough 1971; Talbot and Morgan 1989), though long-term clearance may be more balanced between the two (Hollins and McCullough 1971). Following subcutaneous injection, both $CoCl_2$ and $Co(NO_3)_2$ were cleared rapidly from the body (Rosenberg 1993; Talbot and Morgan 1989), with the urine being the major route of clearance (Talbot and Morgan 1989).

Table 3-9. Summary of Measurements of Retention and Excretion of Cobalt Following Injection of Cobalt Nitrate $Co(NO_3)_2$ Solution (Mean Percent Recovery)[a,b]

Species (strain)	Whole body retention on day			Cumulative urinary excretion on day			Cumulative fecal excretion on day		
	1	7	21	1	7	21	1	7	21
Baboon	No data	No data	No data	57	74	80	5	17	20
Beagle dog	No data	No data	No data	71	86	87	3.4	4.4	4.9
Guinea pig	34	8	3.5	64	82	85	2.2	10	12
Rat (HMT)	18	4.2	1.9	64	72	74	18	24	24
Rat (F-344)	No data	No data	2.9	No data	No data	80	No data	No data	18
Hamster	27	4.3	1.9	55	68	69	17	28	29
Mouse	23	2.9	1.1	59	71	72	18	26	27

[a]Derived from Bailey et al. 1989
[b]Cobalt-57 used as tracer

Following injection, studies have shown that the chemical form of the cobalt compound can affect its elimination. Subcutaneous injection of cobalt protoporphyrin in rats, in which the cobalt atom is chelated within the porphyrin ring, resulted in a slower elimination from the body than cobalt chloride, with significant cobalt levels (~20% of initial injection) still present in the body 14 days after exposure (Rosenberg 1993). Likewise, intramuscular injection of cobalt mesoporphyrin resulted in primarily in fecal excretion, with a high systemic retention (Feng et al. 1998). It therefore appears that a greater solubility leads to fast elimination, mainly in the urine, while a less soluble compound will be retained for longer periods and eliminated to a greater extent in the feces.

3.5.5 Physiologically Based Pharmacokinetic (PBPK)/Pharmacodynamic (PD) Models

Physiologically based pharmacokinetic (PBPK) models use mathematical descriptions of the uptake and disposition of chemical substances to quantitatively describe the relationships among critical biological processes (Krishnan et al. 1994). PBPK models are also called biologically based tissue dosimetry models. PBPK models are increasingly used in risk assessments, primarily to predict the concentration of potentially toxic moieties of a chemical that will be delivered to any given target tissue following various combinations of route, dose level, and test species (Clewell and Andersen 1985). Physiologically based pharmacodynamic (PBPD) models use mathematical descriptions of the dose-response function to quantitatively describe the relationship between target tissue dose and toxic end points.

PBPK/PD models refine our understanding of complex quantitative dose behaviors by helping to delineate and characterize the relationships between: (1) the external/exposure concentration and target tissue dose of the toxic moiety, and (2) the target tissue dose and observed responses (Andersen et al. 1987; Andersen and Krishnan 1994). These models are biologically and mechanistically based and can be used to extrapolate the pharmacokinetic behavior of chemical substances from high to low dose, from route to route, between species, and between subpopulations within a species. The biological basis of PBPK models results in more meaningful extrapolations than those generated with the more conventional use of uncertainty factors.

The PBPK model for a chemical substance is developed in four interconnected steps: (1) model representation, (2) model parametrization, (3) model simulation, and (4) model validation (Krishnan and Andersen 1994). In the early 1990s, validated PBPK models were developed for a number of

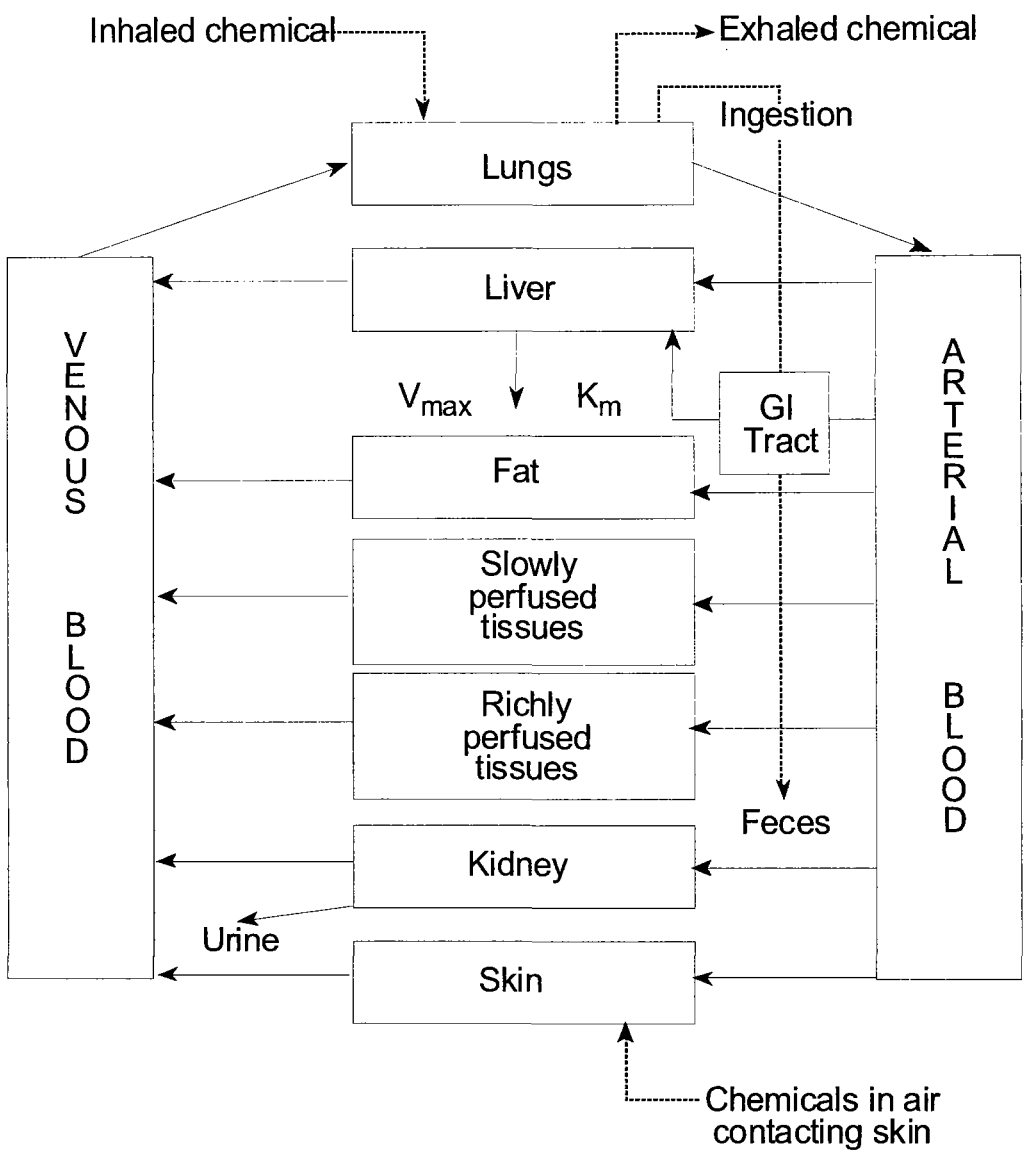
toxicologically important chemical substances, both volatile and nonvolatile (Krishnan and Andersen 1994; Leung 1993). PBPK models for a particular substance require estimates of the chemical substance-specific physicochemical parameters, and species-specific physiological and biological parameters. The numerical estimates of these model parameters are incorporated within a set of differential and algebraic equations that describe the pharmacokinetic processes. Solving these differential and algebraic equations provides the predictions of tissue dose. Computers then provide process simulations based on these solutions.

The structure and mathematical expressions used in PBPK models significantly simplify the true complexities of biological systems. If the uptake and disposition of the chemical substance(s) is adequately described, however, this simplification is desirable because data are often unavailable for many biological processes. A simplified scheme reduces the magnitude of cumulative uncertainty. The adequacy of the model is, therefore, of great importance, and model validation is essential to the use of PBPK models in risk assessment.

PBPK models improve the pharmacokinetic extrapolations used in risk assessments that identify the maximal (i.e., the safe) levels for human exposure to chemical substances (Andersen and Krishnan 1994). Similar models have been developed for radionuclides. These PBPK models provide a scientifically sound means to predict the target tissue dose of chemicals in humans who are exposed to environmental levels (for example, levels that might occur at hazardous waste sites) based on the results of studies where doses were higher or were administered in different species. Figure 3-5 shows a conceptualized representation of a PBPK model. Figures 3-6 through 3-9 show models for radionuclides in general or specifically for cobalt.

The ICRP (1995) developed a Human Respiratory Tract Model for Radiological Protection, which contains respiratory tract deposition and clearance compartmental models for inhalation exposure that may be applied to particulate aerosols of cobalt compounds. The ICRP (1993) also developed a 3-compartment biokinetic model for human oral exposure that applies to cobalt. EPA (1998) has adopted the ICRP (1993, 1995) models for assessment of radiologic cancer risks from cobalt exposures. The National Council on Radiation Protection and Measurement (NCRP) has also developed a respiratory tract model for inhaled radionuclides (NCRP 1997). At this time, the NCRP recommends the use of the

Figure 3-5. Conceptual Representation of a Physiologically Based Pharmacokinetic (PBPK) Model for a Hypothetical Chemical Substance



Source: adapted from Krishnan et al. 1994

Note: This is a conceptual representation of a physiologically based pharmacokinetic (PBPK) model for a hypothetical chemical substance. The chemical substance is shown to be absorbed via the skin, by inhalation, or by ingestion, metabolized in the liver, and excreted in the urine or by exhalation.

ICRP model for calculating doses for radiation workers and the general public. Readers interested in this topic are referred to NCRP Report No. 125; *Deposition, Retention and Dosimetry of Inhaled Radioactive Substances* (NCRP 1997). In the appendix to the report, NCRP provides the animal testing clearance data and equations fitting the data which supported the development of the human model for cobalt

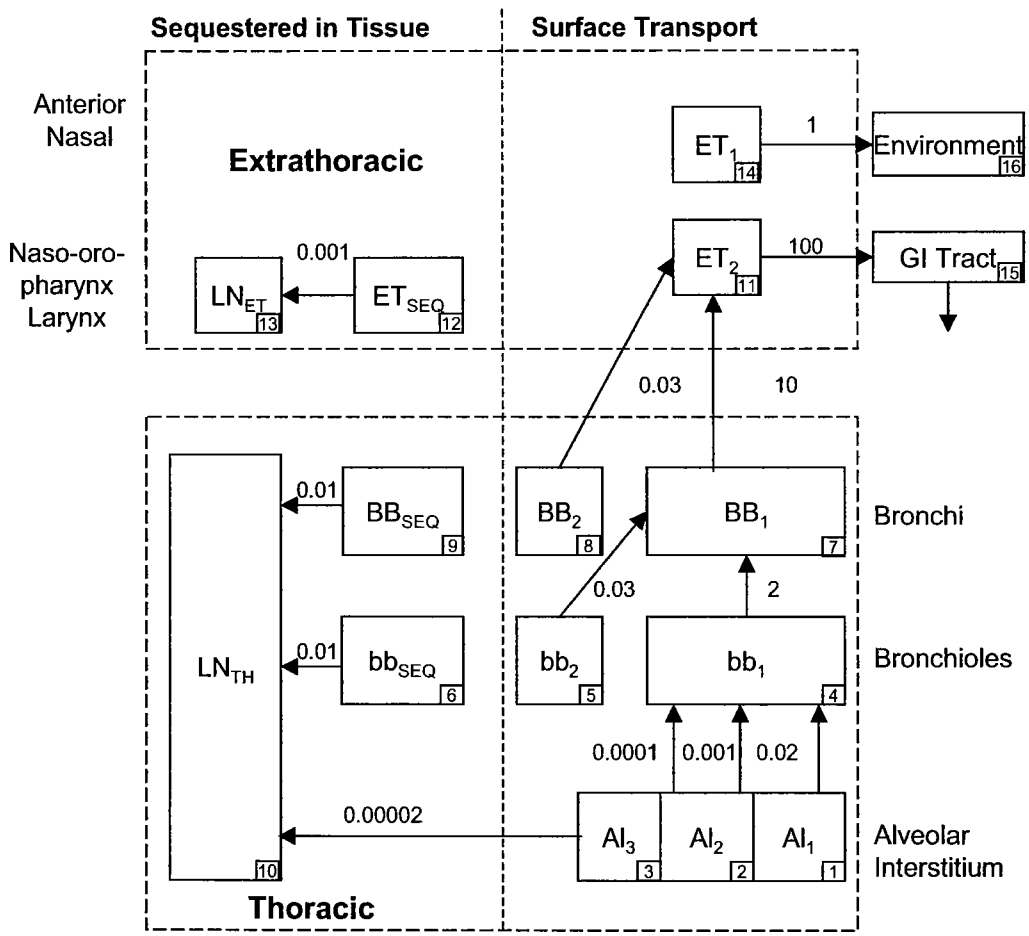
Human Respiratory Tract Model for Radiological Protection (ICRP 1994).

Respiratory Tract Deposition. The ICRP (1994) has developed a physiologically-based pharmacokinetic model for behavior of aerosols and vapors in the respiratory tract. ICRP (1994) provides inhalation dose coefficients that can be used to estimate the committed equivalent and the effective doses to organs and tissues throughout the body based on a unit intake of radioactive material and the anticipated distribution and retention of the material, its radioactive decay, and the energy of the radiationemitted from the material and absorbed by tissues. The model applies to three levels of particle solubility, a wide range of particle sizes (approximately 0.0005–100 μm in diameter), and parameter values that can be adjusted for various segments of the population (e.g., sex, age, level of physical exertion). This model also allows one to evaluate the bounds of uncertainty in deposition estimates. Uncertainties arise from natural biological variability among individuals and the need to interpret some experimental evidence that remains inconclusive. It is applicable to particulate aerosols containing cobalt, and was developed for a wide variety of radionuclides and their chemical forms.

The ICRP deposition model estimates the fraction of inhaled particle mass that initially deposits in each compartment (Figure 3-6). The model was developed with 5 compartments: (1) the anterior nasal passages (ET_1); (2) all other extrathoracic airways (ET_2) (posterior nasal passages, the naso- and oropharynx, and the larynx); (3) the bronchi (BB); (4) the bronchioles (bb); and (5) the alveolar interstitium (AI). Particles deposited in each of the regions may be removed from each region and redistributed either upward into the respiratory tree or to the lymphatic system and blood by different particle removal mechanisms.

For extrathoracic deposition of particles, the model uses experimental data, where deposition is related to particle size and airflow parameters, and scales deposition for women and children from adult male data. Similarly to the extrathoracic region, experimental data served as the basis for lung (bronchi, bronchioles, and alveoli) aerosol transport and deposition. A theoretical model of gas transport and particle deposition

Figure 3-6. Compartment Model to Represent Particle Deposition and Time-Dependent Particle Transport in the Respiratory Tract*



*Compartment numbers shown in lower right corners are used to define clearance pathways. The clearance rates, half-lives, and fractions by compartment, as well as the compartment abbreviations are presented in Table 3-11.

Source: ICRP 1994b

was used to interpret data and to predict deposition for compartments and subpopulations other than adult males. Table 3-10 provides reference respiratory values for the general Caucasian population under several levels of activity.

Deposition of inhaled gases and vapors is modeled as a partitioning process, which depends on the physiological parameters noted above as well as the solubility and reactivity of compound in the respiratory tract (Figure 3-7). The ICRP (1994) model defines three categories of solubility and reactivity: SR-0, SR-1, and SR-2:

- Type SR-0 compounds include insoluble and nonreactive gases (e.g., inert gases such as H2, He). These compounds do not significantly interact with the respiratory tract tissues and essentially all compound inhaled is exhaled. Radiation doses from inhalation of SR-0 compounds are assumed to result from the irradiation of the respiratory tract from the air spaces.

- Type SR-1 compounds include soluble or reactive gases and vapors that are expected to be taken up by the respiratory tract tissues and may deposit in any or all of the regions of the respiratory tract, depending on the dynamics of the airways and properties of the surface mucous and airway tissues, as well as the solubility and reactivity of the compound.

- Type SR-2 compounds include soluble and reactive gases and vapors that are completely retained in the extrathoracic regions of the respiratory tract. SR-2 type compounds include sulfur dioxide (SO2) and hydrogen fluoride (HF).

Respiratory Tract Mechanical (Particle) Clearance. This portion of the model identifies the principal clearance pathways within the respiratory tract. The model was developed to predict the retention of various chemical materials. The compartmental model is linked to the deposition model (see Figure 3-6) and to reference values presented in Table 3-11. This table provides deposition fractions and clearance rates for each compartment for insoluble particles. The table provides rates of insoluble particle transport for each of the compartments, expressed as a fraction of the deposit per day and also as clearance half-time. ICRP (1994) also developed modifying factors for some of the parameters, such as age, smoking, and disease status. Parameters of the clearance model are based on human evidence for the most part, although particle retention in airway walls is based on experimental data from animal experiments.

The clearance of deposited particles from the respiratory tract is a dynamic process. The rate of clearance generally changes with time from each region and by each route. Following deposition of large numbers

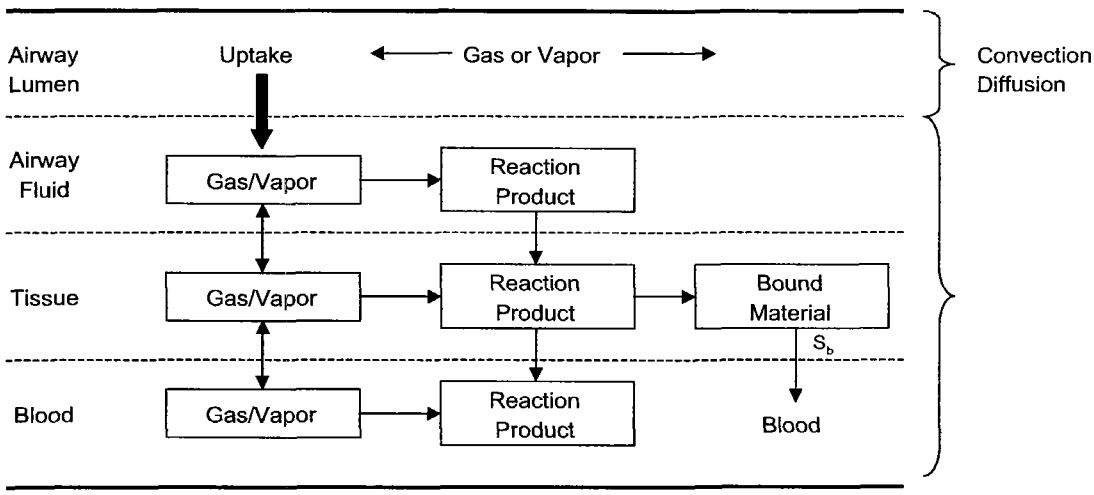
Table 3-10. Reference Respiratory Values for a General Caucasian Population at Different Levels of Activity[a]

Activity:		Resting (sleeping)			Sitting awake			Light exercise			Heavy exercise		
Maximal workload:		8%			12%			32%			64%		
Breathing parameters[b]		V_T (L)	B (m^3h^{-1})	f_R (min^{-1})	V_T (L)	B (m^3h^{-1})	f_R (min^{-1})	V_T (L)	B (m^3h^{-1})	f_R (min^{-1})	V_T (L)	B (m^3h^{-1})	f_R (min^{-1})
Age	Sex												
3 months		0.04	0.09	38	N/A	N/A	N/A	0.07	0.19	48	N/A	N/A	N/A
1 year		0.07	0.15	34	0.1	0.22	36	0.13	0.35	46	N/A	N/A	N/A
5 years		0.17	0.24	23	0.21	0.32	25	0.24	0.57	39	N/A	N/A	N/A
10 years	Male:										0.841	2.22	44
	Female:										0.667	1.84	46
	Both:	0.3	0.31	17	0.33	0.38	19	0.58	1.12	32			
15 years	Male:	0.50	0.42	14	0.533	0.48	15	1.0	1.38	23	1.352	2.92	36
	Female:	0.42	0.35	14	0.417	0.40	16	0.903	1.30	24	1.127	2.57	38
Adult	Male:	0.63	0.45	12	0.750	0.54	12	1.25	1.5	20	1.923	3.0	26
	Female:	0.44	0.32	12	0.464	0.39	14	0.992	1.25	21	1.364	2.7	33

[a]See Annex B (ICRP 1994) for data from which these reference values were derived.
[b]V_T = Tidal volume, B = ventilation rate, f_R = respiration frequency

h = hour; L = liter(s); min = minute(s); N/A = not applicable

Figure 3-7. Reaction of Gases or Vapors at Various Levels of the Gas-Blood Interface



Source: ICRP 1994b

Table 3-11. Reference Values of Parameters for the Compartment Model to Represent Time-dependent Particle Transport from the Human Respiratory Tract

Part A

Clearance rates for insoluble particles				
Pathway	From	To	Rate (d^{-1})	Half-time[a]
$m_{1,4}$	AI_1	bb_1	0.02	35 days
$m_{2,4}$	AI_2	bb_1	0.001	700 days
$m_{3,4}$	AI_3	bb_1	0.0001	7,000 days
$m_{3,10}$	AI_3	LN_{TH}	0.00002	—
$m_{4,7}$	bb_1	BB_1	2	8 hours
$m_{5,7}$	bb_2	BB_1	0.03	23 days
$m_{6,10}$	bb_{seq}	LN_{TH}	0.01	70 days
$m_{7,11}$	BB_1	ET_2	10	100 minutes
$m_{8,11}$	BB_2	ET_2	0.03	23 days
$m_{9,10}$	BB_{seq}	LN_{TH}	0.01	70 days
$m_{11,15}$	ET_2	GI tract	100	10 minutes
$m_{12,13}$	ET_{seq}	LN_{ET}	0.001	700 days
$m_{14,16}$	ET_1	Environment	1	17 hours

See next page for Part B

Table 3-11. Reference Values of Parameters for the Compartment Model to Represent Time-dependent Particle Transport from the Human Respiratory Tract

Part B

Partition of deposit in each region between compartments[b]		
Region or deposition site	Compartment	Fraction of deposit in region assigned to compartment[c]
ET_2	ET_2	0.9995
	ET_{seq}	0.0005
BB	BB_1	$0.993-f_s$
	BB_2	f_s
	BB_{seq}	0.007
bb	bb_1	$0.993-f_s$
	bb_2	f_s
	bb_{seq}	0.007
AI	AI_1	0.3
	AI_2	0.6
	AI_3	0.1

[a]The half-times are approximate since the reference values are specified for the particle transport rates and are rounded in units of day^{-1}. A half-time is not given for the transport rate from AI_3 to LN_{TH}, since this rate was chosen to direct the required amount of material to the lymph nodes. The clearance half-time of compartment AI_3 is determined by the sum of the clearance rates from it.
[b]See paragraph 181, Chapter 5 (ICRP 1994) for default values used for relating f_s to d_{ae}.
[c]It is assumed that f_s is size-dependent. For modeling purposes, f_s is taken to be:

$$f_s = 0.5 \; for \; d_{ae} \leq 2.5\sqrt{\rho / \chi} \; \mu m \; and$$

$$f_s = 0.5e^{0.63(d_{ae}\sqrt{\rho/\chi}-2.5)} \; for \; d_{ae} > 2.5\sqrt{\rho / \chi} \; \mu m$$

where:

f_s	=	fraction subject to slow clearance
d_{ae}	=	aerodynamic particle diameter/(μm)
ρ	=	particle density (g/cm^3)
χ	=	particle shape factor

AI = alveolar-interstitial region; BB = bronchial region; bb = bronchiolar region; BB_{seq} = compartment representing prolonged retention in airway walls of small fraction of particles deposited in the bronchial region; bb_{seq} = compartment representing prolonged retention in airway walls of small fraction of particles deposited in the bronchiolar region; ET = extrathoracic region; Et_{seq} = compartment representing prolonged retention in airway tissue of small fraction of particles deposited in the nasal passages; LN_{ET} = lymphatics and lymph nodes that drain the extrathoracic region; LN_{TH} = lymphatics and lymph nodes that drain the thoracic region

Source: ICRP 1994

of particles (acute exposure), transport rates change as particles are cleared from the various regions. Physical and chemical properties of deposited material determine the rate of dissolution and as particles dissolve; absorption rates tend to change over time. By creating a model with sub-compartments of different clearance rates within each region (e.g., BB_1, BB_2, BBseq), the ICRP model overcomes problems associated with time-dependent functions. Each compartment clears to other compartments by constant rates for each pathway.

Particle transport from all regions is toward both the lymph nodes and the pharynx, and a majority of deposited particles end up being swallowed. In the front part of the nasal passages (ET_1), nose blowing, sneezing, and wiping remove most of the deposited particles. Particles remain here for about a day. For particles with AMADs a few micrometers or greater, the ET_1 compartment is probably the largest deposition site. The majority of particles deposited at the back of the nasal passages and in the larynx (ET_2) are removed quickly by the fluids that cover the airways. In this region, particle clearance is completed within 15 minutes. Ciliary action removes deposited particles from both the bronchi and bronchioles. Though it is generally thought that mucocilliary action rapidly transports most particles deposited here toward the pharynx, a fraction of these particles are cleared more slowly. Evidence for this is found in human studies. For humans, retention of particles deposited in the lungs (BB and bb) is apparently biphasic. The "slow" action of the cilia may remove as many as half of the bronchi- and bronchiole-deposited particles. In human bronchi and bronchiole regions, mucus moves more slowly the closer to the alveoli it is. For the faster compartment, it has been estimated that it takes about 2 days for particles to travel from the bronchioles to the bronchi and 10 days from the bronchi to the pharynx. The second (slower) compartment (BB_2 and bb_2) is assumed to have fractions of the inhaled particles, depending on the particle size, deposited in BB_2 and bb_2; both have clearance half-times estimated at 20 days. A small fraction of particles deposited in the BB and bb regions is retained in the airway wall for even longer periods (BBseq and bbseq).

If particles reach and become deposited in the alveoli, they tend to stay imbedded in the fluid on the alveolar surface or move into the lymph nodes. The one mechanism by which particles are physically resuspended and removed from the AI region is coughing. For modeling purposes, the AI region is divided into three subcompartments to represent different clearance rates, all of which are slow.

Particle clearance from the alveolar-interstitial region has been measured in human subjects. The ICRP model uses 2 half-times to represent clearance: about 30% of the particles have a 30-day half-time, and

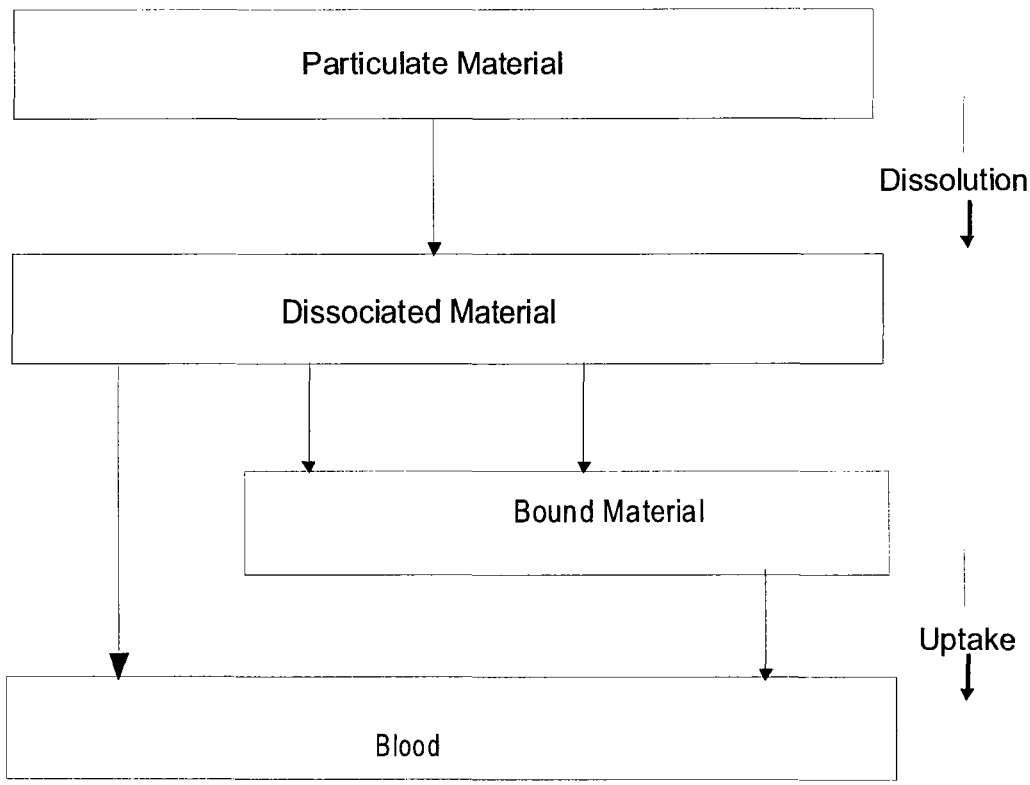
the remaining 70% are given a half-time of several hundred days. Over time, the AI particle transport rate falls and some compounds have been found in lungs 10–50 years after exposure.

Absorption into Blood. The ICRP model assumes that absorption into blood occurs at equivalent rates in all parts of the respiratory tract, except in the anterior nasal passages (ET_1), where no absorption occurs. It is essentially a 2-stage process, as shown in Figure 3-8. First, there is a dissociation (dissolution) of particles; then, the dissolved molecules or ions diffuse across capillary walls and are taken up by the blood. Immediately following dissolution, rapid absorption is observed. For some elements, rapid absorption does not occur because of binding to respiratory-tract components. In the absence of data for specific compounds, the model uses the following default absorption rate values for those compounds that are classified as Types F (fast), M (medium), S (slow), and V (instantaneous):

- For Type F, there is rapid 100% absorption within 10 minutes of the material deposited in the BB, bb, and AI regions, and 50% of material deposited in ET_2. Thus, for nose breathing, there is rapid absorption of approximately 25% of the deposit in ET and 50% for mouth breathing.
- For Type M, about 70% of the deposit in AI reaches the blood eventually. There is rapid absorption of about 10% of the deposit in BB and bb, and 5% of material deposited in ET_2. Thus, there is rapid absorption of approximately 2.5% of the deposit in ET for nose breathing, and 5% for mouth breathing.
- For Type S, 0.1% is absorbed within 10 minutes and 99.9% is absorbed within 7,000 days, so there is little absorption from ET, BB, or bb, and about 10% of the deposit in AI reaches the blood eventually.
- For Type V, complete absorption (100%) is considered to occur instantaneously.

ICRP (1995) considers the experimental and human data to support the following classifications: cobalt chloride and nitrate, Type F; cobalt oxides, Type M or S; cobalt in fused aluminosilicate or polystyrene, Type S; cobalt in mineral dusts such as fly ash and volcanic ash, Type M; cobalt metal and metal alloys, M or S. ICRP (1995) recommends assigning all cobalt aerosols to Type M in the absence of specific information supporting an alternative classification.

Figure 3-8. The Human Respiratory Tract Model: Absorption into Blood



Source: ICRP 1994

ICRP (1993) Cobalt Biokinetics Model.

Description of the model.

ICRP (1979, 1993) developed a 3-compartment model of the kinetics of ingested cobalt in humans that is applicable to infants, children, adolescents, and adults. Absorption of ingested cobalt is assumed to be 60% in infants up to 3 months of age, 30% from 3 months to 15 years of age, and 10% after age 15 years. Absorbed cobalt is assumed to distribute as follows: 50% is excreted (urine and feces combined in a 6:1 ratio), 5% is transferred to the liver, and 45% is transferred to other tissues (Figure 3-9). Elimination from tissue compartments is described by three first order rate constants representing slow, medium, and fast elimination pools with half-times of 6, 60, and 800 days, respectively. The elimination half-times are assumed to be independent of age.

Validation of the model.

The extent to which the ICRP model has been validated is not described in ICRP (1993).

Risk assessment.

The model has been used to establish radiation dose equivalents (Sv/Bq) of ingested ^{57}Co, ^{58}Co, and ^{60}Co for ages 3 months to 70 years (ICRP 1993).

Target tissues.

The model can be used to estimate the radiation dose from cobalt radionuclides to all major organs and can be applied to environmental and occupational exposures.

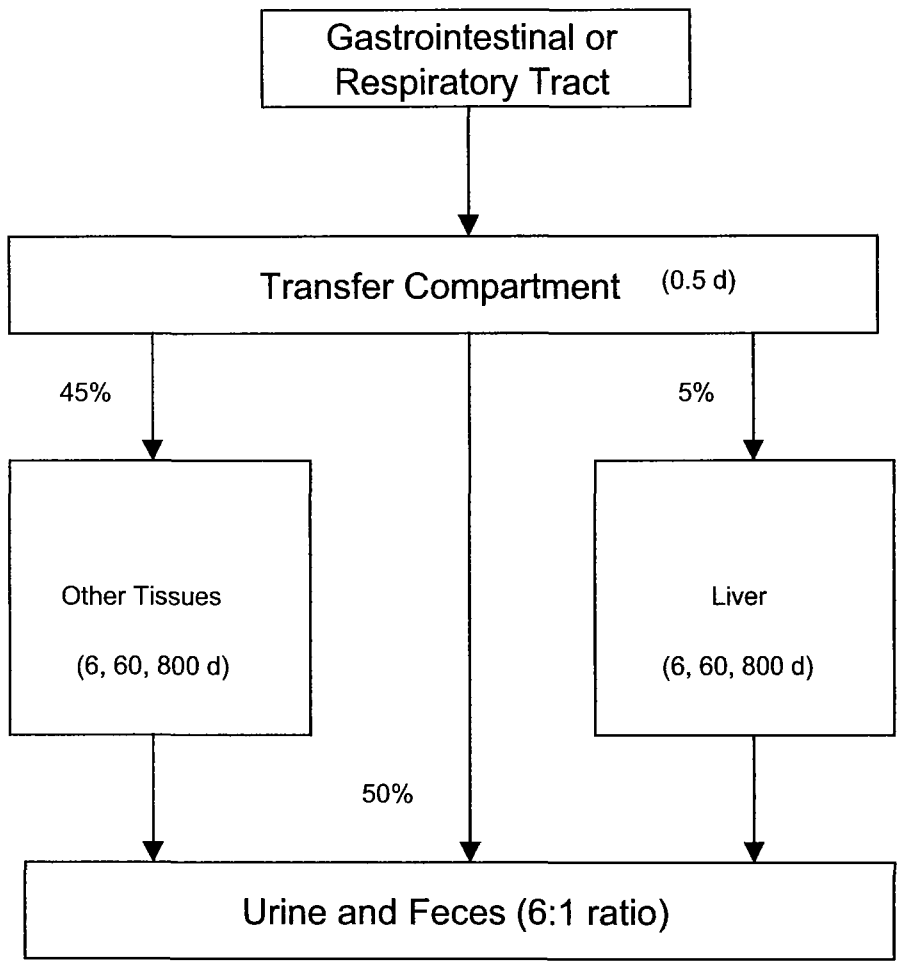
Species extrapolation.

The model is designed for applications to human dosimetry and cannot be applied to other species without modification.

*Inter*route extrapolation.

The model is designed to simulate oral exposures to cobalt and cannot be applied to other routes of exposure without modification.

Figure 3-9. ICRP Biokinetics Model for Cobalt



Absorbed cobalt enters a virtual transfer compartment from which unidirectional transfer to tissues is assumed to occur. Percentages shown are of the initial amounts absorbed. Numbers in parentheses are elimination half-times to urine and feces combined (d=days. Liver other tissues are assumed to have fast, medium, and slow elimination pools.

3.6 MECHANISMS OF ACTION

3.6.1 Pharmacokinetic Mechanisms

Absorption. Following inhalation exposure, the absorption of deposited cobalt compounds seems to be related to their biological solubility. Cobalt compounds deposit in the lungs based on their aerosol characteristics. Physiologically insoluble cobalt particles are generally cleared by phagocytosis and/or mucociliary transport, and thus, have a low systemic absorption. To some extent, cobalt particles may be dissolved within alveolar macrophages (Kreyling et al. 1990). More soluble forms of cobalt may enter the bloodstream through the alveolar or bronchial walls.

Following oral exposure, the absorption of cobalt varies with the amount given, with a greater dose leading to 4- to 20-fold greater fractional absorption (Smith et al. 1972). Nutritional status also seems to be an important factor in cobalt absorption, with both overnight fasting and iron deficiency resulting in increased cobalt absorption (Smith et al. 1972; Sorbie et al. 1971; Valberg et al. 1969). It has been suggested that cobalt and iron share a common absorptive pathway in the intestines, though the cobalt absorption takes place without ferritin (Reuber et al. 1994; Schade et al. 1970; Thomson et al. 1971). Solubility of the cobalt compound is also an important factor regarding the absorption following oral exposure, with increasing solubility resulting in increasing absorption (Christensen et al. 1993). One study in humans showed that oral exposure to cobalt resulted in significantly higher urinary excretion in females relative to males (Christensen et al. 1993), but these results have not been verified by other studies. A complex, specific pathway exists for the absorption of vitamin B_{12}, whereby the molecule interacts with several factors in the stomach and intestine to facilitate absorption (for review, see Russel-Jones and Alpers 1999).

Dermal absorption of cobalt compounds depends greatly on whether the skin is intact or damaged. Absorption through intact skin is comparatively low, while absorption through damaged skin is much higher (Inaba and Suzuki-Yasumoto 1979; Lacy et al. 1996).

Distribution. As a component of vitamin B_{12}, cobalt is found in most body tissues. Absorbed cobalt is transported throughout the body in the blood, with greatest levels found in the liver, followed by the kidney (Ayala-Fierro et al. 1999; Greenberg et al. 1943; Gregus and Klaassen 1986; Patrick et al. 1989).

Following inhalation exposure, significant levels of cobalt are found in the lungs of exposed humans and animals (Barnes et al. 1976; Brune et al. 1980; Collier et al. 1991; Gerhardsson et al. 1984; Hewitt 1988; Hillerdal and Hartung 1983; Kreyling et al. 1986; Kyono et al. 1992; Patrick et al. 1989; Talbot and Morgan 1989; Teraoka 1981). Within the lung, physiologically insoluble cobalt particles tend to be located within macrophages within the bronchial wall or in the interstitium close to the terminal bronchioli (Brune et al. 1980).

Excretion. Following inhalation exposure, the rate of urinary excretion appears to correlate with the rate of translocation of cobalt from the lungs to the blood, and the rate of fecal clearance with the rate of mechanical clearance of cobalt from the lungs to the gastrointestinal tract (Andre et al. 1989; Bailey et al. 1989; Collier et al. 1989; Kerfoot 1975; Kreyling et al. 1986, 1989; Palmes et al. 1959; Patrick et al. 1989; Talbot and Morgan 1989). Likewise, the majority of absorbed cobalt following oral exposure is rapidly removed from the body by excretion in the urine, and to a lesser extent in the bile and feces, with fecal elimination being the primary method of excretion for physiologically insoluble cobalt compounds in both humans and animals (Andre et al. 1989; Bailey et al. 1989; Collier et al. 1989; Harp and Scoular 1952; Paley et al. 1958; Patrick et al. 1989; Smith et al. 1972; Sorbie et al. 1971; Talbot and Morgan 1989; Valberg et al. 1969). The primary route for excretion following dermal exposure is the urine (Lacy et al. 1996; Scansetti et al. 1994).

3.6.2 Mechanisms of Toxicity

Stable Cobalt. The exact mechanisms by which cobalt exerts its effects on cells are not completely understood. However, a number of potential mechanisms have been identified. Several studies have demonstrated that hard metal, a metal alloy with a tungsten carbide and cobalt matrix, is considerably more toxic than either cobalt or tungsten carbide alone. A mechanism by which hard metal may exert its effects has been proposed by a group of Belgian researchers (Lasfargues et al. 1995; Lison et al. 1995, 1996). In this proposed mechanism, tungsten carbide, which is a very good conductor of electrons, facilitates the oxidation of cobalt metal to ionic cobalt (presumably Co^{2+}) by transferring electrons from the cobalt atom to molecular oxygen adjacent to the tungsten carbide molecule. The result is an increased solubility of cobalt, relative to cobalt metal alone, and the generation of active oxygen species. The cobalt ions formed may be absorbed into the blood and transported throughout the body, where they may elicit effects by the above mechanisms. *In vitro* evidence for this mechanism includes the ability of hard

metal particles, but neither cobalt nor tungsten carbide alone, to generate substantial levels of oxidant species and cause significant lipid peroxidation (Lison et al. 1995; Zanetti and Fubini 1997). Hard metal particles have also been shown to increase the levels of inducible nitric oxide synthase (iNOS), a gene responsive to oxidant stress (Rengasamy et al. 1999).

Another potential mechanism for cobalt toxicity is through oxidant-based and free radical-based processes. Exposure to soluble cobalt increases indices of oxidative stress, including diminished levels of reduced glutathione, increased levels of oxidized glutathione, activation of the hexose monophosphate shunt, and free-radical-induced DNA damage (Hoet et al. 2002; Kasprzak et al. 1994; Lewis et al. 1991; Zhang et al. 1998a); hydrogen peroxide appears to be a necessary cofactor for cobalt-induced oxidative DNA damage (Ivancsits et al. 2002). Cobalt has been shown to generate oxygen radicals, including superoxide, both *in vitro* and *in vivo* (Kadiiska et al. 1989; Kawanishi et al. 1994; Moorhouse et al. 1985), through what may be a Fenton-type mechanism (Lloyd et al. 1997). *In vivo* exposure to cobalt in rats and guinea pigs resulted in increased lipid peroxidation in the liver (Christova et al. 2001, 2002; Sunderman and Zaharia 1988), as well as changes in reduced glutathione and hepatic levels of superoxide dismutase, catalase, heme oxygenase, and glutathione peroxidase (Christova et al. 2001, 2002). Exposure to cobalt results in accumulation in cardiac tissues, and is thought to stimulate carotid-body chemoreceptors, mimicking the action of hypoxia (Di Giulio et al. 1990, 1991; Hatori et al. 1993; Morelli et al. 1994). Cobalt administration to a neuroblastoma/glioma cell line resulted in an upregulation of opioid delta receptors, through a mechanism similar to that of hypoxia (Mayfield et al. 1994). Exposure to cobalt also elicits effects on a number of genes known to be sensitive to oxidant status, including hypoxia-inducible factor 1, erythropoietin, vascular endothelial growth factor, catalase, and monooxygenase enzymes (Bunn et al. 1998; Daghman et al. 1999; Dalvi and Robbins 1978; Di Giulio et al. 1991; Goldberg et al. 1988, 1994; Ho and Bunn 1996; Hoet et al. 2002; Ladoux and Frelin 1994; Legrum et al. 1979; Semenza et al. 1994; Yasukochi et al. 1974), and may also lead, through these genes or other pathways, to the induction of apoptosis (Zou et al. 2001).

Soluble cobalt has also been shown to alter calcium influx into cells, functioning as a blocker of inorganic calcium channels (Henquin et al. 1983; Moger 1983; Yamatani et al. 1998). This mechanism has been linked to a reduction of steroidogenesis in isolated mouse Leydig cells (Moger 1983). Additionally, soluble cobalt has been shown to alter the inorganic calcium influx in liver cells after exposure to glucagon (Yamatani et al. 1998), and calcium influx into pancreatic β cells (Henquin et al. 1983) and

isolated rat islets (Henquin and Lambert 1975). Cobalt may also affect neuromuscular transmission though antagonism with calcium (Weakly 1973).

Another potential mechanism of cobalt toxicity is relevant to cobalt cardiomyopathy. As mentioned previously, cobalt accumulated in the heart of beer drinkers. Microscopic analysis revealed fragmentation and degeneration of myofibers and aggregates of abnormal mitochondria (Ferrans et al. 1964). These mitochondrial changes are indicative of disturbances in energy production or utilization possibly related to cobalt effects on lipoic acid. Cobalt irreversibly chelates lipoic acids under aerobic conditions (Webb 1982). Lipoic acid is a required cofactor for oxidative decarboxylation of pyruvate to acetyl CoA and of α-ketoglutarate to succinate (Lehninger 1982). In the myocadrium of rats treated with cobalt, oxidation of pyruvate or fatty acids is impaired (Wiberg 1968).

A number of investigators have reported that cobalt ions can result in increased damage to DNA when co-exposed with oxidants *in vitro*, such as UV radiation or H_2O_2 (De Boeck et al. 1998; Hartwig et al. 1991; Nackerdien et al. 1991). It is believed that cobalt acts by inhibition of DNA repair, particularly the incision and polymerization steps (Asmuß et al. 2000; Kasten et al. 1997), accomplishing this through interaction with zinc finger DNA repair proteins (Asmuß et al. 2000; Sarkar 1995).

Another potentially important mechanism by which cobalt may exert effects is through its effects on heme and heme-containing enzymes. Cobalt is thought to inhibit heme synthesis *in vivo* by acting upon at least two different sites in the biosynthetic pathway: synthesis of 5-aminolevulinate and conversion of 5-aminolevulinate into heme (de Matteis and Gibbs 1977). This inhibitory activity might result in the formation of cobalt protoporphyrin rather than heme (Sinclair et al. 1979). Cobalt treatment also stimulates heme oxidation in many organs, due to the induction of heme oxygenase (for review, see Sunderman 1987). Effects on heme synthesis may potentially affect a wide variety of heme-containing proteins, including monooxygenase enzymes (i.e., cytochromes P450) and catalase (Legrum et al. 1979; Yasukochi et al. 1974). Conversely, cobalt acts, through a mechanism believed to involve a heme-containing protein, to increase erythropoietin, which stimulates the production of red blood cells (Di Giulio et al. 1991; Goldberg et al. 1988; Smith and Fisher 1973). The regulatory mechanisms behind this apparent dichotomy have not been fully elucidated.

Another potential mechanism by which cobalt may exert its effects is through interactions with the immune system. Exposure of humans to cobalt by the inhalation and dermal routes have resulted in

sensitization to cobalt (Alomar et al. 1985; Bencko et al. 1983; Dooms-Goossens et al. 1980; Fischer and Rystedt 1983; Goh et al. 1986; Kanerva et al. 1988; Marcussen 1963; Shirakawa et al. 1988, 1989; Valer et al. 1967). Exposure to inhaled cobalt chloride aerosols can precipitate an asthmatic attack in sensitized individuals (Shirakawa et al. 1989), suggesting cobalt sensitization as one mechanism by which cobalt-induced asthma may be produced. IgE and IgA antibodies specific to cobalt have been reported in humans (Bencko et al. 1983; Shirakawa et al. 1988, 1989). There is evidence that cobalt sensitivity in humans may to be regulated by T-lymphocytes (Katsarou et al. 1997). A human helper T-lymphocyte cell line specific for cobalt (CoCl2) has been established (Löfström and Wigzell 1986). Cobalt may also interact directly with immunologic proteins, such as antibodies or Fc receptors, to result in immunosensitization (Cirla 1994). *In vitro*, cobalt(II) has been shown to reduce the proliferation of both B and T lymphocytes, as well as the release of the cytokines IL-2, IL-6, and IFN-Gamma (Wang et al. 1996). Interrelationships exist between nickel and cobalt sensitization (Bencko et al. 1983; Rystedt and Fisher 1983); however, the extent of any potential interactions between the two metals on immunologic end points is not well understood. In guinea pigs, nickel and cobalt sensitization appear to be interrelated and mutually enhancing (Lammintausta et al. 1985), though cross-reactivity was not reported to occur.

Cobalt has been shown to have a number of effects on glucose metabolism. Treatment of animals with cobalt results in a depression of serum (Eaton and Pommer 1973; Ybarra et al. 1997) or tissue (Wiberg 1968) glucose levels. In rats made diabetic by pretreatment with streptozotocin, this depression was persistent, whereas it was transient in normal rats (Ybarra et al. 1997). Many of the effects of cobalt on glucose metabolism are thought to result from alterations in the expression of the glut family of glucose transport proteins, a family of facilitative Na+-independent transport proteins thought to mediate non-insulin-dependent transport of glucose. Exposure to soluble cobalt results in increased expression of these genes, particularly GLUT1, in cells of the liver, kidney cortex, myocardium, skeletal muscle, and cerebrum (Behrooz and Ismail-Beigi 1997; Ybarra et al. 1997). Cobalt also reduces the amount of glucose produced in liver cells following stimulation with glucagon (Eaton and Pommer 1973; Yamatani et al. 1998), as well as reducing insulin release in isolated rat islets (Henquin and Lambert 1975).

Radioactive Cobalt. Due to the nature of its ionizing radiation, radioactive cobalt can present a health hazard. Highly-penetrating gamma emissions are the major source of damage to tissues and internal organs following external exposure to radioactive cobalt isotopes. If radioactive cobalt is internalized, nearby tissues are at highest risk for damage due to the release of beta particles. In either case, exposure to ionizing radiation results in an increased risk of cellular damage. Both beta and gamma radiations are

capable of producing ionization events when they hit cellular molecules, including DNA, RNA, or lipids. Ionized molecules within irradiated cells may be repaired quickly to prevent further damage. On the other hand, irreparable damage may be imposed on cellular materials, such as DNA, which might ultimately result in either cell death or the formation of cancerous tumors. Very large acute radiation doses can damage or kill enough cells to cause the disruption of organ systems, resulting in acute radiation syndrome or even death. Human and animal data indicate that sufficiently high exposures to cobalt radiation can result in adverse effects such as reduced fertility, abnormal development, genotoxicity, pulmonary fibrosis, gastrointestinal atrophy and fibrosis, hematological and lymphoreticular disorders, cancer, and death (Chang et al. 1999b; Davis et al. 1992; Dinehart et al. 1991; Hashimoto and Mitsuyasu 1967; Klener et al. 1986; Libshitz 1993; Myskowski and Safai 1981; Rauscher and Bauchinger 1983; Roschler and Woodard 1969; Roswit and White 1977; Stavem et al. 1985; Van Oort et al. 1984). For a more complete discussion of the mechanisms associated with the toxic effects of ionizing radiation, refer to Chapter 5 of the Toxicological Profile for Ionizing Radiation (Agency for Toxic Substances and Disease Registry 1999).

3.6.3 Animal-to-Human Extrapolations

Bailey et al. (1989) reported a wide variation across species, including man, in the retention and clearance of inhaled physiologically insoluble 57Co particles (see Table 3-8), noting that this variation illustrates the potential difficulty of extrapolating the results of animal lung retention experiments to human even qualitatively. Species differences in absorption of physiologically insoluble cobalt oxide following oral exposure do not appear to exist (Bailey et al. 1989), although humans were not examined. Absorption of soluble cobalt compounds is greater in rats (13–34%) than in dairy cows (1–2%) and guinea pigs (4–5%) following oral exposure (Ayala-Fierro et al. 1999; Barnaby et al. 1968; Hollins and McCullough 1971; Kirchgessner et al. 1994; Naylor and Harrison 1995; Schade et al. 1970; Taylor 1962; van Bruwaene et al. 1984).

3.7 TOXICITIES MEDIATED THROUGH THE NEUROENDOCRINE AXIS

Recently, attention has focused on the potential hazardous effects of certain chemicals on the endocrine system because of the ability of these chemicals to mimic or block endogenous hormones. Chemicals with this type of activity are most commonly referred to as endocrine disruptors. However, appropriate

terminology to describe such effects remains controversial. The terminology endocrine disruptors, initially used by Colborn and Clement (1992), was also used in 1996 when Congress mandated the Environmental Protection Agency (EPA) to develop a screening program for "...certain substances [which] may have an effect produced by a naturally occurring estrogen, or other such endocrine effect[s]...". To meet this mandate, EPA convened a panel called the Endocrine Disruptors Screening and Testing Advisory Committee (EDSTAC), which in 1998 completed its deliberations and made recommendations to EPA concerning *endocrine disruptors*. In 1999, the National Academy of Sciences released a report that referred to these same types of chemicals as *hormonally active agents*. The terminology *endocrine modulators* has also been used to convey the fact that effects caused by such chemicals may not necessarily be adverse. Many scientists agree that chemicals with the ability to disrupt or modulate the endocrine system are a potential threat to the health of humans, aquatic animals, and wildlife. However, others think that endocrine-active chemicals do not pose a significant health risk, particularly in view of the fact that hormone mimics exist in the natural environment. Examples of natural hormone mimics are the isoflavinoid phytoestrogens (Adlercreutz 1995; Livingston 1978; Mayr et al. 1992). These chemicals are derived from plants and are similar in structure and action to endogenous estrogen. Although the public health significance and descriptive terminology of substances capable of affecting the endocrine system remains controversial, scientists agree that these chemicals may affect the synthesis, secretion, transport, binding, action, or elimination of natural hormones in the body responsible for maintaining homeostasis, reproduction, development, and/or behavior (EPA 1997c). Stated differently, such compounds may cause toxicities that are mediated through the neuroendocrine axis. As a result, these chemicals may play a role in altering, for example, metabolic, sexual, immune, and neurobehavioral function. Such chemicals are also thought to be involved in inducing breast, testicular, and prostate cancers, as well as endometriosis (Berger 1994; Giwercman et al. 1993; Hoel et al. 1992).

The available human and animal data suggest that the endocrine system, particularly the thyroid gland, may be a target of stable and radioactive cobalt toxicity. These effects are discussed in Sections 3.2 and 3.3 under Systemic Effects.

3.8 CHILDREN'S SUSCEPTIBILITY

This section discusses potential health effects from exposures during the period from conception to maturity at 18 years of age in humans, when all biological systems will have fully developed. Potential

effects on offspring resulting from exposures of parental germ cells are considered, as well as any indirect effects on the fetus and neonate resulting from maternal exposure during gestation and lactation. Relevant animal and *in vitro* models are also discussed.

Children are not small adults. They differ from adults in their exposures and may differ in their susceptibility to hazardous chemicals. Children's unique physiology and behavior can influence the extent of their exposure. Exposures of children are discussed in Section 6.6 Exposures of Children.

Children sometimes differ from adults in their susceptibility to hazardous chemicals, but whether there is a difference depends on the chemical (Guzelian et al. 1992; NRC 1993). Children may be more or less susceptible than adults to health effects, and the relationship may change with developmental age (Guzelian et al. 1992; NRC 1993). Vulnerability often depends on developmental stage. There are critical periods of structural and functional development during both prenatal and postnatal life and a particular structure or function will be most sensitive to disruption during its critical period(s). Damage may not be evident until a later stage of development. There are often differences in pharmacokinetics and metabolism between children and adults. For example, absorption may be different in neonates because of the immaturity of their gastrointestinal tract and their larger skin surface area in proportion to body weight (Morselli et al. 1980; NRC 1993); the gastrointestinal absorption of lead is greatest in infants and young children (Ziegler et al. 1978). Distribution of xenobiotics may be different; for example, infants have a larger proportion of their bodies as extracellular water and their brains and livers are proportionately larger (Altman and Dittmer 1974; Fomon 1966; Fomon et al. 1982; Owen and Brozek 1966; Widdowson and Dickerson 1964). The infant also has an immature blood-brain barrier (Adinolfi 1985; Johanson 1980) and probably an immature blood-testis barrier (Setchell and Waites 1975). Many xenobiotic metabolizing enzymes have distinctive developmental patterns. At various stages of growth and development, levels of particular enzymes may be higher or lower than those of adults, and sometimes unique enzymes may exist at particular developmental stages (Komori et al. 1990; Leeder and Kearns 1997; NRC 1993; Vieira et al. 1996). Whether differences in xenobiotic metabolism make the child more or less susceptible also depends on whether the relevant enzymes are involved in activation of the parent compound to its toxic form or in detoxification. There may also be differences in excretion, particularly in newborns who all have a low glomerular filtration rate and have not developed efficient tubular secretion and resorption capacities (Altman and Dittmer 1974; NRC 1993; West et al. 1948). Children and adults may differ in their capacity to repair damage from chemical insults. Children also

have a longer remaining lifetime in which to express damage from chemicals; this potential is particularly relevant to cancer.

Certain characteristics of the developing human may increase exposure or susceptibility, whereas others may decrease susceptibility to the same chemical. For example, although infants breathe more air per kilogram of body weight than adults breathe, this difference might be somewhat counterbalanced by their alveoli being less developed, which results in a disproportionately smaller surface area for alveolar absorption (NRC 1993).

Though human data are lacking, animal studies have suggested several differences in pharmacokinetic behavior of cobalt compounds between children and adults. Following inhalation exposure to Co_3O_4, deposition tended to increase with age, though no significant differences were reported (Collier et al. 1991). The youngest animals exposed (3 weeks postnatal) had the lowest fractional retention 182 days postexposure, though no differences were seen at day 7 or 83. The authors attributed this to a faster rate of translocation of cobalt from the lung to the blood, which could enhance subsequent excretion. Naylor and Harrison (1995) reported that in rats and guinea pigs, fractional absorption of cobalt following oral exposure was highest at 1 day after birth, and diminished rapidly with time thereafter. Collier et al. (1991) reported no difference in absorption of cobalt nitrate following oral exposure to animals aged 3–46 weeks, which is in agreement with the results of the later portion of the Naylor and Harrison (1995) study. No PBPK models specific for cobalt exposures to children were located. However, the ICRP Human Respiratory Tract Model is applicable to children, and may be used for children if the appropriate values for the parameters are used.

Once in the bloodstream, soluble cobalt compounds have been shown, in animal studies, to cross the placenta and enter the fetus. Twenty-four hours after intravenous injection of cobalt chloride in rats, 0.14% of the dose was found in the fetus, 0.19% in the chorioallantoic placenta, and 0.22% in the yolk sac (Zylicz et al. 1975). Several other rat studies (Nishimura et al. 1978; Zylicz et al. 1975, 1976) have demonstrated that the amount of cobalt crossing the placenta following intravenous injection is greater in later gestation stages, though the percent of the maternal dose reaching the fetus is still relatively low (in <1% of the maternal dose). The fetal uptake of cobalt following intravenous administration to the mother was increased when the cobalt was given as cyanocobalmin, relative to cobalt chloride (~5% of the maternal dose for cyanocobalmin, compared to <1% for cobalt chloride) (Nishimura et al. 1978), indicating that the form of the cobalt compound may affect its availability to the fetus.

Cobalt has been detected in human breast milk (Byczkowski et al. 1994; Kratchler et al. 1998). In general, physiological concentrations of cobalt in breast milk are very low, on the order of parts per billion (Byczkowski et al. 1994). Animal studies are in agreement with this observation. By day 70 post-exposure in lactating dairy cows orally exposed to cobalt chloride, the milk contained 0.012% of the dose (van Bruwaene et al. 1984). One to two percent of cobalt given intravenously to mother rats as cyanocobalmin was transferred to offspring via the breast milk (Nishimura et al. 1978).

Health Effects from Exposure to Stable Cobalt. Available data have not clearly defined whether children are at greater risk from exposure to stable cobalt than adults. Studies in adult humans have identified several health effects of cobalt compounds following inhalation, oral, or dermal exposure. Data on effects of cobalt in children following inhalation exposures are lacking. Jacobziner and Raybin (1961) reported on two cases of children who had accidentally ingested unknown amounts of cobalt chloride; a 19-month-old male died approximately 6.5 hours after ingestion, whereas a 3-year-old male was given medical treatment and showed no symptoms after ingestion. Several studies (Chamberlain 1961; Little and Sunico 1958; Sederholm et al. 1968; Washburn and Kaplan 1964) have reported enlarged thyroid glands in children given cobalt chloride for treatment of anemia; removal of cobalt therapy resulted in a return to normal thyroid size. Patch testing of children aged 4–14 years revealed a 13.3% dermal sensitization rate to cobalt chloride (Romaguera and Vilaplana 1998). More girls reacted positively than boys, which the authors attributed to the wearing of costume jewelry, which often contains cobalt, and the resulting exposure.

Offspring of mice intravenously injected with approximately 1.2 mg cobalt/kg at day 8 of gestation, but not at day 3, showed a significant increase in the number of skeletons with delayed ossification (Wilde 1984). Other studies, however, have not shown developmental effects of stable cobalt compounds, or have shown effects only at maternally toxic doses (Domingo et al. 1985b; Paternian et al. 1988; Seidenberg 1986).

Health Effects from Exposure to Radioactive Cobalt. Taiwanese children (48 boys, 37 girls) who were raised in apartments contaminated with ^{60}Co were compared to 21,898 age- and sex-matched non-exposed children from a nationwide surveillance program (Wang et al. 2001). After adjusting for effects from parental heights and body mass index, clear dose-related decreases in height percentile (HP) and age-specific relative height differences (RHD) were seen in exposed boys, but not in exposed girls. Average

cumulative exposures were 120.8 and 129.9 mSv (equivalent to ~12.1 or 13 rad) for the boys and girls, respectively.

No other studies of human children exposed to radioactive cobalt or cobalt radiation were located. As rapidly-dividing cells are more sensitive to radiation, the developing fetus and growing children are expected to be more sensitive to cobalt radiation than adults.

Animal studies have shown that exposures to external radiation from cobalt isotopes (as low as 10 rad [0.1 Gy] in mice) may have a dramatic effect on the developing fetus (see Section 3.2.4.6 and Agency for Toxic Substances and Disease Registry 1999). Exposure duration, gestational day, and dose all influence the effect of cobalt radiation on the developing organism. Radiation exposure to very young dogs (80 rad [0.8 Gy] on day 2 or 70 postpartum) has resulted in an increased incidence of diabetes mellitus, renal disease, and cancer (Benjamin et al. 1998a, 1998b).

3.9 BIOMARKERS OF EXPOSURE AND EFFECT

Biomarkers are broadly defined as indicators signaling events in biologic systems or samples. They have been classified as markers of exposure, markers of effect, and markers of susceptibility (NAS/NRC 1989).

Due to a nascent understanding of the use and interpretation of biomarkers, implementation of biomarkers as tools of exposure in the general population is very limited. A biomarker of exposure is a xenobiotic substance or its metabolite(s) or the product of an interaction between a xenobiotic agent and some target molecule(s) or cell(s) that is measured within a compartment of an organism (NAS/NRC 1989). The preferred biomarkers of exposure are generally the substance itself or substance-specific metabolites in readily obtainable body fluid(s), or excreta. However, several factors can confound the use and interpretation of biomarkers of exposure. The body burden of a substance may be the result of exposures from more than one source. The substance being measured may be a metabolite of another xenobiotic substance (e.g., high urinary levels of phenol can result from exposure to several different aromatic compounds). Depending on the properties of the substance (e.g., biologic half-life) and environmental conditions (e.g., duration and route of exposure), the substance and all of its metabolites may have left the body by the time samples can be taken. It may be difficult to identify individuals exposed to hazardous

substances that are commonly found in body tissues and fluids (e.g., essential mineral nutrients such as copper, zinc, and selenium). Biomarkers of exposure to cobalt are discussed in Section 3.9.1.

Biomarkers of effect are defined as any measurable biochemical, physiologic, or other alteration within an organism that, depending on magnitude, can be recognized as an established or potential health impairment or disease (NAS/NRC 1989). This definition encompasses biochemical or cellular signals of tissue dysfunction (e.g., increased liver enzyme activity or pathologic changes in female genital epithelial cells), as well as physiologic signs of dysfunction such as increased blood pressure or decreased lung capacity. Note that these markers are not often substance specific. They also may not be directly adverse, but can indicate potential health impairment (e.g., DNA adducts). Biomarkers of effects caused by cobalt are discussed in Section 3.9.2.

A biomarker of susceptibility is an indicator of an inherent or acquired limitation of an organism's ability to respond to the challenge of exposure to a specific xenobiotic substance. It can be an intrinsic genetic or other characteristic or a preexisting disease that results in an increase in absorbed dose, a decrease in the biologically effective dose, or a target tissue response. If biomarkers of susceptibility exist, they are discussed in Section 3.11 "Populations That Are Unusually Susceptible".

3.9.1 Biomarkers Used to Identify or Quantify Exposure to Cobalt

Biomonitoring data exist that demonstrate a positive correlation between occupational exposure levels of cobalt and the levels of cobalt in both the urine and blood (Table 3-12) (Alexandersson 1988; Ichikawa et al. 1985; Lison et al. 1994; Nemery et al. 1992; Scansetti et al. 1985). Available studies of unexposed humans have reported cobalt blood levels of 0.05–0.19 μg/dL and urinary cobalt levels of 0.04–2 μg/dL (Alexandersson 1988; Ichikawa et al. 1985). Figure 3-10 graphically presents the cobalt exposure data and cobalt in blood data presented in Table 3-12 (Ichikawa et al. 1985). The highest excretion rate of cobalt in urine occurs during the first 24 hours after short-term exposure; therefore, subjects should be tested quickly to assess whether cobalt exposure has occurred (Alexandersson 1988). Occupational exposure to 0.1 mg/m^3 cobalt resulted in blood levels of cobalt ranging (95% CI) from 0.57 to 0.79 μg/dL, compared to 0.19 μg/dL in unexposed workers, and urinary levels from 59 to 78 μg/L, compared to 2 μg/L in unexposed workers (Ichikawa et al. 1985). Correlations between recent exposure and cobalt levels in the blood or urine are more consistent for soluble cobalt compounds (metal, salts, and hard

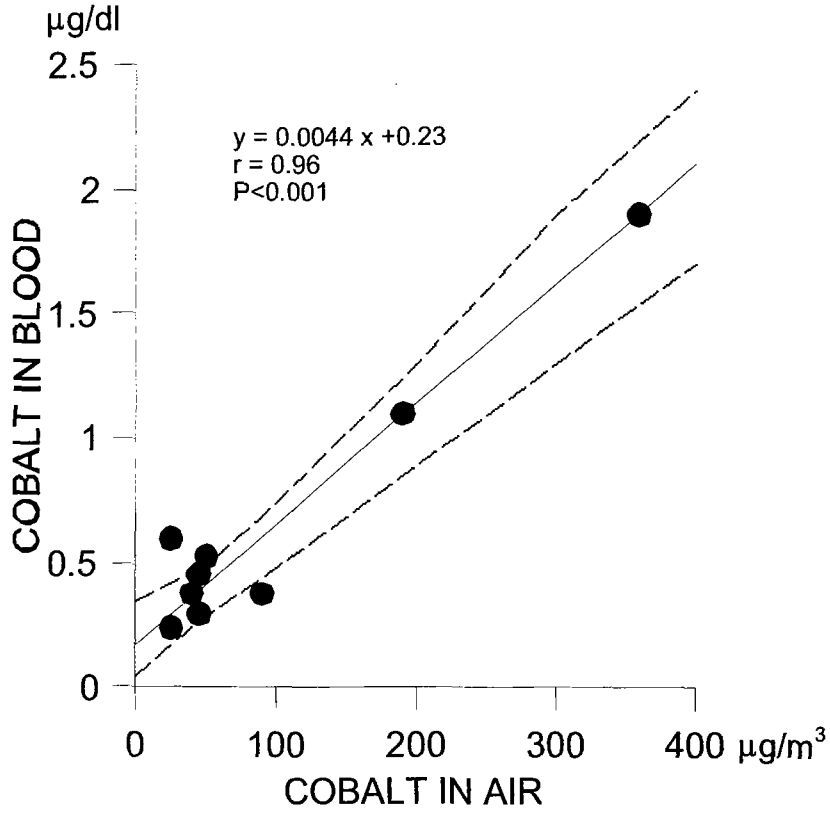
Table 3-12. Cobalt Exposure Concentrations and Amounts in the Blood and Urine of Subjects Examined[a]

Subjects	Number	Cobalt in air[b] mean±SD μg/m^3		Cobalt in blood[b] mean±SD μg/dL		Cobalt in urine[b] mean±SD μg/L	
Powder handlers	2	186±108	(110–262)	1.08±0.28	(0.88–1.28)	148±13	(138–158)
Rubber press operators	6	367±324	(92–859)	1.87±1.96	(0.40–5.30)	235±182	(41–392)
Automatic press operators	11	56±60	(9–210)	0.57±0.53	(0.10–0.95)	34±43	(4–73)
Shapers (lathing)	7	33±15	(15–62)	0.67±0.44	(0.14–1.34)	33±30	(11–95)
Shapers (sawing)	21	50±35	(8–144)	0.52±0.31	(0.15–1.15)	41±60	(6–266)
Sintering workers	21	28±30	(4–145)	0.26±0.10	(0.09–0.45)	10±10	(2–46)
Wet grinders							
A	27	44±48	(4–227)	0.42±0.31	(0.10–1.30)	35±34	(2–180)
B	18	45±50	(3–161)	0.33±0.10	(0.16–0.52)	19±15	(2–67)
C	12	92±92	(15–291)	0.43±0.39	(0.12–1.90)	68±87	(3–265)
D	25	44±54	(3–205)	0.35±0.20	(0.10–1.00)	17±16	(1–69)
Workers using respirators	25	317±307	(7–1,203)	0.65±0.86	(0.20–3.90)	26±30	(1–119)
Office workers	20	No data		0.19±0.11	(0.08–0.40)	2±1	(1–4)

[a]Adapted from Ichikawa et al. 1985
[b]The range of each value is given in parentheses.

SD = standard deviation

Figure 3-10. Relation Between Mean Cobalt Exposure and Mean Blood Concentration of Cobalt in Exposed Workers*



*Adapted from Ichikawa et al. 1985

metals), while blood and/or urinary cobalt levels are less reflective of recent exposure for less soluble compounds (cobalt oxides) (Lison et al. 1994).

Sensitive serum protein responses were found in animals exposed to cobalt at levels below those necessary to produce hematopoietic effects (Stokinger and Wagner 1958). These serum protein responses included an increase in alpha globulin fractions of serum proteins and associated serum neuraminic acid. The responses were observed in rabbits and dogs following both inhalation and injection of cobalt. The authors indicated that this increase was a unique response to cobalt exposure. The characteristics of the response were similar to those of the erythropoietic response found following exposure to higher levels of cobalt; the response is delayed, does not occur in all animals within a given exposure group, is not of great magnitude, and is not persistent (Stokinger and Wagner 1958).

Biomarkers specific for exposure to cobalt radioisotopes have not been reported.

3.9.2 Biomarkers Used to Characterize Effects Caused by Cobalt

Sensitization to cobalt results in cobalt-specific changes in serum antibodies (IgE and IgA) that may be monitored to determine if sensitization, or additional exposure, to cobalt has occurred (Bencko et al. 1983; Shirakawa et al. 1988, 1989).

No biomarkers specific for effects of radioactive cobalt isotopes have been reported. Biomarkers for response to ionizing radiation are discussed in Agency for Toxic Substances and Disease Registry (1999).

3.10 INTERACTIONS WITH OTHER CHEMICALS

A major medical use of cobalt is in combination with bleomycin, an antineoplastic antibiotic, as a tumor-localizing and therapeutic agent (Goodwin and Meares 1976; Hansen et al. 1976; Kapstad 1978, 1979). The anti-tumor effects of the two agents are amplified when given in combination with each other. The complex, wherein cobalt is coordinately bound to the bleomycin molecule, is intravenously injected and acts by binding to and cleaving the DNA in the tumor cells (Kakinuma and Orii 1982).

The interaction of cobalt with various chelators has been investigated in animals for mitigation of the toxicity of cobalt (Baker et al. 1987; Domingo et al. 1983; Llobet et al. 1988). Glutathione, N-acetyl-L-cysteine (NAC) and diethylenetriaminepentaacetic acid (DTPA), administered to rats previously exposed to cobalt, significantly increased urinary excretion of cobalt, while EDTA, NAC, and 2,3-di-mercaptosuccinic acid (DMSA) increased fecal excretion. NAC was the most effective chelator because it increased both urinary and fecal excretion of cobalt while decreasing its levels in liver and spleen (Llobet et al. 1988). Cysteine, also acting as a chelator, mitigated the toxicity of cobalt when both chemicals were given to chicks in the feed (Baker et al. 1987).

A number of studies have suggested an association between cobalt ions and calcium ions. Soluble cobalt has also been shown to alter calcium influx into cells, functioning as a blocker of inorganic calcium channels (Henquin et al. 1983; Moger 1983; Yamatani et al. 1998). This mechanism has been linked to a reduction of steroidogenesis in isolated mouse Leydig cells (Moger 1983). Additionally, soluble cobalt has been shown to alter the inorganic calcium influx in liver cells after exposure to glucagon (Yamatani et al. 1998), and calcium influx into pancreatic β cells (Henquin et al. 1983) and isolated rat islets (Henquin and Lambert 1975). Cobalt may also affect neuromuscular transmission through antagonism with calcium (Weakly 1973).

Hard metal, consisting of 5–10% cobalt with the balance being tungsten carbide, has been shown to be considerably more toxic than cobalt alone, resulting from interactions between particles of cobalt metal and tungsten carbide particles. The mechanisms responsible for this interaction are discussed in Section 3.6.2.

An interrelationship between cobalt and nickel sensitization has been reported in individuals exposed to the two metals (Rystedt and Fisher 1983; Veien et al. 1987), as well as in animal studies (Wahlberg and Lidén 2000). It was concluded that the combination of nickel sensitivity and irritant eczema resulted in a high risk for developing an allergy to cobalt. Studies of cultured alveolar type II cells showed a synergistic (greater than additive) response to co-exposure to cobalt and nickel chlorides (Cross et al. 2001).

3.11 POPULATIONS THAT ARE UNUSUALLY SUSCEPTIBLE

A susceptible population will exhibit a different or enhanced response to cobalt than will most persons exposed to the same level of cobalt in the environment. Reasons may include genetic makeup, age, health and nutritional status, and exposure to other toxic substances (e.g., cigarette smoke). These parameters result in reduced detoxification or excretion of cobalt, or compromised function of organs affected by cobalt. Populations who are at greater risk due to their unusually high exposure to cobalt are discussed in Section 6.7, Populations With Potentially High Exposures.

Individuals who are already sensitized to cobalt may be unusually susceptible because cobalt exposure may trigger asthmatic attacks (Shirakawa et al. 1988, 1989). Sensitization to cobalt results in cobalt-specific changes in serum antibodies (IgE and IgA) (Bencko et al. 1983; Shirakawa et al. 1988, 1989). Potolicchio et al. (1997, 1999) have suggested that individuals with a polymorphism in the HLA-DP gene (presence of glutamate 69 in the β chain) may be more susceptible to hard metal lung disease. Individuals with ongoing respiratory illness may also be more susceptible to the effects of inhaled cobalt. Following oral exposure, individuals with iron deficiency may at greater risk, as animal studies have shown an increased absorption of cobalt compounds in iron-deficient animals (Reuber et al. 1994; Schade et al. 1970). Studies of beer-cobalt cardiomyopathy have suggested that individuals with high alcohol consumption may be more susceptible to health effects of cobalt (Alexander 1969, 1972; Morin et al. 1971).

Ionizing radiation has greater effects on rapidly-dividing cells than on those that divide at a slower rate. The most sensitive population to exposure to cobalt radiation is likely to be the developing fetus, as even moderate exposures to cobalt radiation have been shown to cause dramatic effects on the developing fetus in animal studies (see Section 3.2.4.6), Likewise, growing children are likely to be more susceptible to cobalt radiation than adults, and people who are immunocompromised, have existing lung diseases, or who have defects in genetic repair enzymes would be expected to show an increased susceptibility to cobalt radiation. A detailed discussion on the effects of ionizing radiation in children can be found in Agency for Toxic Substances and Disease Registry (1999).

3.12 METHODS FOR REDUCING TOXIC EFFECTS

This section will describe clinical practice and research concerning methods for reducing toxic effects of exposure to cobalt. However, because some of the treatments discussed may be experimental and unproven, this section should not be used as a guide for treatment of exposures to cobalt. When specific exposures have occurred, poison control centers and medical toxicologists should be consulted for medical advice. The following texts provide specific information about treatment following exposures to cobalt:

Ellenhorn MJ, Schonwald S, Ordog G, et al., eds. 1997. Medical toxicology: Diagnosis and treatment of human poisoning. 2nd edition. Baltimore, MD: Williams & Wilkins, 1682–1723.

Goldfrank, LR, Flomenbaum, NE, Lewin, NA, et al., eds. 1998. Toxicological emergencies. 6th edition. Connecticut: Appleton & Lange, 481t, 489, 490t, 1338–1339.

REAC/TS. Radiation Emergency Assistance Center/Training Site. www.orau.gov/reacts/.

3.12.1 Reducing Peak Absorption Following Exposure

Methods for reducing peak absorption are similar for both the stable and radioactive forms of cobalt. General management and treatment of patients following acute exposure to cobalt includes removal of the victim from the contaminated area, and removal and isolation of contaminated clothing, jewelry, and shoes (Bronstein and Currance 1988; Stutz and Janusz 1988). The excess solid contaminant is gently brushed away, and excess liquids are blotted with absorbent material. If the victim is in respiratory distress, ventilation assistance is provided and oxygen is administered. Measures that are appropriate to the route of exposure are then taken to remove cobalt from the body. Following ocular exposure, the eyes are immediately flushed thoroughly with water. Skin is washed immediately with soap or mild detergent and water. Some evidence has been presented that the use of cheating creams on the skin can reduce the occurrence of symptoms in allergic persons (Wöhrl et al. 2001). Following ingestion of cobalt, two conflicting forms of treatment have been recommended. Stutz and Janusz (1988) recommend that victims over 1 year old be given ipecac, followed by activated charcoal (after vomiting). A cathartic, such as magnesium sulfate in water, is then administered to adults and children. Bronstein and Currance (1988) recommend that the victim be given water for dilution of the cobalt; however, they recommend that emetics not be administered. Following all routes of exposure, victims are monitored for pulmonary edema, circulatory collapse, and shock, and treated as necessary.

3.12.2 Reducing Body Burden

Chelation therapy with EDTA or dimercaprol can be effectively used if necessary (Goldfrank et al. 1990; Haddad and Winchester 1990; Stutz and Janusz 1988). Animal studies have investigated the effectiveness of various chelating agents for mitigating the toxicity of cobalt (Baker et al. 1987; Domingo et al. 1983; Llobet et al. 1988). NAC was found to be the most effective chelator because it increased both urinary and fecal excretion of cobalt as well as decreased the levels of cobalt in the liver and spleen (Llobet et al. 1988). These chelators react chemically with cobalt, so they are effective for both stable and radioactive cobalt isotopes. For more complete information on treatment of specific symptoms, refer to Bronstein and Currance (1988) and Stutz and Janusz (1988).

3.12.3 Interfering with the Mechanism of Action for Toxic Effects

No studies were located in humans or animals regarding interfering with the mechanism of action of stable or radioactive cobalt compounds.

3.13 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of cobalt is available. Where adequate information is not available, ATSDR, in conjunction with the National Toxicology Program (NTP), is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of cobalt.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

3.13.1 Existing Information on Health Effects of Cobalt

The existing data on health effects of inhalation, oral, and dermal exposure of humans and animals are summarized in Figure 3-11 for stable cobalt and in Figure 3-12 for radioactive cobalt. The purpose of these figures is to illustrate the existing information concerning the health effects of cobalt. Each dot in the figure indicates that one or more studies provide information associated with that particular effect. The dot does not necessarily imply anything about the quality of the study or studies, nor should missing information in this figure be interpreted as a "data need". A data need, as defined in ATSDR's Decision Guide for Identifying Substance-Specific Data Needs Related to Toxicological Profiles (Agency for Toxic Substances and Disease Registry 1989), is substance-specific information necessary to conduct comprehensive public health assessments. Generally, ATSDR defines a data gap more broadly as any substance-specific information missing from the scientific literature.

Figures 3-11 and 3-12 represent studies conducted with all forms of cobalt. The effects of cobalt have been studied in humans following both inhalation and oral exposure. Human dermal studies designed to investigate nondermal systemic effects of cobalt have been reported. Similarly, the effects of cobalt in animals have been studied following inhalation and oral exposure. Few dermal studies are available.

3.13.2 Identification of Data Needs

Stable Cobalt. Effects in humans following acute inhalation, oral, and dermal exposures to cobalt have been reported. In humans, the primary targets following acute exposure to cobalt include the respiratory system following inhalation exposure (Kusaka et al. 1986a), the thymus following oral exposure (Roche and Layrisse 1956), and the immunological system following dermal exposure (Alomar et al. 1985; Fischer and Rystedt 1983; Kanerva et al. 1988). Acute oral studies in animals have also identified the cardiovascular and hematopoietic systems as targets of cobalt toxicity (Domingo and Llobet 1984; Speijers et al. 1982). Although acute exposure levels associated with some of these effects in humans have been reported, the minimal acute exposure levels required to produce these effects are not known because few acute human studies exist. The results of animal studies of the acute toxicity of cobalt have been used to determine dose levels that produce death and respiratory effects following inhalation exposure, death and various systemic effects following oral exposure, and dermal and immunological effects following dermal exposure.

Figure 3-11. Existing Information on Health Effects of Stable Cobalt

	Death	Acute	Intermediate	Chronic	Immunologic/Lymphoretic	Neurologic	Reproductive	Developmental	Genotoxic	Cancer
	Systemic									
Inhalation	●	●		●	●	●				●
Oral	●		●		●			●		
Dermal				●	●					

Human

	Death	Acute	Intermediate	Chronic	Immunologic/Lymphoretic	Neurologic	Reproductive	Developmental	Genotoxic	Cancer
	Systemic									
Inhalation	●	●	●	●	●	●	●			●
Oral	●	●	●		●	●	●	●		
Dermal	●	●			●					

Animal

● Existing Studies

Figure 3-12. Existing Information on Health Effects of Radioactive Cobalt

	Death	Acute	Intermediate	Chronic	Immunologic/Lymphoretic	Neurologic	Reproductive	Developmental	Genotoxic	Cancer
	Systemic									
Inhalation										
Oral										
Dermal										
External	●	●	●	●	●	●			●	●

Human

	Death	Acute	Intermediate	Chronic	Immunologic/Lymphoretic	Neurologic	Reproductive	Developmental	Genotoxic	Cancer
	Systemic									
Inhalation										
Oral										
Dermal										
External	●	●	●	●	●	●	●	●	●	●

Animal

● Existing Studies

There were insufficient data for derivation of inhalation or oral acute MRLs because reported effects were severe and occurred at levels above those reported in the few human studies. Animal studies that identify minimally effective inhalation and oral exposure levels for the various cobalt compounds would be useful in estimating acute MRLs for each cobalt compound. Acute dermal studies would enable the determination of hazardous levels for this route of exposure. Because a small portion of the cobalt taken into the body is retained for a relatively long time, studies on the long-term consequences of acute exposure on the heart, respiratory tract, hematological system, and immune response could provide information about the potential for chronic effects of acute exposures in humans. Knowledge about the acute toxicity of cobalt is important because people living near hazardous waste sites might be exposed for brief periods.

Radioactive Cobalt. Data on health effects following acute exposure to radioactive cobalt by the inhalation, oral, or dermal routes are lacking. Because all cobalt radioisotopes are man-made (see Chapter 5), only low-level exposure to radioactive cobalt in the environment by these routes is likely to occur. A number of health effects have been seen following cases of accidental acute exposure to high levels of external cobalt radiation in humans, including death, gastrointestinal disorders, hematological alterations, and dermal lesions (Klener et al. 1986; Stavem et al. 1985). Acute-exposure animal studies have shown pronounced effects, including death, cardiovascular changes, gastrointestinal effects, kidney effects, and neurobehavioral changes (Brady and Hayton 1977b; Bruner 1977; Cockerham et al. 1986; Darwezah et al. 1988; Down et al. 1986; Gomez-d-Segura et al. 1998; Hanks et al. 1966; King 1988a; Mele et al. 1988; Page et al. 1968; Robbins 1989a, 1989b, 1989c, 1991a). The most pronounced effects in animals following acute exposure to cobalt radiation have been reproductive and developmental effects (see Sections 3.2.4.5 and 3.2.4.6). Agency for Toxic Substances and Disease Registry (1999) has derived an acute MRL for external exposure to ionizing radiation, which is applicable to external exposures to cobalt radiation, so additional data for the derivation of an MRL are not needed.

Intermediate-Duration Exposure.

Stable Cobalt. Information on oral exposure of humans to cobalt, in the form of cobalt chloride added to beer as a foam stabilizer, provides the only human data available for exposure of intermediate duration (Alexander 1969, 1972; Morin et al. 1971). Inhalation and dermal data in humans were not located for this duration of exposure. The cardiac and hematopoietic systems are the primary targets in humans following oral exposure to cobalt. Some exposure levels associated with cardiomyopathy have been

reported following oral exposure, but the minimal exposure level required to produce this effect in humans is not known (Alexander 1969, 1972; Morin et al. 1971). Oral studies in animals reported dose levels associated with death, various systemic and neurological effects, and effects on reproduction and development (Domingo et al. 1984, 1985b; Krasovskii and Fridlyand 1971; Mohiuddin 1970; Mollenhauer et al. 1985; Pedigo et al. 1988). Intermediate-duration inhalation studies in animals reported that the respiratory tract is the target of the toxicity of inhaled cobalt (Bucher et al. 1990; Johansson et al. 1987; Kerfoot 1975; NTP 1991; Palmes et al. 1959). Animal studies were insufficient for derivation of an intermediate-duration MRL for oral exposure, since the reported effects were severe and the effects occurred at levels above those reported in the few human studies. Dermal data in animals were not located. Animal studies that investigate the possible toxic interaction between cobalt and alcohol may be helpful in understanding the role of cobalt in the cardiomyopathy reported in the heavy beer drinkers (Alexander 1969, 1972; Morin et al. 1971). One such study in guinea pigs already exists (Mohiuddin et al. 1970), but this study used a single, high dose of cobalt. Studies using a series of lower doses, both with and without alcohol preexposure, would be helpful in determining the threshold for the cardiac effects. Intermediate-duration dermal studies would enable determination of hazardous levels for this route of exposure. Intermediate-duration toxicity information is important because people living near hazardous waste sites might be exposed for corresponding time periods.

Radioactive Cobalt. Data on health effects following acute exposure to radioactive cobalt by the inhalation, oral, or dermal routes are lacking. Because cobalt radioisotopes are man-made (see Chapter 5), only low-level exposure to radioactive cobalt in the environment by these routes is likely to occur. Substantial human data exist concerning intermediate-duration exposure to external radiation, as radiotherapy treatment regimens fall into this duration category. Animal data from intermediate-duration external exposure also exist, but are less numerous. Additional intermediate-duration studies are not likely to provide substantial additions to our knowledge of radiation-induced toxic effects.

Chronic-Duration Exposure and Cancer.

Stable Cobalt. Chronic inhalation exposure levels in humans associated with respiratory effects have been reported (Gennart and Lauwerys 1990; Nemery et al. 1992; Shirakawa et al. 1988; Sprince et al. 1988). In humans, the respiratory system is the primary target following chronic inhalation exposure. A chronic-duration inhalation MRL was derived from a NOAEL for decreased ventilatory function in exposed workers (Nemery et al. 1992). Wehner et al. (1977) reported no adverse effects in hamsters

exposed chronically to cobalt oxide. NTP (1998; Bucher et al. 1999) exposed rats and mice to cobalt sulfate for 2 years, reporting pronounced effects on the respiratory tract, including hyperplasia, inflammation, fibrosis, and metaplasia; an increased incidence of cancer was also reported. Chronic oral or dermal studies have not been reported in either humans or animals. Animal studies that identify minimally effective chronic oral exposure levels would be useful for estimating a chronic MRL. Chronic dermal studies would enable determination of hazardous levels for this route of exposure. Chronic toxicity information is important because people living near hazardous waste sites might be exposed to cobalt for many years.

Several studies of hard metal exposure in humans have reported increases in lung cancer mortality from occupational inhalation exposure to hard metal (Lasfargues et al. 1994; Moulin et al. 1998; Wild et al. 2000). In humans, cancer has not been reported following exposure to cobalt by the oral or dermal routes. An increased incidence of alveolar/bronchiolar neoplasms was noted following lifetime exposure of male rats to 1.14 mg cobalt/m^3 and in female rats to 0.38 mg cobalt/m^3 as cobalt sulfate, with tumors occurring in both sexes with significantly positive trends (Bucher et al. 1999; NTP 1998). Similarly, mice of both sexes exposed to 1.14 mg cobalt/m^3 showed an increase in alveolar/bronchiolar neoplasms, again with lung tumors occurring with significantly positive trends. Parenteral exposure to cobalt has been found to induce tumors (Gilman 1962; Gilman and Ruckerbauer 1962; Heath 1956, 1969; Heath and Daniel 1962; Shabaan et al. 1977). Further chronic exposure studies by the oral and dermal routes may determine the actual carcinogenic potential of cobalt. Also, studies examining the effect of cobalt speciation (i.e., cobalt metal vs. cobalt sulfate) would add to our understanding of the carcinogenic potential of cobalt.

Radioactive Cobalt. Data on health effects following chronic exposure to radioactive cobalt by the inhalation, oral, or dermal routes are lacking. Because cobalt radioisotopes are man-made (see Chapter 5), only low-level exposure to radioactive cobalt in the environment by these routes is likely to occur. Limited data exist on chronic exposure to cobalt radiation in humans, with genotoxicity, immunologic effects, and cancer being the primary end points examined. Animal data are similarly limited. Additional human or animal data following chronic exposure to external cobalt radiation would be useful in further identifying possible long-term health effects or susceptible populations. Agency for Toxic Substances and Disease Registry (1999) has derived a chronic-duration MRL for external radiation exposure, which is applicable to external exposures to cobalt radiation, so additional data for the derivation of an MRL are not needed.

Genotoxicity.

Stable Cobalt. Gennart et al. (1993) reported an increase in sister-chromatid exchanges in workers exposed to a mixture of cobalt, chromium, nickel, and iron. De Boeck et al. (2000) reported no significant change in the comet assay on lymphocytes from nonsmoking workers who were occupationally exposed to cobalt or hard metal dusts; a positive association was found between hard metal exposure and increased micronucleus formation in smokers only.

Data regarding the mutagenic action of cobalt in bacterial cell lines and mammalian cell lines have been reported in the literature (Hamilton-Koch et al. 1986; Kharab and Singh 1985; Ogawa et al. 1986). *In vivo* mutagenicity studies in animals following inhalation, oral, or dermal exposure to cobalt would be helpful in ascertaining its true mutagenic potential. Further studies examining the differences in genotoxicity between different valence states of cobalt would also be useful.

Radioactive Cobalt. Data on genotoxic effects following exposure to radioactive cobalt by the inhalation, oral, or dermal routes are lacking. Because cobalt radioisotopes are man-made (see Chapter 5), only low-level exposure to radioactive cobalt in the environment by these routes is likely to occur. Several studies have demonstrated genotoxic effects in humans exposed to external cobalt radiation (Chang et al.1999c; House et al. 1992; Rauscher and Bauchinger 1983). Numerous data from animal studies exist demonstrating the genotoxic effects of ionizing radiation, including cobalt radiation.

Reproductive Toxicity.

Stable Cobalt. No studies were located regarding the reproductive effects of cobalt in humans following exposure by any route. Inhalation and oral studies in male animals have demonstrated adverse effects on reproductive organs (Anderson et al. 1992, 1993; Bucher et al. 1990; Corrier et al. 1985; Domingo et al. 1985b; Mollenhauer et al. 1985; NTP 1991; Pedigo et al. 1988). One study also reported effects on the estrous cycle in mice following inhalation exposure (Bucher et al. 1990; NTP 1991). Multigenerational studies would be helpful in assessing the significance of these effects on reproductive performance.

Radioactive Cobalt. Data on reproductive effects following exposure to radioactive cobalt by the inhalation, oral, or dermal routes are lacking. Because cobalt radioisotopes are man-made (see Chapter 5), only low-level exposure to radioactive cobalt in the environment by these routes is likely to occur. Human data on reproductive effects following external exposure to cobalt radiation are lacking,

but are sufficiently understood for gamma radiation. Available animal studies are limited, but have demonstrated radiation-induced deficits on reproductive ability in both genders (Cunningham and Huckins 1978; Laporte et al. 1985; Searl et al. 1976, 1980). Additional data in humans and animals would be helpful in refining minimal effective doses for radiation effects on reproduction.

Developmental Toxicity.

Stable Cobalt. No developmental effects were observed in the children of 78 women given cobalt chloride orally during pregnancy for treatment of anemia (Holly 1955); however, only a limited examination of offspring was reported, and details of examined end points were not reported. No studies of developmental effects by other routes of exposure in humans were located. Developmental effects in animals following oral exposure during gestation, however, have been observed (Domingo et al. 1985b). Further developmental studies in animals by all relevant routes of exposure (inhalation, oral, dermal) may clarify the potential developmental effects of cobalt in humans.

Radioactive Cobalt. Data on developmental effects following exposure to radioactive cobalt by the inhalation, oral, or dermal routes are lacking. Because cobalt radioisotopes are man-made (see Chapter 5), only low-level exposure to radioactive cobalt in the environment by these routes is likely to occur. No human studies describing developmental effects of exposure to external cobalt radiation were located. Extensive data from animal studies have shown that even acute exposures to small amounts of cobalt radiation may elicit profound effects on the developing organism (see Section 3.2.4.6). The effects of ionizing radiation on the developing organism are also described in the Agency for Toxic Substances and Disease Registry Toxicological Profile for Ionizing Radiation (1999).

Immunotoxicity.

Stable Cobalt. Humans have been shown to develop sensitivity to cobalt following occupational exposure (Bencko et al. 1983; Shirakawa et al. 1988, 1989). No immunological effects were observed following oral exposure of humans to cobalt. Similar evidence of sensitization has been reported in animals (Lammintausta et al. 1985). Studies examining the mechanism of sensitization might be helpful in fully understanding and treating this effect in humans. A battery of immune function tests would further assess the immunotoxicity of cobalt in humans and animals.

Radioactive Cobalt. Data on immunotoxic effects following exposure to radioactive cobalt by the inhalation, oral, or dermal routes are lacking. Because cobalt radioisotopes are man-made (see Chapter 5), only low-level exposure to radioactive cobalt in the environment by these routes is likely to occur. Following external exposure to cobalt radiation, above levels normally encountered except for medical procedures, decreases in white blood cell counts have been seen in both humans and animals. Further studies on the immunotoxic effects of external cobalt radiation would be useful in refining the minimum effective dose.

Neurotoxicity.

Stable Cobalt. No studies were located regarding neurotoxic effects of cobalt in humans following oral or dermal exposure. Two occupational inhalation exposure studies have reported memory deficits, optic atrophy, or nerve deafness in humans exposed to cobalt (Jordan et al. 1990; Meecham and Humphrey 1991). In animals, alterations in several neurologic parameters were found following oral exposure (Bourg et al. 1985; Krasovskii and Fridlyand 1971; Mutafova-Yambolieva et al. 1994; Nation et al. 1983; Singh and Junnarkar 1991; Vassilev et al. 1993; Wellman et al. 1984). Additional studies in animals would assist in determining whether these neurological effects have any relevance to potential effects in humans.

Radioactive Cobalt. Data on neurotoxic effects following exposure to radioactive cobalt by the inhalation, oral, or dermal routes are lacking. Because cobalt radioisotopes are man-made (see Chapter 5), only low-level exposure to radioactive cobalt in the environment by these routes is likely to occur. Human data following cobalt radiotherapy have demonstrated effects believed to result from neurological damage, but data are limited, doses were extreme, and effects have not been well-characterized. Several animal studies have shown neurobehavioral or neurophysiological changes following exposure to cobalt radiation (Bassant and Court 1978; Maier and Landauer 1989; Mele et al. 1988).

Epidemiological and Human Dosimetry Studies.

Stable Cobalt. Epidemiological studies relating to cobalt exposure are available in the literature. Studies of persons exposed to cobalt occupationally are available (Kusaka et al. 1986a, 1986b; Shirakawa et al. 1988, 1989; Sprince et al. 1988), dietetically (beer drinkers) (Alexander 1969, 1972; Morin et al. 1971), and medically (cobalt given to alleviate anemia) (Davis and Fields 1958; Holly 1955; Taylor et al. 1977).

Further studies assessing the cause/effect relationship between cobalt exposure and human health effects would be helpful in monitoring individuals living near a hazardous waste site to verify that documented exposure levels are not associated with adverse health effects.

Radioactive Cobalt. Epidemiological data on exposure to radioactive cobalt by the inhalation, oral, or dermal routes are lacking. Because cobalt radioisotopes are man-made (see Chapter 5), only low-level exposure to radioactive cobalt in the environment by these routes is likely to occur. Human external exposures to cobalt radiation have been documented in the literature. Radiotherapy exposures, though to extremely high radiation doses, are generally well-controlled and documented, whereas environmental and accidental workplace exposures are less frequent and less well-documented.

Biomarkers of Exposure and Effect.

Exposure.

Stable Cobalt. Information is available on the monitoring of cobalt exposure by the quantification of cobalt in urine and blood (Alexandersson 1988; Ichikawa et al. 1985; Scansetti et al. 1985). A portion of inhaled cobalt is rapidly excreted in the feces, and the amount retained in the body tends to be steadily excreted over time. Levels in body fluids, therefore, can be monitored up to several days after exposure. Many different methods for the detection of cobalt in body fluids have been reported (Section 7.1).

Radioactive Cobalt. No information is available regarding biomarkers specific for exposure to cobalt radionuclides by the inhalation, oral, dermal, or external exposure routes. Biomarkers for exposure to ionizing radiation are discussed in Agency for Toxic Substances and Disease Registry (1999). Personal dosimeters (film or luminescent) are an artificial surrogate to measure the amount of exposure to external beta or gamma radiation, though these are not specific for radiation from cobalt radionuclides.

Effect.

Stable Cobalt. Alterations in serum proteins and changes in serum antibodies have been found that are specific for cobalt exposure (Stokinger and Wagner 1958). These changes may be the earliest indication of the effects of cobalt exposure. Further studies may reveal other cobalt-specific biomarkers that, in

combination with these changes, may alert health professionals to cobalt exposure before serious toxicological effects occur.

Radioactive Cobalt. While in many cases radioactive cobalt itself can be measured following exposure, no information is available regarding biomarkers specific for effects of cobalt radionuclides following exposure by the inhalation, oral, dermal, or external exposure routes. Biomarkers for effects of ionizing radiation are discussed in Agency for Toxic Substances and Disease Registry (1999), and include changes in levels of formed elements of the blood as some of the most sensitive indicators. These biomarkers are believed to be suitable for monitoring exposure to cobalt radiation.

Absorption, Distribution, Metabolism, and Excretion. Pharmacokinetic data in humans indicate that cobalt is absorbed through the lungs (Foster et al. 1989) and the gastrointestinal tract (Harp and Scoular 1952; Sorbie et al. 1971; Valberg et al. 1969), that cobalt is well distributed in the body with the highest concentration being found in the lungs following inhalation (Gerhardsson et al. 1984; Hewitt 1988; Hillerdal and Hartung 1983; Teraoka 1981), and that some of the inhaled or ingested cobalt is rapidly excreted in the feces, with the amount retained in the body being excreted slowly, primarily in the urine (Foster et al. 1989; Paley et al. 1958; Smith et al. 1972). Pharmacokinetic studies in animals following inhalation and oral exposure have demonstrated similar responses (Andre et al. 1989; Bailey et al. 1989; Collier et al. 1989; Foster et al. 1989; Patrick et al. 1989; Talbot and Morgan 1989). Few data exist regarding the pharmacokinetics of cobalt following dermal exposure, though what data are available demonstrate that cobalt can be absorbed in small quantities through human (Scansetti et al. 1994) and animal (Inaba and Suzuki-Yasumoto 1979; Lacy et al. 1996) skin, with greater absorption occurring through damaged than intact skin.

Comparative Toxicokinetics. Several inhalation and oral studies have compared the toxicokinetics of cobalt in several different species of animals, including humans (Andre et al. 1989; Bailey et al. 1989; Collier et al. 1989; Foster et al. 1989; Patrick et al. 1989; Talbot and Morgan 1989). No comparative pharmacokinetic studies following dermal exposure were located. These studies would be useful because humans are exposed via the skin in the workplace and may potentially be exposed via this route at waste sites.

Methods for Reducing Toxic Effects.

Stable and Radioactive Cobalt. Chelation therapy is expected to apply equally well to stable and radioactive cobalt isotopes. EDTA or British anti-lewisite (BAL) has been shown to effectively mitigate the toxicity of cobalt in humans (Goldfrank et al. 1990; Haddad and Winchester 1990; Stutz and Janusz 1988). In animal studies examining the effectiveness of various chelators, n-acetyl cysteine (NAC) was shown to be the most effective (Llobet et al. 1988). It would be useful to determine the effective dose of NAC in humans. Studies examining the effectiveness of other chelating agents may be helpful in determining the most effective chelation therapy for humans.

Children's Susceptibility.

Stable Cobalt. Data comparing the susceptibility of children to cobalt compounds are limited. Animal studies have suggested that absorption following inhalation or oral exposure may be greater in very young animals, resulting in increased systemic dose. Data are not available on the differences between children and adults following dermal exposure. Further studies on the susceptibility of young animals relative to adult animals may be useful in determining whether children are at greater risk from exposure to cobalt in the environment than adults.

Radioactive Cobalt. No data are available on whether children are more susceptible to the effects of radioactive cobalt compounds than adults. Animal studies have shown that exposure *in utero* to even moderate amounts of cobalt radiation can cause dramatic effects in the developing organism. It would be expected that children would be more susceptible to the effects of external cobalt radiation, due to the greater percentage of rapidly-dividing cells during growth.

Child health data needs relating to exposure are discussed in 6.8.1 Identification of Data Needs: Exposures of Children.

3.13.3 Ongoing Studies

Relevant ongoing studies were not located for cobalt.

4. CHEMICAL, PHYSICAL, AND RADIOLOGICAL INFORMATION

4.1 CHEMICAL IDENTITY

Cobalt is a naturally-occurring element that appears in the first transition series of Group 9 (VIII) of the periodic table along with iron and nickel. There is only one stable isotope of cobalt, ^{59}Co. There are about 26 known radioactive isotopes of cobalt, of which only two are of commercial importance, ^{60}Co and ^{57}Co. ^{60}Co, a commonly-used source of gamma radiation, is the most important radionuclide. It may be a low-level contaminant of cooling water released by nuclear reactors. Table 4-1 summarizes information on the chemical identity of elemental cobalt and some common cobalt compounds.

4.2 PHYSICAL, CHEMICAL, AND RADIOLOGICAL PROPERTIES

Cobalt commonly occurs in the 0, +2, and +3 valence states. Compounds containing cobalt in the -1, +1, +4, and +5 oxidation state are few and uncommon (Cotton and Wilkinson 1980). Cobalt (II) is much more stable than Co(III), and Co^{3+} is a sufficiently powerful oxidizing agent to oxidize water, liberating oxygen. Table 4-2 summarizes important physical and chemical properties of elemental cobalt and some common cobalt compounds. These properties are similar to those of its neighbors in Group 9 of the periodic table, iron and nickel. Metallic cobalt, Co(0), occurs as two allotropic forms, hexagonal and cubic; the hexagonal form is stable at room temperature. A biochemically important cobalt compound is vitamin B_{12}, or cyanocobalamin, in which cobalt is complexed with four pyrrole nuclei joined in a ring called the corrinoid ligand system (similar to porphyrin).

The Chemical Abstract Service (CAS) registry numbers, decay modes, half-lives, and specific activity of the three principal radioactive cobalt isotopes, ^{57}Co, ^{58}Co, and ^{60}Co, are presented in Table 4-3. ^{60}Co (half-life of 5.27 years) decays by beta decay to nickel-60, a stable isotope (ICRP 1983; Lide 1998).

$$^{60}_{27}\mathrm{Co} \rightarrow ^{60}_{28}\mathrm{Ni} + ^{0}_{-1}e + \gamma$$

Table 4-1. Chemical Identity of Cobalt and Selected Compounds

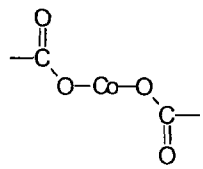
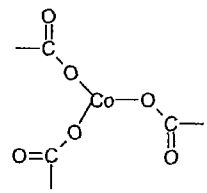
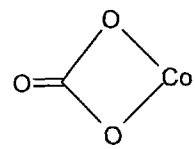
Characteristic	Cobalt	Cobalt(II) acetate	Cobalt(III) acetate	Cobalt(II) carbonate
Synonym(s)	Cobalt-59, cobalt metal	Cobaltous acetate, cobalt diacetate	Cobaltic acetate, cobalt triacetate	Cobaltous carbonate; carbonic acid; cobalt (+2) salt
Registered trade name(s)	No data	No data	No data	No data
Chemical formula	Co	$Co(C_2H_4O_2)_2$	$Co(C_2H_4O_2)_3$	$CoCO_3$
Chemical structure	Co			
Identification numbers:				
CAS registry	7440-48-4	71-48-7	917-69-1	513-79-10
NIOSH RTECS	GF8750000	AG3150000	No data	FF9450050
EPA hazardous waste	No data	No data	No data	No data
OHM/TADS	No data	No data	No data	No data
DOT/UN/NA/IMCO shipping[a]	UN1318	No data	No data	No data
HSDB	519	997	No data	No data
NCI	C60311	No data	No data	No data

Table 4-1. Chemical Identity of Cobalt and Selected Compounds

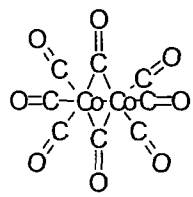
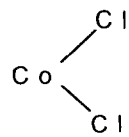
Characteristic	Cobalt carbonyl	Cobalt(II) chloride	Cobalt(II) hydroxide	Cobalt(II) meso-porphyrin
Synonym(s)	Dicobalt octa-carbonyl; cobalt tetracarbonyl	Cobalt dichloride; cobaltous chloride	Cobaltous hydr-oxide; cobalt dihydroxide	Cobalt meso-porphyrin IX Cobalt-iprotoporphyrin
Registered trade name(s)	No data	No data	No data	No data
Chemical formula	$Co_2(CO)_8$	$CoCl_2$	$Co(OH)_2$	$C_{34}H_{34}CoN_4O_4$
Chemical structure			HO—Co—OH	No data
Identification numbers:				
CAS registry	10210-68-1	7646-79-9	21041-93-0	21158-51-0
NIOSH RTECS	GG0300000	GF9800000	No data	No data
EPA hazardous waste	No data	No data	No data	No data
OHM/TADS	No data	7217328	No data	No data
DOT/UN/NA/IMCO shipping	No data	No data	No data	No data
HSDB	6345	1000	No data	No data
NCI	No data	No data	No data	No data

Table 4-1. Chemical Identity of Cobalt and Selected Compounds

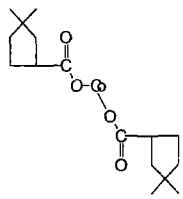
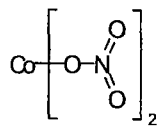
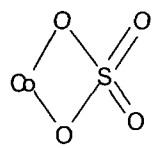
Characteristic	Cobalt(II) naphthenate	Cobalt(II) nitrate	Cobalt(II) oxide	Cobalt(III) oxide
Synonym(s)	Naftolite; naphthenic acid, cobalt salt	Cobaltous nitrate	Black 13; C.I. 77322; cobalt monoxide; cobaltous oxide	Cobalt black; cobaltic oxide; cobalt sesquioxide; cobalt trioxide; C.I. 77323
Registered trade name(s)	No data	No data	C.I. Pigment Black 13; Zaffre	No data
Chemical formula	$Co(C_{11}H_{10}O_2)_2$	$Co(No_3)_2{\bullet}6H_20$	CoO	Co_2O_3
Chemical structure			Co═O	O═Co—O—Co═O
Identification numbers:				
CAS registry	10210-68-1	7646-79-9	21041-93-0	21158-51-0
NIOSH RTECS	GG0300000	GF9800000	No data	No data
EPA hazardous waste	No data	No data	No data	No data
OHM/TADS	No data	7217328	No data	No data
DOT/UN/NA/IMCO shipping	No data	No data	No data	No data
HSDB	6345	1000	No data	No data
NCI	No data	No data	No data	No data

Table 4-1. Chemical Identity of Cobalt and Selected Compounds

Characteristic	Cobalt(II, III) oxide	Cobalt(II) sulfate
Synonym(s)	Cobaltic-cobaltous oxide; cobalt tetra-oxide, tricobalt tetraoxide, cobaltosic oxide; cobalt black; C.I. Pigment Black 13	Cobalt sulfate; cobaltous sulfate
Registered trade name(s)	No data	No data
Chemical formula	Co_3O_4	$CoSO_4$
Chemical structure	Co=OO=Co–O–Co=O	
Identification numbers:		
CAS registry	1308-06-1	10124-43-3
NIOSH RTECS	No data	GG3100000
EPA hazardous waste	No data	No data
OHM/TADS	No data	7217330
DOT/UN/NA/IMCO shipping	No data	No data
HSDB	No data	240
NCI	No data	No data

[a]The identification number for radioactive materials is UN2910

CAS = Chemical Abstract Service; DOT/UN/NA/IMCO = Department of Transportation/United Nations/North America/International Maritime Dangerous Goods Code; EPA = Environmental Protection Agency; HSDB = Hazardous Substances Data Bank; NCI = National Cancer Institute; NIOSH = National Institute for Occupational Safety and Health; OHM/TADS = Oil and Hazardous Materials/Technical Assistance Data System; RTECS = Registry of Toxic Effects of Chemical Substances

Source: Budavari 1996; HSDB 2001; RTECS 1987

Table 4-2. Physical and Chemical Properties of Cobalt and Selected Compounds

Property	Cobalt	Cobalt(II) acetate	Cobalt(III) acetate	Cobalt(II) carbonate
Molecular weight	58.93	177.03	236.07	118.94
Color	Silvery gray	Light pink	Dark green	Red
Physical state	Solid	Solid	Solid	Solid
Melting point, °C	1,495	No data	Decomposes at 100 °C	Decomposes
Boiling point, °C	2,870	No data	Not relevant	Not relevant
Density, g/cm^3	8.9 (20 °C)	No data	No data	4.13
Odor	No data	No data	No data	No data
Odor threshold:				
Water	No data	No data	No data	No data
Air	No data	No data	No data	No data
Solubility:				
Water	Insoluble	Soluble	Soluble	0.18 g/100 g H_2O at 15 °C
Organic solvent(s)	Insoluble	2.1 g/100 g methanol at 15 °C	soluble in alcohol, acetic acid	Insoluble in ethanol
Partition coefficients:				
Log K_{ow}	No data	No data	No data	No data
Log K_{oc}	No data	No data	No data	No data
Vapor pressure	1 mmHg at 1,910 °C	No data	No data	No data
Henry's law constant	No data	No data	No data	No data
Autoignition temperature	760 °C for dust cloud	No data	No data	No data
Flashpoint	No data	No data	No data	No data
Flammability limits	No data	No data	No data	No data
Conversion factors	Not relevant[a]	Not relevant[a]	Not relevant[a]	Not relevant[a]
Explosive limits	No data	No data	No data	No data

Table 4-2. Physical and Chemical Properties of Cobalt and Selected Compounds

Property	Cobalt carbonyl	Cobalt(II) chloride	Cobalt(II) hydroxide	Cobalt(II) mesoporphyrin
Molecular weight	341.9	129.84	92.95	621.2[b]
Color	Orange (white when pure)	Blue	Rose red or blue green	No data
Physical state	Solid	Solid	Solid	No data
Melting point, °C	51	724	No data	No data
Boiling point, °C	Decomposes	1,049	No data	No data
Density, g/cm^3	1.73 at 18 °C	3.356 (36 °C)	3.597 at 15 °C	No data
Odor	No data	No data	No data	No data
Odor threshold:				
Water	No data	No data	No data	No data
Air	No data	No data	No data	No data
Solubility:				
Water	Insoluble	450 g/L at 7 °C	0.0032 g/L	No data
Organic solvent(s)	Soluble in ether; insoluble in naphtha	544 g/L in ethanol; 86 g/L in acetone	No data	No data
Partition coefficients:				
Log K_{ow}	No data	No data	No data	No data
Log K_{oc}	No data	No data	No data	No data
Vapor pressure	199.5 at 25 °C	No data	No data	No data
Henry's law constant	No data	No data	No data	No data
Autoignition temperature, °C	No data	No data	No data	No data
Flashpoint, °C	No data	No data	No data	No data
Flammability limits	No data	No data	No data	No data
Conversion factors	Not relevant[a]	Not relevant[a]	Not relevant[a]	Not relevant[a]
Explosive limits	No data	No data	No data	No data

Table 4-2. Physical and Chemical Properties of Cobalt and Selected Compounds

Property	Cobalt(II) naphthenate	Cobalt(II) nitrate	Cobalt(II) oxide	Cobalt(III) oxide
Molecular weight	407	182.94	74.93	165.86
Color	No data	Red	Pink	Black-gray
Physical state	Solid	Solid	Solid	Solid
Melting point, °C	140	Decomposes at 100–105[b]	1,795	895 (decomposes)
Boiling point, °C	No data	Not relevant	No data	Not relevant
Density g/cm^3	0.9	2.49[b]	6.45	5.18
Odor	No data	No data	No data	No data
Odor threshold:				
Water	No data	No data	No data	No data
Air	No data	No data	No data	No data
Solubility:				
Water	Insoluble	133.8 at 0 °C[c]	Insoluble	Insoluble
Organic solvent(s)	No data	Soluble in ethanol, acetone	Insoluble in alcohol	Insoluble in ethanol
Partition coefficients:				
Log K_{ow}	No data	No data	No data	No data
Log K_{ow}	No data	No data	No data	No data
Vapor pressure	No data	No data	No data	No data
Henry's law constant	No data	No data	No data	No data
Autoignition temperature	No data	No data	No data	No data
Flashpoint	No data	No data	No data	No data
Flammability limits	No data	No data	No data	No data
Conversion factors	Not relevant[a]	Not relevant[a]	Not relevant[a]	Not relevant[a]
Explosive limits	No data	No data	No data	No data

Table 4-2. Physical and Chemical Properties of Cobalt and Selected Compounds

Property	Cobalt(II, III) oxide	Cobalt(II) sulfate
Molecular weight	250.80	154.99
Color	Black	Dark blue
Physical state	Solid	Solid
Melting point, °C	-O_2 at 900–950	Decomposes at 735 °C
Boiling point, °C	Not relevant	Not relevant
Density g/cm^3	6.07	3.71
Odor	No data	No data
Odor threshold:		
Water	No data	No data
Air	No data	No data
Solubility:		
Water	Insoluble	3.83 g/100 mL H_2O at 25 °C
Organic solvent(s)	No data	1.04 g/100 mL methanol at 18 °C
Partition coefficients:		
Log K_{ow}	No data	No data
Log K_{ow}	No data	No data
Vapor pressure	No data	No data
Henry's law constant	No data	No data
Autoignition temperature	No data	No data
Flashpoint	No data	No data
Flammability limits	No data	No data
Conversion factors	Not relevant[a]	Not relevant[a]
Explosive limits	No data	No data

[a]Substances exist in the atmosphere in the particulate state, and the concentration is expressed in weight per cubic meter
[b]CAS Online
[c]Hexahydrate

Source: Budavari 1996; HSDB 2001, 2004; Lide 1994; Stockinger 1981; Weast 1985

Table 4-3. Principal Radioactive Cobalt Isotopes

				Beta radiation		Gamma radiation		
Isotope	CAS registry no.	Decay mode (product)	Decay mode energy (MeV)	Energy (MeV)	Intensity (percent)	Energy (MeV)	Intensity (percent)	Half-life
^{55}Co	13982-25-7	E.C. β^+ (^{55}Fe)	3.452	1.498	46	0.9312	75	17.53 hours
				1.021	25.6	0.4772	20	
				2.043	10.7	1.408	16.88	
^{57}Co	13981-50-5	E.C. (^{57}Fe)	0.836	0.700	99.8	0.1221	85.6	271.8 days
						0.1365	10.7	
						0.014	9.2	
^{58}Co	13981-38-9	E.C. β^+ (^{58}Fe)	2.30	1.4966	83.9	0.811	99	70.86 days
				0.4746	14.9			
^{60}Co	10198-40-0	β^- (^{60}Ni)	2.824	0.3181	99.9	1.173	100	5.271 years
						1.332	100	

B^- = negative beta emission; β^+ = positron emission; E.C. = orbital electron capture

Source: ICRP 1983; LBNL 2000; Lide 1998

The decay is accompanied by the emission of 1.173 and 1.332 MeV gamma rays. ^{57}Co (half-life of 271.8 days) and ^{58}Co (half-life of 70.9 days) decay by electron capture and electron capture/position (β+) emission to ^{57}Fe and ^{58}Fe, respectively. These decay processes are also accompanied by gamma emissions (Table 4-3).

5. PRODUCTION, IMPORT/EXPORT, USE, AND DISPOSAL

5.1 PRODUCTION

Cobalt is the 33rd most abundant element, comprising approximately 0.0025% of the weight of the earth's crust. It is often found in association with nickel, silver, lead, copper, and iron ores and occurs in mineral form as arsenides, sulfides, and oxides. The most import cobalt minerals are: linnaeite, Co_3S_4; carrolite, $CuCo_2S_4$; safflorite, $CoAs_2$; skutterudite, $CoAs_3$; erythrite, $Co_3(AsO_4)_2 \cdot 8H_2O$; and glaucodot, CoAsS (Hodge 1993; IARC 1991; Merian 1985; Smith and Carson 1981). The largest cobalt reserves are in the Congo (Kinshasa), Cuba, Australia, New Caledonia, United States, and Zambia. Most of the U.S. cobalt deposits are in Minnesota, but other important deposits are in Alaska, California, Idaho, Missouri, Montana, and Oregon. Cobalt production from these deposits, with the exception of Idaho and Missouri, would be as a byproduct of another metal (USGS 2004). Cobalt is also found in meteorites and deep sea nodules.

The production of pure metal from these ores depends on the nature of the ore. Sulfide ores are first finely ground (i.e., milled) and the sulfides are separated by a floatation process with the aid of frothers (i.e., C_5–C_8 alcohols, glycols, or polyethylene or polypropylene glycol ethers). The concentrated product is subjected to heating in air (roasting) to form oxides or sulfates from the sulfide, which are more easily reduced. The resulting matte is leached with water and the cobalt sulfate leachate is precipitated as its hydroxide by the addition of lime. The hydroxide is dissolved in sulfuric acid, and the resulting cobalt sulfate is electrolyzed to yield metallic cobalt. For the cobalt-rich mineral cobaltite, a leaching process with either ammonia or acid under pressure and elevated temperatures has been used to extract cobalt. The solution is purified to remove iron and is subsequently reduced by hydrogen in the presence of a catalyst under elevated temperature and pressure to obtain fine cobalt powder (Duby 1995; Nagaraj 1995; Planinsek and Newkirk 1979).

Except for a negligible amount of byproduct cobalt produced from some mining operations, no cobalt is presently mined or refined in the United States. In addition to byproduct production, U.S. production is derived from scrap (secondary production). In 2003, an estimated 2,200 metric tons of cobalt were recycled from scrap (USGS 2004). Since 1993, production has been supplemented by sales of excess cobalt from the National Defense Stockpile (NDS), which the government maintains for military,

industrial, and essential civilian use during national emergencies. In fiscal year 2002, 2,720 metric tons of cobalt were released from the NDS. In 2001, the United States did not mine or refine cobalt, with the exception of small amounts of byproduct cobalt produced from mining operations in Missouri and Montana. The 2002 U.S. consumption of cobalt metal, organic and inorganic cobalt compounds, and purchased scrap (in terms of cobalt content) was 3,870, 1,270, and 2,800 metric tons, respectively (USGS 2002).

Current U.S. manufacturers of selected cobalt compounds are given in Table 5-1. Table 5-2 lists facilities in each state that manufacture, process, or use cobalt or cobalt compounds, the intended use, and the range of maximum amounts of these substances that are stored on site. In 2000, there were 618 reporting facilities that produced, processed, or used cobalt or cobalt compounds in the United States. The data listed in Table 5-2 are derived from the Toxics Chemicals Release Inventory (TRI) (TRI01 2004). Only certain types of facilities were required to report. Therefore, this is not an exhaustive list.

^{60}Co is produced by irradiating stable cobalt, ^{59}Co, with thermal neutrons in a nuclear reactor: ^{59}Co(n,γ)^{60}Co. The neutron flux employed is 10^{12}–10^{15} *n*/cm^2-sec and the conversion is 99%. The maximum specific activity obtained is 3.7×10^{13} Bq/g (1,000 Ci/g). Commercial ^{60}Co sources used for bacterial sterilization are made into rods with double metal shielding. The individual sources have an activity of about 2×10^{14}–6×10^{14} Bq (6–15 kCi). The annual output of ^{60}Co was about 2×10^{18}–3×10^{18} Bq (50–80 MCi) in the early 1990s. In 1991, there were 170 gamma irradiation systems operating in 45 countries having a total activity of about 6×10^{18} Bq (160 MCi) (Zyball 1993). Producers of ^{60}Co include MDS Nordion in Canada, AEA Technology (formerly Amersham QSA) in the United Kingdom, and Neutron Products in Dickerson, Maryland.

^{58}Co is not produced commercially. It can be produced by irradiating ^{58}Ni, a stable isotope, with neutrons, followed by positron decay: ^{58}Ni(n,γ)^{58}Co. It can be produced in a nuclear reactor or a cyclotron. Both ^{60}Co and ^{58}Co may be produced unintentionally in reactors. These are the dominant sources of residual radiation in the primary circuit outside the reactor core of nuclear plants and are formed by neutron absorption of ^{59}Co and ^{58}Ni, both stable isotopes commonly used in plant construction materials (Taylor 1996). ^{60}Co is commonly found as one of the radionuclides present in the low-level radioactive waste discharges from many nuclear power plants; however, amounts rarely make a significant contribution to the radiation exposure of the public (Leonard et al. 1993a). The geometric

Table 5-1. Current U.S. Manufacturers of Cobalt Metal and Selected Cobalt Compounds[a]

Company	Location
Cobalt metal[b]:	
Kennametal, Inc.	Latrobe, Pennsylvania
OM Group, Inc.	Cleveland, Ohio
Cobalt (II) acetate:	
The IMC Group	Shelby, North Carolina
McGean-Rohco, Inc., McGean Specialty Chemicals Division	Cleveland, Ohio
OM Group, Inc.	Franklin, Pennsylvania
The Shepard Chemical Company	Cincinnati, Ohio
Cobalt (II) carbonate:	
The IMC Group	Shelby, North Carolina
McGean-Rohco, Inc., McGean Specialty Chemicals Division	Cleveland, Ohio
OMG Apex	St. George, Utah
OM Group, Inc.	Franklin, Pennsylvania
The Shepherd Chemical Co.	Cincinnati, Ohio
Cobalt (II) chloride:	
The IMC Group	Shelby, North Carolina
Johson Matthey, Inc., Alfa Aesar	Ward Hill, Massachusetts
McGean-Rohco, Inc., McGean Specialty Chemicals Division	Cleveland, Ohio
OM Group. Inc.	Franklin, Pennsylvania
The Shepard Chemical Company	Cincinnati, Ohio
Cobalt (II) hydroxide:	
McGean-Rohco, Inc., McGean Specialty Chemicals Division	Cleveland, Ohio
OM Group, Inc.	Franklin, Pennsylvania
The Shepard Chemical Company	Cincinnati, Ohio
Cobalt (II) nitrate:	
The IMC Group	Shelby, North Carolina
Johnson Matthey, Inc., Alfa Aesar	Ward Hill, Massachusetts
McGean-Rohco, Inc., McGean Specialty Chemicals Division	Cleveland, Ohio
OMG Apex	St. George, Utah
OM Group, Inc.	Franklin, Pennsylvania
The Shepard Chemical Company	Cincinnati, Ohio
Umicore USA, Inc., Cobalt Products	Laurinburg, North Carolina
Cobalt (II) oxide:	
OMG Apex	St. George, Utah
The Shepard Chemical Company	Cincinnati, Ohio

Table 5-1. Current U.S. Manufacturers of Cobalt Metal and Selected Cobalt Compounds[a]

Cobalt (III) oxide:	
Johnson Matthey, Inc., Alfa Aesar	Ward Hill, Massachusetts
Mallinckrodt Baker, Inc.	Phillipsburg, New Jersey
McGean-Rohco, Inc., McGean Specialty Chemicals Division	Cleveland, Ohio
OM Group, Inc.	Franklin, Pennsylvania
Osram Sylvania Inc.	Towanda, Pennsylvania
Cobalt (II) sulfate:	
The IMC Group	Shelby, North Carolina
McGean-Rohco, Inc., McGean Specialty Chemicals Division	Cleveland, Ohio
OMG Apex	St. George, Utah
OM Group, Inc.	Franklin, Pennsylvania
The Shepard Chemical Company	Cincinnati, Ohio

[a]Derived from Stanford Research Institute (SRI) 2003, except where otherwise noted. SRI reports production of chemicals produced in commercial quantities (defined as exceeding 5,000 pounds or $10,000 in value annually) by the companies listed

[b]U.S. members of The Cobalt Development Institute that are listed as producers of cobalt powder or hard metal products

Table 5-2. Facilities that Produce, Process, or Use Cobalt and Cobalt Compounds

State[a]	Number of facilities	Minimum amount on site in pounds[b]	Maximum amount on site in pounds[b]	Activities and uses[c]
AK	2	10,000	999,999	1, 5, 7, 12
AL	22	100	999,999	1, 2, 3, 4, 5, 6, 7, 8,9, 10, 12, 13
AR	9	100	99,999	1, 5, 7, 8,9
AZ	12	1,000	49,999,999	1, 2, 3, 5, 7, 8,9, 10, 12, 13, 14
CA	28	0	9,999,999	1, 2, 3, 5, 6, 7, 8,9, 10, 11, 12
CO	1	10,000	99,999	12
CT	9	0	999,999	2, 3, 7, 8
DE	2	1,000	9,999	1, 5,9, 13
FL	11	0	99,999	1, 2, 3, 4, 5, 6, 7, 8,9, 12, 13, 14
GA	17	100	999,999	1, 2, 3, 4, 5, 6, 7, 8,9, 11, 12, 13
IA	6	100	99,999	3, 4, 7, 8, 12
ID	2	100,000	999,999	1, 3, 5, 12
IL	24	100	9,999,999	1, 2, 3, 4, 5, 6, 7, 8,9, 10, 11, 12, 13, 14
IN	42	100	999,999	1, 2, 3, 4, 5, 6, 7, 8,9, 10, 11, 12, 13, 14
KS	5	10,000	99,999	1, 3, 5, 6, 7, 8,9, 10, 11, 12, 13
KY	22	100	999,999	1, 2, 3, 4, 5, 6, 7, 8,9, 12, 13, 14
LA	15	1,000	999,999	1, 2, 3, 4, 5, 6, 8, 10, 12, 13, 14
MA	11	100	99,999	1, 5, 8,9, 12
MD	6	1,000	99,999	1, 2, 3, 4, 5, 6, 7,9, 13
ME	2	100	99,999	1, 5, 8
MI	24	0	999,999	1, 2, 3, 4, 5, 7, 8,9, 11, 12, 13, 14
MN	6	100	99,999	1, 2, 5, 7, 8,9, 10, 12, 13, 14
MO	5	1,000	999,999	1, 2, 3, 4, 5, 6, 8,9, 12, 13, 14
MS	7	100	99,999	1, 5, 6, 7, 8, 10
MT	1	10,000	99,999	1, 5, 12, 14
NC	24	0	9,999,999	1, 2, 3, 4, 5, 6, 7, 8,9, 10, 12, 13, 14
ND	4	1,000	99,999	1, 5, 7, 12, 13, 14
NE	1	1,000	9,999	8, 11
NH	1	100	999	8
NJ	14	1,000	9,999,999	1, 3, 4, 5, 6, 7, 8, 10, 12, 14
NM	6	1,000	9,999,999	1, 3, 4, 5, 7, 8,9, 11, 12, 13
NV	8	1,000	10,000,000,000	1, 5, 6, 8, 12, 13, 14
NY	12	1,000	99,999	1, 2, 3, 4, 5, 7, 8,9, 11, 12
OH	44	0	9,999,999	1, 2, 3, 4, 5, 6, 7, 8,9, 11, 12, 13, 14
OK	14	100	999,999	1, 2, 3, 5, 6, 7, 8,9, 10, 11, 12
OR	6	1,000	99,999	1, 5, 7, 8, 12
PA	44	100	9,999,999	1, 2, 3, 4, 5, 6, 7, 8,9, 10, 11, 12, 13, 14

Table 5-2. Facilities that Produce, Process, or Use Cobalt and Cobalt Compounds

State[a]	Number of facilities	Minimum amount on site in pounds[b]	Maximum amount on site in pounds[b]	Activities and uses[c]
PR	2	1,000	99,999	8,9
RI	1	100,000	999,999	8
SC	26	100	9,999,999	1, 2, 3, 4, 5, 6, 7, 8,9, 10, 11, 12, 13
SD	1	10,000	99,999	7
TN	18	0	999,999	1, 2, 3, 4, 5, 6, 7, 8,9, 10, 12, 13
TX	45	0	9,999,999	1, 2, 3, 4, 5, 6, 7, 8,9, 10, 11, 12, 13, 14
UT	6	1,000	9,999,999	1, 3, 4, 5, 7, 8,9, 12, 13
VA	9	10,000	999,999	1, 2, 3, 4, 5, 6, 7, 8
VI	1	10,000	99,999	10
WA	2	10,000	99,999	1, 3, 4, 5,9, 10, 11, 12, 13
WI	20	100	999,999	1, 3, 4, 5, 7, 8,9, 11, 12, 13, 14
WV	13	100	999,999	1, 2, 3, 4, 5, 7, 8,9, 11, 12, 13, 14
WY	2	0	99,999	1, 5,9, 12, 13

Source: TRI01 2004

[a]Post office state abbreviations used
[b]Amounts on site reported by facilities in each state
[c]Activities/Uses:

1. Produce
2. Import
3. Onsite use/processing
4. Sale/Distribution
5. Byproduct
6. Impurity
7. Reactant
8. Formulation Component
9. Article Component
10. Repackaging
11. Chemical Processing Aid
12. Manufacturing Aid
13. Ancillary/Other Uses
14. Process Impurity

mean release of ^{60}Co in liquid effluents of light-water nuclear power stations was reported as in the early 1970s as 0.0805 Ci/year (3.0 GBq) (Morgan 1976).

The ^{60}Co activities for a representative pressurized-water reactor (PWR) and boiling water reactor (BWR) fuel assemblies are 1,100 and 170 Ci (41 and 6.3 TBq), respectively. There are 78 PWR and 40 BWR reactors in the United States, several of which have ceased operation. The postirradiation cobalt content of typical PWR and BWR reactor fuel assemblies are 38 g (0.01%) and 26 g (0.01%), respectively (DOE 2002).

^{55}Co may be produced by applying 12 MeV indirect deuteron energy to ^{54}Fe ($^{54}Fe(d,n)^{55}Co$), 40 MeV protons to natural iron ($^{56}Fe(p,2n)^{55}Co$), or 20 MeV protons to natural nickel foil ($^{58}Ni(p,\alpha)^{55}Co$) followed by separation of the ^{55}Co on an ion exchange column (Wolf 1955). Due to the short half-life (17.5 hours), however, ^{55}Co would not be persistent in the environment or in waste sites. ^{57}Co (half-life of 270 days) is produced by AEA Technology (formerly Amersham QSA) in the United Kingdom (Web Research Co. 1999).

5.2 IMPORT/EXPORT

In 2002, 8,450 metric tons of cobalt were imported into the United States compared with 7,670, 8,150, 8,770, and 9,410 metric tons in 1998, 1999, 2000, and 2001 (USGS 2002). Between 1999 and 2002, Finland, Norway, Russia, and Canada supplied 24, 18, 13, and 10% of cobalt, respectively (USGS 2004). Imports for 2002 by form included (form, metric tons cobalt content): metal, 6,800; oxides and hydroxides, 936; acetates, 84; carbonates, 60; chlorides, 22; and sulfates 545. Cobalt exports for 1999, 2000, 2001, and 2002 were 1,550, 2,630, 3,210, and 2,080 metric tons, respectively; exports estimated in 2002 are 2,500 metric tons (USGS 2002; 2004).

^{60}Co and ^{57}Co are produced in Canada and in the United Kingdom and are imported from these countries. No import and export quantities for cobalt radioisotopes were available.

5.3 USE

The United States is the world's largest consumer of cobalt. Cobalt is used in a number of essential military and industrial applications. The largest use of metallic cobalt is in superalloys that are used in gas turbines aircraft engines. Superalloys are alloys developed for applications where elevated temperatures and high mechanical stress are encountered. It is also used in magnetic alloys and alloys that are required for purposes requiring hardness, wear resistance, and corrosion resistance. Cobalt is used as a binder for tungsten carbide (cemented carbides) cutting tools to increase impact strength. Cobalt compounds are used as pigments in glass, ceramics, and paints; as catalysts in the petroleum industry; as paint driers; and as trace element additives in agriculture and medicine.

Over 40% of nonmetallic cobalt is used in catalysis, and most cobalt catalysts are used in hydrotreating/desulfurization in the oil and gas industry, the production of terephthalic acid and dimethylterephthalate, and the production of aldehydes using the high pressure oxo process (hydroformylation). Cobalt chemicals primarily used as catalysts include cobalt(III) acetate, cobalt(II) bromide, carbonate, manganate, oxalate, and sulfide, cobalt carbonyl, and cobalt naphthenate. Cobalt carbonate and chromate are mainly used as pigments and cobalt(II) acetate, 2-ethylhexanoate, linoleate, naphthenate, nitrate, oleate, and stearate are mainly used as driers. Cobalt has been used for hundreds of years as a blue colorant in glass, ceramics, and paints (Richardson 1993).

A growing use for cobalt is as an addition to the Ni/Cd, Ni-metal hydride battery or as the main component of the lithium ion cell ($LiCoO_2$). In 2002, the reported U.S. cobalt consumption was 7,930 metric tons with a use pattern of (end use, metric tons cobalt content, percent): superalloys, 3,700, 46.7%; steel alloys, 555, 7.0%; other alloys, including magnetic alloys, 1,050, 13.2%; cemented carbides, 617, 7.8%; chemical and ceramic use, 1,950, 24.6%; and miscellany, 63, 0.8%. Cobalt is also used a target material in electrical x-ray generators (Cobalt Development Institute 2004; Donaldson 1986; Hodge 1993; IARC 1991; Richardson 1993; USGS 2002).

Gamma rays from ^{60}Co are used medically to treat cancer and industrially to sterilize medical and consumer products, to crosslink, graft and degrade plastics, and as an external source in radiography and radiotherapy. ^{60}Co, along with iridium 192 (^{192}Ir), are the most commonly used isotopes in industrial radiography. In this application, ^{60}Co is used for nondestructive testing of high-stress alloy parts, such as pipeline weld joints, steel structures, boilers, and aircraft and ship parts. Radiography may be conducted

at permanent, specially shielded facilities or temporary sites in the field (USNRC 1999). ^{60}Co is used in chemical and metallurgical analysis and as a tracer in biological studies. In 1990, about 95% of installed ^{60}Co activity was used for the sterilization of medical devices; about 45% of medical devices were sterilized using radiation. ^{60}Co is also a source of gamma rays used for food irradiation; depending on the dose levels, irradiation may be used to sterilize food, destroy pathogens, extend the shelf-life of food, disinfest fruits and grain, delay ripening, and retard sprouting (e.g., potatoes and onions). Sludge, waste water, and wood may also be treated with gamma rays to kill harmful organisms.

^{57}Co decays to an excited state of ^{57}Fe, the most widely used x-ray source in Mössbauer spectroscopy (Hodge 1993; Richardson 1993). It is also made into standards and sources for dose calibrators, gamma cameras, and gauges, and is used as markers and rulers to help estimate organ size/location. It is also used in *in vitro* diagnostic kits for the study of anemia related to vitamin B_{12} deficiency/malabsorption (MDS Nordion 2000). ^{55}Co-bleomycin has been used for scanning malignant tumors (e.g., lung and brain cancer) and is a practical isotope for positron emission tomography (PET) studies because it mainly (81%) decays by positron emission.

5.4 DISPOSAL

There is a paucity of data on the methods of disposal of cobalt and its compounds. Due to the lack of natural sources of economically extractable ores in the United States, cobalt is entirely imported in the United States, and it is considered a strategic mineral. It is economical to recycle certain cobalt wastes rather than to dispose of them. Recycling of superalloy scrap is an important method for the recovery of cobalt. About 2,200 metric tons of cobalt were recycled from purchased scrap in 2003. This was about 28% of reported consumption for the year (USGS 2004). According to TRI (TRI01 2004), 7.14 and 1.42 million pounds of cobalt and cobalt compounds combined were recycled onsite and offsite, respectively, in 2001. Waste containing cobalt dust and, presumably, waste containing cobalt in the solid state may be placed in sealed containers and disposed of in a secured sanitary landfill (HSDB 1989). Waste water containing cobalt can be treated before disposal, for instance, by precipitation of carbonate or hydroxide of cobalt or by passage through an ion-exchange resin (Clifford et al. 1986). According to TRI (TRI01 2004), 1,619,874 pounds of cobalt and cobalt compounds, were transferred offsite for disposal, in processes such as solidification/stabilization and waste water treatment, including publicly operated treatment plants (POTWs). The amount of cobalt so transferred by state is shown in Table 6-1.

In August 1998, EPA issued a final rule listing spent hydrotreated and hydrorefined catalysts as hazardous waste under the Resource Conservation and Recovery Act (USGS 1998). Listing under this act requires that releases of these substances will be subject to certain management and treatment standards and emergency notification requirements. Information regarding effluent guidelines and standards for cobalt may be found in Title 40 of the Code of Federal Regulations, Parts 421.230, 421.310, and 471.30.

^{60}Co sources used for irradiation purposes are valuable and are not to be discarded. However, some radioactive cobalt isotopes may occur in waste material from nuclear reactors. Radioactive waste is categorized according to origin, type of waste present, and level of activity. Radioactive cobalt isotopes may be commingled with other radioactive isotopes. The first distinction in radioactive waste is between defense waste and commercial waste, the former being generated during and after World War II principally at the Department of Energy (DOE) facilities at Hanford, Washington; Savannah River, South Carolina; and Idaho Falls, Idaho, where plutonium and other isotopes were separated from production reactor spent fuel or nuclear-powered naval vessels. Commercial wastes are produced predominantly by nuclear power plants as well as the long defunct commercial reprocessing facility at West Valley, New York and manufacturers of radioisotopes used in nuclear medicine for the treatment and diagnosis of disease. Nuclear waste is also classified as high-level waste (HLW), transuranic waste (TRU), and low-level waste (LLW). LLW is further differentiated into three classes, A, B, and C, according to increasing of the level of activity. A fourth category, commercial greater-than-class-C LLW (listed in 10 CFR 61.55 Tables 1 and 2 for long and short half-life radionuclides, respectively) is not generally suitable for near-surface disposal. This could include operating and decommissioning waste from nuclear power plant and sealed radioisotope sources. The final disposition for this waste has not been determined. If LLW also contains nonradioactive hazardous material (i.e., that which is toxic, corrosive, inflammable, or explosive), it is termed mixed waste. Mine tailings from uranium mining is yet another category of radioactive waste (DOE 1999; Murray 1994). While radioactive cobalt would not ordinarily be found in HLW or TRU, the definitions of these are included below for completion.

TRUs are those containing isotopes, like plutonium, that are above uranium in the periodic table and whose half-lives are >20 years. If their level of activity is <100 nanocuries (nCi) (<3,700 becquerels[Bq]) of alpha-emitters per gram of waste material (up from 10 nCi/g in 1982), the waste could be disposed of by shallow burial. Otherwise, the waste must be placed in retrievable storage for eventual transfer to a

permanent repository. The level of radioactivity in TRUs is generally low; they generate very little heat and can be handled by ordinary means without remote control (Eisenbud 1987, Murray 1994).

HLW includes spent fuels that are contained in fuel rods that have been used in a nuclear reactor. These may contain small amounts of transuranic elements. After removal from the reactor, these rods are placed into pools adjacent to the commercial nuclear power plants and DOE facilities where they were produced. It was originally intended that the fuel rods remain in these pools for only about 6 months to allow for a reduction in short-lived radioactivity and rate of heat production temperature and then be transferred to a reprocessing or storage facility. There is no commercial reprocessing facility or permanent disposal facility for HLW operating in the United States. The U.S. Nuclear Regulatory Commission (USNRC) has issued standards for the disposal of HLW (10 CFR 60), and the DOE is pursuing the establishment of an HLW facility in Yucca Mountain, Nevada. Efforts to establish an HLW facility, which began over 2 decades ago, have experienced many delays (Eisenbud 1987; Murray 1994). However, in July, 2002, the U.S. Congress and the President selected Yucca Mountain, Nevada as the nation's first long-term repository for HLW. The facility is projected to begin operation in 2010, and efforts are underway to consider establishing a nearby interim facility (DOE 2002b).

LLWs are officially defined as wastes other than those previously defined. These wastes come from certain reactor operations and from manufacturers of radioisotopes used in nuclear medicine and institutions such as hospitals, universities, and research centers. Most LLW contain very little radioactivity and contain practically no transuranic elements. It requires little or no shielding or special handling and may be disposed of by shallow burial. However, some LLW contains sufficient radioactivity as to require special treatment. Although USNRC regulations for LLW disposal (10 CFR 61) permit shallow land burial, many states have enacted more stringent regulations that require artificial containment of the waste in addition to natural containment (Eisenbud 1987; Murray 1994). The EPA has the authority to set generally acceptable environmental standards for LLW that would be implemented by the US NRC and DOE (EPA 2004). The Manifest Information Management System (MIMS), maintained by the Idaho National Engineering and Environmental Laboratory (INEEL), contains information on low-level radioactive waste shipments received at commercial low-level radioactive waste disposal facilities at Barnwell, South Carolina (1/1/86–present), Beaty, Nevada (4/1/86–12/31/92), Richland, Washington(1/1/86–present), and Envirocare, Utah (1/1/98–12/31/99). In 1999, 17 Ci (0.63 TBq) of ^{57}Co, 1,300 Ci (48 TBq) of ^{58}Co, and 1.08×10^6 Ci (4.00×10^4 TBq) of ^{60}Co contained in LLW was received at these facilities from academic, industrial, government, and utility generators throughout the United

States (INEL 2000). In addition, 4.26 Ci (0.158 TBq) of ^{57}Co of NARM ("naturally occurring and accelerator-related waste") was received.

At present, DOE stores most of its spent fuel at three primary locations: the Hanford site, Washington, the Idaho National Engineering and Environmental Laboratory (INEEL), Idaho, and the Savannah River site, South Carolina. Some spent fuel is also stored at the dry storage facility at Fort St. Vrain in Colorado. Much smaller amounts of spent nuclear fuel stored at other sites were to be shipped to the three prime sites for storage and preparation for ultimate disposal (DOE 1999). The DOE National Spent Fuel Program maintains a spent nuclear fuel database that lists the total volume, mass, and metric tons heavy metal (MTHM) of 16 DOE categories of spent nuclear fuel stored in each of the three locations. The categories having the highest ^{60}Co activities per spent nuclear fuel canister (decayed to 2030) are 'naval surface ship fuel' and 'naval submarine fuel'. The ^{58}Co and ^{60}Co solid wastes stored on the Hanford site in 1998 as LLW were 2,600 and 6,900 Ci (96 and 260 TBq), respectively (Hanford 1999). In addition, 40 Ci (1.5 TBq) of ^{60}Co was included in TRU.

In commercial irradiators, additional quantities of ^{60}Co are added, usually once a year to maintain preferred radiation levels of the source (MDS Nordion 2000). ^{60}Co sources are removed from the facility at the end of their useful life, which is typically 20 years. In general, manufacturers of ^{60}Co sources guarantee to accept the sources they originally supplied. These old sources may be reencapsulated, reprocessed, or recycled when technically, environmentally, and economically feasible.

6. POTENTIAL FOR HUMAN EXPOSURE

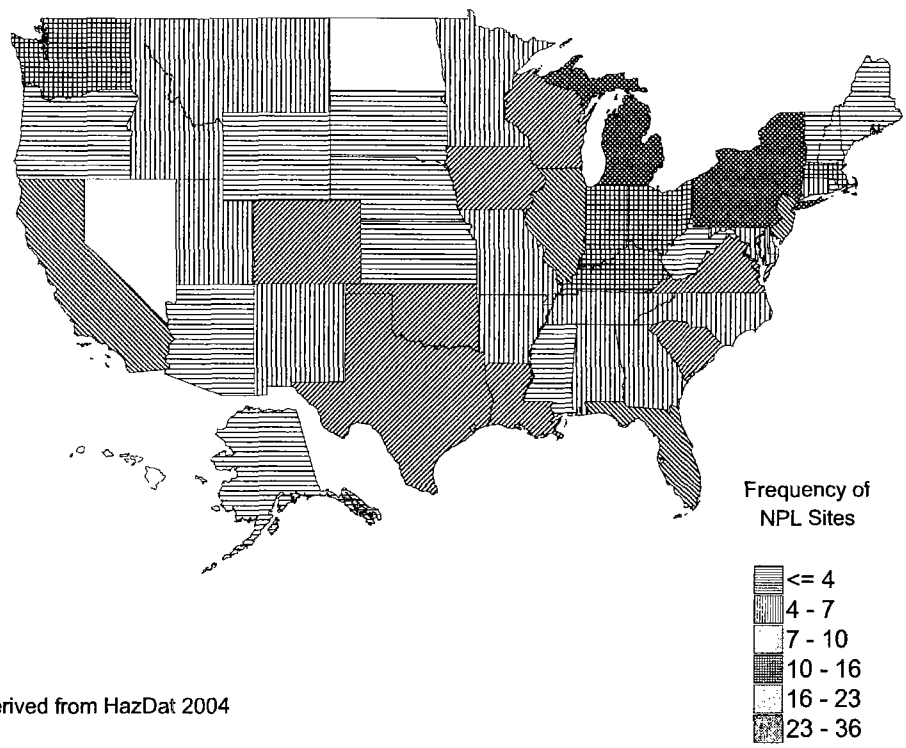
6.1 OVERVIEW

Stable cobalt has been identified in at least 426 of the 1,636 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL) (HazDat 2004). Radioactive cobalt as ^{60}Co has been identified in at least 13 of the 1,636 hazardous waste sites that have been proposed for inclusion on the EPA NPL (HazDat 2004). However, the number of sites evaluated for stable cobalt and ^{60}Co is not known. The frequency of these sites can be seen in Figures 6-1 and 6-2, respectively. Of the cobalt sites, 421 are located within the United States, 1 is located in Guam (not shown), 3 are located in the Commonwealth of Puerto Rico (not shown), and 1 is located in the Virgin Islands (not shown). All of the sites at which ^{60}Co has been identified are located within the United States.

Cobalt occurs naturally in the earth's crust, and therefore, in soil. Low levels of cobalt also occur naturally in seawater and in some surface water and groundwater (Smith and Carson 1981). However, elevated levels of cobalt in soil and water may result from anthropogenic activities such as the mining and processing of cobalt-bearing ores, the application of cobalt-containing sludge or phosphate fertilizers to soil, the disposal of cobalt-containing wastes, and atmospheric deposition from activities such as the burning of fossil fuels and smelting and refining of metals (Smith and Carson 1981). Cobalt is released into the atmosphere from both anthropogenic and natural sources. However, emissions from natural sources are estimated to slightly exceed those from manufactured sources. Natural sources include windblown soil, seawater spray, volcanic eruptions, and forest fires. Primary anthropogenic sources include fossil fuel and waste combustion, vehicular and aircraft exhausts, processing of cobalt and cobalt-containing alloys, copper and nickel smelting and refining, and the manufacture and use of cobalt chemicals and fertilizers derived from phosphate rocks (Barceloux 1999; Lantzy and Mackenzie 1979; Nriagu 1989; Smith and Carson 1981). ^{60}Co and ^{58}Co, both radioactive forms of cobalt, may be released to the environment as a result of nuclear research and development, nuclear accidents, operation of nuclear power plants, and radioactive waste dumping in the sea or in radioactive waste landfills.

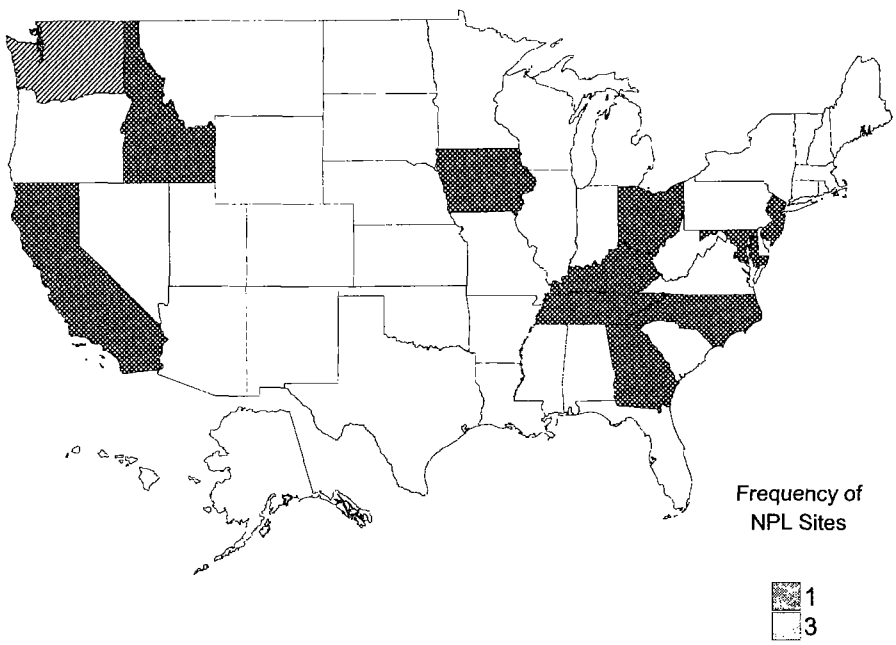
Cobalt compounds are nonvolatile and cobalt will be emitted to the atmosphere only in particulate form. Their transport in air depends on their form, particle size and density, and meteorological conditions. Cobalt so released will return to land or surface water as wet or dry deposition. Coarse particles, those

Figure 6-1. Frequency of NPL Sites with Cobalt Contamination



Derived from HazDat 2004

Figure 6-2. Frequency of NPL Sites with ^{60}Cobalt Contamination



Derived from HazDat 2004

with aerodynamic diameters >2 μm (such as those obtained during ore processing), may deposit within 10 km from the point of emission; finer particles (such as is obtained from thermal processes) may travel longer distances. It is generally assumed that anthropogenic cobalt originating from combustion sources exists primarily as the oxide; arsenides or sulfides may be released during mining and ore processing (Schroeder et al. 1987). Frequently, sediment and soil are the ultimate sinks for cobalt; however, this process is dynamic, and cobalt can be released into the water depending upon conditions. Soluble cobalt released into waterways will sorb to particles and may settle into the sediment or be sorbed directly by sediment. It may precipitate out as carbonates and hydroxides or with mineral oxides. It may also sorb to or complex with humic acid substances in the water. These processes are sensitive to environmental factors such as pH and the proportion of dissolved cobalt will be higher at low pH. In the case of ^{60}Co released into an experimental lake in northwestern Ontario, cobalt's half-life in the water column was 11 days; 5% of added ^{60}Co remained in the water after 100 days (Bird et al. 1998a). Cobalt can also be transported in dissolved form or as suspended sediment by rivers to lakes and the sea or by ocean currents. The proportion of cobalt transported in each form is highly variable (Smith and Carson 1981). In deep sediment where water is anoxic and hydrogen sulfide is present, some mobilization of cobalt from sediment may occur, probably due to the formation of bisulfides and polysulfides (Bargagli 2000; Brügmann 1988; Finney and Huh 1989; Glooschenko et al. 1981; Knauer et al. 1982; Nriagu and Coker 1980; Shine et al. 1995; Smith and Carson 1981; Szefer et al. 1996; Windom et al. 1989). Cobalt adsorbs rapidly and strongly to soil and sediment in which it is retained by metal oxides, crystalline minerals, and natural organic matter. The mobility of cobalt sediment depends on the nature of the soil or sediment; it increases with decreasing pH and redox potential (Eh) and in the presence of chelating/complexing agents (Brooks et al. 1998; Buchter et al. 1989; King 1988b; McLaren et al. 1986; Schnitzer 1969; Smith and Carson 1981; Swanson 1984; Yashuda et al. 1995).

While cobalt may be taken up from soil by plants, the translocation of cobalt from roots to above-ground parts of plants is not significant in most soils; the transfer coefficient (concentration in plant/concentration in soil) for cobalt is generally 0.01–0.3 (Mascanzoni 1989; Mermut et al. 1996, Smith and Carson 1981). However, in highly acidic soils (pH as low as 3.3) and in some higher plants (plants excluding algae), significantly higher transfer has been observed (Boikat et al. 1985; Francis et al. 1985; Jenkins 1980; Kloke et al. 1984; Mejstrik and Svacha 1988; Palko and Yli-Hala 1988; Tolle et al.1983; Watabe et al. 1984). The bioaccumulation factors (dry weight basis) for cobalt in marine fish and freshwater fish are ~100–4,000 and <10–1,000, respectively; accumulation is largely in the viscera and on the skin, as

opposed to the edible parts of the fish. Cobalt does not biomagnify up the food chain (Barceloux 1999; Evans et al. 1988; Freitas et al. 1988; Smith and Carson 1981).

Atmospheric cobalt is associated with particulate matter. Mean cobalt levels in air at unpolluted sites are generally <1–2 ng/m^3. In several open-ocean environments, geometric mean concentrations ranged from 0.0004 to 0.08 ng/m^3 (Chester et al. 1991). However, in source areas, cobalt levels may exceed 10 ng/m^3; the highest average cobalt concentration recorded was 48 ng/m^3 at the site of a nickel refinery in Wales (Hamilton 1994; Smith and Carson 1981). By comparison, the Occupational Safety and Health Administration (OSHA) limit for airborne stable cobalt is 100,000 ng/m^3. While ^{60}Co has been detected in some air samples at the Hanford, Washington site and Oak Ridge National Laboratories, Tennessee, levels were not reported (HazDat 2004; PNNL 1996).

The concentrations of stable cobalt in surface and groundwater in the United States are generally low; <1 μg/L in pristine areas and 1–10 μg/L in populated areas (Hamilton 1994; Smith and Carson 1981). However, cobalt levels may be considerably higher in mining or agricultural areas. Cobalt concentrations in surface water and groundwater samples collected in 1992 from area creeks near the Blackbird Mine in Idaho, one of the large deposits of cobalt in North America where mining occurred from the late 1800s to 1982, were reported to range from <1 to 625,000 μg/L, and from not detected to 315,000 μg/L, respectively (ATSDR 1995). Cobalt levels in most drinking water is <1–2 μg/L although levels as high as 107 μg/L have been recorded (Greathouse and Craun 1978; Meranger et al. 1981; NAS 1977; Smith and Carson 1981).

Little data are available on the levels of ^{60}Co in water. In 1989, subsequent to the largest effluent discharge from the Steam Generating Heavy Water Reactor at Winfrith on the south coast of England, ^{60}Co levels in offshore seawater from 18 sites contained 0.06–2.22 mBq/L (1.6–69 fCi) of particulate ^{60}Co, 0.30–10.3 mBq/L (8–280 fCi) of soluble ^{60}Co(II), and 0.12–1.55 mBq/L (3.2–42 fCi) of soluble ^{60}Co(III) (Leonard et al. 1993a). The U.S. NRC discharge limit is 111,000 mBq/L (3x10^6 fCi/L) (USNRC 1991).

The average concentrations of cobalt in the earth's crust are 20–25 mg/kg (Abbasi et al. 1989; Merian 1985; Smith and Carson 1981). Most soils contain 1–40 mg cobalt/kg; the average cobalt concentration in U.S. soils is 7.2 mg/kg (Smith and Carson 1981). Soils containing <0.5–3 mg cobalt/kg are considered cobalt-deficient because plants growing on them have insufficient cobalt (<0.08–0.1 mg/kg) to meet the

dietary requirements of cattle and sheep. Cobalt-deficient soils are found in some areas of the southeastern and northeastern United States. Soils near ore deposits, phosphate rocks, or ore smelting facilities, and soils contaminated by airport traffic, highway traffic, or other industrial pollution may contain high concentrations of cobalt; concentrations up to 800 mg/kg have been detected in such areas (Kloke et al. 1984; Smith and Carson 1981). Cobalt concentrations in 28 samples collected from surface deposits in the Big Deer and Blackbird Creek drainage basins near a site of former cobalt mining in Idaho ranged from 26.5 to 7,410 mg/kg (ATSDR 1995).

The level of cobalt in most foods is low. However, food is the largest source of exposure to cobalt in the general population. The estimated average daily dietary intake of cobalt in Canada was 11 μg/day. Food groups contributing most heavily to this intake were bakery goods and cereals (29.8%) and vegetables (21.9%) (Dabeka and McKenzie 1995). No estimates of the average dietary input of cobalt in the United States were located. People living near mining and smelting facilities or metal shops where cobalt is used in grinding tools may be exposed to higher levels of cobalt in air or soil. Similarly, people living near hazardous waste sites may be exposed to higher levels of cobalt in these media. Contaminated soils pose a hazardous exposure pathway to children because of both hand-to-mouth behavior and intentional ingestion of soil (pica) that contain metals and other contaminants (Hamel et al. 1998). However, much of the cobalt in soil may not be in a form that is available for uptake by the body. People who work in the hard metal industry, metal mining, smelting, and refining or other industries that produce or use cobalt and cobalt compounds may be exposed to substantially higher levels of cobalt, mainly from dusts or aerosols in air. Workers at nuclear facilities, irradiation facilities, or nuclear waste storage sites may be exposed to radioisotopes of cobalt. Exposure would generally be to radiation produced by these isotopes (e.g., gamma radiation from ^{60}Co).

6.2 RELEASES TO THE ENVIRONMENT

Stable cobalt has been identified in a variety of environmental media (air, surface water, leachate, groundwater, soil, and sediment) collected at 426 of 1,636 current or former NPL hazardous waste sites (HazDat 2004). ^{60}Co has been identified in a variety of environmental media (air, surface water, leachate, groundwater, soil, and sediment) collected at 13 of 1,636 current or former NPL hazardous waste sites (HazDat 2004).

According to the Toxic Chemical Release Inventory (TRI), in 2001, total releases of cobalt and cobalt compounds to the environment (including air, water, soil, and underground injection) from 605 reporting facilities that produced, processed, or used cobalt or cobalt compounds were 16,443,429 pounds (TRI01 2004). Table 6-1 lists amounts released from these facilities grouped by state. In addition, 1,619,874 pounds of cobalt and cobalt compounds were transferred offsite by these facilities (TRI01 2004). Starting in 1998, metal mining, coal mining, electric utilities, and Resource Conservation and Recovery Act (RCRA)/solvent recovery industries are required to report, to the TRI, industries with potentially large releases of cobalt and cobalt compounds. Industrial sectors producing, processing, or using cobalt that contributed the greatest environmental releases in 2001 were primary metals and RCRA/solvent recovery with 141,554 and 531,427 pounds, respectively. Industrial sectors producing, processing, or using cobalt compounds that contributed the greatest environmental releases in 2001 were metal mining and electrical utilities with 10,228,193 and 3,652,398, pounds, respectively. The TRI data should be used with caution because only certain types of facilities are required to report. This is not an exhaustive list.

6.2.1 Air

The sources of cobalt in the atmosphere are both natural and anthropogenic (Barceloux 1999). Natural sources include wind-blown continental dust, seawater spray, volcanoes, forest fires, and continental and marine biogenic emissions. The worldwide emission of cobalt from natural sources has been estimated to range from 13 to 15 million pounds/year (Lantzy and Mackenzie 1979; Nriagu 1989). The global atmospheric emission of cobalt from anthropogenic sources is an estimated 9.7 million pounds/year. Therefore, natural sources contribute slightly more to cobalt emissions in the atmosphere than anthropogenic sources (Lantzy and Mackenzie 1979). The primary anthropogenic sources of cobalt in the atmosphere are the burning of fossil fuels and sewage sludge, phosphate fertilizers, mining and smelting of cobalt-containing ores, processing of cobalt-containing alloys, and industries that use or process cobalt compounds. Small amounts of cobalt are found in coal, crude oils, and oil shales. Therefore, burning of these fossil fuels for power generation will emit cobalt into the atmosphere. The cobalt contents of the fly ash and flue gases of a coal-burning power plant are approximately 25 mg/kg and 100–700 μg/L, respectively. Gasoline contains <0.1 mg cobalt/kg, but catalytic converters may contain cobalt; therefore, emissions from vehicular exhaust are also a source of atmospheric cobalt (Abbasi et al. 1989; Holcombe

Table 6-1. Releases to the Environment from Facilities that Produce, Process, or Use Cobalt and Cobalt Compounds[a]

		Reported amounts released in pounds per year[b]						
State[c]	Number of facilities	Air[d]	Water	Under-ground injection	Land	Total on-site release[e]	Total off-site release[f]	Total on and off-site release
AK	2	23	0	16,000	546,463	562,486	0	562,486
AL	21	5,893	8,612	0	315,853	330,358	30,040	360,398
AR	9	921	142	0	8,301	9,364	2,015	11,379
AZ	12	1,029	0	0	1,061,035	1,062,064	2,266	1,064,330
CA	26	646	20	0	307,654	308,320	7,463	315,783
CO	1	3	1	0	12,026	12,030	0	12,030
CT	9	632	65	0	0	697	4,133	4,830
DE	2	1,265	52	0	52	1,369	27,444	28,813
FL	11	2,397	345	0	93,049	95,791	15,464	111,255
GA	17	3,508	268	0	282,610	286,386	12,461	298,847
IA	6	566	0	0	0	566	2,123	2,689
ID	2	74	5	0	395,424	395,503	0	395,503
IL	23	1,630	1,278	0	16,999	19,907	102,088	121,995
IN	42	7,005	351	0	279,122	286,478	64,293	350,771
KS	5	4,269	0	0	10,200	14,469	3,859	18,328
KY	22	3,184	542	0	478,855	482,581	13,269	495,850
LA	15	385	8,477	2,700	66,858	78,420	91,274	169,694
MA	11	794	780	0	5	1,579	17,403	18,982
MD	6	2,472	15	0	6,629	9,116	45,382	54,498
ME	2	66	0	0	0	66	700	766
MI	24	4,699	559	0	125,405	130,663	33,737	164,400
MN	5	255	No data	0	0	255	7,666	7,921
MO	5	1,457	8	0	559,401	560,866	0	560,866
MS	7	386	120	12,000	44	12,550	3,044	15,594
MT	1	250	No data	0	31,000	31,250	505	31,755
NC	24	6,593	8,257	0	194,974	209,824	216,849	426,673
ND	4	1,165	21	0	108,300	109,486	39,842	149,328
NE	1	0	27	0	0	27	3,982	4,009
NH	1	0	No data	0	0	0	No data	0
NJ	12	1,191	26	0	413	1,630	26,894	28,524
NM	6	498	1	0	4,257,140	4,257,639	69	4,257,708
NV	8	678	0	0	4,099,136	4,099,814	9,950	4,109,764
NY	12	755	44	0	11,843	12,642	14,322	26,964
OH	44	4,977	771	1,100	310,653	317,501	96,116	413,617
OK	13	1,357	158	0	5,677	7,192	20,760	27,952

Table 6-1. Releases to the Environment from Facilities that Produce, Process, or Use Cobalt and Cobalt Compounds[a]

		Reported amounts released in pounds per year[b]						
State[c]	Number of facilities	Air[d]	Water	Under-ground injection	Land	Total on-site release[e]	Total off-site release[f]	Total on and off-site release
OR	6	1,262	20	0	16,487	17,769	2,862	20,631
PA	44	6,169	3,176	0	51,350	60,695	221,662	282,357
PR	2	2	No data	0	0	2	2,871	2,873
RI	1	1	1	0	0	2	50	52
SC	25	1,579	10,970	0	43,488	56,037	70,316	126,353
SD	1	0	No data	0	0	0	0	0
TN	18	5,560	4,013	0	330,615	340,188	36,520	376,708
TX	44	8,126	784	3,730	150,470	163,110	95,840	258,950
UT	6	278	No data	0	23,350	23,628	126,502	150,130
VA	9	1,451	518	0	89,388	91,357	9,683	101,040
VI	1	0	0	0	0	0	0	0
WA	2	72	91	0	106,618	106,781	5,112	111,893
WI	20	1,098	5	0	8	1,111	95,996	97,107
WV	13	1,341	566	0	212,254	214,161	37,047	251,208
WY	2	898	No data	0	38,927	39,825	0	39,825
Total	605	88,860	51,089	35,530	14,648,076	14,823,555	1,619,874	16,443,429

Source: TRI01 2004

[a]The TRI data should be used with caution since only certain types of facilities are required to report. This is not an exhaustive list. Data are rounded to nearest whole number.
[b]Data in TRI are maximum amounts released by each facility.
[c]Post office state abbreviations are used.
[d]The sum of fugitive and stack releases are included in releases to air by a given facility.
[e]The sum of all releases of the chemical to air, land, water, and underground injection wells.
[f]Total amount of chemical transferred off-site, including to publicly owned treatment works (POTW).

et al. 1985; Ondov et al. 1982; Smith and Carson 1981). Cobalt has been detected in cigarette tobacco and therefore, smoking is a potential source of atmospheric cobalt that could impact on indoor air quality (Munita and Mazzilli 1986).

Stable cobalt has been identified in air samples collected at 5 of the 426 current or former NPL hazardous waste sites where it was detected in some environmental media (i.e., air, soil, sediment, or water) (HazDat 2004). ^{60}Co has been identified in air samples collected at 2 of the 13 current or former NPL hazardous waste sites where it was detected in some environmental media (HazDat 2004).

Air sampling data were used to estimate ^{60}Co release from the Savannah River Site (SRS) from the plant's start up in 1954 to 1989 (DOE 1991). From this monitoring, it was estimated that 0.092 Ci (3.4 GBq) of ^{60}Co was released to the atmosphere between 1968 and 1986. Total releases of ^{60}Co to the atmosphere from the SRS between 1968 and 1996 were 0.092 Ci (3.4 GBq) (DOE 1998). Data were not reported for all years in this interval. In 1999, atmospheric releases of ^{57}Co, ^{58}Co, and ^{60}Co as particulates were 4.71×10^{-8}, 1.27×10^{-4}, and 1.30×10^{-4} Ci (0.00174, 4.70, and 4.81 MBq), respectively (DOE 1999). The SRS was a major production facility to the U.S. defense program and included five nuclear reactors, a fuel fabrication plant, a naval fuel materials facility, two chemical separation plants, a heavy water production plant, and a laboratory. ^{60}Co has also been detected in air samples at the Hanford site and Oak Ridge National Laboratories (HazDat 2004; PNNL 1996).

According to the TRI, in 2001, releases of 88,860 pounds of cobalt and cobalt compounds to air from 605 reporting facilities accounted for 0.5% of the total onsite environmental releases of these substances (TRI01 2004). The industrial sectors contributing the largest release of cobalt and cobalt compounds to air were electrical utilities, chemicals, and primary metals. Table 6-1 lists the amounts of cobalt and cobalt compounds released to air from these facilities grouped by state. The TRI data should be used with caution, however, since only certain types of facilities are required to report. This is not an exhaustive list.

6.2.2 Water

Compounds of cobalt occur naturally in seawater and in some surface, spring, and groundwater (Smith and Carson 1981). Cobalt is also released into water from anthropogenic sources. While there has been

no mine production of cobalt in the United States in recent years, cobalt is a byproduct or coproduct of the refining of other mined metals such as copper and nickel. Historic mining operations that processed cobalt containing ores may continue to release cobalt into surface water and groundwater. Waste water from the recovery of cobalt from imported matte or scrap metal, refining of copper and nickel, or during the manufacture of cobalt chemicals are sources of cobalt in water (Smith and Carson 1981). Process water and effluent from coal gasification and residue from solvent-refined coal contain cobalt. The accidental discharge of activated sludge and sewage may be important sources of cobalamins in waterways, together with bioconcentration by benthic organisms (Smith and Carson 1981). The discharge of waste water by user industries, such as paint and pigment manufacture, also contributes to the release of cobalt into water. In one case, manufacturers of nickel-cadmium batteries operating between 1953 and 1979 discharged cobalt from a battery factory to the Hudson River in Foundry Cove, New York, of which 1.2 metric tons are estimated to be present in the eastern cove (Knutson et al. 1987). Atmospheric deposition is an additional source of cobalt in water. Lake Huron receives an estimated 76% of its cobalt input from natural sources and 24% from anthropogenic sources. The corresponding estimated values for Lake Superior are 85.4 and 14.6% (Smith and Carson 1981). In these Great Lakes, it therefore appears that natural inputs of cobalt far exceed anthropogenic ones.

Cobalt has been identified in groundwater and surface water at 255 and 106 sites, respectively, of the 426 NPL hazardous waste sites, where it was detected in some environmental media (i.e., air, soil, sediment, or water) (HazDat 2004). ^{60}Co has been identified in groundwater and surface water at 4 and 2 sites, respectively, of the 13 NPL hazardous waste sites, where it was detected in some environmental media (HazDat 2004).

According to the TRI, in 2001, the reported releases of 51,089 pounds of cobalt and cobalt compounds to water from 605 reporting facilities accounted for 0.3% of the total onsite environmental releases of these substances (TRI01 2004). Table 6-1 lists the amounts of cobalt and cobalt compounds released to water from these facilities grouped by state. As of 1998, TRI no longer separately collects data on substances released indirectly to Publicly-Owned Treatment Works (POTWs), part of which may ultimately be released to surface waters. The TRI data should be used with caution, however, since only certain types of facilities are required to report. This is not an exhaustive list.

^{60}Co is present in the low-level aqueous radioactive waste discharges from many nuclear power plants. Alloys that contain stable cobalt (^{59}Co), such as stellite, used in piping of nuclear reactors corrode and

may be activated, producing ^{60}Co, which accumulates in the reactor and must be periodically decontaminated. A common decontaminating agent includes a reducing metal ion (e.g., vanadium(II)) and a chelating agent (e.g., picolinate) resulting in low-level discharges of uncomplexed $^{60}Co(II)$ and complexed $^{60}Co(III)$. While soluble ionic and particulate forms predominate, at some sites stable, nonionic trivalent complexes of cobalt are present (Leonard et al. 1993a, 1993b; USNRC 2000d). For example, in 1987–1989 samples of treated effluent from the Steam Generating Heavy Water Reactor at Winfrith on the south coast of England, the percent of ^{60}Co as Co(III) picolinate ranged from 6.2 to 75.4%. Between 1978 and 1988, 12 TBq (320 Ci) of ^{60}Co was released into the Irish Sea by the British Nuclear Fuels reprocessing plant at Sellafield, United Kingdom (McCartney et al. 1994). These discharges are believed to be Co(II) (Leonard et al. 1993a). Both ^{58}Co and ^{60}Co are discharged into the Rhone River by the nuclear power plant at Bugey, France. This facility, which consists of a natural Uranium-Graphite-Gas unit and four pressurized water reactor (PWR) units, two of which are cooled by Rhone River water, discharged about 406 and 280 GBq (11.0 and 7.56 Ci) of ^{58}Co and ^{60}Co, respectively, in liquid waste during 1986–1990 (Beaugelin-Seiler et al. 1994).

Water sampling data were used to estimate effluent release from the SRS from the plant's start up in 1954 to 1989 (DOE 1991). From this monitoring, it was estimated that 17.8 Ci (659 GBq) of ^{60}Co were released into seepage basins and 66.4 Ci (2,460 GBq) were released into streams between 1955 and 1988. In addition, 2.7 Ci (100 GBq) of ^{58}Co were released into seepage basins between 1971 and 1988; no ^{58}Co was released into streams. Total releases of ^{60}Co to streams from the SRS for 1954–1995 were 66 Ci (2,400 GBq) (DOE 1998). No data were reported from 1985 to 1994. In 1999, 4.94×10^{-4} Ci (0.0183 GBq) of ^{60}Co was released to surface waters at the SRS (DOE 1999). ^{60}Co has also been reported in surface water at, Hanford, Washington, and Oak Ridge National Laboratories, and groundwater at Brook Industrial Park, New Jersey, the Hanford site and Oak Ridge National Laboratories, Tennessee (HazDat 2004). The Columbia River receives discharges from the unconfined aquifer underlying the Hanford Site via subsurface and surface (riverbank springs) discharges. This aquifer is contaminated by leachate from past waste-disposal practices at the site.

6.2.3 Soil

Cobalt occurs naturally in the earth's crust, and therefore, in soil. However, elevated levels of cobalt in soil may result from anthropogenic activities such as the mining and processing of cobalt-bearing ores,

the application of cobalt-containing sludge or phosphate fertilizers to soil, the disposal of cobalt-containing wastes, and atmospheric deposition from activities such as burning of fossil fuels, smelting, and metal refining (Smith and Carson 1981).

Cobalt has been identified in soil at 219 sites and sediment at 143 sites collected from 426 NPL hazardous waste sites, where it was detected in some environmental media (i.e., air, soil, sediment, or water) (HazDat 2004). ^{60}Co has been identified in soil at 8 sites and sediment at 2 sites collected from 13 NPL hazardous waste sites, where it was detected in some environmental media (HazDat 2004). ^{60}Co has been detected onsite in soils at the Hanford Site, Washington; INEEL, Idaho; Lawrence Livermore National Laboratory, Main Site, California; and Robins Air Force Base, Georgia at maximum concentrations of 87.7, 570, 0.21, and 0.07 pCi/g (3.24, 21, 0.0078, and 0.003 Bq/g) (HazDat 2004).

According to the TRI, in 2001, reported releases of 14,646,076 pounds of cobalt and cobalt compounds to land from 605 reporting facilities accounted for 98.8% of the total onsite environmental releases of these substances (TRI01 2004). An additional 35,530 pounds, accounting for 0.2% of the total onsite environmental releases were injected underground (TRI01 2004). Industrial sectors contributing the largest releases of cobalt and cobalt compounds to land were metal mining and electrical utilities with 10,210,508 and 3,197,209 pounds, respectively. Table 6-1 lists the amounts of cobalt and cobalt compounds released on land from these facilities grouped by state. The TRI data should be used with caution, however, since only certain types of facilities are required to report. This is not an exhaustive list.

6.3 ENVIRONMENTAL FATE

6.3.1 Transport and Partitioning

Cobalt compounds are nonvolatile, and thus, cobalt is emitted to the atmosphere in particulate form. The transport of cobalt in air depends on its particle size and density, and meteorological conditions; it can be returned to land or surface water by rain or it may settle to the ground by dry deposition. In nonarid areas, wet deposition may exceed dry deposition (Arimoto et al. 1985; Erlandsson et al. 1983). Coarse particles, with aerodynamic diameters >2 μm (such as those obtained during ore processing), may deposit within 10 km from the point of emission; finer particles may travel longer distances. It is the larger

particles that may be responsible for elevated local concentrations around emission sources. The mass median diameter for cobalt particles emitted from a power generator with a stack emission controlled by an electrostatic precipitator or scrubber ranged from <2 to 12 μm. The mass median diameter of cobalt in the ambient atmosphere is about 2.6 μm (Milford and Davidson 1985). Golomb et al. (1997) report average total (wet+dry) deposition rates of cobalt to Massachusetts Bay during the period September 15, 1992 to September 16, 1993. The total deposition rate was 58 $\mu g/m^2$-year, of which 47 $\mu g/m^2$-year was dry deposition and 12 $\mu g/m^2$-year was wet deposition. Total cobalt deposition flux at a site in the Rhone delta in southern France in 1988–1989 was 0.42±0.23 kg/km^2-year with 0.15 kg/km^2-year in the form of wet deposition (Guieu et al. 1991).

As with most metals, sediment and soil are frequently the final repository for cobalt released into the environment, although the process is dynamic, and cobalt can be released into the water depending upon conditions. Cobalt released into waterways may sorb to particles and settle into the sediment or be sorbed directly into the sediment. However, complexation cobalt to dissolved organic substances can significantly reduce sorption to sediment particles (Albrecht 2003). Studies by Jackman et al. (2001) suggest that interparticle migration of cobalt can influence the transport of metal ions, including cobalt, in sediments. For example, migration of a metal ion from a highly mobile sediment particle, such as clay, to less mobile gravels will slow the transport of that metal. Cobalt can also be transported in dissolved form or as suspended sediment by rivers to lakes and the sea or by ocean currents. Sediment in areas of active sedimentation would receive a large portion of the suspended sediment. In the case of the Peach Bottom Atomic Power Plant where ^{60}Co is released into the Conowingo Reservoir, an impoundment of the lower Susquehanna River, <20% of the radionuclide is trapped in the reservoir sediment, the rest being transported downstream and into the Chesapeake Bay (McLean and Summers 1990). It is often assumed that the primary mode of transport of heavy metals in aquatic systems is as suspended solids (Beijer and Jernelov 1986). However, in the case of cobalt, the percent that is transported by suspended solids is highly variable. Examples of the percentage of cobalt transported in suspended solids include (water body, percent): Main River (Germany), 33.4–42.2%; Susquehanna River (near its source in New York), 9%; New Hope River (North Carolina), 92%; Yukon River, >98%; Danube Rive (1961–1970), 27.4–85.9%; Columbia River (^{60}Co, downstream of the Hanford site), 95–98%; Strait of Juan de Fuca (Puget Sound, Washington), 11–15%; North Sea, 34%; and Lake Washington (Washington), 0% (Smith and Carson 1981).

In the oxic zones of many surface waters, dissolved cobalt levels decrease with increasing depth. This may be due to cobalt's continuous input into surface water from discharges or to increased adsorption and precipitation of the soluble forms with increasing depth. The fact that cobalt concentration profiles in deep water follow manganese and aluminum profiles strongly suggests that dissolved cobalt is precipitated in the adsorbed state with oxides of iron and manganese and with crystalline sediments such as aluminosilicate and goethite. A part of the cobalt may also precipitate out as carbonate and hydroxide in water. The higher concentration of organic pollutants in polluted water probably results in the formation of higher concentrations of soluble organic complexes. In a deep sediment where the water was anoxic and contained hydrogen sulfide, some mobilization of cobalt was observed, probably due to the formation of bisulfide and polysulfide complexes (Bargagli 2000; Brügmann 1988; Finney and Huh 1989; Glooschenko et al. 1981; Knauer et al. 1982; Nriagu and Coker 1980; Shine et al. 1995; Smith and Carson 1981; Szefer et al. 1996; Windom et al. 1989).

Cobalt strongly binds to humic substances naturally present in aquatic environments. Humic acids can be modified by UV light and bacterial decomposition, which may change their binding characteristics over time. The lability of the complexes is strongly influenced by pH, the nature of the humic material, and the metal-to-humic substance ratio. The lability of cobalt-humate complexes decreases in time ("aging effect") (Burba et al. 1994). The "aging effect" indicates that after a period of time (~12 hours), complexes that were initially formed are transformed into stronger ones from which the metal ion is less readily dislodged. In the Scheldt Estuary and the Irish Sea, between 45 and 100% of dissolved cobalt was found to occur in these very strong complexes (Zhang et al. 1990). Aquifer material from the contaminated aquifer at a low-level infiltration pit at the Chalk River Nuclear Laboratories in Canada was analyzed to assess the nature of the adsorbed ^{60}Co using sequential leaching techniques (Killey et al. 1984). Of the sediment-bound ^{60}Co, <10% was exchangeable, 5–35% was retained by iron oxide, and 55–>90% was fixed. Over 80% of the dissolved ^{60}Co was present as weakly anionic hydrophilic organic complexes. The average K_d for ^{60}Co between particulate matter and Po River (Italy) water was 451 m^3/kg over a 2-year monitoring period (Pettine et al. (1994). The mean K_d for ^{60}Co in Arctic surface sediment (Kara Sea) where large quantities of radioactive waste by the former Soviet Union was disposed was $1x10^5$ L/kg (range $1x10^3$–$7x10^5$), which is comparable to that in temperate coastal regions, $2x10^5$ L/kg (range, $2x10^4$–$1x10^6$) (Fisher et al. 1999).

The distribution coefficient of cobalt may vary considerably in the same sediment in response to conditions affecting the pH, redox conditions, ionic strength, and amount of dissolved organic matter

(Mahara and Kudo 1981b). Uptake of ^{60}Co from the water by sediment increased rapidly as the pH was increased from 5 to 7–7.5 and then slightly decrease (Benes et al. 1989a, 1989b). Therefore, pH would be an important factor affecting the migration of cobalt in surface water. Uptake was little affected by changes in liquid-to-solids ratio and ionic strength. ^{60}Co is more mobile in anaerobic marine aquatic environments than in freshwater aerobic ones (Mahara and Kudo 1981b). Therefore, ^{60}Co waste is most suitably stored underground in aerated zones away from possible seawater intrusions. In seawater-sediment systems under anaerobic conditions ^{60}Co was 250 times more mobile than ^{60}Co in freshwater-sediment systems under aerobic conditions. Under anaerobic conditions, 30% of the ^{60}Co added to a sediment-freshwater system was 'exchangeable' and therefore potentially mobile, while under aerobic conditions, 98% of the ^{60}Co was permanently fixed. Most of the mobile ^{60}Co produced under anaerobic conditions in seawater consisted of nonionic cobalt associated with low molecular weight organic substances that were stable to changes in pH; the exchangeable ^{60}Co appeared to be mostly ionic.

Bird et al. (1998b) added ^{60}Co to the anoxic hypolimnion of a Canadian Shield lake to simulate a nuclear waste scenario where radionuclides entered the bottom waters of a lake, and evaluated its behavior over 5 years. This situation was considered to be a likely pathway by which nuclear fuel waste stored deep underground in the plutonic (igneous) rock of this region would reach the surface environment via deep groundwater flow into the bottom waters of a lake. It was felt that adding a redox sensitive element such as cobalt to the anoxic hypolimnion might be different from adding it to the epilimnion. Monitoring vertical profiles in the lake established that the cobalt remained confined to the anoxic hypolimnion prior to the fall turnover (first 72 days) when mixing occurred throughout the water column. After 358 days, only about 4% of the ^{60}Co remained in the water. After the second year, approximately 2% of the ^{60}Co remained and after 5 years, only 0.4%. These results mirror previous experiments in which the ^{60}Co was added to the epilimnion, therefore establishing that there is little difference in the overall behavior of cobalt when added to the epilimnion or hypolimnion. The loss rate coefficient of ^{60}Co was 0.036/day (half-life=19 days) between days 90 and 131 (lake mixing) during which time, the cobalt sorbed to the suspended sediment and bottom sediment under anoxic conditions. Loss was to the sediment as there was no hydrological loss from the lake. In the previous experiment in which ^{60}Co was added to the epilimnion, the initial loss rate coefficient was somewhat higher, 0.056/day (half-life=12 days). Following the initial loss, ^{60}Co continued to be slowly removed from the water (loss rate coefficient 0.002/day; half-life=347 days); after 328 days, ^{60}Co was no longer detectable in the epilimnion. The half-life of ^{60}Co in the water column of an experimental lake in northwestern Ontario was 11 days; 5% of added ^{60}Co remained in the water after 100 days (Bird et al. 1998b). The redox potential also affects the

behavior of cobalt in sediment. Under moderately reducing conditions, cobalt is released from sediment as Co^{2+} and forms CoS in the presence of sulfide. The concentration of cobalt in the bottom water increases as the water becomes more anoxic (Brügmann 1988; Smith and Carson 1981).

The mobility of cobalt in soil is inversely related to how strongly it is adsorbed by soil constituents. Cobalt may be retained by mineral oxides such as iron and manganese oxide, crystalline materials such as aluminosilicate and goethite, and natural organic substances in soil. Sorption of cobalt to soil occurs rapidly (within 1–2 hours). Soil-derived oxide materials were found to adsorb greater amounts of cobalt than other materials examined, although substantial amounts were also adsorbed by organic materials. Clay minerals sorbed relatively smaller amounts of cobalt (McLaren et al. 1986). In addition, little cobalt was desorbed from soil oxides while substantial amounts desorbed from humic acids and montorillonite. In clay soil, adsorption may be due to ion exchange at the cationic sites on clay with either simple ionic cobalt or hydrolyzed ionic species such as $CoOH^+$. Adsorption of cobalt onto iron and manganese increases with pH (Brooks et al. 1998). In addition, as pH increases, insoluble hydroxides or carbonates may form, which would also reduce cobalt mobility. Conversely, sorption onto mobile colloids would enhance its mobility. In most soils, cobalt is more mobile than lead, chromium (II), zinc, and nickel, but less mobile than cadmium (Baes and Sharp 1983; King 1988b; Mahara and Kudo 1981b; Smith and Carson 1981). In several studies, the K_d of cobalt in a variety of soils ranged from 0.2 to 3,800. The geometric mean, minimum, median, and maximum K_ds of ^{60}Co in 36 Japanese agricultural soils were 1,840, 130, 1,735, and 104,000 L/kg, respectively (Yasuda et al. 1995). The soil properties showing the highest correlation with K_d were exchangeable calcium, pH, water content, and cation exchange capacity (CEC). In 11 U.S. soils, the mean Freundlich K_F and n values were 37 L/kg and 0.754, respectively; K_F values ranged from 2.6 to 363 L/kg and correlated with soil pH and CEC (Buchter et al. 1989). In 13 soils from the southeastern United States whose soil pH ranged from 3.9 to 6.5, cobalt sorption ranged from 15 to 93%; soil pH accounted for 84–95% of the variation in sorption (King 1988b).

Organic complexing agents such as ethylenediaminetetraacetic acid (EDTA), which are used for decontamination operations at nuclear facilities, greatly enhance the mobility of cobalt in soil. Other organic complexing agents, such as those obtained from plant decay, may also increase cobalt mobility in soil. However, both types of complexes decrease cobalt uptake by plants (Killey et al. 1984; McLaren et al. 1986; Toste et al. 1984). Addition of sewage sludge to soil also increases the mobility of cobalt, perhaps due to organic complexation of cobalt (Gerritse et al. 1982; Williams et al. 1985).

Leaching of cobalt has been observed from municipal and low-level radioactive waste sites (Cyr et al. 1987; Czyscinski et al. 1982; Friedman and Kelmers 1988). The mobility of cobalt was assessed in two soils from the Cabriolet and Little Feller event sites at the Nevada Test site as a function of various parameters such as pH, ionic strength, cobalt concentrations, soil solids concentrations, and particle size distribution (DOE 1996). Cobalt was quantitatively sorbed on these soils (at least 90% sorbed) when the pH was above 7 and the solid concentration was at least 20 g/L. The experiments suggest that binding is principally on amphoteric surface-hydroxyl surfaces. Since the pH of these soils is around 8, cobalt would bind strongly under normal environmental conditions. Migration would be severely retarded under all but the most extreme conditions, e.g., pH of 4 or below and high ionic strength soil solutions (approximately 0.1 M). In addition, unrealistically large quantities of water would be needed to displace cobalt from the upper layers of the soil profile.

Cobalt may be taken up from soil by plants. Surface deposition of cobalt on leaves of plants from airborne particles may also occur. Elevated levels of cobalt have been found in the roots of sugar beets and potato tubers in soils with high cobalt concentrations (e.g., fly ash-amended soil) due to absorption of cobalt from soil. However, the translocation of cobalt from roots to above-ground parts of plants is not significant in most soils, as indicated by the lack of cobalt in seeds of barley, oats, and wheat grown in high-cobalt soil (Mermut et al. 1996; Smith and Carson 1981). Mermut et al. (1996) found 0.01–0.02 mg/kg in 10 samples of durum wheat grain from different areas of Saskatchewan where surface soil cobalt levels ranged from 3.7 to 16.4 mg/kg. The enrichment ratio, defined as the concentration in a plant grown in amended soil (fly ash) over the concentration in unamended soil, was about 1. Other authors have determined the transfer coefficient (concentration in plant/concentration in soil) for cobalt to be 0.01–0.3. The mean ^{57}Co soil-plant transfer factors obtained for clover from eight soils over a 4-year period ranged from 0.02 to 0.35, in good agreement with results of other investigators (Mascanzoni 1989). However, in highly acidic soil (pH as low as 3.3), significantly higher than normal concentrations of cobalt were found in rye grass foliage, oats, and barley. For example, cobalt concentrations in rye grass grown in unlimed soil (pH<5.0) was 19.7 mg/kg compared with 1.1 mg/kg in rye grass grown in limed soil (pH>5.0) (Boikat et al. 1985; Francis et al. 1985; Kloke et al. 1984; Mejstrik and Svacha 1988; Palko and Yli-Hala 1988; Tolle et al. 1983; Watabe et al. 1984). Soil and plant samples taken in the 30-km zone around Chernobyl indicated that ^{60}Co was not accumulated by plants and mushrooms (Lux et al. 1995). Transfer factors obtained in 1992 ranged from 0.005 to 0.16 and those obtained in 1993 ranged from <0.001 to 0.008. Studies investigating the uptake of ^{60}Co by tomato plants watered with ^{60}Co-contaminated water showed that tomato plants absorbed <2% of the activity available from the soil. The

absorption was 6 times higher if the plants were watered aerially rather than ground watering. Using either watering method, >90% of the activity was absorbed by the stems and leaves (Sabbarese et al. 2002). Soil to plant transfer factors for ^{60}Co were determined for plants grown in containers with soil contaminated with ^{65}Zn and ^{60}Co over a 3-year period under outdoor tropical conditions. Average transfer factors for ^{60}Co over the 3-year period ranged from a high for spinach (1.030) to a low for rice (0.087) (Mollah and Begum 2001).

^{60}Co is taken up by phytoplankton and unicellular algae (*Senenastrum capricornutum*) with concentration factors (dry weight) ranging from 15,000 to 40,000 and 2,300 to 18,000, respectively (Corisco and Carreiro 1999). Elimination experiments with the algae indicate a two component biological half-life, 1 hour and 11 days, respectively, and suggest that the cobalt might be absorbed not only on the surface, but also intracellularly. Since these organisms are at the bottom of the food chain, they could play an important role in the trophic transfer of ^{60}Co released into waterways by nuclear facilities. However, cobalt levels generally diminish with increasing trophic levels in a food chain (Smith and Carson 1981).

The low levels of cobalt in fish may also reflect cobalt's strong binding to particles and sediment. The bioaccumulation factors (dry weight basis) for cobalt in marine and freshwater fish are ~100–4,000 and <10–1,000, respectively; accumulation in the muscle of marine fish is 5–500 (Smith and Carson 1981). Cobalt largely accumulates in the viscera and on the skin, as opposed to the edible parts of the fish. In carp, accumulation from water accounted for 75% of ^{60}Co accumulated from both water and food; accumulation from water and food was additive (Baudin and Fritsch 1989). Depuration half-lives were 53 and 87 days for fish contaminated from food and water, respectively. In the case of an accidental release of ^{60}Co into waterways, the implication is that effects would manifest themselves rapidly since the primary route of exposure is from water rather than food. Uptake of ^{60}Co by biota in lakes in northwestern Ontario was not affected by the tropic status of the lakes (Bird et al. 1998a). Uptake of ^{60}Co was very low in whitefish, with concentrations being highest in kidney and undetectable in muscle. Similarly, while accumulation of ^{60}Co by carp from food was dependent on food type, the transfer factor was very low, approximately 0.01, and no long-term bioaccumulation of the radionuclide occurred (Baudin and Fritsch 1987; Baudin et al. 1990). Accumulation of ^{60}Co from food for rainbow trout showed that after the 42-day exposure period, the highest concentrations of ^{60}Co were found in the kidneys, secondary gut, and viscera, and the trophic transfer factor was 0.0186. After 73 days of depuration, residual ^{60}Co concentrations were the highest in the kidneys, viscera, and fins (Baudin et al. 2000). In the experiment described above in which Bird et al. (1998a) added ^{60}Co to the anoxic

hypolimnion of a Canadian Shield lake to simulate a nuclear waste scenario where radionuclides entered the bottom waters of a lake, ^{60}Co levels in biota were low because of the rapid loss of cobalt to the sediment. Levels in forage fish, minnows, and sculpins were low, <0.3 Bq/g (8 pCi/g) dry weight; an occasional high level, ~4 Bq/g (110 pCi/g) dry weight, in slimy sculpin was thought to reflect the presence of detritus in the gut of the fish. Epilimnion additions of ^{60}Co in an earlier study resulted in lower maximum concentrations in fish, 0.07, 0.11, and 0.01 Bq/g (2, 3.0, and 0.3 pCi/g) dry weight in pearl dace, fathead minnows, and slimy sculpins, respectively, when similar quantities of radioactive cobalt were added to the lake.

Concentration factors have also been reported for various other aquatic organisms. Freshwater mollusks have concentration factors of 100–14,000 (~1–300 in soft tissue). Much of the cobalt taken up by mollusks and crustacae from water or sediment is adsorbed to the shell or exoskeleton; very little cobalt is generally accumulated in the edible parts (Amiard and Amiard-Triquet 1979; Smith and Carson 1981). A concentration factor for ^{60}Co of 265 mL/g (wet weight) was determined for *Daphnia magna* in laboratory studies. The rapid decrease in radioactivity during the depuration phase indicated that adsorption to the surface was the major contamination process (Adam et al. 2001). However, the digestive glands of crustaceans, which are sometimes eaten by humans, may accumulate high levels of ^{60}Co. Five different species of marine mollusks had whole-body ^{60}Co concentration factors between 6.3 and 84 after 1-month exposure to ^{60}Co in seawater (Carvalho 1987). The shell accounted for more than half of the body-burden. Among the soft tissue, the gills and viscera had the highest concentrations factors and the muscle had the lowest. Fisher et al. (1996) studied the release of ^{60}Co accumulated in mussels from water and ingested phytoplankton. In each case, there was a slow and fast component to the release; the rapid release was in the form of fecal pellets if uptake was from food and from desorption from the shell if uptake was from the dissolved phase. Biological half-lives obtained in laboratory studies were about 12–21 days from both the shell and soft parts. Higher absorption efficiencies and lower efflux rates were obtained for cobalamins than for inorganic cobalt, suggesting that it is a more bioavailable form of cobalt for mussels. Cobalt from fecal pellets is rapidly released into the overlying water and may play a role in its geochemical cycling (Fisher et al. 1996). The concentration of cobalt in clams in the Indian River Lagoon, Florida did not correlate with levels found in either water or sediment (Trocine and Trefry 1996). Kinetics of bioaccumulation of ^{57}Co from water and depuration by starfish (*Asterias rubens*) were carried out in laboratory studies. After 32 days of exposure to seawater containing ^{57}Co, whole body uptake from seawater reached a concentration factor of 23 (wet weight). ^{57}Co was released with a half-life of 27 days after removal to uncontaminated water. Comparison of the kinetics of loss of ^{57}Co following exposure to

^{57}Co-contaminated food versus exposure from ^{57}Co-contaminated water indicate that *A. rubens* accumulates ^{57}Co predominately from seawater rather than from food (Warnau et al. 1999).

6.3.2 Transformation and Degradation

6.3.2.1 Air

There is a paucity of data in the literature regarding the chemical forms of cobalt in air and their transformations in the atmosphere. It is generally assumed that anthropogenic cobalt originating from combustion sources exists primarily as the oxide (Schroeder et al. 1987). In addition, cobalt may be released into the atmosphere as its arsenide or sulfide during ore extraction processes. It is not clear if these species are transformed in the atmosphere. Should a relatively insoluble species such as the oxide be transformed into a more soluble form such as the sulfate, one would expect greater quantities to be washed out of the atmosphere in rain.

6.3.2.2 Water

Many factors control the speciation and fate of cobalt in natural waters and sediments. These include the presence of organic ligands (e.g., humic acids, EDTA), the presence and concentration of anions (Cl^-, OH^-, CO_3^{-2}, HCO_3^-, SO_4^{-2}), pH, and redox potential (Eh). Modeling the chemical speciation of a metal in water depends upon the environmental factors assumed and the stability constants of the various complexes. Mantoura et al. (1978) predicted the equilibrium levels of Co^{2+} species in fresh water to follow the order: free $Co^{+2} \geq CoCO_3 > CoHCO_3^+ >> CoSO_4 \geq$ Co•humic acid. However, the mole percent of various cobalt species in a Welsh lake was found to be: free Co^{+2}, 76%; $CoCO_3$, 9.8%; $CoHCO_3^+$, 9.6%; humate complexes, 4.0%; and $CoSO_4$, 0.4%. The rank order of species concentration in seawater was estimated to be: $CoCO_3$>free Co^{+2}>$CoSO_4 \geq CoHCO_3^+$. In another model, the speciation of cobalt was completely different with $CoCl^+$>free Co^{+2}>$CoCO_3$>$CoSO_4$ (Smith and Carson 1981). More recently, Tipping et al. (1998) estimated the equilibrium speciation of cobalt in riverine, estuarine, and marine surface water of the Humber system (England). In all but seawater, cobalt complexes with carbonate (HCO_3^- and CO_3^{2-}) constituted about 70% of dissolved cobalt while the free Co^{2+} ion, was a major

species, ~25%, which is much lower than the 61% predicted by Mantoura et al. (1978). As the alkalinity of the water increases, the proportion of cobalt complexed with carbonate increases at the expense of free Co^{2+}. The proportion, but not the concentration, of cobalt that exists as the free ion and the carbonate complexes in river water is independent of the level of fulvic acid in the water. In seawater, the carbonate species and the free aqua species assume roughly equal importance. The proportion of dissolved cobalt complexed with fulvic acid decreased with increasing salinity. About 20% of cobalt in seawater was estimated to be present as complexes with sulfate. In a bioconcentration study in which $CoCl_2$ was initially added to the seawater, at month's end, the cationic form of cobalt was progressively converted into anionic and neutral forms, possibly as a result of complexation with organic ligands (Carvalho 1987). Addition of humic acid to natural waters may merely increase the concentration of colloidal dispersed metal rather than form truly soluble humic complexes. In water that contains high organic wastes such as was the case in the Rhone River in France, cobalt was almost completely complexed. A recent study determined that the distribution of ^{60}Co in the Rhone River sampled at Arles, France was 45% in the particulate phase, 30% in the dissolved phase, and 25% in the colloidal phase (Eyrolle and Charmasson 2001). Cobalt forms complexes with EDTA that are very stable environmentally. EDTA is often used in agriculture, food and drug processing, photography, and textile and paper manufacturing, and therefore, it is a likely constituent of industrial discharges.

Acidity and redox potential have an effect on the behavior of cobalt in water. The adsorption of cobalt by particulate matter decreases with decreasing pH, since the increasing H^+ concentration competes with metal binding sites. This may lead to increased concentrations of dissolved cobalt at low pH. The effect of Eh (redox potential) on the speciation of cobalt has been shown by the increase in the concentration of dissolved cobalt by orders of magnitude with increasing depth in certain parts of Baltic waters. The increase in the concentration of dissolved cobalt may be due to the formation of soluble bisulfide and polysulfide complexes in the anoxic zones. The residence time of soluble cobalt in seawater has been estimated to range from <1 to 52 years (Brugmann 1988; Knauer et al. 1982; Smith and Carson 1981).

Vitamin B_{12}, which contains cobalt, is synthesized by 58 species of seven genuses of bacteria as well as blue-green algae and actinomycetes (mold-like bacteria). Consequently, vitamin B_{12} levels in marine water range from very low levels in some open ocean water to much higher levels in some coastal waters. Freshwater environments have comparable levels of vitamin B_{12}. The high level of cobalamins in coastal

water appears to be related to the occurrence of macrophytes in these areas with their high concentrations of vitamin B_{12}. Cobalamins are released into the water when the organisms die (Smith and Carson 1981).

Alkaline thermal groundwater in granitic areas have been studied as possible waste disposal sites for radioactive waste (Alaux-Negrel et al. 1993). Water in these areas is characterized by high pH, low CO_2 partial pressure, and generally low redox potential; sulfide concentrations are in the range of 10^{-4} to 10^{-3} mol/L. The solubility of cobalt is controlled by the solubility of CoS (log K_1 and log K_2 being 5.7 and 8.7 at 25°C) and therefore, levels of cobalt are very low, 10^{-8}–10^{-10} mol/L.

The ^{60}Co (III) picolinate complex that is released into water by some nuclear reactors does not break down immediately on release into seawater, but rather can coexist with the ^{60}Co (II) forms for lengthy periods in the environment (Leonard et al. 1993a, 1993b). Studies indicate that several processes occur to the Co(III) organic complexes, including reduction to the inorganic form, sorption of both species to particulate matter, and transformations of the uncomplexed species. It is possible that this more soluble and uncharged form of radioactive cobalt will increase the dispersion of ^{60}Co from its point of discharge.

6.3.2.3 Sediment and Soil

The speciation of cobalt in soil or sediment depends on the nature of the soil or sediment, concentration of chelating/complexing agents, pH, and redox potential (Eh) of the soil. Dissolved cobalt may be absorbed by ion exchange and other mechanisms, or may form complexes with fulvic acids, humic acid, or other organic ligands in soil. The humic and fulvic complexes of cobalt are not very stable compared with those of copper, lead, iron, and nickel. The speciation of cobalt in sediment from nine sites in the Red Sea, a sea that is unique in that it has no permanent streams flowing into it, was assessed using a sequential extraction technique (Hanna 1992). The mean percentages contained in the various fractions were: exchangeable, 5.5%; carbonate, 5%; Fe/Mn oxides, 24%; organic, 30.4%; sulfides, 13%; and lithogenous, 22%. While the mean concentration of cobalt in the sediment increased from 0.003 to 0.006 ppb between 1934 and 1984, its distribution among the different phases did not change appreciably.

The reduction of soil Eh, which may occur when soil is flooded or in deeper layers of soil that are oxygen-depleted, may change the speciation of cobalt. This may result in the reduction of soil iron and manganese and the subsequent release of adsorbed cobalt from the mineral oxides. Similarly, a decrease

in soil pH may result in the solubilization of precipitated cobalt and desorption of sorbed cobalt, resulting in increased cobalt mobility (Smith and Carson 1981). Co^{2+} may also be oxidized to Co^{3+} by manganese oxides, a common component of soils and aquifer material, with subsequent surface precipitation (Brusseau and Zachara 1993). This process may affect transport of cobalt in the subsurface environment.

EDTA complexes of cobalt are very stable and are likely to form in soils containing EDTA. EDTA is widely used as a decontaminating agent at nuclear facilities. Although cobalt-EDTA complexes are adsorbed by some soils, the mobility of cobalt in soil may increase as a result of complex formation (Schnitzer 1969; Smith and Carson 1981; Swanson 1984). ^{60}Co that is disposed of in shallow land trenches have sometimes been found to migrate more rapidly than expected from the disposal sites. Organic chelating agents are frequently present at these sites and would possibly increase the solubility and transport of the radionuclide.

Bacterial action can affect the mobility of a substance by mediating reactions or by participating in reactions that lower the pH. Another way of influencing radionuclide mobility is by degrading complexing agents used in cleaning reactors (e.g., citric acid), thereby releasing the radionuclide. However, experiments on the fate and transport of cobalt released upon the biodegradation of the complexing ligand indicate that results are not always predictable; the means of ligand removal and the geochemical environment are important factors that must be considered (Brooks et al. 1998).

6.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

Cobalt concentrations in environmental media, including food and human tissue, have been exhaustively tabulated by Smith and Carson (1981) and Young (1979). The International Agency for Research on Cancer (IARC 1991) contains reviews of more recent studies, but is primarily focused on occupational exposures and body burdens of cobalt.

6.4.1 Air

Atmospheric cobalt is associated with particulate matter. Mean cobalt levels in air at unpolluted sites are generally <1–2 ng/m^3 (Hamilton 1994; Smith and Carson 1981). At the South Pole, cobalt levels of 0.00049±0.00015 ng/m^3 were recorded in 1974–1975 (Maenhaut et al. 1979). Geometric mean cobalt

levels in several open-ocean environments ranged from 0.0004 to 0.08 ng/m^3 (Chester et al. 1991). The average annual PM-10 (particles with diameters ≤10 μm) cobalt concentration at Nahant, Massachusetts (near Boston) in 1992–1993 was 1.7 ng/m^3 (Golomb et al. 1997). Half of the cobalt was contained in fine particles (<2.5 μm) and half in coarse particles (2.5–10 μm). The mean cobalt level in southern Norway in 1985–1986 (n=346) was 0.10 ng/m^3 with 35% of the samples falling below the detection limit of 0.04 ng/m^3 (Amundsen et al. 1992). Atmospheric cobalt levels in industrial settings may exceed 10 ng/m^3. The highest recorded average cobalt concentration in air was 48 ng/m^3 in Clydach, Wales at the site, where nickel and cobalt were refined (Smith and Carson 1981). Some ambient atmospheric levels of cobalt are given in Table 6-2. These data show the contribution of anthropogenic sources in increasing the level of cobalt in the ambient air. Typical occupational cobalt levels are 1.0×10^4–1.7×10^6 ng/m^3 (Barceloux 1999; IARC 1991). While ^{60}Co has been detected in air samples at the Hanford site and Oak Ridge National Laboratories, levels were not reported (HazDat 2004; PNNL 1996). In 1995, the concentration of ^{60}Co in air at the Hanford site was below the detection limit in over 88% of the air samples.

6.4.2 Water

The concentrations of cobalt in surface water and groundwater in the United States are generally low, <1 μg/L in pristine areas and 1–10 μg/L in populated areas (Hamilton 1994; Smith and Carson 1981). However, cobalt levels may be considerably higher in mining or agricultural areas. Levels as high as

4,500 μg/L were reported in Mineral Creek, Arizona, near a copper mine and smelter; levels of 6,500 μg/L were reported in the Little St. Francis River, which receives effluent from cobalt mining and milling operations (Smith and Carson 1981). Mining at Blackbird Mine in Idaho, one of the large deposits of cobalt in North America, occurred from the late 1800s to 1982. Cobalt concentration in surface water and groundwater samples collected in 1992 from area creeks near this mine were reported to range from <1 to 625,000 μg/L, and from not detected to 315,000 μg/L respectively (ATSDR 1995), Eckel and Jacob (1988) analyzed U.S. Geological Survey (USGS) data for 6,805 ambient surface water stations and estimated the geometric mean and median dissolved cobalt concentration as 2.9 and 2.0 μg/L, respectively. Mean cobalt levels reported in seawater range from 0.078 μg/L in the Caribbean Sea to 0.39 μg/L in the Indian Ocean (Hamilton 1994). Vitamin B_{12} is synthesized by bacteria, macrophytes,

Table 6-2. Concentration of Cobalt in the Atmosphere

Location	Possible source/activity	Concentration[a]	Units	Type	Reference
Ambient levels—remote					
South Pole, 1974–1975	Crustal material	0.00049±0.00015	ng/m^3	Mean±SD	Maenhaut et al. 1979
Open-ocean		0.0004–0.08	ng/m^3	Geomean range	Chester et al. 1991
North Atlantic		0.006–0.09	ng/m^3	Range	Smith and Carson 1981
Baltic Sea, 1983		0.09, 0.01–0.43	ng/m^3	Mean, range	Hasanen etal. 1990
Remote sites		0.001–0.9	ng/m^3	Range	Schroeder et al. 1987
Ambient levels—rural/suburban/urban					
Rural sites		0.08–10.1	ng/m^3	Range	Schroeder et al. 1987
Massachusetts, Nahant, 1992–1993		1.7	ng/m^3	Annual mean	Golomb et al. 1997
Urban sites					Schroeder et al. 1987
United States		0.2–83	ng/m^3	Range	
Canada		1–7.9			
Europe		0.4–18.3			
Texas state average (1978–1982)		2.0	ng/m^3	Mean	Wiersema et al. 1984
Illinois, urban air (<2.5 μm; 2.5–10 μm)					Sweet et al. 1993
Bondville, Ill (rural)	Background	0.2; 0.1	ng/m^3	Mean (fine; coarse)	
Southeast Chicago	Steel mills	0.4; 0.4			
East St. Louis	Smelters	0.5; 0.4			
Washington, DC (1974)	Urban area	1.1	ng/m^3	Mean	Smith and Carson 1981
Ambient levels—industrial					
Maryland, Baltimore Harbor Tunnel (1973–1974)					Ondov et al. 1982
Air outside	Vehicular exhaust	0.8–1.9	ng/m^3	Range	
Air inside	Vehicular exhaust	2.2–5.3			
Ohio, Cleveland	Be-Cu alloy and other industrial activities	610	ng/m^3	Maximum	Smith and Carson 1981
Texas, El Paso (1978–1982)	Industrial	127	ng/m^3	Maximum	Wiersema et al. 1984

Table 6-2. Concentration of Cobalt in the Atmosphere

Location	Possible source/activity	Concentration[a]	Units	Type	Reference
Texas, Houston (1978–1982)	Urban area	81	ng/m^3	Maximum	Wiersema et al. 1984
Arizona, Tucson					Smith and Carson 1981
Urban	Copper smelting	1.9	ng/m^3	Mean	
Rural		0.7			
Maryland, Chalk Point Generator	Coal-burning power plant	3.86	ng/m^3	Mean	Smith and Carson 1981
Wales, Clydach	Nickel refining	48, 3–300	ng/m^3	Mean, range	Smith and Carson 1981
Wales, Llausamlet and Trebanos	Towns near Clydach	3.8		Mean	Smith and Carson 1981
Occupational air levels					
Northern Italy, exposure survey, 1991, area monitoring (n=259)	Diamond abrasive mfg.				Mosconi et al. 1994a
	Mould-filling	220, 47–960	ng/m^3	Median, range	
	Sintering	101.5, 32–240			
	Grinding	22, 15–45			
	Mechanical-working	20, 12–44			
	Grinding	5, 2.5–94			
	Tool production	6, 5–47			
	Hard metal alloy filing	2, 0.8–3			
	Other	2.7, 2.3–15			
Northern Italy, exposure survey, 1991, personal sampling (n=259)	Diamond abrasive mfg.				Mosconi et al. 1994a
	Mould-filling	382, 76–2,600	ng/m^3	Median, range	
	Sintering	309, 238–413			
	Grinding	230, 82–690			
	Mechanical-working	40, 7.1–65			
	Grinding	9.3, 1.5–178			
	Tool production	17, 4–28			
	Hard metal alloy filling	5, 1–107			
	Other	50, 10–290			

Table 6-2. Concentration of Cobalt in the Atmosphere

Location	Possible source/activity	Concentration[a]	Units	Type	Reference
Japan, personal sampling, hard metal tool manufacture, 8-hour TWA, 356 workers (n=935)	Powder preparation				Kumagai et al. 1996
	Rotation	459, 7–6,390	$\mu g/m^3$	Mean, range	
	Full-time	147, 26–378			
	Press				
	Rubber	339, 48–2,910			
	Steel	47, 6–248			
	Shaping	97, 4–1,160			
	Sintering	24, 1–145			
	Blasting	2, 1–4			
	Electron discharging	3, 1–23			
	Grinding	45, 1–482			

geomean = geometric mean; SD = standard deviation; TWA = time weighted average

blue-green algae, and actinomycetes, and cobalt levels in oceans often correlate with biological productivity. In the Baltic Sea, dissolved cobalt levels that are 1.0 ng/L near the surface, increase to 71.0 ng/L at a depth of 200 m (Brügmann 1988). The rise in dissolved cobalt is coincident with the onset of anoxic conditions and the presence of hydrogen sulfide, indicating that soluble bisulfide and polysulfide complexes may be present. Some cobalt levels reported in water are given in Table 6-3.

In a 1962–1967 survey, cobalt was detected in 2.8% of 1,577 U.S. raw surface waters from which drinking water is derived; the detection limit was 1 μg/L and the maximum concentration was 48 μg/L (NAS 1977). Of 380 U.S. finished drinking waters, only 0.5% contained cobalt levels exceeding 1 μg/L; the maximum concentration found was 29 μg/L (NAS 1977). These values are higher than the respective median and maximum levels of <2.0 and 6.0 μg/L found in Canadian finished drinking water (Meranger et al. 1981). Meranger et al. (1981) tested source water and drinking water in 71 municipalities across Canada and concluded that, in general, both surface water and groundwater used for drinking water supplies contain negligible amounts of cobalt. Greathouse and Craun (1978) analyzed 3,834 grab samples of household tap water from 35 geographical areas in the United States for 28 trace elements. Cobalt was found in 9.8% of the samples at concentrations ranging from 2.6 to 107 μg/L. It is not clear whether these higher levels could indicate that cobalt was picked up in the distribution system. In the earlier National Community Water Supply Study (2,500 samples), 62% of the samples contained <1 μg Co/L; the average and maximum cobalt concentrations were 2.2 and 19 μg/L, respectively (Smith and Carson 1981). Cobalt was not detected (detection limit 8 μg/L) in a 1982–1983 survey of drinking water in Norway that covered 384 waterworks serving 70.9% of the Norwegian population (Flaten 1991).

The mean concentrations of cobalt in rain is around 0.03–1.7 μg/L, with levels generally ranging from 0.002 μg/L at Enewetak Atoll to about 2.9 μg/L in the Swansea Valley, Wales (Arimoto et al. 1985; Dasch and Wolff 1989; Hansson et al. 1988; Heaton et al. 1990; Helmers and Schrems 1995; Nimmo and Chester 1993; Nimmo and Fones 1997; Smith and Carson 1981). The highest recorded level of cobalt in precipitation was 68.9 μg/L in the vicinity of a nickel smelter in Monchegorsk in the Russian Arctic (Reimann et al. 1997). An analysis of rain in the Mediterranean and urban and coastal sites in northwest England showed that about 33–44% of the cobalt occurred as very stable dissolved organic complexes (Nimmo and Chester 1993; Nimmo and Fones 1997).

As it was pointed out in Section 6.3.2.2, ^{60}Co discharged from the Steam Generating Heavy Water Reactor at Winfrith on the south coast of England was shown to be largely in the form of the nonionic

Table 6-3. Cobalt Levels in Water

Nature/location of water	Level	Units	Type	Reference
Sea water				
Florida (Indian River Lagoon) (43 sites)	0.031, 50	μg/L	Mean, range	Trocine and Trefry 1996
California (Baja) 2–45 km offshore (n=11)	0.022–0.17	nM	Range	Sañudo-Wilhelmy and Flegal 1996
<100 m off shore (n=11)	0.11–0.59			
Agean Sea, 1994; 8 sites (dissolved)	0.168–0.632, 1.917	nM	Range of means, maximum	Voutsinou-Taliadouri 1997
Baltic Sea (Gotland Deep site)				Brügmann 1988
10 m	1.0	ng/L	Mean (dissolved Co)	
50 m	1.0			
100 m	3.5			
150 m	4.2			
200 m (anoxic)	71.0			
235 m (anoxic)	49.2			
Seawater background	0.04	μg/L		Bargagli 2000
Seawater	0.27	μg/L	Mean	Abbasi et al. 1989
Fresh surface water				
Freshwater background	0.05	μg/L		Bargagli 2000
U.S. ambient surface water (6,805 stations)	<2.9, 2.0	μg/L	Mean, median	Eckel and Jacob 1988
Five Great Lakes waters	ND–0.09	μg/L	Range	Rossmann and Barres 1988
Japan, unpolluted lake	<0.004	μg/L		Nojiri et al. 1985
Norway, 11 rivers	0.94	μg/L	Maximum	Flaten 1991
Streams near populated areas	1–10	μg/L	Range	Smith and Carson 1981
Streams in agricultural and mining areas	11–50	μg/L	Range	Smith and Carson 1981
Suspended solids in rivers	7–94	mg/kg	Range	Smith and Carson 1981
Groundwater				
Canada (Chalk River nuclear waste site)	0.0001–0.002	μg/L		Cassidy et al. 1982
Colorado (Denver)–shallow groundwater, (n=30)	<1 (<1–9)	μg/L	Median, range	Bruce and McMahon 1996

Table 6-3. Cobalt Levels in Water

Nature/location of water	Level	Units	Type	Reference
Drinking water				
Canadian drinking water (71 municipalities)				Meranger et al. 1981
Raw:	<2.0	μg/L	Median	
Treated:	<2.0			
Distributed:	≤2.0			
Precipitation				
Massachusetts, 1984 (12 events)	0.045 (0.008), 0.02–0.12	μg/L	Mean (SD), range	Dasch and Wolff 1989
Rhode Island (rain/snow), 1985 (n=269)	0.038 (0.067)	ppb	Median (mean)	Heaton et al. 1990
	0.001–0.80		Range	
Western Mediterranean, 1988–1989				Nimmo and Chester 1993
Total cobalt	0.029–0.134, 0.043	μg/L	Range, mean	
Labile cobalt	0.009–0.104, 0.025			
Organic cobalt	ND–0.613, 0.019			
Arctic (7 sites in Finland, Norway, Russia)	<0.02–1.07, 3.32	μg/L	Median range, maximum	Reimann et al. 1997
Russia (Monchegorsk), nickel smelter	11.8, 68.9		Median, maximum	

ND = not detected; SD = standard deviation

trivalent complex, ^{60}Co(III) picolinate. The ^{60}Co(III) species is not immediately reduced to the more particle-reactive divalent form, and both oxidation states may coexist for long periods of time in the environment. The proportion of the more soluble and mobile ^{60}Co(III) would be expected to increase with time and distance from the point of discharge. Shoreline water samples (n=22) taken in 1987–1988 at two locations in the vicinity of the discharge from the Steam Generating Heavy Water Reactor at Winfrith contained 0.3–16.2 mBq/L (8–437 fCi/L) of particulate ^{60}Co, 2.8–44.4 mBq/L (76–1,200 fCi/L) of soluble ^{60}Co(II), and 0.2–4.8 mBq/L (5–130 fCi/L) of soluble ^{60}Co(III) (Leonard et al. 1993). The percent of the soluble ^{60}Co present as Co(III) ranged from 4.3 to 18.6%. In 1989, in conjunction with the largest discharge of effluent from the plant, offshore seawater samples from 18 sites contained 0.06–2.22 mBq/L (2–60 fCi/L) of particulate ^{60}Co, 0.30–10.3 mBq/L (8.1–278 fCi/L) of soluble ^{60}Co(II), and 0.12–1.55 mBq/L (3.2–41.9 fCi/L) of soluble ^{60}Co(III). The percent of the soluble ^{60}Co present as Co(III) ranged from 6.0 to 28.6%.

6.4.3 Sediment and Soil

Cobalt is the 33rd most abundant element in the earth's crust. Its average concentrations in the earth's crust and in igneous rocks are 20–25 and 18 mg/kg, respectively (Abbasi et al. 1989; Merian 1985; Smith and Carson 1981). Trace metals in soils may originate from parent rock or from anthropogenic sources, primarily fertilizers, pesticides, and herbicides. Most soils contain 1–40 mg cobalt/kg. The average cobalt concentration in U.S. soils is 7.2 mg/kg (Smith and Carson 1981). Soils containing <0.5–3 mg cobalt/kg are considered cobalt-deficient because plants growing on them have insufficient cobalt (<0.08–0.1 mg/kg) to meet the dietary requirements of cattle and sheep. Cobalt-deficient soils include the humus podzols of the southeastern United States, and the podzols, brown podzolic soils, and humus groundwater podzols in the northeastern parts of the United States. (Podzols are generally coarse-textured soils.) The cobalt content of surface soils from 13 sites in the brown and dark brown soil zones of southwestern Saskatchewan ranged from 3.7 to 16.0 mg/kg and only in one case was the soil appreciably elevated above the corresponding parent material (Mermut et al. 1996). Fertilizers used in this agricultural area contained 0.12–102 mg Co/kg, with a median of 5.7 mg/kg.

Mean cobalt concentrations in surface soil from nine sites on two active volcanic islands off of Sicily ranged from 5.1 to 59.0 mg/kg (Bargagli et al. 1991). Soils near ore deposits, phosphate rocks, or ore

smelting facilities, and soils contaminated by airport traffic, highway traffic, or other industrial pollution may contain much higher concentrations of cobalt; concentrations up to 800 mg/kg have been detected in such areas (Kloke et al. 1984; Smith and Carson 1981). Cobalt concentrations from 28 samples collected from surface deposits in the Big Deer and Blackbird Creek drainage basins in Idaho near the Blackbird Mine ranged from 26.5 to 7,410 mg/kg (Agency for Toxic Substances and Disease Registry 1995). Soils around the large copper-nickel smelters in Sudbury, Ontario have been shown to contain high levels of cobalt. Fifty kilometers from the smelters, cobalt levels in surface soil were 19 mg/kg. These levels increased to 48 mg/kg at 19 km, 33 mg/kg at 10 km, and 42–154 mg/kg between 0.8 and 1.3 km from the smelter (Smith and Carson 1981). Soils around a cemented tungsten carbide tool grinding factory contained cobalt levels as high as 12,700 mg/kg, almost 2,000 times the average in U.S. soils (Abraham and Hunt 1995). However, neighborhood soils between 30 and 160 meters from the factory only contained 12–18 mg Co/kg.

Unpolluted freshwater sediment contains about the same levels of cobalt as does cobalt-sufficient soil, generally <20 mg/kg (Smith and Carson 1981). In the Hudson River Estuary, cobalt levels in suspended sediment were an order of magnitude higher than in bottom sediment (Gibbs 1994). This can be attributed to the finer grain size of suspended sediment or local sources. Cobalt levels in core samples (surface to 42 cm deep) from the Upper St. Lawrence Estuary were independent of depth, indicating the lack of any recent significant anthropogenic releases (Coakley et al. 1993). Cobalt levels in sediment are shown in Table 6-4.

No broad-based monitoring studies of ^{60}Co or other radioactive cobalt isotopes in soil or sediment were found in the literature. Soil samples from the O-horizon taken from three sites in the 30-km zone around Chernobyl in 1992 and again in 1993 contained 14–290 and 4.5–245 Bq/kg (380–7,800 and 120–6,620 pCi/kg) dry weight of ^{60}Co, respectively (Lux et al. 1995). The Columbia River receives radiological contaminants along the Hanford Reach primarily through seepage of contaminated groundwater. The regional median concentration of ^{60}Co in sediment was highest along the Hanford reach, approximately 0.09 pCi/g (0.003 Bq/g) (PNNL 1996). ^{60}Co activity in a sediment cores in water off of Southampton in southern England contained up to 28 Bq/kg (760 pCi/kg) in the upper 3 cm; no activity was found below 12.5 cm (Croudace and Cundy 1995). Discharges of treated effluent occurred on closing a steam generating heavy water reactor west of where the sampling was done. The maximum discharge occurred in 1980–1981; however, no value was reported (Croudace and Cundy 1995).

Table 6-4. Cobalt Levels in Sediment

Nature/location of sediment	Level	Units	Type	Reference
Freshwater				
Polluted lakes and rivers	0.16–133	mg/kg	Range	Smith and Carson 1981
Lake Ontario near Miesissaqua, Canada	4.1–19.8	mg/kg	Range	Glooschenko et al. 1981
Hudson River, Foundry Cove, 1983, Ni-Cd battery plant, 1953–1979, surficial (0–5 cm) sediment, 16 sites	18–700	mg/kg	Range	Knutson et al. 1987
Estuaries and Marine				
Hudson River Estuary (0–80 km from ocean), 1991				Gibbs 1994
Bottom sediment	1–13	mg/kg	Range	
Suspended sediment (near surface)	30–140			
Upper St. Lawrence Estuary, 1989–1990				Coakley et al. 1993
Core C168	3.1 (0.6)	mg/kg	Mean (SD)	
Cores LE and LO	2.7 (0.5)			
Massachusetts, New Bedford Harbor- core (0–25 cm)				Shine et al. 1995
Outer Harbor	7.03, 3.64–9.79, range			
Inner Harbor	6.38, 2.62–10.52			
Buzzards Bay (control site)	4.76, 1.64–8.19			
Indian River Lagoon, Florida (43 sites)	2.3, 0.4–6.3	mg/kg	Mean, range	Trocine and Trefry 1996
Gulf of Mexico				Villanueva and Botello 1998
Coastal areas (11 sites)	12.30–36.26	mg/kg	Range of means	
Continental shelf (3 sites)	6.39–21.00			
Antarctica (Ross Sea) continental shelf (n=12)	19, 0.10–13	mg/kg	Mean, range	Bargagli 2000
Northern Arctic Alaska, continental shelf (n=136)	9, 3.3–18	mg/kg	Mean, range	Bargagli 2000
Chukchi Sea, northeast Alaska (31 stations, surficial sediment)	32.7, 19–74	mg/kg	Mean, range	Naidu et al. 1997
Baltic Sea, southern, off Poland (surficial sediment)	0.69–18.10	mg/kg	Range	Szefer et al. 1996
Baltic Sea (Gotland Deep site)	19, 11–33	mg/kg	Mean, range	Brügmann 1988

SD = standard deviation

Sediment samples were analyzed from the Peconic River system on Long Island, New York, downstream of Brookhaven National Laboratory (BNL). Near the sewage treatment plant, closest to the BNL, mean concentrations of ^{60}Co from three locations at the depth intervals 0.00–0.06, 0.06–0.15, 0.15–0.24, and 0.24–0.37 meters were 9.6, 6.7, 9.6, and 10.5 Bq/kg (0.25, 0.18, 0.25, and 0.28 nCi/kg) dry weight, respectively. At one location at the BNL property boundary, mean concentrations of ^{60}Co, using the same depth intervals, were 5.8 Bq/kg (0.16 nCi/kg) dry weight for the 0.00–0.06 m depth and <4 Bq/kg (<0.11 nCi/kg) dry weight for the remaining depth intervals. Sediment samples from a control river, Connetquot River, were <4 Bq/kg (<0.11 nCi/kg) in two locations at two depths (0.00–0.06 and 0.06–0.15 m) (Rapiejko et al. 2001).

Mururoa and Fangataufa Atolls were used for underground testing of nuclear weapons from 1975 to 1996. ^{60}Co was detected in the particle fraction of water in measurable levels at two of the nine Mururoa Atoll sites, Aristee and Ceto, at 0.58 and 1.06 mBq/L (0.016 and 0.029 pCi/L), respectively. ^{60}Co levels were found at levels below the detection limit, <0.1 mBq/L (<0.003 pCi/L), at the two Fangataufa Atoll sites and at the seven other Muruoa Atoll sites (Mulsow et al. 1999). Concentrations of ^{60}Co of soil samples used for growing onion, potatoes, tomatoes, cabbage, and maize in the Bulgarian village, Ostrov, in the vicinity (approximately 25–30 km) of the "Kozloduy" nuclear power plant were <8, 3, 320, 330, and 180 mBq/kg (2, 8.1, 8.6, 8.9, and 4.9 pCi/kg), respectively (Djingova and Kuleff 2002).

6.4.4 Other Environmental Media

The cobalt content of plants depends on the plant, the cobalt content of the soil, and numerous environmental factors. The mean cobalt concentration reported for terrestrial plants was 0.48 μg/g, while the mean and median levels for freshwater vascular plants were 0.48 and 0.32 μg/g, respectively (Outridge and Noller 1991). The median cobalt level in freshwater vascular plants from polluted waters was about the same as in unpolluted waters, 0.37 μg/g, although extremely high levels of cobalt, up to 860 μg/g, was reported in one species, *Myriophyllum verticillatum*, from central Ontario lakes. Grasses normally contain 0.2–0.35 μg/g of cobalt, but grasses from cobalt-deficient regions contain only 0.02–0.06 μg/g of cobalt (Hamilton 1994). Durum wheat grown in southeastern Saskatchewan contained 0.01–0.02 mg/kg dry weight (Mermut et al. 1996). In view of the cobalt content of the soil and the fact that almost half of the cobalt in fertilizers used in the area was in a readily available form, the uptake of cobalt by wheat was negligible.

^{60}Co levels in plants and mushrooms in the 30-km zone around Chernobyl were mostly below the detection limit in samples obtained in 1992 and 1993; the highest activity recorded was 3.9 Bq/kg (110 pCi/kg) dry weight in *Athyrium filix femina* (Lux et al. 1995).

Cobalt concentrations have been reported in various aquatic animals and seabirds. Eel and a freshwater fish from three Dutch polder lakes contained 2.5–25.0 and 2.50–5.63 mg cobalt/kg wet weight, respectively, (Badsha and Goldspink 1988). Muscle tissue of ocean fish and rock crabs caught near dump sites off New York City, New Haven, Connecticut, and Delaware Bay contained 10–40 and 16.0 μg/kg, respectively (Greig and Jones 1976). In a study of the levels and distribution of 14 elements in oceanic seabirds, the concentration of cobalt, an essential element, appeared to be highly regulated, with over 80% of the body burden residing in the skeleton. The mean cobalt concentration in the livers of 11 seabird species ranged from 0.048 to 0.078 μg/g dry weight, and cobalt had the lowest coefficient of variation in the different species of the elements studied (Kim et al. 1998a). In another study in Antarctica, mean cobalt levels in fish and amphipods were 0.11–0.14 and 1.01 μg/g dry weight, respectively, while those in the tissue of penguin and other sea birds ranged from 0.09 to 0.11 μg/g (Szefer et al. 1993). The concentration of cobalt in the tissue of 14 bluefin tuna caught by various commercial fishing vessels off Newfoundland was essentially the same, 0.01±0.004 μg/g (Hellou et al. 1992a). Similarly, in a broad survey of contaminant levels in nine species of fish and fiddler crabs from 11 sites in the lower Savannah River, Georgia and the Savannah National Wildlife Refuge, mean cobalt levels among different species and sites were statistically indistinguishable (Winger et al. 1990). These and other studies indicate that cobalt does not biomagnify up the food chain (Smith and Carson 1981). While high levels of cobalt were found in sediment from the Tigris River in Turkey and low levels in the water, cobalt was not detected in two species of fish, *Cyprinion macrostomus* and *Garra rufa* (Gümgüm et al. 1994). Cobalt was detected in two other species of fish collect between 1995 and 1996 in the upper Sakarya river basin, Turkey. Cobalt concentrations ranged from 0.038 to 0.154 μg/g dry weight for *Cyprinus caprio* and from 0.045 to 0.062 μg/g dry weight for *Barbus plebejus* (Barlas 1999).

^{60}Co was not detected in fish and mussel samples analyzed from the Peconic River system on Long Island, New York, downstream of the BNL. The lower detection limit for ^{60}Co was 0.4 Bq/kg (10 pCi/kg). ^{60}Co had been detected in sediment samples from this area (Section 6.4.3) (Rapiejko et al. 2001).

Some female birds sequester metals into their eggs under certain conditions, a phenomenon that may jeopardize the developing embryos. The geometric mean concentrations of cobalt in tern eggs collected from coastal New Jersey in 1971 and 1982 were 0.48 and 0.50 mg/kg, respectively. Unlike the levels of seven other common metals (e.g., mercury, cadmium, copper, lead, manganese, nickel, and zinc), the level of cobalt in tern eggs (and in the environment) showed no decline over the 11-year period (Burger and Gochfeld 1988).

Table 6-5 shows the levels of cobalt in food items and food categories from different countries. The level of cobalt in most Canadian foods was low; items with the highest concentrations in this study were waffles (0.076 μg/g), corn cereal (0.074 μg/g), and potato chips (0.070 μg/g) (Dabeka and McKenzie 1995). Green leafy vegetables and fresh cereals are the richest sources of cobalt (0.2–0.6 μg/g dry weight), while dairy products, refined cereals, and sugar contain the least cobalt (0.1–0.3 μg/g dry weight) (Barceloux 1999). The levels of cobalt were determined in 50 different food items, mainly meat, fish, fruit, vegetables, pulses, and cereals on the Swedish market during the years 1983–1990 (Jorhem and Sundström 1993). Beef liver and seeds were fairly high in cobalt and fish, fruit, and root and leafy vegetables were under 0.01 μg cobalt/g fresh weight. The cobalt levels in μg/g fresh weight were highest in alfalfa seeds, 0.86; linseed, 0.56; milk chocolate, 0.34; dark chocolate, 0.24; white poppy seeds, 0.30; blue poppy seeds, 0.15; soya beans, 0.084; green lentils, 0.054; and beef liver, 0.043. The cobalt content of 20 brands of alcoholic and nonalcoholic beer widely consumed in Spain ranged from 0.16 to 0.56 μg/L with a median of 0.39 μg/L (Cameán et al. 1998). Cobalt, which was at one time added to beer to increase the foam head, has been associated with cardiomyopathies (heart disease) in heavy beer drinkers.

A study of radionuclide levels in various foods and drinks in Hong Kong found that the ^{60}Co content in nearly all foods and drinks used in the study were below the minimal detection limit (Yu and Mao 1999). Analysis of wild plants in Bulgaria in villages near the "Kozloduy" nuclear power plant showed that the concentrations of ^{60}Co were below the detection limit. Mean activity concentrations of ^{60}Co in edible plants in this region were mostly <0.04 Bg/kg (<1 pCi/kg) (Djingova and Kuleff 2002).

Stable cobalt is present in various consumer products including cleaners, detergents, and soaps, which have resulted in dermatitis in sensitive individuals (Kokelj et al. 1994; Vilaplana et al. 1987). Tobacco contains about <0.3–2.3 μg Co/g dry weight and approximately 0.5% of the cobalt appears in mainstream smoke (Barceloux 1999; Munita and Mazzilli 1986; Ostapczuk et al. 1987; Stebbens et al. 1992).

Table 6-5. Cobalt Levels in Food

Food item	Level	Units[a]	Type	Reference
Infant formulas/milk				
Evaporated milk (n=21)	0.74, 0.52–2.6	μg/kg[b]	Median, range	Dabeka 1989
Ready-to-use formula (n=49)	0.53, 0.21–5.2	μg/kg[b]	Median, range	Dabeka 1989
Milk-based (n=33)	0.40, 0.21–0.99			
No added iron (n=6)	0.36, 0.21–0.61			
Added iron (n=27)	0.87, 0.41–0.99			
Soy-based (n=16)	2.27, 1.71–5.2			
Concentrated liquid formula (n=50)	2.27,0.25–11.8	μg/kg[b]	Median, range	Dabeka 1989
Milk-based (n=34)	1.57, 0.25–3.11			
No added iron (n=20)	1.06, 0.25–1.77			
Added iron (n=14)	2.59, 2.03–3.11			
Soy-based (n=16)	4.33, 2.7–11.8			
Powdered formula (n=64)	9.54, 2.6–53	μg/kg[b]	Median, range	Dabeka 1989
Milk-based (n=36)	4.96, 2.6–10.6			
No added iron (n=23)	4.24, 2.6–9.6			
Added iron (n=13)	8.26, 5.1–10.6			
Soy-based (n=28)	20.0, 10.6–53			
Agricultural crops				
Cabbage, United States	0.2	mg/kg[c]	Typical level	NAS 1977
Corn seed, United States	0.01	mg/kg[c]	Typical level	NAS 1977
Fruits, 12 types, Poland	0.01–0.02	mg/kg	Range	Bulinski et al. 1986
Lettuce, Sweden 1983–1990 (n=7)	0.002, 0.006	mg/kg	Mean, maximum	Jorhem and Sundström 1993
Lettuce, United States	0.2	mg/kg[c]	Typical level	NAS 1977
Onions, 11 Danish sites (n=110)	1.51, 0.119–5.1	μg/kg	Median, range	Bibak et al. 1998a
Peas, 10 Danish sites (n=93)	4.6, 0.57–17	μg/kg	Median, range	Bibak et al. 1998b
Potatoes, Sweden (n=8)	0.008, 0.017	mg/kg	Mean, maximum	Jorhem and Sundström 1993
Spinach, United States	0.4–0.6	mg/kg[c]	Typical range	NAS 1977
Strawberries, Sweden (n=10)	0.004, 0.010	mg/kg	Mean, maximum	Jorhem and Sundström 1993
Vegetables, 30 types, Poland	0.008–0.032	mg/kg	Range	Bulinski et al. 1986
White flour, United States	0.003	mg/kg[c]	Typical level	NAS 1977
Meat, fish, beverages				
Beef, Sweden (n=3)	0.001, 0.001	mg/kg	Range, maximum	Jorhem and Sundström 1993
Beef liver, Sweden (n=3)	0.043, 0.074	mg/kg	Range, maximum	Jorhem and Sundström 1993
Beef kidney, Sweden (n=3)	0.008, 0.010	mg/kg	Range, maximum	Jorhem and Sundström 1993

Table 6-5. Cobalt Levels in Food

Food item	Level	Units[a]	Type	Reference
Beer, Spain, 20 brands	0.39, 0.16–0.56	μg/L	Median, range	Cameán et al. 1998
Cocoa, Germany	1.31	mg/kg[c]		Ostapczuk et al. 1987
Coffee (whole), South Africa	0.93	mg/kg[c]		Horwitz and Van der Linden 1974
Coffee (whole), Germany (61% water extractable)	0.11–0.31	mg/kg[c]	Range	Ostapczuk et al. 1987
Fish, Sweden, 10 varieties (n=40)	<0.001–.008, 0.020	mg/kg	Range of mean, maximum	Jorhem and Sundström 1993
Pork, Sweden (n=36)	0.001, 0.012	mg/kg	Range, maximum	Jorhem and Sundström 1993
Pork liver, Sweden (n=36)	0.010, 0.023	mg/kg	Range, maximum	Jorhem and Sundström 1993
Pork kidney, Sweden (n=36)	0.004, 0.011	mg/kg	Range, maximum	Jorhem and Sundström 1993
Tea (whole), South Africa	0.2	mg/kg[c]		Horwitz and Van der Linden 1974
Tea (whole), Germany (40% water extractable)	0.18–6.7	mg/kg[c]	Range	Ostapczuk et al. 1987
Food categories				
Bakery good/ cereals, Canada (n=24)	10.9, 75.7	μg/kg	Median, maximum	Dakeba and McKenzie 1995
Beverages, Canada (n=7)	5.9, 9.1	μg/kg	Median, maximum	Dakeba and McKenzie 1995
Fats and oils, Canada (n=3)	<2.6, 37.6	μg/kg	Median, maximum	Dakeba and McKenzie 1995
Fish, Canada (n=6)	18.6, 14.3–29.4	μg/kg	Median, range	Dakeba and McKenzie 1995
Fruits and fruit juices, Canada (n=25)	<6.6, 35.7	μg/kg	Median, maximum	Dakeba and McKenzie 1995
Meat and poultry, Canada (n=18)	<5.5, 38.2	μg/kg	Median, maximum	Dakeba and McKenzie 1995
Milk and milk products, Canada (n=13)	<1.4, 18.9	μg/kg	Median, maximum	Dakeba and McKenzie 1995
Soups, Canada (n=4)	5.6, 8.5	μg/kg	Median, maximum	Dakeba and McKenzie 1995
Sugar and candy, Canada (n=7)	<0.4, 3.5	μg/kg	Median, maximum	Dakeba and McKenzie 1995
Vegetables, Canada (n=38)	2.4, 18.1	μg/kg	Median, maximum	Dakeba and McKenzie 1995

[a]Produce on a fresh weight basis, unless otherwise specified
[b]As sold
[c]Dry weight basis

The cobalt content of sewage sludge, incinerator ash, fertilizers, soil amendments, and other substances appears in Table 6-6. The concentration of cobalt in U.S. coal averages about 5 mg/kg, levels in crude oil and fuel oil are 0.001–10 and 0.03–0.3 mg/kg, respectively, and those in gasoline are <0.1 mg/kg (Smith and Carson 1981). Cobalt levels were below the detection limit of 0.05 ppm dry weight in all but 1 of 26 samples of composted yard waste, sewage sludge, and municipal solid waste samples nationwide in 1991. The one positive sample of composted yard waste contained 1.53 ppm of cobalt (Lisk et al. 1992).

6.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

Exposure of the general population to cobalt occurs through inhalation of ambient air and ingestion of food and drinking water. In general, intake from food is much greater than from drinking water, which in turn, is much greater than from air. From the limited monitoring data available, the average concentration of cobalt in ambient air in the United States is approximately 0.4 ng/m^3. However, levels may be orders of magnitude higher in source areas. Therefore, intake to cobalt in air will vary substantially from nonsource areas to areas with cobalt-related industries. Similarly, the median cobalt concentration in U.S. drinking water is <2.0 μg/L; however, values as high as 107 μg/L have been reported in surveys of water supplies (Smith and Carson 1981). Therefore, exposure from drinking water may vary considerably from one location to another. In Canada, the daily cobalt intake of the average adult from drinking water is ≤2.6 μg; this could increase to 10 μg for those living in areas with the highest cobalt levels (Meranger et al. 1981).

General population exposure to cobalt from food is highly variable and normally higher than intake from drinking water. Most of the cobalt ingested is inorganic; vitamin B_{12}, which occurs almost entirely in food of animal origin, constitutes only a very small fraction of cobalt intake. The cobalt intake in food has been estimated to be 5.0–40.0 μg/day (Jenkins 1980). The daily cobalt intake, including food, water, and beverages of two men that were followed for 50 weeks was much higher, 310 and 470 μg (Smith and Carson 1981). The estimated average daily cobalt intake from diet in Canada was 11 μg/day; the intake varied from 4 to 15 μg/day between the various age/sex groups (Table 6-7) (Barceloux 1999; Dabeka and McKenzie 1995). The contributions of various food groups to cobalt intake in this study were (category, contribution of dietary intake): bakery goods and cereals, 29.8%; vegetables, 21.9%; beverages, 9.8%; milk and milk products, 9.4%; meat and poultry, 9.1%; soups, 6.4%; fruit and fruit juices, 5.0%; sugar

Table 6-6. Cobalt Content of Miscellaneous Substances

Substance/source	Level	Units	Type	Reference
Bituminous coal used for power generation	6.4	mg/kg	Median	Rubin 1999
Coal, United States	~5	mg/kg	Mean	Smith and Carson 1981
Fly ash	~25	mg/kg	Mean	Smith and Carson 1981
MSW Incinerator ash, Mississippi				Buchholz and Landsberger 1995
Fly ash (n=30)	11.3–13.5	µg/g	Range	
Bottom ash (n=30)	65.2–90.3			
Combined ash (n=30)	24.8–30.5			
MSW Incinerator ash, United States, 1987				Mumma et al. 1990
Fly ash (n=5)	18.2–54.0	µg/g	Range	
Bottom ash (n=7)	13.5–35.1			
Combined ash (n=8)	11.2–43.4			
Compost, Toronto				Evans and Tan 1998
Residential compost	8.1, 3.2–12	mg/kg	Median, range	
Greenhouse finished compost	6.1±1.03		Mean ± SD	
Sewage sludge				
16 large U.S. cities	11.3, 6.08–29.1	mg/kg	Median, range	Gutenmann et al 1994
32 U.S. cities	7.2, 2.4–30.1	mg/kg	Median, range	Mumma et al. 1984
Cow manure (comparison)	6.1	mg/kg		Mumma et al. 1984
Miscellaneous soil amendments[a]				Raven and Loeppert 1997
Compost	3.55, 3.57	mg/kg	Individual means	
Diammonium phosphate	3.24, 0.68			
Dolomite	0.33			
Manure	2.23			
Monoammonium phosphate	0.78, 3.38			
Rock phosphate, Tilemsi	19.6			
Rock phosphate, North Carolina	<0.08			
Sewage sludge, Austinite	4.10			
Sewage sludge, Milorganite	4.07			
Triple superphosphate	6.61, 2.24			
Street dust, New York City	8.7–12.9	µg/g	Range	Fergusson and Ryan 1984

[a]The rest of the 24 fertilizers and soil amendments tested were below the detection limit (typically <0.07 ppm)

MSW = municipal solid waste; SD = standard deviation

Table 6-7. Mean Daily Dietary Intake of Cobalt for Selected Population Groups in Canada

Group	Mean daily intake (μg/day)
1–4 years	7
5–11 years	10
12–19 years; male	14
12–19 years; female	10
20–39 years; male	15
20–39 years; female	9
40–65 years; male	12
40–65 years; female	9
65+; male	10
65+; male	8

Source: Dabeka and McKenzie 1995

and candies, 2.8%; fish, 2.7%; fats and oils, 2.2%; and miscellaneous, 1.1%. The average daily intake of cobalt in France was estimated to be 29 μg/day (Biego et al. 1998). In this study, foods were divided into nine categories. The foods accounting for the greatest contributions of cobalt intake were milk and dairy products, fish-crustaceans, and condiments-sugar oil, respectively contributing 32, 20, and 16% to the daily intake. The U.S. Department of Agriculture (USDA) conducted a special exploratory study in 1985–1986 to determine the concentration of trace metals in tissue of health livestock and poultry randomly selected from those slaughtered. Between 0.6 and 5.9% of samples in the 11 production classes had levels of cobalt that exceeded the lowest reliable quantitation level of 0.15 ppm (0.15 mg/kg) and the mean of positive samples ranged from 0.20 to 0.23 ppm in all classes but heifer/steer, which had a level of 1.92 ppm (Coleman et al. 1992). Cobalt, which has been added to beer to increase the foam head, has been associated with cardiomyopathies (heart disease) in heavy beer drinkers. However, according to a recent Spanish study, the low levels of cobalt presently found in beer do not make a significant contribution to the total cobalt intake in heavy beer drinkers (Cameán et al. 1998). Smokers may be exposed to cobalt in mainstream smoke, but the level of exposure has not been assessed (Barceloux 1999).

Since cobalt and other heavy metals have been used on hand-painted china, a study was conducted to see whether these metals are released into food under acidic conditions. Forty-six samples of porcelain dinnerware from Europe or Asia that were manufactured before the mid-1970s and had hand-painted designs over the glaze were filled with 4% acetic acid to within 7 mm of the rim and analyzed after 24 hours (Sheets 1998). Of these, 36 samples released <0.02 μg/mL of cobalt and 10 released 0.020–2.9 μg/mL. The Food and Drug Administration (FDA) has not established dinnerware extraction limits for cobalt.

Data are lacking on the levels of cobalt in tissues and fluids of the general populations in the United States; values from various countries are given in Table 6-8. This table shows that cobalt concentrations are greatest in nail, hair, and bone. The differences in cobalt levels in similar human tissues (e.g., hair, nail) in different countries may be due to differences in dietary and living habits and levels of cobalt in food (Takagi et al. 1988). The total amount of cobalt in the body of an adult as vitamin B_{12} is about 0.25 mg, of which 50–90% in contained in the liver (IARC 1991).

A recent study in the United States determined the concentrations of trace metals in seminal plasma in industrial workers in a petroleum refinery, smelter, and chemical plant as compared with those of hospital

Table 6-8. Cobalt Levels in Human Tissues and Fluids

Tissue or fluid	Level	Units[a]	Type	Reference
Urine, U.S., NHANES, representative population (n=1007)	0.36, 0.11–0.89	μg/L	Geomean, 10th–90th percentile	CDC 2001
Urine, U.S., NHANES 1999–2000, Total, age 6 and older (n=2,465)	0.372, 0.130–1.32	μg/L	Geomean, 10th–95th percentile	CDC 2003
6–11 years (n=340)	0.498, 0.130–1.32			
12–19 years (n=719)	0.517, 0.200–1.52			
20 years and older (n=1,406)	0.339, 0.120–1.28			
Males (n=1,227)	0.369, 0.150–1.01			
Females (n=1,238)	0.375, 0.120–1.49			
Mexican Americans (n=884)	0.415, 0.130–1.47			
Non-Hispanic blacks (n=568)	0.433, 0.160–1.45			
Non-Hispanic whites (n=822)	0.365, 0.120–1.29			
Urine, The Netherlands	<0.2–1.2	μg/L	Range	Bouman et al. 1986
Urine, Sweden	0.5, 0.1–2.2	μg/L	Mean, range	Alexandersson 1988
Urine, Denmark (3 reference groups)				Poulsen et al. 1994
Unexposed control females (n=46)	1.5, LOD–20.5	nmol[b]	Mean, range	
Unexposed males (n=12)	0.9, LOD–2.31			
Unexposed females (n=11)	5.9, LOD–25.02			
Urine, hip arthroplasty patients, observed 7–15 years (n=17)	0.9–1.05	μg/L	Range	IARC 1991
Urine, hip arthroplasty patients, observed 5–15.5 years (n=10)	3.8	μg/L	Mean	IARC 1991
Urine, 48 metal sharpening workers in 12 Italian factories	0–40.3, 86	μg/L	Range of means, maximum	Imbrogno et al. 1994
Urine, 12 female cobalt powder sintering workers, Italy				Ferdenzi et al. 1994
Monday, before shift	25, 1–51	μg/L	Mean, range	
Friday, before shift	29, 3–159			
Friday, end-of shift	85, 6–505			
After 3-week holiday	11, 4–34			
Urine, Italian workers wet grinding of hard metal tools (end of shift)				Sesana et al. 1994
Factory A no local exhausts (n=3)	138.3 (108), 123.7 (74)	μg/L	Mean (SD) Monday, Friday	
Factory B local exhausts (n=5)	15.3 (7.7), 24.4 (14.1)			
Factory C local exhausts (n=3)	48.2 (7.3), 74.7 (13)			

Table 6-8. Cobalt Levels in Human Tissues and Fluids

Tissue or fluid	Level	Units[a]	Type	Reference
Urine, Northern Italy, 1991, occupational exposure survey, 314 exposed people				Mosconi et al. 1994
Diamond abrasive production				
Mould-filling	320, 587, 39–2,100	µg/L	Median, mean, range	
Sintering	168, 193, 02–390			
Grinding	61, 151, 34–520			
Mechanical-working	50, 67, 143–165			
Grinding	15, 32, 0.8–730			
Tool production	12, 19, 0.8–100			
Hard metal alloy filling	5, 5, 0.8–18			
Other	1, 2.9, 0.8–72			
Blood, Denmark, porcelain factory				Raffn et al. 1988
Plate painters, off work for 6 weeks (n=46)	8.05, 1.70–22.1	nmol/L	Mean, range	
Plate painters, working 4 weeks (n=46)	36.7, 3.40–407			
Top glaze painters (unexposed) (n=51)	4.04, <1.70–10.2			
Urine, Denmark, porcelain factory				Raffn et al. 1988
Plate painters, off work for 6 weeks (n=46)	81.8, <1.70–445	nmol/L	Mean, range	
Plate painters, working 4 weeks (n=46)	1,308, 37.4–14,397			
Top glaze painters (unexposed) (n=51)	16.0, <1.70–234			
Plasma, Sweden	0.1–1.2	µg/L	Range	Alexandersson 1988
Whole Blood, Denmark (3 Reference groups)				Poulsen et al. 1994
Unexposed control females (n=46)	4.1, <1.7–10.2	nmol/L	Mean, range	
Unexposed males (n=12)	3.1, <1.7–6.8			
Unexposed females (n=11)	7.6, <1.7–30.5			
Lung, Sweden				Gerhardsson et al. 1988
Rural	0.007	mg/kg	Mean	
Urban	0.011			
Liver Tissue, United Kingdom, newborns and infants that died from SIDS (n=157)	17.4±11.3 (15.9)	ng/g wet mass	mean±SD (median)	Patriarca et al. 1999
Liver, New Zealand (n=96)	0.120	mg/kg	Mean	IARC 1991

Table 6-8. Cobalt Levels in Human Tissues and Fluids

Tissue or fluid	Level	Units[a]	Type	Reference
Tissue, Japan				Yamagata et al. 1962
Pectoral muscle	0.016	mg/kg	Mean	
Rib bone	0.036			
Stomach	0.021			
Liver	0.017			
Brain	0.0055			
Urinary bladder	0.0055			
Kidney	0.012			
Aorta	0.021			
Nails				Takagi et al. 1988
Canada (n=40)	0.09	mg/kg	Mean	
India (n=100)	0.06			
Japan (n=252)	0.17			
Poland (n=49)	0.04			
U.S. (n=71)	0.06			
Adipose tissue	0.035–0.078	mg/kg	Range	EPA 1986
Hair				Takagi et al. 1986
Canada (n=92)	0.043	mg/kg	Mean	
India (n=255)	0.051			
Japan (n=457)	0.18			
Poland (n=46)	0.022			
United States (n=55)	0.047			
Hair, Italy				Vienna et al. 1995
Male biology students (n=20)	0.007, 0.001–0.07	mg/kg	Geomean, range	
Female biology students (n=20)	0.017, 0.001–0.28			
Hair, Pakistan				Ashraf et al. 1995
Rural (n=28)	2.05, 0.10–4.80	mg/kg	Mean, range	
Urban (n=39)	3.86, 1.10–5.90			

[a]fresh weight, unless otherwise specified
[b]creatinine basis

geomean = geometric mean; LOD = limit of detection; NHANES = Nation Health and Nutrition Examination Survey; SD = standard deviation; SIDS = sudden infant death syndrome

workers (control group). There were four groups each with 50 adult men. The mean cobalt concentrations (μg/dL), including standard errors, were determined to be 31±2 (hospital workers), 25±0.8 (metal ore smelter workers), 19±0.6 (petroleum refinery workers), and 22±1 (chemical workers) (Dawson et al. 2000).

Surgical implants for knee and hip replacements often use cobalt-containing alloys, which may lead to elevated cobalt levels in body fluids. Indeed, cobalt levels in serum and urine have been used as an index of prosthesis wear. In some cases, significant increases in cobalt levels have been observed, while in other cases, elevations were much lower or only sporadic (IARC 1991). These differences have been ascribed to greater release rates from metal to metal than metal to polyethylene articular surfaces as well to differences in the cobalt-containing alloys.

There are several reports of cobalt exposure among occupational groups. The concentrations of cobalt in the air of hard metal manufacturing, welding, and grinding factories may range from 1 to 300 $\mu g/m^3$, compared to normal atmospheric levels of 0.4–2.0 ng/m^3 (Burr and Sinks 1989; Haddad and Zikovsky 1985; Koponen et al. 1982; Lichtenstein et al. 1975). The maximum OSHA permissible level is 100 $\mu g/m^3$. The concentration of cobalt in the dust of an electric welding factory was 4.2 μg/g compared to its normal dust level of 0.1–1.0 μg/g (Baumgardt et al. 1986). The higher rate of exposure to cobalt for occupational groups is also reflected in the higher cobalt content in tissues and body fluids of living and deceased workers in this group. The levels of cobalt in the urine of workers in the hard metal industry varied with the levels of cobalt concentration in the working atmosphere. At a concentration of 0.09 mg/m^3, the urinary excretion of cobalt exceeded normal values by orders of magnitude. When the cobalt concentration in the working atmosphere was 0.01 mg/m^3 or lower, urinary cobalt excretion was 4–10 times higher than normal level (Alexandersson 1988; Scansetti et al. 1985). At high exposure levels, the cobalt concentration in blood was 20 times higher than normal; in the low exposure group, it was only slightly higher than in the control group (Alexandersson 1988).

An extensive survey of workers potentially exposed to cobalt in the Bergamo Province in northern Italy in 1991 identified 403 exposed workers in different production areas (Mosconi et al. 1994a). Significant cobalt exposure occurred especially for operators working in diamond abrasive production, and in particular, in mold filling and sintering units where environmental limits are regularly exceeded. Exposure in tool production, tool sharpening, and hard metal alloy filling is much more restrained.

Occupational cobalt air levels and urinary excretion levels recorded in the survey appear in Tables 6-2 and 6-8.

Several studies of cobalt concentrations in air in the hard metal industry have been reported. In the hard metal industry in Japan, Kumagai et al. (1996) found that mean 8-hour time weighted averages (TWAs) of airborne cobalt were >50 μg/m^3 for workers involved in powder preparation (rotation), powder preparation (full-time), rubber press, and shaping operations; mean atmospheric concentrations were 459, 147, 339, and 97 μg/m^3, respectively. Workers involved in the manufacture and maintenance of hard metal and stellite blades in Finland were exposed to breathing zone cobalt concentrations ranging from 2 to 240 μg/m^3, with a geometric mean of 17 μg/m^3 (Linnainmaa et al. 1996). The average proportion of water soluble cobalt in airborne cobalt was 68% (range 14–100%). Wet grinding was not sufficient to adequately control cobalt levels and coolant cobalt levels were high. In a group of 12 factories in Italy in which 48 workers were tested who had been exposed to cobalt in operations such as sharpening with diamond grinding stones, the mean concentration of cobalt in air was 21.2 and 137.7 μg/m^3 (Permissible exposure limit [PEL]-TWA 100 μg/m^3) in work places with and without dust ventilation, respectively (Imbrogno et al. 1994).

Urine concentrations have been used to monitor workers' exposure to airborne cobalt. Ferdenzi et al. (1994) obtained a correlation between Friday TWA air cobalt levels and Friday end-of-shift urine levels among women in the powder sintering industry. Median urinary cobalt concentrations were 25 (range: 1–51) and 29 (3–159) μg/L, on Monday and Friday before the shift, respectively, and 85 (6–505) μg/L on Friday after the shift. Imbrogno and Alborghetti (1994) evaluated the levels of occupational exposure to cobalt during dry and/or wet hard metal sharpening. The mean urine cobalt level in the workers in 12 factories was found to range from 0 to 40.3 μg/L and the maximum was 86 μg/L. The average urinary cobalt level among workers using wet/mixed sharpening methods was 4 times higher than those using dry sharpening methods; 21.38 μg/L as compared to 5 μg/L, respectively. Gallorini et al. (1994) found that the ratio of inorganic to organic cobalt in the urine of hard metal workers was 2.3 compared to 1.01 in controls; the ratio was constant over the range of urinary cobalt levels analyzed (180–1,254 μg/L). Exposure to cobalt during the wet grinding of hard metal tools (Widia tools) used in the wood industry produced exposure to cobalt above the PEL-TWA of 100 μg/m^3 (Sesana et al. 1994). However, exhausts near the grinding wheels were shown to substantially reduce exposure levels (see Table 6-8). In the processing department of a small company producing carbide tip saw blades for the woodworking industry, area air sampling showed that exposure levels were low in all departments except tip grinding

where wet and dry tip grinding areas contained 55 and 21 μg/m^3 of cobalt, respectively, for the total collection method (Stebbins et al. 1992). For the method collecting respirable particles, cobalt levels ranged from 2 to 28 μg/m^3. Wet grinding is a traditional method for controlling dust during grinding. However, some coolants may contain significant concentrations of cobalt (in this case, 61–538 mg/mL) that can contribute to exposure during grinding (Stebbins et al. 1992). Among cobalt blue dye plate painters in a porcelain factory in Denmark, the blood and urine cobalt levels were, respectively, 2–4 and 5–15 times higher than in control groups (Raffn et al. 1988). Similarly, lungs taken from deceased, occupationally exposed workers also had higher levels of cobalt than lungs from control groups. Lungs of deceased hard metal industry workers in Sweden contained 2.5–4 times higher levels of cobalt than control lungs (Gerhardsson et al. 1988). Similarly, the lungs of coal miners from England contained 6 times higher cobalt levels than control lungs (Hewitt 1988).

Exposure to radioactive cobalt can occur through various means. Workers at nuclear facilities, irradiation facilities, or nuclear waste storage sites may be accidentally exposed to radioisotopes of cobalt. Also, workers using cobalt isotopes in tracer studies, in calibration or other devices, or ^{57}Co in Mössbauer spectroscopy, may be exposed to radioactive cobalt. Exposure would generally be to radiation produced by these isotopes (e.g., gamma radiation from ^{60}Co). Patients receiving ^{60}Co radiotherapy will obviously be exposed to its radiation. According to the USNRC (1999), the collective intake of ^{60}Co by ingestion and inhalation at power reactors in 1998 was 352 μCi (13 MBq) for 25 intake records and 27,000 μCi (1,000 MBq) for 281 intake records (USNRC 1999). The collective intake at fuel fabrication facilities was 0.486 μCi (0.180 MBq) for 502 intake records. The USNRC occupational inhalation annual limits of intake (ALIs) for ^{60}Co are 200 μCi (7.4 MBq) for all compounds, except oxides, hydroxides, halides, and nitrates, and 30 μCi (1.1 MBq) for compounds of oxides, hydroxides, halides, and nitrates (USNRC 2001k).

6.6 EXPOSURES OF CHILDREN

This section focuses on exposures from conception to maturity at 18 years in humans. Differences from adults in susceptibility to hazardous substances are discussed in 3.8 Children's Susceptibility.

Children are not small adults. A child's exposure may differ from an adult's exposure in many ways. Children drink more fluids, eat more food, breathe more air per kilogram of body weight, and have a

larger skin surface in proportion to their body volume. A child's diet often differs from that of adults. The developing human's source of nutrition changes with age: from placental nourishment to breast milk or formula to the diet of older children who eat more of certain types of foods than adults. A child's behavior and lifestyle also influence exposure. Children crawl on the floor, put things in their mouths, sometimes eat inappropriate things (such as dirt or paint chips), and spend more time outdoors. Children also are closer to the ground, and they do not use the judgment of adults to avoid hazards (NRC 1993).

As with adults, most children are exposed to cobalt largely through their diet. Dabeka and McKenzie (1995) estimated that the dietary cobalt intake by Canadian children ages 1–19 ranged from 7 to 14 mg/day (see Table 6-7). Milk constitutes a larger part of children's diets than that of adults, and infants may consume infant formula. Cobalt concentrations ranging from 0.3 to 0.8 ng/g in cow's milk were reported by Iyengar (1982). The levels of cobalt in human milk from Nigeria, Zaire, Guatemala, Hungary, Philippines, and Sweden ranged from 150 (Hungary) to 1,400 ng/g (Philippines), median 320 ng/g (Nriagru 1992). Garg et al. (1993) reported much lower cobalt levels in three samples of human milk in India, 2.42 ng/g, and reported a cobalt concentration of 5.07 ng/g in cow's milk in India. Dakeba (1989) determined cobalt levels in various infant formulas (see Table 6-5). Milk-based infant formulas and evaporated milk contained <1 ng/g of cobalt on a "ready-to-use" basis. Milk-based formulas with added iron contained about twice the cobalt as those with no added iron and soy-based formulas contained about 5 times more cobalt. The influence of added iron suggests that the cobalt in formula is not primarily from vitamin B_{12}. Using literature values of cobalt in food, Dakeba also estimated that infants 0–12 months old ingest an average of 0.52 μg Co/kg-day (3.93 μg/day) from food and water and that for an infant, 0–12 months old, the total dietary cobalt intake would range from 0.42 μg/kg-day (3.39 μg/day) for a breast or milk-based formula fed infant to 1.0 μg/kg-day (7.33 μg/day) for an infant fed soy-based formula powder. The recommended dietary allowance for Canadian infants is 0.012 μg/day cobalt as vitamin B_{12}. In a 1967 study of the total dietary intake of some trace elements, excluding drinking water, of institutionalized children aged 9–12 in 28 U.S. cities, cobalt intake ranged from 0.297 to 1.767 mg/day with a mean value of 1.024 mg/day (Murthy et al. 1971).

Exposure to stable cobalt in communities near mining and smelting facilities or metal shops where cobalt is used in grinding tools is a public health concern, especially for infants and children. Since cobalt remains in the surface soil indefinitely and long past land uses may be forgotten, people may not realize that they are living in areas where high levels of cobalt may occur in soil. Contaminated soils pose a particular hazard to children because of both hand-to-mouth behavior and intentional ingestion of soil that

contain metals and other contaminants (Hamel et al. 1998). In these communities, cobalt may have been tracked in from outdoors and contaminate carpeting. Cobalt-containing dust may be brought home in the clothing of parents working in industries where they are exposed to cobalt. Children may be exposed to this cobalt while crawling around or playing on contaminated carpeting. Exposure may also result from dermal contact with soil, or by inhaling dust and then swallowing it after mucociliary transport up out of the lungs. Because there is little absorption of cobalt through the skin following dermal exposure, and because much of the cobalt in soil is embedded in or adsorbed to soil particles or insoluble, it may not be in a form accessible for uptake by the body, and therefore may not pose a serious health hazard.

6.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

In addition to workers in the hard metal industry (tool production, grinding, etc.) and industries such as coal mining, metal mining, smelting and refining, cobalt dye painters, and the cobalt chemical production, the general population living near these industrial sites may be exposed to high levels of cobalt in air and in soil. Exposure to cobalt during the wet grinding of hard metal tools is especially high when local exhausts are not in use (Sesana et al. 1994). People living near hazardous waste sites may be exposed to cobalt by inhaling dust from contaminated sites or through dermal contact with cobalt-contaminated soil. In the case of children playing in and around unrestricted landfill sites, exposure via dermal and ingestion routes is possible. The general populations in agricultural areas that use sewage sludge or cobalt-containing fertilizers or other soil amendments may be exposed to higher levels of cobalt via inhalation of dust or dermal contact with the soil. However, no experimental evidence of higher than normal exposures for these population groups was found in the literature. People who live in areas that naturally contain higher levels of cobalt minerals may also be exposed to higher levels of cobalt from both the inhalation and dermal contact routes.

The higher exposure of cobalt in patients with cobalt-chromium knee implants has been demonstrated by the slightly higher levels of cobalt in whole blood, serum, and urine, and by very high levels of cobalt in bone of these patients (IARC 1991; Ostapczuk et al. 1985; Sunderman et al. 1989). Prosthetic devices that contain polyethylene components to avoid metal-to-metal contact do not appear to cause elevated levels of cobalt in tissues and body fluids (IARC 1991; Ostapczuk et al. 1985; Sunderman et al. 1989). People who use cobalt supplements as a treatment for anemia and those who take large amounts of vitamin B-12 as a dietary supplement would have higher intakes of cobalt than the general population.

Workers at nuclear facilities and nuclear waste storage sites may be exposed to potentially high levels of radiation exposure from ^{60}Co and ^{58}Co. Workers at irradiation facilities using ^{60}Co may be exposed to potentially high levels of gamma radiation exposure from this isotope. Patients receiving ^{60}Co radiotherapy will intentionally be exposed to high levels of gamma radiation.

6.8 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of cobalt is available. Where adequate information is not available, ATSDR, in conjunction with the National Toxicology Program (NTP), is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of cobalt.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

6.8.1 Identification of Data Needs

Physical, Chemical and Radiological Properties. As can be seen from Table 4-2 and Section 4.2, the relevant physical and chemical properties of cobalt and its compounds are sufficiently known to enable prediction of environmental fate and transport of cobalt compounds (Budavari 1996; Lide 1994; Stokinger 1981; Weast 1985). Information on the radiological properties of important cobalt isotopes are also well known (see Table 4-3) (ICRP 1983; Lide 1994). No data needs were identified.

Production, Import/Export, Use, Release, and Disposal. Information on the production, import/export, use, release, and disposal of a chemical is important because it is an indicator of possible environmental contamination and human exposure. Large releases and consumer use would indicate

higher general population exposure from environmental sources (e.g., air, drinking water, and food) and use of consumer products. Occupational exposure may also increase with increased production and use. U.S. production of cobalt is derived primarily from scrap (secondary production). Information is available on cobalt consumption derived from secondary production, import/export, and release of cobalt from the National Defense Stockpile (USGS 1998, 1999, 2002). However, production volumes of individual cobalt compounds are not available and information on the production of individual compounds would be useful in assessing exposure to specific cobalt compounds. Radioactive cobalt isotopes, primarily ^{60}Co and ^{57}Co, are not commercially produced in the United States, but rather are imported from Canada and the United Kingdom; consumption amounts are not available. Information on the uses of cobalt is available (Cobalt Development Institute 2004; Donaldson 1986; Hodge 1993; IARC 1991; Richardson 1993; USGS 1998, 2002). Cobalt-containing products are mostly used in the workplace, although some consumer products contain cobalt.

According to the Emergency Planning and Community Right-to-Know Act of 1986, 42 U.S.C. Section 11023, industries are required to submit chemical release and offsite transfer information to the EPA. The TRI for 2001 is currently available (TRI01 2004). Starting in 1998, metal mining, coal mining, electric utilities, and RCRA/solvent recovery industries were required to report to the TRI. These sectors include those contributing greatest environmental releases of cobalt and cobalt compounds, giving us a much more complete picture of cobalt releases to the environment. The TRI also contains information on the onsite and offsite disposal and management of wastes (e.g., recycling, treatment, transfer to publicly owned treatment works [POTWs]). EPA guidelines address the disposal of hazardous cobalt wastes. The TRI database will be updated yearly and provides a list of industrial production facilities and emissions. The TRI data should be used with caution since the 1987 data represent first-time reporting by these facilities. Only certain types of facilities were required to report. This is not an exhaustive list.

Environmental Fate. There are data that permit assessment of the environmental fate and transport of cobalt in water and soil (Section 6.3). Frequently, sediment and soil are the ultimate sinks for cobalt; however, this process is dynamic, and cobalt can be released into the water depending upon conditions. There is a paucity of data in the literature regarding the chemical forms of cobalt released to the atmosphere and their transformations in air and this information would facilitate the determination of the transport and persistence of cobalt in the atmosphere. Additional data elucidating the mode of speciation

of cobalt in water and soil would also be desirable. For example, under what circumstances Co(III) compounds might be formed in the environment and how long.

Bioavailability from Environmental Media. Absorption by the inhalation and oral routes in humans has been studied, but the results vary considerably (see Section 3.5.1) (Foster et al. 1989; Harp and Scoular 1952; Sedlet et al. 1958; Sorbie et al. 1971; Valberg et al. 1969). These variations were attributed to differences in the types and doses of cobalt compounds given, to the nutritional status of the subjects following oral exposure, and to particle size differences following inhalation exposure. Additional data assessing the absorption of cobalt following soil ingestion by children may be helpful. Data in animals are plentiful for both inhalation and oral routes and correlate well with the human data (Andre et al. 1989; Bailey et al. 1989; Collier et al. 1989; Kreyling et al. 1986; Patrick et al. 1989; Talbot and Morgan 1989). Data in animals following dermal exposure suggested that cobalt is not absorbed well through intact skin, but is rapidly taken up through damaged skin. Data regarding the bioavailability of cobalt following dermal exposure are important because dermal exposure to cobalt in the workplace is probable.

Food Chain Bioaccumulation. Bioaccumulation in the food chain is important in assessing the human exposure to cobalt from the consumption of food. Data are available that indicate that cobalt is not taken up appreciably by plants and does not biomagnify up the food chain (Baudin and Fritsch 1987; Baudin et al. 1990; Boikat et al. 1985; Francis et al. 1985; Kloke et al. 1984; Lux et al. 1995; Mascanzoni 1989; Mejstrik and Svacha 1988; Mermut et al. 1996; Palko and Yli-Hala 1988; Smith and Carson 1981; Tolle et al. 1983; Watabe et al. 1984).

Exposure Levels in Environmental Media. Monitoring data on levels of cobalt in air, water, and food permits the estimation of exposure from these sources. Data are available on the cobalt levels in ambient air (Golomb et al. 1997; Hasanen et al. 1990; Schroeder et al. 1987; Smith and Carson 1981; Sweet et al. 1993; Wiersema et al. 1984). However, the data are not sufficiently recent or broad-based for estimating the current levels of exposure to cobalt in the general U.S. population and particularly those living near cobalt-containing hazardous waste sites. In addition, in only isolated studies was there an assessment of the concentration of cobalt associated with coarse and fine particles (Sweet et al. 1993) or an average annual level obtained at a site (Golomb et al. 1997). Similarly, levels of cobalt in ambient water, while generally low, are also not sufficiently broad-based or recent to be satisfactory (Bargagli 2000; Bruce and McMahon 1996; Cassidy et al. 1982; Eckel and Jacob 1988; Flaten 1991; Nojiri et al.

1985; Rossmann and Barres 1988; Smith and Carson 1981). This deficiency may be satisfied when the EPA's improved and updated STORET database becomes available. Cobalt levels in Canadian drinking water are ≤2.0 mg/L (Meranger et al. 1981). However, U.S. drinking water levels have not been reported and would be useful. The levels of cobalt in sediment are available (Bargagli 2000; Coakley et al. 1993; Gibbs 1994; Glooschenko et al. 1981; Knutson et al. 1987; Naidu et al. 1997; Shine et al. 1995; Smith and Carson 1981; Trocine and Trefry 1996; Villanueva and Botello 1998), but more data on levels in soil and in the vicinity of industrial and hazardous waste sites would be useful. Few data on the levels of cobalt in U.S. foods are available, although studies from Canada and Sweden are available that indicate that cobalt levels in food items are generally low (Barceloux 1999; Dabeka and McKenzie 1995; Jorhem and Sundström 1993). In particular, total diet studies of cobalt in U.S. food is lacking. A Canadian total diet study estimated average daily cobalt intake to range from 7 to 15 μg/day for different age-sex groups (Dabeka and McKenzie 1995).

Few data are available on levels of ^{60}Co and other cobalt isotopes in environmental media.

Exposure Levels in Humans. The levels of cobalt in hair, nail, and adipose tissues of the general U.S. population are known (EPA 1986; Takagi et al. 1986, 1988). No reliable data on the levels of this substance in blood (or plasma) and urine of the general U.S. population were found, although such data are available for certain European populations including occupationally-exposed groups (Table 6-8). These data may be important for establishing the background exposure level of cobalt. No data on the levels of cobalt in any body tissue or fluid for populations living near hazardous waste sites are available. Such data would be important in assessing the exposure levels of this group of people.

Exposures of Children. Dabeka (1989) reported the levels of cobalt in various formulas and milk products consumed by children in Canada, and Dabeka and McKenzie (1995) determined the mean dietary intake of Canadian children as young as 1–4 years of age. Nriagru (1992) reported levels of cobalt in human milk from several countries. No analogous U.S. studies were found. Cobalt levels is the tissue and body fluids of children have not been found.

Child health data needs relating to susceptibility are discussed in 3.13.2 Identification of Data Needs: Children's Susceptibility.

Exposure Registries. No exposure registries for cobalt were located. This compound is not currently one of the compounds for which a subregistry has been established in the National Exposure Registry. The compound will be considered in the future when chemical selection is made for subregistries to be established. The information that is amassed in the National Exposure Registry facilitates the epidemiological research needed to assess adverse health outcomes that may be related to the exposure to cobalt and its compounds.

6.8.2 Ongoing Studies

The Federal Research in Progress (FEDRIP 2002, 2004) database provides additional information obtainable from a few ongoing studies that may fill in some of the data needs identified in Section 6.8.1. These studies are summarized in Table 6-9.

Remedial investigations and feasibility studies conducted at the NPL sites known to be contaminated with cobalt, such as the Blackbird Mine in Idaho, will add to the available database on exposure levels in environmental media, exposure levels in humans, and exposure registries, and will increase the current knowledge regarding the transport and transformation of cobalt in the environment.

The Cobalt Development Institute (CDI) is implementing a research program to assess environmental risks posed by the manufacture and use of cobalt and cobalt compounds. Studies that are underway include the assessment of seasonal and background variability of cobalt compounds in aquatic environment and a literature survey for existing data on the effects of cobalt and cobalt compounds in soils and sediment. Environmental studies proposed for 2002 included the assessment of seasonal and background variability of cobalt compounds in soils and sediments and a literature survey for existing data on the effects of cobalt and cobalt compounds on marine environments.

Table 6-9. Ongoing Studies on Cobalt

Investigator	Affiliation	Research description	Sponsor
Hamilton, JW	Dartmouth College, Hanover, New Hampshire	The overall goal of the Dartmouth Superfund Basic Research Program (SBRP) Project, Toxic Metals in the Northeast: From Biological to Environmental Implications is to determine the impact of toxic metals found at waste sites, including Superfund sites on human health and the environment. The program-wide focus of this research program is on toxic metals, particularly on arsenic, and also chromium, nickel, cadmium, mercury, cobalt, and lead.	NIH
Jones, BT	Wake Forest University, Winston-Salem, North Carolina	The investigators developing a novel, low-cost, portable instrument for the simultaneous determination of trace radioactive elements in nuclear forensic samples. At issue is the routine, inexpensive sampling for radioactivity that could be released on transport or storage of potential "dirty bomb" material. The instrument to be developed is expected to provide analytical figures comparable to inductively coupled plasma mass spectrometry, but the instrument is much lower cost and more portable. The specific objectives of the project include determination of the analytical figures of merit for elements including cobalt, cesium, and strontium, and analysis of real samples such as soil, urban dust, water and agricultural materials.	NSF
Kpomblekou-Ademawou, K Ankumah, RO	Tuskegee University, Tuskegee, Alabama	This project will investigate if excessive accumulation of some trace elements, added to poultry diet and excreted through feces, affects nitrogen transformation in broiler (chicken) litter amended soils and if this compromises safe food and feed production. The goals of this work are (1) to study the effects of concentrations of key trace elements (e.g., As, Cd, Co, Cr, Cu, Mn, Ni, Pb, Se, and Zn) found in broiler litter on nitrogen transformation in litter amended soils, (2) to assess the effects of temperature on the nitrogen transformation in the presence of trace elements and (3) to assess the fate of trace elements in sudax (Sorghum bicolor) grown in trace element-enriched broiler litter amended soils.	USDA
Longnecker, M	NIEHS, NIH	Evaluate the use of toenail levels as a measure of exposure by analyzing toenail and whole-diet homogenates by neutron activation analysis. Toenails reflect exposure over a longer period of time than do blood or urine measures, and are less likely to be influenced by contamination than hair.	NIEHS

Table 6-9. Ongoing Studies on Cobalt

Investigator	Affiliation	Research description	Sponsor
Saito, MA	Woods Hole Oceanographic Institution, Woods Hole, MA	This research will examine the influence of cobalt and cadmium speciation on *Synechococcus* and *Crocosphaera* at two sites in the Pacific Ocean. In addition, the distribution of cobalt across transects in the Eastern Equatorial Pacific will be determined to improve understanding of the global biogeochemical cycle of cobalt.	NSF
Tavlarides, LL	Syracuse University, Syracuse, New York	This work will be towards the development of sol-gel synthesis methods for organo-ceramic adsorbants for the extraction of toxic and valuable metal ions, such as cobalt, chromium, and arsenic ions from aqueous streams.	NSF

NIEHS = National Institute of Environmental Health Sciences; NIH = National Institute of Health; NSF = National Science Foundation USDA = U.S. Department of Agriculture; USDOE = U.S. Department of Energy

7. ANALYTICAL METHODS

The purpose of this chapter is to describe the analytical methods that are available for detecting, measuring, and/or monitoring cobalt, its metabolites, and other biomarkers of exposure and effect to cobalt. The intent is not to provide an exhaustive list of analytical methods. Rather, the intention is to identify well-established methods that are used as the standard methods of analysis. Many of the analytical methods used for environmental samples are the methods approved by federal agencies and organizations such as EPA and the National Institute for Occupational Safety and Health (NIOSH). Other methods presented in this chapter are those that are approved by groups such as the Association of Official Analytical Chemists (AOAC) and the American Public Health Association (APHA). Additionally, analytical methods are included that modify previously used methods to obtain lower detection limits and/or to improve accuracy and precision.

7.1 BIOLOGICAL MATERIALS

Entry of cobalt and its radioisotopes into the human body can be gained through ingestion, inhalation, or penetration through skin. The quantities of cobalt within the body can be assessed through the use of bioassays that are comprised of either *in vivo* and/or *in vitro* measurements. *In vivo* measurements can be obtained through techniques that directly quantitate internally deposited cobalt using, for example, whole body counters. These *in vivo* measurement techniques are commonly used to measure body burdens of cobalt radioisotopes (i.e., ^{60}Co), but cannot be used to assess the stable isotope of cobalt (^{59}Co). Instead, *in vitro* measurements provide an estimate of internally deposited cobalt (both the stable and radioactive isotopes), utilizing techniques that measure cobalt in body fluids, feces, or other human samples. Examples of these analytical techniques are given in NRCP Report No. 87 (1987) and are also listed in Tables 7-1 and 7-2.

7.1.1 Internal Cobalt Measurements

In vivo measurement techniques are the most direct and widely used approach for assessing the burden of cobalt radioisotopes within the body. The *in vivo* measurement of these radioisotopes within the body is

Table 7-2. Analytical Methods for Determining Radioactive Cobalt in Biological Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit[a]	Percent recovery	Reference
Urine	Direct count of sample	γ-spectrometry with NaI detector	No data (<MDL)	No data	Miltenberger et al. 1981
Soft tissue	Sample wet-ashed	γ-spectrometry (NaI)	No data	No data	Baratta et al. 1969
	Sample directly counted in detector	γ-spectrometry	5 pCi/g	No data	Rabon and Johnson 1973
	Sample digested in acid, oxidized with $HClO_4$, concentrated by precipitation with AMP, purified by resin column, precipitated with hexachloroplatinic acid	-counter	0.1 pCi/g	40–85%	Nevissi 1992
Feces	Direct count of sample	γ-spectrometry	No data	No data	Smith et al. 1972
Blood	Red cells separated from plasma and washed	γ-spectrometry with NaI detector	No data	No data	Smith et al. 1972

[a]1 Bq=2.7×10^{-11} Ci=27 pCi

AMP = ammonium molybdophosphate; MDL = minimum detectable level; NaI = sodium iodide

performed with various radiation detectors and associated electronic devices that are collectively known as whole body counters. These radiation detectors commonly utilize sodium iodide (NaI), hyperpure germanium, and organic liquid scintillation detectors to measure the 1,172 and 1,332 keV gamma rays from the decay of ^{60}Co. Because of the relatively low attenuation of the high energy gamma rays emitted from ^{60}Co by most tissues, cobalt radioisotopes can easily be detected and quantified using whole body counting techniques (Lessard et al. 1984; NCRP 1987; Raghavendran et al. 1978; Smith et al. 1972; Sun et al. 1997). Many configurations of the whole body counter and scanning methods have been utilized, ranging from unshielded single-crystal field detectors to shielded, multi-detector scanning detectors (IAEA 1962, 1970, 1972, 1976, 1985; NCRP 1987). Where appropriate, shielding of the room that houses the whole body counter and/or the detector is often used to increase the detection sensitivity of the equipment by minimizing background radiation. Additionally, care must be exercised to insure that external contamination with radioactive cobalt or other gamma-emitting radioisotopes on the clothing or skin of the individual to be scanned has been removed. Also, *in vitro* measurements of cobalt (see Section 7.1.2) are often used in conjunction with whole body counting when monitoring individuals working with cobalt, especially in conjunction with the assessment of individuals who have experienced accidental exposures to cobalt (Bhat et al. 1973).

Calibration of whole body counters is achieved through the use of tissue-equivalent phantoms. These phantoms are constructed to mimic the shape and density of the anatomical structure using tissue equivalent materials such as water-filled canisters or masonite (Barnaby and Smith 1971; Bhat et al. 1973; Sun et al. 1997). For example, the bottle mannequin absorber (BOMAB) consists of a series of water-filled polyethylene canisters constructed into seated or reclined human forms (Sun et al. 1997). ^{60}Co standards are measured either as point sources along the phantom or dissolved within the water-filled canisters. Comparisons of the actual counts obtained from the phantom to the known activity of the cobalt standards are used to determine the efficiency of the counting technique and, thus, provide the basis for calibrating the technique. Even so, differences in whole body measurement techniques, calibration methods, and background radiation count calculations between different laboratories can complicate the direct comparisons of body burden measurements and clearance rates for cobalt radioisotopes and should be taken into consideration when comparing data obtained from independent laboratories.

7.1.2 External Measurements

In vitro analyses of cobalt are routinely performed in situations where *in vivo* analyses can not be obtained or in support of an *in vivo* monitoring program. Urine and feces are the preferred samples for *in vitro* analyses of cobalt, although other sample types, such as tissue, bone, or blood, can also be used on a more limited basis. Urine provides for an analysis of soluble (inorganic) cobalt, fecal analysis can be used to assess the cobalt (organic) that is eliminated into the gut or the fraction of ingested cobalt not absorbed by the gut, and tissue/blood/bone are used to assess whole or regional body burdens of cobalt (NCRP 1987; Smith et al. 1972).

The analytical methods for determining the stable cobalt isotope, ^{59}Co, in biological matrices are given in Table 7-1. For accurate determination of cobalt, contamination of samples during sample collection, storage, and treatment must be avoided, particularly for biological samples containing low levels of cobalt. Cobalt contamination in blood samples has been reported from disposable syringes and technical-grade anticoagulants. Menghini needles, often used for liver biopsy, and mortar, pestles, and grinding devices used for homogeneous mixing may contaminate samples. Other sources of contamination may be collection and storage containers and chemical reagents used for preparing samples. In fact, sample contamination was responsible for erroneous reports in the earlier literature of grossly high levels of cobalt in biological specimens of unexposed persons. Therefore, blanks should always be run with the samples.

The commonly used classical methods for determining stable cobalt in biological samples are polarographic and colorimetric methods. Details about these methods are given by Saltzman and Keenan (1957). Since these older methods have interference problems and are unsuitable for determining low levels of cobalt in many biological samples, the samples are pretreated before quantification. Precipitation, chelation, chromatography, and ion-exchange are some of the methods used for this purpose. In recent years, the two single-element instrumental techniques most frequently used methods for determining cobalt are graphite furnace-atomic absorption spectrometry (GF-AAS) (also called electrothermal atomic absorption spectrometry) and differential pulse anodic stripping voltammetry (DPAVS). Multi-element techniques commonly used for cobalt determination are neutron activation analysis and inductively coupled plasma-atomic emission spectrometry (ICP-AES). Several other methods are available for determining stable cobalt in biological samples; these include x-ray fluorescence and Spark source mass spectrometry (Adeloju et al. 1985; Smith and Carson 1981).

For the *in vitro* analysis of cobalt radioisotopes in human samples, the majority of the analytical methods measure the cobalt radioisotopes directly in the samples, without the requirement for an extensive sample preparation procedure, using gamma spectrometry techniques. Of the cobalt radioisotopes that have been detected in the environment (e.g., ^{57}Co, ^{58}Co, and ^{60}Co), ^{60}Co is the most common. Consequently, most of the analytical methods that will be described in this chapter are those developed for the detection and quantitation of ^{60}Co in biological (see Table 7-2) and environmental samples (see Table 7-4).

The radiochemical analysis of ^{60}Co in urine has been used in conjunction with whole body scanning methods to assess acute and long-term body burdens of this isotope. The analysis of ^{60}Co in urine is the same as that described for a standardized method of analysis of cesium radioisotopes in urine (Gautier 1983). A urine sample of approximately 2 L is collected (either over 24 hours or before and after bedtime) and a 1-L aliquot is transferred to a Marinelli beaker for counting in a gamma-ray spectrometer (Gautier 1983). This simple procedure offers high recoveries of cobalt (98%) and the minimum detection sensitivity (100 pCi/L [3.7 Bq/L]) that is required to evaluate individuals for exposures to radioactive cobalt (Gautier 1983). Direct counting methods are also used for the analysis of cobalt radioisotopes in tissues, feces, and blood (Smith et al. 1972, Table 7-2). However, some of these methods may require sample preparation to reduce volume or increase concentration.

Accuracy of *in vivo* and *in vitro* measurements of cobalt is determined through the use of standard, certified solutions or radioactive sources with known concentrations or activities of cobalt. Certified standards for stable cobalt can be obtained through a number of commercial sources. The primary source of certified cobalt radioisotope standards is the National Institute of Standards and Technology (NIST). Gamma ray point sources for ^{60}Co (SRM 4200, 60,000 Bq [1.6 μCi] and SRM 4207, 300,000 Bq [56 μCi]) and standard solutions of ^{60}Co (SRM 4233, 600,000 Bq/g [16 μCi/g]) are available from NIST. Also, the determination of accuracy of a method often requires standard reference materials (SRMs). Unfortunately, very few biological SRMs are available. An SRM for cobalt in animal muscle is available from the International Atomic Energy Agency (IAEA), Vienna; an SRM for bovine liver (SRM-1577) is available from NIST (formerly the National Bureau of Standards) (Adeloju et al. 1985; Smith and Carson 1981).

7.2 ENVIRONMENTAL SAMPLES

There are two common approaches for measuring cobalt in the environment. Cobalt radioisotopes can either be measured directly in the field (*in situ*) using portable survey instruments or samples can be procured from the field and returned to the laboratory for quantitation. However, quantitation of the stable cobalt isotope ^{59}Co in environmental samples is generally conducted in the laboratory.

7.2.1 Field Measurements of Cobalt

In situ measurement techniques are extremely useful for the rapid characterization of radionuclide contamination in the environment, such as soils, sediments, and vegetation, or when monitoring personnel for exposure to radionuclides. The measurement of gamma-ray-emitting radionuclides, like cobalt, in the environment is conducted with portable survey instruments such as Gieger-Mueller detectors, sodium iodide scintillation detectors, and gamma-ray spectrometers. However, the use of gamma-ray spectrometers in field survey equipment is preferred for measuring cobalt in the field because of its selectivity and sensitivity. The relatively high energy and penetrability of the gamma ray that is emitted during the decay of ^{60}Co provides an advantage for assessing the level of cobalt both on and below the surface using portable field survey instruments such as the gamma-ray spectrometer. These gamma-ray spectrometers are equipped with a high purity germanium detector that is able to selectively and sensitively differentiate the 1,173 and 1,332 keV gamma rays emitted from ^{60}Co from the gamma-rays emitted from other radionuclides, for example ^{40}K or ^{137}Cs (USNRC 1997). Minimum detectable activities (MDAs) of 0.005 Bq/g (0.05 pCi/g) for ^{60}Co are routinely achieved using p-type germanium gamma-ray spectrometers with 10-minute counting times (USNRC 1997). However, counting errors can occur where the simultaneous detection of the 1,173 and 1,332 keV gamma rays produces a sum peak at 2,505 keV or a count in the continuum between the individual peaks and the sum peak (APHA 1998; USNRC 1997). These errors can be minimized by changing the geometry of the detector or the distance of the detector from the source of radioactivity. Computational methods have been derived to aid in determining the concentrations and distributions of ^{60}Co in different soil types and depths (USNRC 1997). The concentrations and distributions of ^{60}Co that have been derived from the computational analysis of the survey data are often verified by laboratory-based analyses of soil samples procured from the survey area.

7.2.2 Laboratory Analysis of Environmental Samples

Analytical methods for quantifying stable cobalt and cobalt radioisotopes in environmental samples (e.g. air, water, soil, and biota) are summarized in Tables 7-3 (^{59}Co) and 7-4 (^{60}Co). The methods that are commonly used in the analysis of stable cobalt are based on instrumental analytical techniques, such as atomic absorption spectrometry (AAS), instrumental neutron activation analysis (INAA), and mass spectrometry (MS). The analysis of ^{60}Co can be determined either as total mass or total activity, depending on the analytical technique that is used. Typically, radiochemical methods of analysis employing gamma-ray spectrometry techniques are used to quantitate ^{60}Co in environmental samples.

Analytical methods for determining stable cobalt in environmental samples are given in Table 7-3. Since cobalt exists in the particulate form in the atmosphere, it is sampled by drawing air through a metal-free filter (usually cellulose ester membrane), and the metal is quantified in the collected particles. Sample treatment prior to quantification is important for environmental samples. For example, the use of sodium carbonate for dry ashing plant materials results in poor cobalt recovery. Low-temperature ashing may be inadequate for some samples, and losses may occur during rigorous dry ashing. Wet ashing is the preferred method when sample treatment is necessary. Wet extraction with dilute nitric acid is most suitable for analyzing cobalt in dust samples. In some samples, the determination of soluble and insoluble cobalt is important, and analytical methods used to determine cobalt in filtered and unfiltered samples are available for this purpose.

As in the case of biological samples, contamination of environmental samples during sample collection, storage, and treatment should be avoided. Loss of cobalt from aqueous samples due to adsorption on storage containers should be avoided by using polyethylene or similar containers and acidifying the solution to the proper pH (Smith and Carson 1981). Because of its rapidity, accuracy, and low detection limit, GF-AAS with Zeeman background correction is the most commonly used method for quantifying cobalt in environmental samples. To meet the detection limits of the available analytical methods, preconcentration prior to quantification may be necessary for some samples (e.g., seawater). A few commonly used methods for determining cobalt in environmental samples are given in Table 7-3. Other less frequently used methods are inductively coupled plasma-mass spectrometry (ICP-MS) (Henshaw et al. 1989; McLaren et al. 1985), gas, liquid, and ion chromatography with colorimetric, electron capture, and electrochemical detection (Bond and Wallace 1984; Carvajal and Zienius 1986; Cheam and Li 1988; King and Fritz 1987; Schaller and Neeb 1987), photoacoustic spectroscopy with colorimetry (Kitamori

Table 7-3. Analytical Methods for Determining Stable Cobalt in Environmental Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Air (workplace)	Weighed filter irradiated in a reactor	INAA	0.17 μg/m^3	No data	Haddad and Zikovsky 1985
	Sample filter digested by wet acid ashing	Flame-AAS with background correction (NIOSH method 7027)	0.4 μg/m^3	98% with 12–96 μg spiked filter	NIOSH 1984
	Sample filter digested by wet acid ashing	ICP-AES (NIOSH method 7300)	0.5 μg/m^3	95–100% with 2.5–1,000 spiked filter	NIOSH 1984
Water (low ionic strength)	Direct injection	GF-AAS with Zeeman or deuterium background correction	<0.5 μg/L	93–115% at 8.5–30 μg/L	Fishman et al. 1986
Lake water	Sample complexed with 8-hydroxyquinoline absorbed on a column, desorbed and digested with acid	ICP-AES	<0.004 μg/L	No data	Nojiri et al. 1985
Rainwater	Sample preconcentrated onto polystyrene films by spray-drying	PIXE	0.08 μg/L	No data	Hansson et al. 1988
Seawater	Sample complexed with 8-hydroxyquinoline absorbed on a column, desorbed and digested with acid	GF-AAS with Zeeman background correction	0.0002 μg/L	90%	Nakashima et al. 1988
Water and waste water	Direct aspiration of sample	Flame-AAS (EPA method 219.1)	0.05 mg/L	97–98% at 0.2–5.0 mg/L	EPA 1983
	Direct injection	GF-AAS with background correction (EPA method 219.2)	1 μg/L	No data	EPA 1983
Groundwater or leachate	Direct aspiration	Flame-AAS with background correction (EPA method 7200)	0.05 mg/L	97–98% at 0.2–5.0 mg/L	EPA 1986b
Groundwater or leachate	Direct injection	GF-AAS with background correction (EPA method 7201)	1 μg/L	No data	EPA 1986b

Table 7-3. Analytical Methods for Determining Stable Cobalt in Environmental Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Food	Sample digested with acid	GF-AAS with background correction	1.88 μg/L in dissolved extract	100–107% at 0.2–0.6 mg/kg (leaves, liver)	Barbera and Farre 1988
Milled Wheat	Wet ashing (HNO_3), preconcentration and chelation	ET-AAS	20 ng/L	approximately 100%	González et al. 2000

AAS = atomic absorption spectrometry; EPA = Environmental Protection Agency; ET-AAS = electrothermal atomic absorption spectrometry; GF-AAS= graphite furnace atomic absorption spectrometry; ICP-AES = inductively coupled plasma-atomic emission spectrometry; INAA = instrumental neutron activation analysis; NIOSH = National Institute for Occupational Safety and Health; PIXE = photon induced x-ray emission

Table 7-4. Analytical Methods for Determining Radioactive Cobalt in Environmental Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit[a]	Percent recovery	Reference
Air	Direct count of sample collected on paper filter	γ-spectrometry with Ge/Li detector	0.001 pCi/m^3	No data	USAEC 1974a
Air	Sample filter ashed	Scintillation counter with NaI detector	No data	No data	De Franceschi et al. 1974
Drinking water	Direct count of sample	γ-spectrometry with Ge detector	<2 pCi/L	99%	APHA 1998
Drinking water	Direct count of sample	γ-spectrometry	2 pCi/L	No data	USAEC 1974b
Water	Direct count of sample	γ-spectrometry with Ge/Li detector	2 pCi/L	No data	ASTM 1999
Water	Direct count of sample	γ-spectrometry	10 pCi/L	No data	Cahill et al. 1972
Seawater	Sample concentrated using continuous-flow coprecipitation-flotation separation technique	Scintillation detector	50 fCi/L	92–95%	Hiraide et al. 1984
Sediments	Sample dried and ground	γ-spectrometry	0.04 pCi/g	No data	Cahill et al. 1972
Fish	Samples dried and ashed	γ-spectrometry	0.001 pCi/g (DW)	No data	Cushing et al. 1981
Mollusc	Samples dried and ashed	γ-spectrometry	<0.01 pCi/g	No data	De Franceschi et al. 1976

[a]1 Bq=2.7x10^{-11} Ci=27 pCi

DW = dry weight; Ge/Li = lithium drifted geranium; NaI = sodium iodide

et al. 1986), electrothermal vaporization with ICP-AES (Malinski et al. 1988) and chemiluminescence with spectrofluorimetry (Jones et al. 1989).

Analytical methods for determining cobalt radioisotopes in the environment are shown in Table 7-4. The analysis of cobalt in air is based on quantifying cobalt within aerosols or particles that become trapped on cellulose (paper) or glass fiber filters after a calibrated amount of air is passed through the filters. Since the cobalt radioisotopes do not occur naturally, but may be released as a result of nuclear weapons testing (which has been discontinued for several years), neutron-activation of specific materials (e.g., cobalt containing alloys used in piping of nuclear reactors), or a severe core damage accident in a nuclear plant, the amounts of these isotopes within the ambient environment are near or below the minimum detectable levels for these isotopes (DOE 1995). However, trace amounts of ^{60}Co can be detected in air, water, and sediments within or near nuclear weapons or fuel production facilities, nuclear reactors, and nuclear waste storage sites (DOE 1995; Boccolini et al. 1976; USAEC 1973). Analysis of cobalt radioisotopes in air filters, water, sediments, vegetation, and biota can be performed directly using gamma-ray spectrometry, or following some sample preparation (e.g., drying, ashing, or extraction) (Boccolini et al. 1976; Cahill et al. 1972; Cushing 1981; Hiraid et al. 1984; Windham and Phillips 1973).

The detection limits, accuracy, and precision of any analytical methodology are important parameters in determining the appropriateness of a method for quantifying a specific analyte at the desired level of sensitivity within a particular matrix. The Lower Limit of Detection (LLD) has been adopted to refer to the intrinsic detection capability of a measurement procedure (sampling through data reduction and reporting) to aid in determining which method is best suited for the required sample quantitation (USNRC 1984). Several factors influence the LLD, including background, size or concentration of sample, detector sensitivity and recovery of desired analyte during sample isolation and purification, level of interfering contaminants, and, particularly, counting time. Because of these variables, the LLDs between laboratories, utilizing the same or similar measurement procedures, will vary.

The accuracy of a measurement technique in determining the quantity of a particular analyte in environmental samples is greatly dependent on the availability of standard reference materials. Several SRMs for cobalt in environmental samples are also available. Some of these are coal, fly ash, diet, and orchard leaf SRMs available from NIST. The Community Bureau of Reference, European Communities offers SRMs for cobalt in sludges, and an SRM for cobalt in thin polymer films is available from NIST for x-ray fluorescence analysis in aerosol particle samples (Dzubay et al. 1988; Miller-Ihli and Wolf

1986; Schramel 1989; Smith and Carson 1981; Tinsley et al. 1983). Gamma ray point sources for ^{60}Co (SRM 4200, 60,000 Bq [1.6 μCi] and SRM 4207, 300,000 Bq [56 μCi]) and standard solutions of ^{60}Co (SRM 4233, 600,000 Bq/g [16 μCi/g]) are available from NIST.

7.3 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of cobalt is available. Where adequate information is not available, ATSDR, in conjunction with the National Toxicology Program (NTP), is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of cobalt.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

7.3.1 Identification of Data Needs

Methods for Determining Biomarkers of Exposure and Effect. Cobalt concentrations in blood or urine can serve as exposure indicator (Alexandersson 1988; Ichikawa et al. 1985; Scansetti et al. 1985). The available analytical methods are capable of determining the levels of cobalt in both the blood and urine of normal and occupationally exposed persons (Table 7-1). For the quantitation of cobalt radioisotopes, whole body counters can be used to assess radioactive cobalt body burdens that have occurred both from acute and chronic exposures to cobalt radioisotopes (Bhat et al. 1973; NCRP 1987). *In vitro* analytical methods are available for analyzing cobalt radioisotopes in urine, feces, and tissues obtained from normal and occupationally exposed persons (Table 7-2).

Sensitive serum protein responses were found in animals exposed to cobalt at levels below those that produce hematopoietic effects. This unique serum protein response to cobalt exposure includes an increase in alpha globulin fractions of serum proteins and associated serum neuraminic acid. Details of this effect are given in Chapters 2 and 3. If similar changes occur in humans, this measurement may provide the earliest indications of effects of cobalt exposure. The available analytical methods are capable of determining these effects of cobalt exposure.

Methods for Determining Parent Compounds and Degradation Products in Environmental Media. Analytical methods with good sensitivity and specificity are available for determining cobalt in air, water, soil, and other environmental media (Table 7-3). Analytical methods for cobalt, like those for most metals, measure total metal content rather than the particular compound. Therefore, analytical methods do not generally differentiate between the parent compound and a transformation product as would be the case, for example, were cobalt oxide to be converted to cobalt sulfate. (An exception to this would be the case of radioactive decay in which the parent could be readily distinguished from the decay product.) Analytical methods with the capability of distinguishing between different cobalt species would be important an important tool for assessing the fate of cobalt compounds in the environment. However, methods for quantifying specific cobalt compounds were not found in the literature.

The levels of the parent compound or its reaction products in different environmental media can be used to assess the exposure to cobalt by humans through the inhalation of air and ingestion of food and drinking water. In the case of cobalt, a correlation between its levels in environmental media (e.g., occupational air) and in biological tissues and body fluids has been found (Alexandersson 1988; Ichikawa et al. 1985; Scansetti et al. 1985). Therefore, it is possible to estimate the total body burden of cobalt in workers exposed to airborne cobalt vapor and fumes from its concentration in workplace air.

For cobalt radioisotopes, analytical methods also exist that have good sensitivity and specificity for determining radioactive cobalt in air, water, soil, and other environmental media are available (Table 7-4). Because ^{60}Co decays to the stable element ^{60}Ni, there is no need to develop methods to detect and quantify the decay products.

7.3.2 Ongoing Studies

Two studies involving analytical techniques for cobalt was listed in the Federal Research in Progress database (FEDRIP 2002, 2004). N.J. Miller-Ihli and co-workers of the Agricultural Research Service in Beltsville, Maryland are developing single and multielement methods for the determination of trace elements of nutritional and health concern. This work will develop new/improved methods permitting direct analysis of solids by GF-AAS and ICP-MS, as well as methods for the determination of different chemical forms of these elements by coupling capillary zone electrophoresis with inductively coupled plasma mass spectrometry (ICP-MS). This research is supported by the U.S. Department of Agriculture (USDA) Agricultural Research Service. B.T. Jones of Wake Forest University in Winston-Salem, North Carolina along with C. Calloway of Winthrop College, South Carolina, are working to develop a novel, low-cost, portable instrument for the simultaneous determination of trace radioactive elements in nuclear forensic samples. The instrument to be developed is expected to provide analytical figures comparable to ICP-MS, but the instrument is much lower cost and more portable. The specific objectives of the project include determination of the analytical figures of merit for elements including cobalt, cesium, and strontium, and analysis of real samples such as soil, urban dust, water, and agricultural materials.

8. REGULATIONS AND ADVISORIES

International and national guidelines and state regulations regarding exposure to stable cobalt and its compounds are summarized in Table 8-1. The regulations regarding radioactive cobalt are summarized in Table 8-2.

Stable Cobalt. An MRL of $1x10^{-4}$ mg cobalt/m^3 has been derived for chronic-duration inhalation exposure. The MRL is based on a NOAEL of 0.0053 mg cobalt/m^3 for decreased respiratory function in exposed workers (Nemery et al. 1992). An MRL of $1x10^{-2}$ mg/kg-day has been derived for intermediate-duration oral exposure, based on a LOAEL of 1 mg/kg-day for polycythemia in human volunteers (Davis and Fields 1958). No other inhalation or oral MRLs were derived.

The EPA has not derived an RfC or RfD for cobalt and compounds. Similarly, no cancer classification has been performed by the EPA (IRIS 2000). The American Conference of Governmental Industrial Hygienists (ACGIH) has given cobalt a classification of A3, *confirmed animal carcinogen with unknown relevance to humans,* and established an 8-hour time-weighted average (TWA) of 0.02 mg/m^3 for occupational exposure (ACGIH 2000). The Occupational Safety and Health Administration (OSHA) has promulgated an 8-hour permissible exposure limit (PEL) of 0.1 mg/m^3 (OSHA 2001e), and the National Institute for Occupational Safety and Health (NIOSH) recommends an 8-hour TWA of 0.05 mg/m^3 (NIOSH 2001). IARC (2001b) reports that cobalt and cobalt compounds are *possibly carcinogenic to humans* (Group 2B), based on sufficient evidence for cobalt metal and cobalt oxides and limited evidence for cobalt chloride and cobalt sulfate.

Cobalt and its compounds are regulated by the Clean Water Effluent Guidelines for the following industrial point sources: nonferrous metal manufacturing, asbestos, timber products processing, paving and roofing, paint formulating, ink formulating, gum and wood, carbon black, and battery manufacturing (EPA 1988).

Radioactive Cobalt. No MRLs were derived for inhalation or oral exposure to radioactive cobalt. MRLs for acute and chronic exposure to ionizing radiation exist (Agency for Toxic Substances and Disease Registry 1999) and are applicable to cobalt. The EPA has not derived an RfC or RfD for radioactive

Table 8-1. Regulations and Guidelines Applicable to Stable Cobalt

Agency	Description	Information	Reference
INTERNATIONAL			
Guidelines:			
IARC	Carcinogenicity classification		IARC 2001b
	Cobalt and cobalt compounds[a]	Group 2B[b]	
NATIONAL			
Regulations and Guidelines:			
a. Air			
ACGIH	TLV-TWA		ACGIH 2000
	Cobalt, elemental, and inorganic compounds (as Co)	0.02 mg/m^3	
NIOSH	REL (TWA)		NIOSH 2001
	Cobalt metal, dust, and fumes (as Co)	0.05 mg/m^3	
	IDLH		
	Cobalt metal, dust, and fumes (as Co)	20 mg/m^3	
OSHA	PEL (8-hour TWA) for general industry		OSHA 2001e 29CFR1910.1000 Table Z
	Cobalt metal, dust, and fumes (as Co)	0.1 mg/m^3	
	PEL (8-hour TWA) for construction industry		OSHA 2001d 29CFR1926.55
	Cobalt metal, dust, and fumes (as Co)	0.1 mg/m^3	
	PEL (8-hour TWA) for shipyard industry		OSHA 2001c 29CFR1915.1000
	Cobalt metal, dust, and fumes (as Co)	0.1 mg/m^3	
USC	HAP (cobalt compounds)		USC 2001a 42USC7412
b. Water			
EPA	NPDES permit application testing requirements; conventional and nonconventional pollutants required to be tested by existing dischargers if expected to be present		EPA 2001g 40CFR122 Appendix D Table IV
	BPT effluent limitations		EPA 2001b 40CFR415.652
	Maximum for 1 day	$3x10^{-4}$ kg/kkg	
	Average of daily values for 30 consecutive days	$1.2x10^{-4}$ kg/kkg	
	Groundwater monitoring		EPA 2001d 40CFR264 Appendix IX
	Suggested method	PQL	
	6010	70 µg/L	
	7200	500 µg/L	
	7201	10 µg/L	

Table 8-1. Regulations and Guidelines Applicable to Stable Cobalt

Agency	Description	Information	Reference
NATIONAL (cont.)			
c. Food			
FDA	Drug products withdrawn or removed from the market for reasons of safety or effectiveness	All drug products containing cobalt salts (except radioactive forms of cobalt and its salts and cobalamin and its derivatives)	FDA 2000a 21CFR216.24
	New drug status accorded through rulemaking procedures	Cobalt preparations intended for use by man	FDA 2000b 21CFR310.502 (a)(7)
	Over-the-counter drugs; recommended warning and caution statement for cobalt as a cobalt salt	Required on articles containing ≥0.5 μg per dose and ≥2 μg per 24-hour period	FDA 2000e 21CFR369.20
	Substances generally recognized as safe; trace minerals added to animal feeds	Cobalt acetate Cobalt carbonate Cobalt chloride Cobalt oxide Cobalt sulfate	FDA 2000f 21CFR582.20
	Substances prohibited from use in human food	Cobaltous salts and its derivatives	FDA 2000g 21CFR189.120
d. Other			
ACGIH	Carcinogenicity classification Cobalt, elemental, and inorganic compounds (as Co)	 A3[c]	ACGIH 2000
	BEI Cobalt in urine—end of shift at end of workweek Cobalt in blood—end of shift at end of workweek	 15 μg/L 1 μg/L	
EPA	Carcinogenicity classification RfC RfD	No data	IRIS 2000
	Toxic chemical release reporting; Community Right-to-Know; effective date	01/01/87	EPA 2001c 40CFR372.65(a)
	Hazardous waste; identification and listing	Contain ≤1 ppmv in synthesis gas fuel generated from hazardous waste	EPA 2001e 40CFR261.38 (b)(5)
	TSCA; health and safety data reporting		EPA 2001j 40CFR716.120
EPA	Municipal solid waste landfills; hazardous constituent for detection monitoring Suggested method 6010 7200 7201	 PQL 70 μg/L 500 μg/L 10 μg/L	EPA 2001f 40CFR258 Appendix I and II
	Reportable quantity (cobalt compounds)	1 pound	EPA 2001h 40CFR302.4

Table 8-1. Regulations and Guidelines Applicable to Stable Cobalt

Agency	Description	Information	Reference
NATIONAL (cont.)			
USC	Superfund imposition of tax on cobalt	$4.45 per ton	USC 2001c 26USC4661
	Exemption of tax imposed on recycled cobalt		USC 2001b 26USC4662
STATE Regulations and Guidelines			
a. Air			
Alabama	HAP (cobalt compounds)		BNA 2001
Alaska	Air contaminant standard (TWA)		BNA 2001
	Cobalt metal, dust, and fumes	0.05 mg/m^3	
California	Airborne contaminant (cobalt metal, dust, and fumes)		BNA 2001
	HAP (cobalt compounds)		BNA 2001
	Toxic air contaminant (cobalt compounds)		CA Air Resources Board 2000
Colorado	HAP (cobalt metal, dust, and fumes)		BNA 2001
	"High-concern" pollutant (cobalt and compounds)		BNA 2001
	Reportable pollutants (cobalt metal, dust, and fumes)		CO Dept. of Public Health and Environment 2000
Connecticut	HAP—hazard limiting value (cobalt metal, dust, and fumes)		BNA 2001
	8 hours	2 $\mu g/m^3$	
	30 minutes	10 $\mu g/m^3$	
Delaware	Reportable quantities		DE Air Quality Management 2000
	Cobalt carbonyl	1 pound	
	Cobaltous sulfamate	1,000 pounds	
	Cobalt, ((2,2'-(ethane-diylbis(nitrilomethylidyne)	1 pound	
Hawaii	Air contaminant limit (PEL-TWA)		BNA 2001
	Cobalt metal, dust, and fumes	0.05 mg/m^3	
	HAP (cobalt compounds)		BNA 2001
Idaho	TAP non-carcinogenic increments		ID Dept. of Environmental Quality 2000
	Cobalt carbonyl and cobalt hydrocarbonyl (as Co)		
	OEL	$1x10^{-1}$ mg/m^3	
	EL	$7x10^{-3}$ pounds/hour	
	AAC (24-hour average)	$5x10^{-3}$ mg/m^3	
	Cobalt metal, dust, and fumes		
	OEL	$5x10^{-2}$ mg/m^3	
	EL	$3.3x10^{-3}$ pounds/hour	
	AAC (24-hour average)	$2.5x10^{-3}$ mg/m^3	
Illinois	Toxic air contaminant (cobalt)		IL EPA 2000a
Kansas	HAP (cobalt compounds)		KS Dept. of Health and Environment 2000

Table 8-1. Regulations and Guidelines Applicable to Stable Cobalt

Agency	Description	Information	Reference
STATE (cont.)			
Kentucky	HAP (cobalt compounds)		BNA 2001
Louisiana	Toxic air pollutant (cobalt compounds		BNA 2001
Maine	Emissions standards	2,000 pounds	BNA 2001
Maryland	Toxic air pollutant (cobalt compounds)		BNA 2001
Michigan	High concern toxic air pollutants (cobalt compounds)		BNA 2001
Minnesota	HAP threshold (cobalt metal and cobalt carbonyl)	0.1 tons/year	BNA 2001
Missouri	HAP (cobalt compounds)		BNA 2001
Montana	Occupational air contaminant (cobalt metal, dust, and fumes)	0.1 mg/m^3	BNA 2001
Nebraska	HAP (cobalt compounds and cobalt)		BNA 2001
New Mexico	Toxic air pollutant (cobalt metal, dust, and fumes [as Co])		BNA 2001
	OEL	1x10^{-1} mg/m^3	
	Emissions	6.67x10^{-3} pounds/hour	
New York	Annual guideline concentrations	5x10^{-3} µg/m^3	NYS Dept. of Environmental Conservation 2000
	Dangerous air contaminants (TLV) for cobalt metal, dust, and fumes	0.1 mg/m^3	BNA 2001
	HAP (cobalt compounds)		BNA 2001
	Transition limits (PEL)		BNA 2001
	Cobalt metal, dust, and fumes	0.1 mg/m^3	
	Final rule limits (TWA)		
	Cobalt metal, dust, and fumes	0.05 mg/m^3	
North Carolina	PEL-TWA (cobalt metal, dust, and fumes)	0.05 mg/m^3	BNA 2001
Ohio	TRI		Ohio EPA 2000
Oregon	Air contaminant (cobalt metal, dust, and fumes)	0.1 mg/m^3	BNA 2001
Rhode Island	HAP (cobalt compounds)		BNA 2001
South Carolina	Toxic air emissions (MAC) for cobalt compounds	0.25 µg/m^3	BNA 2001
Texas	HAP (cobalt metal, dust, and fumes)	0.1 mg/m^3	BNA 2001
Vermont	HAP (cobalt compounds)		BNA 2001
	Hazardous ambient air standards		BNA 2001
	Cobalt compounds		
	Annual average	0.12 µg/m^3	
	Averaging time	24 hours	
	Action level	6.2x10^{-3} pounds/8 hours	
Washington	Class B TAP and ASIL (24-hour average)		WA Dept. of Ecology 2000
	Cobalt metal, dust and fumes	0.17 µg/m^3	
	Cobalt carbonyl and cobalt hydrocarbonyl	0.33 µg/m^3	

Table 8-1. Regulations and Guidelines Applicable to Stable Cobalt

Agency	Description	Information	Reference
STATE (cont.)			
	Thresholds for HAPs		BNA 2001
	Cobalt carbonyl	0.1 tons/year	
	Cobalt metal, dust, and fumes	0.1 tons/year	
Wisconsin	HAP—existing sources		WI Dept. of Natural Resources 1999
	AAC <25 feet	4.08×10^{-3} pounds/hour	
	AAC ≥25 feet	1.704×10^{-2} pounds/hour	
b. Water			
Alabama	Groundwater monitoring (cobalt)		BNA 2001
	Suggested methods	PQL	
	6010	70 µg/L	
	7200	500 µg/L	
	7201	10 µg/L	
Arizona	Drinking water guideline	0.70 µg/L	FSTRAC 1999
Arkansas	Groundwater monitoring (cobalt)		BNA 2001
	Suggested methods	PQL	
	6010	70 µg/L	
	7200	500 µg/L	
	7201	10 µg/L	
California	Chemicals known to cause cancer or reproductive toxicity; date of initial appearance on the list		Cal/EPA 2000
	Cobalt metal powder		
	Cobalt[II] oxide	07/01/92	
	Cobalt sulfate heptahydrate	07/01/92	
		06/02/00	
Colorado	Groundwater standard (cobalt)	0.05 mg/L	BNA 2001
Delaware	Groundwater monitoring (cobalt)		BNA 2001
	Suggested methods	PQL	
	6010	70 µg/L	
	7200	500 µg/L	
	7201	10 µg/L	
Illinois	Groundwater quality standards for Class II	1 mg/L	IL EPA 2000b
Kentucky	Hazardous waste constituent for groundwater monitoring (cobalt)		BNA 2001
Louisiana	Groundwater monitoring (cobalt)		BNA 2001
	Suggested methods	PQL	
	6010	70 µg/L	
	7200	500 µg/L	
	7201	10 µg/L	
Massachusetts	Groundwater monitoring (cobalt)		BNA 2001
	Suggested methods	PQL	
	6010	70 µg/L	
	7200	500 µg/L	
	7201	10 µg/L	
Minnesota	Drinking water guideline	2 µg/L	FSTRAC 1995
	Groundwater protection hazardous constituent for cobalt (total)		BNA 2001

Table 8-1. Regulations and Guidelines Applicable to Stable Cobalt

Agency	Description	Information	Reference
STATE (cont.)			
Missouri	Water quality standards		BNA 2001
	Livestock, wildlife watering	$1x10^3$ µg/L	
	Groundwater	$1x10^3$ µg/L	
New Mexico	Standards for groundwater of 10,000 mg/L TDS concentration or less (cobalt)	0.05 mg/L	BNA 2001
New York	Groundwater monitoring (cobalt)		BNA 2001
	Suggested methods	PQL	
	6010	70 µg/L	
	7200	500 µg/L	
	7201	10 µg/L	
Tennessee	Effluent limitations—daily maximum concentration (cobalt)	10 mg/L	BNA 2001
Wisconsin	Drinking water guideline	40 µg/L	FSTRAC 1999
	Groundwater standards (cobalt)		BNA 2001
	Enforcement standard	40 µg/L	
	Preventive action limit	8 µg/L	
c. Food		No data	
d. Other			
Alabama	Detection limit values for comparable fuel specification for cobalt; concentration limit	4.6 mg/kg at 10,000 BTU/pound	BNA 2001
Arizona	Soil remediation levels (cobalt)		BNA 2001
	Residential	$4.6x10^3$ mg/kg	
	Non-residential	$9.7x10^4$ mg/kg	
Arkansas	Detection limit values for comparable fuel specification for cobalt; concentration limit	4.6 mg/kg at 10,000 BTU/pound	BNA 2001
	Solid waste management (cobalt)		BNA 2001
	Suggested methods	PQL	
	6010	70 µg/L	
	7200	500 µg/L	
	7201	10 µg/L	
California	Characteristics of toxicity for cobalt and cobalt compounds		BNA 2001
	STLC	80 mg/L	
	TTLC	8,000 mg/kg (wet-weight)	
	Chemicals known to cause cancer or reproductive toxicity (cobalt metal powder); initial appearance on the list	07/01/92	BNA 2001
	Hazardous substance (cobalt, cobalt carbonyl, and cobalt hydrocarbonyl)		BNA 2001
Delaware	Detection limit values for comparable fuel specification for cobalt; concentration limit	4.6 mg/kg at 10,000 BTU/pound	BNA 2001
Florida	Toxic substance in the workplace (cobalt metal, dust, and fumes)		BNA 2001
Georgia	Soil concentration (cobalt)	20 mg/kg	BNA 2001

Table 8-1. Regulations and Guidelines Applicable to Stable Cobalt

Agency	Description	Information	Reference
STATE (cont.)			
Illinois	Analytical parameters and required quantitation limits for cobalt Water Soil Method	 50 µg/L 10 mg/kg 6010A	BNA 2001
Indiana	Constituent subject to assessment monitoring (cobalt [total and dissolved])		BNA 2001
Maine	Screening standards for beneficial use (cobalt)	5,875 mg/kg (dry weight)	BNA 2001
Michigan	Identification and listing of hazardous waste (cobalt)	When in the form of 100 microns or less	BNA 2001
Minnesota	Hazardous substance Cobalt metal, dust, and fumes (as Co) Cobalt carbonyl (as Co) Cobalt, elemental and inorganic compounds (as Co) Cobalt hydrocarbonyl (as Co)		BNA 2001
Missouri	Hazardous constituent (cobalt [total])		BNA 2001
New Jersey	Hazardous substance Cobalt Cobalt carbonyl Cobalt compounds		BNA 2001
New York	Occupational lung disease; hard metal disease	Cobalt	BNA 2001
Ohio	Toxic release inventory		BNA 2001
Oklahoma	Fertilizer labels and labeling; minimum percentage accepted for registration (cobalt)	$5x10^{-4}$ percent	BNA 2001

Table 8-1. Regulations and Guidelines Applicable to Stable Cobalt

Agency	Description	Information	Reference
Oregon	Toxic substance (cobalt)		BNA 2001
Pennsylvania	Hazardous substance (cobalt and cobalt fumes)		BNA 2001

[a]Cobalt compounds: includes cobalt(II) carbonate, cobalt(II) chloride, cobalt(II) nitrate, cobalt(II) oxide, cobalt(II,III) oxide, cobalt(III) oxide, and cobalt(II) sulfate
[b]Group 2B: possibly carcinogenic to humans
[c]A3: confirmed animal carcinogen with unknown relevance to humans

AAC = acceptable ambient concentrations; ACGIH = American Conference of Governmental Industrial Hygienists; ASIL = acceptable source impact level; BEI = biological exposure indices; BNA = Bureau of National Affairs; BPT = best practicable control technology; BTU = British thermal unit; CFR = Code of Federal Regulations; EL = emissions levels; EPA = Environmental Protection Agency; FDA = Food and Drug Administration; FSTRAC = Federal-State Toxicology and Risk Analysis Committee; HAP = hazardous air pollutant; IARC = International Agency for Research on Cancer; IDLH = immediately dangerous to life and health; IRIS = Integrated Risk Information System; MAC = maximum allowable concentration; NIOSH = National Institute for Occupational Safety and Health; NPDES = National Pollutant Discharge Elimination System; OEL = occupational exposure limit; OSHA = Occupational Safety and Health Administration; PEL = permissible exposure limit; PQL = practical quantitation limit; REL = recommended exposure limit; RfC = reference concentration; RfD = reference dose; STLC = soluble threshold limit concentrations; TAP = toxic air pollutant; TDS = total dissolved solids; TLV = threshold limit value; TRI = Toxic Release Inventory; TSCA = Toxic Substances Control Act; TTLC = total threshold limit concentrations; TWA = time-weighted averages; USC = United States Code

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cobalt

Agency	Description	Information	Reference
INTERNATIONAL Guidelines:			
IARC	Carcinogenicity classification	Group 1 (carcinogenic to humans)	IARC 2001b
ICRP	Occupational dose limits; effective dose	20 mSv per year, averaged over defined periods of 5 years	ICRP 1991
	Annual equivalent dose		
	Lens of the eye	150 mSv	
	Skin	500 mSv	
	Hands and feet	500 mSv	
	General population dose limits; effective dose	1 mSv in a year	ICRP 1991
	Annual equivalent dose		
	Lens of eye	15 mSv	
	Skin	50 mSv	
WHO	Drinking water quality	No data	
NATIONAL Regulations and Guidelines:			
a. Air			
ACGIH	All radiation exposures must be kept as low as reasonably achievable		ACGIH 2000
	Effective dose		ACGIH 2000
	Any single year	50 mSv	
	Averaged over 5 years	20 mSv per year	
	Annual equivalent dose		
	Lens of the eye	150 mSv	
	Skin	500 mSv	
	Hands and feet	500 mSv	
	Embryo-fetus exposures once the pregnancy is known		
	Monthly equivalent dose	0.5 mSv	
	Dose to the surface of women's abdomen (lower trunk)	2 mSv for the remainder of the pregnancy	
	Intake of radionuclide	1/20 of the ALI	

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cobalt

Agency	Description	Information		Reference
NATIONAL *(cont.)*				
DOE	Radiation standards			DOE 2000
	Inhalation DAC (μCi/mL)	Class Wa	Class Yb	10CFR835
	^{55}Co	$1x10^{-6}$	$1x10^{-6}$	Appendix A
	^{56}Co	$1x10^{-7}$	$8x10^{-8}$	
	^{57}Co	$1x10^{-6}$	$3x10^{-7}$	
	^{58}mCo	$4x10^{-5}$	$3x10^{-5}$	
	^{58}Co	$5x10^{-7}$	$3x10^{-7}$	
	^{60}mCo	$2x10^{-3}$	$1x10^{-3}$	
	^{60}Co	$7x10^{-8}$	$1x10^{-8}$	
	^{61}Co	$3x10^{-5}$	$2x10^{-5}$	
	^{62}mCo	$7x10^{-5}$	$7x10^{-5}$	
	Radiation standards for air immersion DACc (μCi/mL) for ^{60}mCo	1x10-3		DOE 2000 10CFR835 Appendix C
NIOSH	REL	No data		
USNRC	Effluent concentrations—air			USNRC 2001k
	^{55}Co	ALI (μCi/mL)		10CFR20
	Class Wd	$4x10^{-9}$		Appendix B
	Class Ye	$4x10^{-9}$		Table 2
	^{56}Co			
	Class Wd	$4x10^{-10}$		
	Class Ye	$3x10^{-10}$		
	^{57}Co			
	Class Wd	$4x10^{-9}$		
	Class Ye	$9x10^{-10}$		
	^{58}Co			
	Class Wd	$2x10^{-9}$		
	Class Ye	$1x10^{-9}$		
	^{58}mCo			
	Class Wd	$1x10^{-7}$		
	Class Ye	$9x10^{-8}$		
	^{60}Co			
	Class Wd	$2x10^{-10}$		
	Class Ye	$5x10^{-11}$		
	^{60}mCo			
	Class Wd	$6x10^{-6}$		
	Class Ye	$4x10^{-6}$		
	^{61}Co			
	Class Wd	$9x10^{-8}$		
	Class Ye	$8x10^{-8}$		
	^{62}mCo			
	Class Wd	$2x10^{-7}$		
	Class Ye	$2x10^{-7}$		

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cobalt

Agency	Description	Information		Reference
NATIONAL *(cont.)*				
USNRC	Occupational values			USNRC 2001k
	Inhalation			10CFR20
	^{55}Co	ALI (μCi)	DAC (μCi/mL)	Appendix B
	Class Wd	$3x10^3$	$1x10^{-6}$	Table 1
	Class Ye	$3x10^3$	$1x10^{-6}$	
	^{56}Co			
	Class Wd	$3x10^2$	$1x10^{-7}$	
	Class Ye	$2x10^2$	$8x10^{-8}$	
	^{57}Co			
	Class Wd	$3x10^3$	$1x10^{-6}$	
	Class Ye	$7x10^2$	$3x10^{-7}$	
	^{58}Co			
	Class Wd	$1x10^3$	$5x10^{-7}$	
	Class Ye	$7x10^2$	$3x10^{-7}$	
	^{58}mCo			
	Class Wd	$9x10^4$	$4x10^{-5}$	
	Class Ye	$6x10^4$	$3x10^{-5}$	
	^{60}Co			
	Class Wd	$2x10^2$	$7x10^{-8}$	
	Class Ye	$3x10^1$	$1x10^{-8}$	
	^{60}mCo			
	Class Wd	$4x10^6$	$2x10^{-3}$	
	Class Ye	$3x10^6$	$1x10^{-3}$	
	^{61}Co			
	Class Wd	$6x10^4$	$3x10^{-5}$	
	Class Ye	$6x10^4$	$2x10^{-5}$	
	^{62}mCo			
	Class Wd	$2x10^5$	$7x10^{-5}$	
	Class Ye	$2x10^5$	$6x10^{-5}$	
OSHA	Safety and health regulations for construction—ionizing radiation			OSHA 2001e 29CFR1926.53
	Toxic and hazardous substances—ionizing radiation			OSHA 2001d 29CFR1910.1096
b. Water				
EPA	Drinking water standards			EPA 2000
	Beta particle and photon activity (formerly man-made radionuclides)			
	MCL	4 mrem		
	Caner risk at 10^{-4}	4 mrem/year		
	Gross alpha particle activity			
	MCL	15 pCi/L		
	Caner risk at 10^{-4}	15 pCi/L		
	Carcinogenic classification	Group A (human carcinogen)		

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cobalt

Agency	Description	Information	Reference
NATIONAL *(cont.)*			
USNRC	Effluent concentrations		USNRC 2001k
	Water		10CFR20
	^{55}Co	ALI (μCi/mL)	Appendix B
	Class Wd	$2x10^{-5}$	Table 2
	^{56}Co		
	Class Wd	$6x10^{-6}$	
	^{57}Co		
	Class Wd	$6x10^{-5}$	
	^{58}Co		
	Class Wd	$2x10^{-5}$	
	^{58}mCo		
	Class Wd	$8x10^{-4}$	
	^{60}Co		
	Class Wd	$3x10^{-6}$	
	^{60}mCo		
	Class Wd	$2x10^{-2}$	
	^{61}Co		
	Class Wd	$3x10^{-4}$	
	^{62}mCo		
	Class Wd	$7x10^{-4}$	
	Releases to sewers—monthly average concentration		USNRC 2001k 10CFR20
	^{55}Co	ALI (μCi/mL)	Appendix B
	Class Wd	$2x10^{-4}$	Table 3
	^{56}Co		
	Class Wd	$6x10^{-5}$	
	^{57}Co		
	Class Wd	$6x10^{-4}$	
	^{58}Co		
	Class Wd	$2x10^{-4}$	
	^{58}mCo		
	Class Wd	$8x10^{-3}$	
	^{60}Co		
	Class Wd	$3x10^{-5}$	
	^{60}mCo		
	Class Wd	$2x10^{-1}$	
	^{61}Co		
	Class Wd	$3x10^{-3}$	
	^{62}mCo		
	Class Wd	$7x10^{-3}$	
c. Food and Drug			
FDA	Ionizing radiation for the treatment of poultry feed and poultry feed ingredients (energy sources)	Ioninzing radiation is limited to gamma rays from sealed units of ^{60}CO	FDA 1999 21CFR579.40
	Requirements regarding certain radioactive drugs for ^{58}Co or ^{60}Co	Labeled cyanocobalamin for use in intestinal absorption studies	FDA 2000d 21CFR310.503(c)

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cobalt

Agency	Description	Information			Reference
NATIONAL *(cont.)*					
FDA	Sources of radiation used for inspection of food, packaged food, and controlling food processing				FDA 2000c 21CFR179.21 (a)(2)
d. Other					
DOE	Values for establishing sealed radioactive source accountability and radioactive material posting and labeling requirements				DOE 2000 10CFR835 Appendix E
		Activity (μCi)			
	^{56}Co	4.0×10^{1}			
	^{57}Co	2.3×10^{2}			
	^{58}Co	1.4×10^{2}			
	^{60}Co	1.8×10^{1}			
DOT	Activity values (Ci)	A1	A2		DOT 2001a 49CFR173.435 Table
	^{55}Co	13.5	13.5		
	^{56}Co	8.11	8.11		
	^{57}Co	216	216		
	^{58}mCo	1080	1080		
	^{58}Co	27.0	27.0		
	^{60}Co	10.8	10.8		
	Superfund, reportable quantity (Ci) (pounds)				DOT 2001b 49CFR172.101 Appendix A Table 2
	^{55}Co	10			
	^{56}Co	10			
	^{57}Co	100			
	^{58}Co	10			
	^{58}mCo	1,000			
	^{60}Co	10			
	^{60}mCo	1,000			
	^{61}Co	1,000			
	^{62}mCo	1,000			
EPA	Carcinogenicity classification	No data			IRIS 2000
	RfC				
	RfD				
	Annual possession quantities for environmental compliance (Ci/year)	Gas	Liquid/ Powder	Solid	EPA 2001a 40CFR61 Appendix E Table 1
	^{56}Co	2.3×10^{-6}	2.3×10^{-3}	2.3	
	^{57}Co	1.8×10^{-2}	1.8×10^{1}	1.8×10^{4}	
	^{58}Co	2.5×10^{-6}	2.5×10^{-3}	2.5	
	^{58m}Co	2.3×10^{-6}	2.3×10^{-3}	2.3	
	^{60}Co	4.6×10^{-2}	4.6×10^{1}	4.6×10^{4}	
	^{60m}Co	7.0	7.0×10^{3}	7.0×10^{6}	
	^{61}Co	9.8×10^{-1}	9.8×10^{2}	9.8×10^{5}	

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cobalt

Agency	Description	Information	Reference
NATIONAL *(cont.)*			
EPA	Concentration levels for environmental compliance (Ci/m^3)		EPA 2001a 40CFR61 Appendix E Table 2
	^{56}Co	$1.8x10^{-13}$	
	^{57}Co	$1.3x10^{-12}$	
	^{58}Co	$6.7x10^{-13}$	
	^{58m}Co	$1.2x10^{-10}$	
	^{60}Co	$1.7x10^{-14}$	
	^{60m}Co	$4.3x10^{-9}$	
	^{61}Co	$4.5x10^{-9}$	
	Carcinogenicity—slope factors		EPA 2002
	Lifetime risk per pCi—ingestion		EPA 2002
	Water		
	^{57}Co	$1.04x10^{-12}$	
	^{58m}Co	$2.95x10^{-12}$	
	^{58}Co	$1.26x10^{-13}$	
	^{60}Co	$1.57x10^{-11}$	
	Lifetime risk per pCi—ingestion		EPA 2002
	Food		
	^{57}Co	$1.49x10^{-12}$	
	^{58m}Co	$4.18x10^{-12}$	
	^{58}Co	$1.83x10^{-13}$	
	60Co	$2.23x10^{-11}$	
	Lifetime risk per pCi—ingestion		EPA 2002
	Soil		
	^{57}Co	$2.78x10^{-12}$	
	^{58}mCo	$7.44x10^{-12}$	
	^{58}Co	$3.47x10^{-13}$	
	^{60}Co	$4.03x10^{-11}$	
	Lifetime risk per pCi—inhalation		EPA 2002
	^{57}Co	$2.09x10^{-12}$	
	^{58}mCo	$5.99x10^{-12}$	
	^{58}Co	$6.88x10^{-14}$	
	^{60}Co	$3.58x10^{-11}$	
	External exposure—risk/year per pCi/g soil		EPA 2002
	^{57}Co	$3.55x10^{-7}$	
	^{58}mCo	$4.48x10^{-6}$	
	^{58}Co	$1.00x10^{-12}$	
	^{60}Co	$1.24x10^{-5}$	

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cobalt

Agency	Description	Information		Reference
NATIONAL *(cont.)*				
EPA	Superfund, reportable quantities (Ci) (pounds)			EPA 2001i 40CFR302.4 Appendix B
	^{55}Co	10		
	^{56}Co	10		
	^{57}Co	100		
	^{58}mCo	1,000		
	^{58}Co	10		
	^{60}mCo	1,000		
	^{60}Co	10		
	^{61}Co	1,000		
	^{62}mCo	1,000		
NCRP	Occupational exposures			NCRP1993
	Effective dose limits			
	Annual	50 mSv		
	Cummulative	10 mSv x age		
	Equivalent dose annual limits	150 mSv		
	Lens of eye	500 mSv		
	Skin, hands, and feet			
	Public exposures (annual)			
	Effective dose limits, continuous or frequent exposure	1 mSv		
	Effective dose limits, infrequent exposures	5 mSv		
	Equivalent dose limits			
	Lens of eye	15 mSv		
	Skin, hands, and feet	50 mSv		
	Embryo and fetus exposures (monthly)			
	Effective dose limit	0.5 mSv		
USNRC	Activity values for radionuclides (Ci)	A1	A2	USNRC 2001a 10CFR71
	^{55}Co	13.5	13.5	
	^{56}Co	8.11	8.11	
	^{57}Co	216	216	
	^{58}mCo	1080	1080	
	^{58}Co	27.0	27.0	
	^{60}Co	10.8	10.8	
	Byproduct material listing; exempt concentrations			USNRC 2001e 10CFR30.70 Schedule A
	Liquid and solid concentration ($\mu Ci/mL^2$)			
	^{57}C	$5x10^{-3}$		
	^{58}C	$1x10^{-3}$		
	^{60}C	$5x10^{-4}$		

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cobalt

Agency	Description	Information		Reference
NATIONAL *(cont.)*				
USNRC	Byproduct material listing (μCi)			USNRC 2001b
	^{58}mCo			10CFR30.71
	^{58}Co	10		Schedule B
	^{60}Co	10		
		1		
	Byproduct material listing (Ci)	Column If	Column IIg	USNRC 2001c
	^{58}mCo	100	1.0	10CFR33.100
	^{58}Co	1.0	0.01	Schedule A
	^{60}Co	0.1	$1x10^{-4}$	
	Items containing byproduct material listing—^{60}Co (μCi)			USNRC 2001d 10CFR30.15(a)(8)
	Electron tubes	1.0		
	Spark gap irradiators	1.0		
	Medical use—^{60}Co as a source for brachytherapy	As a sealed source in needles and applicator cells for topical, interstitial, and intracavitary treatment of cancer		USNRC 2001h 10CFR35.400
	Occupational values—oral ingestion			USNRC 2001k 10CFR20
	^{55}Co	ALI (μCi)		Appendix B
	Class Wd	$1x10^3$		Table 1
	^{56}Co			
	Class Wd	$5x10^2$		
	Class Ye	$4x10^2$		
	^{57}Co			
	Class Wd	$8x10^3$		
	Class Ye	$4x10^3$		
	^{58}Co			
	Class Wd	$2x10^3$		
	Class Ye	$1x10^3$		
	^{58}mCo			
	Class Wd	$6x10^4$		
	^{60}Co			
	Class Wd	$5x10^2$		
	Class Ye	$2x10^2$		
	^{60}mCo			
	Class Wd	$1x10^6$		
	St. wall	$1x10^6$		
	^{61}Co			
	Class Wd	$2x10^4$		
	Class Ye	$2x10^4$		
	^{62}mCo			
	Class Wd	$5x10^4$		
	St. wall	$4x10^4$		

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cobalt

Agency	Description	Information	Reference
NATIONAL *(cont.)*			
USNRC	Quantities of radioactive material requiring labeling (μCi)		USNRC 2001g 10CFR30 Appendix B
	^{58}mCo	10	
	^{58}Co	10	
	^{60}Co	1	
	Quantities of licensed material requiring labeling (μCi)		USNRC 2001i 10CFR20 Appendix C
	^{55}Co		
	^{56}Co	100	
	^{57}Co	10	
	^{58}mCo	100	
	^{58}Co	1,000	
	^{60}mCo	100	
	^{60}Co	1,000	
	^{61}Co	1	
	^{62}mCo	1,000	
		1,000	
	Quantities of radioactive materials requiring need for an emergency plan		USNRC 2001j 10CFR30.72 Schedule C
	Release fraction	0.001%	
	Quantity (Ci)	5,000	
	Radioactive waste classification		USNRC 2001l 10CFR61.55
	Class A (Ci/m3)		
	60Co	≤700	
	Reports of individual monitoring—processing or manufacturing for distribution, byproduct material in quantities exceeding		USNRC 2001f 10CFR20.2206 (a)(7)
	^{60}Co (Ci)	1.0	
STATE Regulations and Guidelines:			
a. Air			
Alabama	HAP—radionuclides		BNA 2001
California	HAP—radionuclides		BNA 2001
Hawaii	HAP—radionuclides		BNA 2001
Illinois	Toxic air contaminant—radionuclides		BNA 2001
Kansas	HAP—radionuclides		BNA 2001
Kentucky	HAP—radionuclides		BNA 2001
Minnesota	HAP—radionuclides		BNA 2001
Missouri	HAP—radionuclides		BNA 2001

Table 8-2. Regulations and Guidelines Applicable to Radioactive Cobalt

Agency	Description	Information	Reference
STATE *(cont.)*			
Nebraska	HAP—radionuclides		BNA 2001
New York	HAP—radionuclides		BNA 2001
Rhode Island	HAP—radionuclides		BNA 2001
Wyoming	HAP—radionuclides		BNA 2001

[a]Class W: refers to the approximate length of retention in the pulmonary region which is 10–100 days for this class
[b]Class Y: refers to the approximate length of retention in the pulmonary region which is greater than 100 days for this class
[c]Air immersion DAC values: based on a stochastic dose limit of 5 rems (0.05 Sv) per year or a nonstochastic (organ) dose limit of 50 rems (0.5 Sv) per year
[d]Class W: all compounds except those given for Y
[e]Class Y: oxides, hydroxides, halides, and nitrates
[f]Column I: gas concentration
[g]Column II: liquid and solid concentration

ACGIH = American Conference of Governmental Industrial Hygienists; ALI = annual limits on intake; BNA = Bureau of National Affairs; CFR = Code of Federal Regulations; DAC = derived air concentrations; DOE = Department of Energy; DOT = Department of Transportation; EPA = Environmental Protection Agency; FDA = Food and Drug Administration; IARC = International Agency for Research on Cancer; ICRP = International Commission on Radiological Protection; IRIS = Integrated Risk Information System; mSv = millisievert; NCRP = National Council on Radiation Protection; NIOSH = National Institute for Occupational Safety and Health; OSHA = Occupational Safety and Health Administration; PEL = permissible exposure limit; REL = recommended exposure limit; RfC = reference concentration; RfD = reference dose; TLV = threshold limit value; TWA = time-weighted averages; USNRC = U.S. Nuclear Regulatory Commission; WHO = World Health Organization

cobalt (IRIS 2000). Slope factors have been derived for exposure to cobalt radioisotopes (EPA 2002). The slope factors for ^{60}Co are $1.57x10^{-11}$, $2.23x10^{-11}$, and $4.03x10^{-11}$/pCi for ingestion of water, food, and soil, respectively. The slope factor for inhalation exposure is $3.58x10^{-11}$/pCi, and $1.24x10^{-5}$/year/pCi/g soil for external exposure. The slope factors for ^{58}Co are $1.26x10^{-13}$, $1.83x10^{-13}$, and $3.47x10^{-13}$/pCi for ingestion of water, food, and soil, respectively. The slope factor for inhalation exposure is $6.88x10^{-14}$/pCi for inhalation exposure, and $1.00x10^{-12}$/year/pCi/g soil for external exposure. The slope factors for ^{58m}Co are $2.95x10^{-12}$, $4.18x10^{-12}$, and $7.44x10^{-12}$/pCi for ingestion of water, food, and soil, respectively. The slope factor for inhalation exposure is $5.99x10^{-14}$/pCi for inhalation exposure, and $4.48x10^{-6}$/year/pCi/g soil for external exposure. The slope factors for ^{57}Co are $1.04x10^{-12}$, $1.49x10^{-12}$, and $2.78x10^{-12}$/pCi for ingestion of water, food, and soil, respectively. The slope factor for inhalation exposure is $2.09x10^{-12}$/pCi for ingestion, and $3.55x10^{-7}$/year/pCi/g soil for external exposure.

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10. GLOSSARY

Some terms in this glossary are generic and may not be used in this profile.

Absorbed Dose, Chemical—The amount of a substance that is either absorbed into the body or placed in contact with the skin. For oral or inhalation routes, this is normally the product of the intake quantity and the uptake fraction divided by the body weight and, if appropriate, the time, expressed as mg/kg for a single intake or mg/kg/day for multiple intakes. For dermal exposure, this is the amount of material applied to the skin, and is normally divided by the body mass and expressed as mg/kg.

Absorbed Dose, Radiation—The mean energy imparted to the irradiated medium, per unit mass, by ionizing radiation. Units: rad (rad), gray (Gy).

Absorbed Fraction—A term used in internal dosimetry. It is that fraction of the photon energy (emitted within a specified volume of material) which is absorbed by the volume. The absorbed fraction depends on the source distribution, the photon energy, and the size, shape and composition of the volume.

Absorption—The process by which a chemical penetrates the exchange boundaries of an organism after contact, or the process by which radiation imparts some or all of its energy to any material through which it passes.

Absorption Coefficient—Fractional absorption of the energy of an unscattered beam of x- or gamma-radiation per unit thickness (linear absorption coefficient), per unit mass (mass absorption coefficient), or per atom (atomic absorption coefficient) of absorber, due to transfer of energy to the absorber. The total absorption coefficient is the sum of individual energy absorption processes (see Compton Effect, Photoelectric Effect, and Pair Production).

Absorption Coefficient, Linear—A factor expressing the fraction of a beam of x- or gamma radiation absorbed in a unit thickness of material. In the expression $I=I_0e^{-\mu x}$, I_0 is the initial intensity, I the intensity of the beam after passage through a thickness of the material x, and μ is the linear absorption coefficient.

Absorption Coefficient, Mass—The linear absorption coefficient per cm divided by the density of the absorber in grams per cubic centimeter. It is frequently expressed as μ/ρ, where μ is the linear absorption coefficient and ρ the absorber density.

Absorption Ratio, Differential—Ratio of concentration of a nuclide in a given organ or tissue to the concentration that would be obtained if the same administered quantity of this nuclide were uniformly distributed throughout the body.

Activation—The process of making a material radioactive by bombardment with neutrons or protons.

Activity—The number of radioactive nuclear transformations occurring in a material per unit time (see Curie, Becquerel). The term for activity per unit mass is specific activity.

Activity Median Aerodynamic Diameter (AMAD)—The diameter of a unit-density sphere with the same terminal settling velocity in air as that of the aerosol particle whose activity is the median for the entire size distribution of the aerosol.

Acute Exposure, Chemical—Exposure to a chemical for a duration of 14 days or less, as specified in the Toxicological Profiles.

Acute Exposure, Radiation—The absorption of a relatively large amount of radiation (or intake of a radioactive material) over a short period of time.

Acute Radiation Syndrome—The symptoms which taken together characterize a person suffering from the effects of intense radiation. The effects occur within hours or days.

Ad libitum—Available in excess and freely accessible.

Adsorption Coefficient (K_{oc})—The ratio of the amount of a chemical adsorbed per unit surface area or per unit weight of organic carbon of a specific particle size in the soil or sediment to the concentration of the chemical in solution at equilibrium.

Adsorption Ratio (K_d)—See Distribution Coefficient.

Alpha Particle—A positively charged particle ejected spontaneously from the nuclei of some radioactive elements. It is identical to a helium nucleus, i.e., 2 neutrons and two protons, with a mass number of 4 and an electrostatic charge of +2.

Alpha Track—The track of ionized atoms (pattern of ionization) left in a medium by an alpha particle that has traveled through the medium.

Annihilation (Positron-Electron)—An interaction between a positive and a negative electron in which they both disappear; their rest mass, being converted into electromagnetic radiation (called annihilation radiation) with two 0.51 MeV gamma photons emitted at an angle of 180° to each other.

Annual Limit on Intake (ALI)—The derived limit for the amount of radioactive material taken into the body of an adult worker by inhalation or ingestion in a year. It is the smaller value of intake of a given radionuclide in a year by the reference man that would result in a committed effective dose equivalent of 5 rem or a committed dose equivalent of 50 rem to any organ or tissue.

Atom—The smallest particle of an element that cannot be divided or broken up by chemical means. It consists of a central core called the *nucleus*, which contains *protons* and *neutrons* and an outer shell of *electrons*.

Atomic Mass (u)—The mass of a neutral atom of a nuclide, usually expressed in terms of "atomic mass units." The "atomic mass unit" is one-twelfth the mass of one neutral atom of carbon-12; equivalent to 1.6604×10^{-24} g.

Atomic Mass Number—See Mass Number.

Atomic Number—The number of protons in the nucleus of an atom. The "effective atomic number" is calculated from the composition and atomic numbers of a compound or mixture. An element of this atomic number would interact with photons in the same way as the compound or mixture. (Symbol: Z).

Atomic Weight—The weighted mean of the masses of the neutral isotopes of an element expressed in atomic mass units.

Attenuation—A process by which a beam from a source of radiation is reduced in intensity by absorption and scattering when passing through some material.

Attenuation Coefficient—The fractional reduction in the intensity of a beam of radiation as it passes through an absorbing medium. It may be expressed as reduction per unit distance, per unit mass thickness, or per atom, and is called the linear, mass, or atomic attenuation coefficient, respectively.

Auger Effect—The emission of an electron from the extranuclear portion of an excited atom when the atom undergoes a transition to a less excited state.

Background Radiation—The amount of radiation to which a member of the general population is exposed from natural sources, such as terrestrial radiation from naturally occurring radionuclides in the soil, cosmic radiation originating from outer space, and naturally occurring radionuclides deposited in the human body.

Becquerel (Bq)—International System of Units unit of activity and equals that quantity of radioactive material in which one transformation (disintegration) occurs per second (see Units).

Terabecquerel (TBq)—One trillion becquerel.
Gigabecquerel (GBq)—One billion becquerel.
Megabecquerel (MBq)—One million becquerel.
Kilobecquerel (kBq))—One thousand becquerel.
Millibecquerel (mBq)—One-thousandth of a becquerel.
Microbecquerel (μBq)—One-millionth of a becquerel.

Beta Particle—An electron that is emitted from the nucleus of an atom during one type of radioactive transformation. A beta particle has a mass and charge equal in magnitude to that of the electron. The charge may be either +1 or -1. Beta particles with +1 charges are called positrons (symbolized β^+), and beta particles with -1 charges are called negatrons (symbolized β^-).

Bioconcentration Factor (BCF)—The quotient of the concentration of a chemical in aquatic organisms at a specific time or during a discrete time period of exposure divided by the concentration in the surrounding water at the same time or during the same period.

Biologic Effectiveness of Radiation—See Relative Biological Effectiveness.

Biological Half-time—The time required for a biological system, such as that of a human, to eliminate by natural process half of the amount of a substance (such as a chemical substance, either stable or radioactive) that has entered it.

Biomagnification—The progressive increase in the concentration of a bioaccumulated chemical in organisms as that chemical is passed from the bottom to the top of the food web.

Biomarkers—Broadly defined as indicators signaling events in biologic systems or samples. They have been classified as markers of exposure, markers of effect, and markers of susceptibility.

Body Burden, Chemical—The total amount of a chemical found in an animal or human body.

Body Burden, Radioactivity—The amount of radioactive material found in an animal or human body.

Bone Seeker—Any compound or ion which migrates in the body and preferentially deposits into bone.

Branching—The occurrence of two or more modes by which a radionuclide can undergo radioactive decay. For example, ^{214}Bi can undergo alpha or beta minus decay, ^{64}Cu can undergo beta minus, beta plus, or electron capture decay. An individual atom of a nuclide exhibiting branching disintegrates by one mode only. The fraction disintegrating by a particular mode is the "branching fraction" for that mode. The "branching ratio" is the ratio of two specified branching fractions (also called multiple disintegration).

Bremsstrahlung—X rays that are produced when a charged particle accelerates (speeds up, slows down, or changes direction) in the strong field of a nucleus.

Buildup Factor—The ratio of the radiation intensity, including both primary and scattered radiation, to the intensity of the primary (unscattered) radiation.

Cancer Effect Level (CEL)—The lowest dose of chemical or radiation in a study, or group of studies, that produces significant increases in the incidence of cancer (or tumors) between the exposed population and its appropriate control.

Capture, Electron—A mode of radioactive decay involving the capture of an orbital electron by its nucleus. Capture from a particular electron shell, e.g., K or L shells, is designated as "K-electron capture" or "L-electron capture."

Capture, K-Electron—Electron capture from the K shell by the nucleus of the atom. Also loosely used to designate any orbital electron capture process.

Carcinogen—A chemical or radiation that is capable of inducing cancer.

Carcinoma—Malignant neoplasm composed of epithelial cells, regardless of their derivation.

Case-Control Study—A type of epidemiological study which examines the relationship between a particular outcome (disease or condition) and a variety of potential causative agents (such as toxic chemicals). In a case-controlled study, a group of people with a specified and well-defined outcome is identified and compared to a similar group of people without outcome.

Case Report—Describes a single individual with a particular disease or exposure. These may suggest some potential topics for scientific research but are not actual research studies.

Cataract—A clouding of the crystalline lens of the eye which obstructs the passage of light.

Ceiling Value—A concentration of a substance that should not be exceeded, even temporarily.

Charged Particle—A nuclear particle, atom, or molecule carrying a positive or negative charge.

Chronic Exposure—A long-term, continuous exposure to a chemical or radioactive material. For example, exposure to a chemical for 365 days or more, as specified in the Toxicological Profiles.

Cohort Study—A type of epidemiological study of a specific group or groups of people who have had a common insult (e.g., exposure to an agent suspected of causing disease or a common disease) and are followed forward from exposure to outcome. At least one exposed group is compared to one unexposed group.

Collective Dose—The sum of the individual doses received in a given period of time by a specified population from exposure to a specified source of radiation. Collective dose is expressed in units such as man-rem and person-sievert.

Compton Effect—An attenuation process observed for x- or gamma radiation in which an incident photon interacts with an orbital electron of an atom to produce a recoil electron and a scattered photon whose energy is less than the incident photon.

Containment—The confinement of a chemical or radioactive substance in such a way that it is prevented from being dispersed from its container or into the environment, or is released only at a specified rate.

Contamination—Deposition of a stable or radioactive substance in any place where it is not desired.

Cosmic Rays—High-energy particulate and electromagnetic radiations that originate outside the earth's atmosphere and interact with the atmosphere to produce a shower of secondary cosmic rays.

Count (Radiation Measurements)—The external indication of a radiation-measuring device designed to enumerate ionizing events. It refers to a single detected event. The term "count rate" refers to the total number registered in a given period of time. The term is sometimes erroneously used to designate a disintegration, ionizing event, or voltage pulse.

Counter, Gas-flow Proportional (GPC)—An instrument for detecting beta particle radiation. Beta particles are detected by ionization of the counter gas which results in an electrical impulse at an anode wire.

Counter, Geiger-Mueller (GM counter)—Highly sensitive, gas-filled radiation-measuring device that detects (counts) individual photons or particulate radiation.

Counter, Scintillation—The combination of a crystal or phosphor, photomultiplier tube, and associated circuits for counting light emissions produced in the phosphors by ionizing radiation. Scintillation counters generally are more sensitive than GM counters for gamma radiation.

Counting, Cerenkov—Relatively energetic β-particles pass through a transparent medium of high refractive index and a highly-directional, bluish-white light ("Cerenkov" light) is emitted. This light is detected using liquid scintillation counting equipment.

Cross-sectional Study—A type of epidemiological study of a group or groups which examines the relationship between exposure and outcome to a chemical or to chemicals at one point in time.

Curie (Ci)—A unit of radioactivity. One curie equals that quantity of radioactive material in which there are 3.7×10^{10} nuclear transformations per second. The activity of 1 gram of radium is approximately 1 Ci.

> **Attocurie (aCi)**—One-thousandth of a femtocurie (3.7×10^{-8} disintegrations per second).
> **Femtocurie (fCi)**—One-billionth of a microcurie (3.7×10^{-5} disintegrations per second).

Megacurie (MCi)—One million curies ($3.7x10^{16}$ disintegrations per second).
Microcurie (μCi)—One-millionth of a curie ($3.7x10^{4}$ disintegrations per second).
Millicurie (mCi)—One-thousandth of a curie ($3.7x10^{7}$ disintegrations per second).
Nanocurie (nCi)—One-billionth of a curie ($3.7x10^{1}$ disintegrations per second).
Picocurie (pCi)—One-millionth of a microcurie ($3.7x10^{-2}$ disintegrations per second).

Daughter Products—See Progeny and Decay Product

Decay Chain or Decay Series—A sequence of radioactive decays (transformations) beginning with one nucleus. The initial nucleus, the parent, decays into a daughter or progeny nucleus that differs from the first by whatever particles were emitted during the decay. If further decays take place, the subsequent nuclei are also usually called daughters or progeny. Sometimes, to distinguish the sequence, the daughter of the first daughter is called the granddaughter, etc.

Decay Constant (λ)—The fraction of the number of atoms of a radioactive nuclide which decay in unit time (see Disintegration Constant).

Decay Product, Daughter Product, Progeny—A new nuclide formed as a result of radioactive decay. A nuclide resulting from the radioactive transformation of a radionuclide, formed either directly or as the result of successive transformations in a radioactive series. A decay product (daughter product or progeny) may be either radioactive or stable.

Decay, Radioactive—Transformation of the nucleus of an unstable nuclide by spontaneous emission of radiation, such as charged particles and/or photons (see Disintegration).

Delta Ray—An electron removed from an atom of a medium that is irradiated, or through which radiation passes, during the process of ionization (also called secondary electron). Delta rays cause a track of ionizations along their path.

Derived Air Concentration (DAC)—The concentration of radioactive material in air that, if breathed by the reference man for a working year of 2000 hours under conditions of light work (at a rate of 1.2 liters of air per hour), would result in an intake of one ALI (see Annual Limit on Intake).
Deterministic Effect—A health effect, the severity of which varies with the dose and for which a threshold is believed to exist (also called a non-stochastic effect).

Developmental Toxicity—The occurrence of adverse effects on the developing organism that may result from exposure to a chemical or radiation prior to conception (either parent), during prenatal development, or postnatally to the time of sexual maturation. Adverse developmental effects may be detected at any point in the life span of the organism.

Disintegration Constant—Synonymous with decay constant. The fraction of the number of atoms of a radioactive material that decays per unit time (see Decay Constant.)

Disintegration, Nuclear—A spontaneous nuclear transformation (radioactivity) characterized by the emission of energy and mass from the nucleus. When large numbers of nuclei are involved, the process is characterized by a definite half-life (see Transformation, Nuclear).

Distribution Coefficient (K_d)—Describes the distribution of a chemical between the solid and aqueous phase at thermodynamic equilibrium, is given as follows:

$K_d = \frac{[C]_s}{[C]_w}$, Units = (L solution)/(kg solid),
where $[C]_s$ is the concentration of the chemical associated with the solid phase in units of (mg)/(kg solid), and $[C]_w$ is the concentration of the chemical in the aqueous phase in units of (mg)/(L solution). As the magnitude of K_d decreases, the potential mobility of the chemical to groundwater systems increases and vice versa.

Dose—A general term denoting the quantity of a substance, radiation, or energy absorbed. For special purposes it must be appropriately qualified. If unqualified, it refers to radiation absorbed dose.

> **Absorbed Dose**—The energy imparted to matter by ionizing radiation per unit mass of irradiated material at the place of interest. The unit of absorbed dose is the rad. One rad equals 100 ergs per gram. In SI units, the absorbed dose is the gray which is 1 J/kg (see Rad).
>
> **Cumulative Dose (Radiation)**—The total dose resulting from repeated or continuous exposures to radiation.

Dose Assessment—An estimate of the radiation dose to an individual or a population group usually by means of predictive modeling techniques, sometimes supplemented by the results of measurement.

Dose Equivalent (DE)—A quantity used in radiation safety practice to account for the relative biological effectiveness of the several types of radiation. It expresses all radiations on a common scale for calculating the effective absorbed dose. The NRC defines it as the product of the absorbed dose, the quality factor, and all other modifying factors at the location of interest. ICRP has changed its definition to be the product of the absorbed dose and the radiation weighting factor. (The unit of dose equivalent is the rem. In SI units, the dose equivalent is the sievert, which equals 100 rem.)

Dose, Fractionation—A method of administering therapeutic radiation in which relatively small doses are given daily or at longer intervals.

Dose, Protraction—A method of administering therapeutic radiation by delivering it continuously over a relatively long period at a low dose rate.

Dose, Radiation—The amount of energy imparted to matter by ionizing radiation per unit mass of the matter, usually expressed as the unit rad, or in SI units, the gray. 100 rad=1 gray (Gy) (see Absorbed Dose).

> **Committed Dose Equivalent ($H_{T,50}$)**—The dose equivalent to organs or tissues of reference (T) that will be received from an intake of radioactive material by an individual during the 50 years following the intake.
>
> **Committed Effective Dose Equivalent ($H_{E,50}$)**—The sum of the products of the weighting factors applicable to each of the body organs or tissues that are irradiated and the committed dose equivalent to those organs or tissues.
>
> **Effective Dose** —A dose value that attempts to normalize the detriment to the body (for cancer mortality and morbidity, hereditary effects, and years of life lost) from a non-uniform exposure to

that of a uniform whole body exposure. Effective dose is calculated as the sum of products of the equivalent dose and the tissue weighting factor (w_T) for each tissue exposed. ($E = \sum D_{T,R}\, w_R\, w_T$).

Effective Dose Equivalent (H_E)—This dose type is limited to internal exposures and is the sum of the products of the dose equivalent to the organ or tissue (H_T) and the weighting factors (w_T) applicable to each of the body organs or tissues that are irradiated. ($H_E = \sum w_T\, H_T$).

Equivalent Dose—A dose quantity that places the biological effect of all radiation types on a common scale for calculating tissue damage. Alpha particles, for example, are considered to cause 20 times more damage than gamma rays. Equivalent dose is calculated as the sum of products of the average absorbed dose (in gray) in an organ or tissue ($_{DT,R}$) from each type of radiation and the radiation weighting factor (w_R) for that radiation ($\sum D_{T,R}\, w_R$).

External Dose—That portion of the dose equivalent received from radiation sources outside the body.

Internal Dose—That portion of the dose equivalent received from radioactive material taken into the body.

Limit—A permissible upper bound on the radiation dose.

Maximum Permissible Dose (MPD)—The greatest dose equivalent that a person or specified part thereof shall be allowed to receive in a given period of time.

Median Lethal Dose (MLD)—Dose of radiation required to kill, within a specified period (usually 30 days), 50% of the individuals in a large group of animals or organisms. Also called the LD_{50}, or $LD_{50/30}$ if for 30 days.

Threshold Dose—The minimum absorbed dose that will produce a detectable degree of any given effect.

Tissue Dose—Absorbed dose received by tissue in the region of interest, expressed in rad (see Dose, Gray, and Rad).

Dose Rate—The amount of radiation dose delivered per unit time. Generically, the rate at which radiation dose is delivered to any material or tissue.

Dose-Response Relationship—The quantitative relationship between the amount of exposure to a toxicant and the incidence of the adverse effects.

Dosimetry—Quantification of radiation doses to cells, tissues, organs, individuals or populations resulting from radiation exposures.

Early Effects (of radiation exposure)—Effects that appear within 60 days of an acute exposure.

Electron—A stable elementary particle having an electric charge equal to $\pm 1.60210 \times 10^{-19}$ C (Coulombs) and a rest mass equal to 9.1091×10^{-31} kg. A positron is a positively charged "electron" (see Positron).

Electron Volt—A unit of energy equivalent to the energy gained by an electron in passing through a potential difference of one volt. Larger multiple units of the electron volt are frequently used: keV for thousand or kilo electron volts; MeV for million or mega electron volts (eV). 1 eV=1.6×10^{-12} erg.

Embryotoxicity and Fetotoxicity—Any toxic effect on the conceptus as a result of prenatal exposure to a chemical; the distinguishing feature between the two terms is the stage of development during which the insult occurred. The terms, as used here, include malformations and variations, altered growth, and *in utero* death.

Energy—Capacity for doing work. Gravitationally, "potential energy" is the energy inherent in a mass because of its spatial relation to other masses. Chemically or radiologically, "potential energy" is the energy released when a chemical reaction or radiological transformation goes to completion. "Kinetic energy" is the energy possessed by a mass because of its motion (SI unit: joules):

> **Binding Energy (Electron)**—The amount of energy that must be expended to remove an electron from an atom.
>
> **Binding Energy (Nuclear)**—The energy represented by the difference in mass between the sum of the component parts and the actual mass of the nucleus. It represents the amount of energy that must be expended to break a nucleus into its component neutrons and protons.
>
> **Excitation Energy**—The energy required to change a system from its ground state to an excited state. Each different excited state has a different excitation energy.
>
> **Ionizing Energy**—The energy required to knock an electron out of an atom. The average energy lost by electrons or beta particles in producing an ion pair in air or in soft tissue is about 34 eV.
>
> **Radiant Energy**—The energy of electromagnetic radiation, such as radio waves, visible light, x and gamma rays.

Enrichment, Isotopic—An isotopic separation process by which the relative abundances of the isotopes of a given element are altered, thus producing a form of the element that has been enriched in one or more isotopes and depleted in others. In uranium enrichment, the percentage of uranium-235 in natural uranium can be increased from 0.7% to >90% in a gaseous diffusion process based on the different thermal velocities of the constituents of natural uranium (^{234}U, ^{235}U, ^{238}U) in the molecular form UF_6.

EPA Health Advisory—An estimate of acceptable drinking water levels for a chemical substance based on health effects information. A health advisory is not a legally enforceable federal standard, but serves as technical guidance to assist federal, state, and local officials.

Epidemiology—Refers to the investigation of factors that determine the frequency and distribution of disease or other health-related conditions within a defined human population during a specified period.

Equilibrium, Radioactive—In a radioactive series, the state which prevails when the ratios between the activities of two or more successive members of the series remains constant.

> **Secular Equilibrium**—If a parent element has a very much longer half-life than the daughters (so there is not appreciable change in its amount in the time interval required for later products to attain equilibrium) then, after equilibrium is reached, equal numbers of atoms of all members of

the series disintegrate in unit time. This condition is never exactly attained, but is essentially established in such a case as ^{226}Ra and its transformation series to stable ^{206}Pb. The half-life of ^{226}Ra is about 1,600 years; of ^{222}Rn, approximately 3.82 days, and of each of the subsequent members, a few minutes. After about a month, essentially the equilibrium amount of radon is present; then (and for a long time) all members of the series disintegrate the same number of atoms per unit time. At this time, the activity of the daughter is equal to the activity of the parent.

Transient Equilibrium—If the half-life of the parent is short enough so the quantity present decreases appreciably during the period under consideration, but is still longer than that of successive members of the series, a stage of equilibrium will be reached after which all members of the series decrease in activity exponentially with the period of the parent. At this time, the ratio of the parent activity to the daughter activity is constant.

Equilibrium, Electron—The condition in a radiation field where the energy of the electrons entering a volume equals the energy of the electrons leaving that volume.

Excitation—The addition of energy to a system, thereby transferring it from its ground state to an excited state. Excitation of a nucleus, an atom, or a molecule can result from absorption of photons or from inelastic collisions with other particles. The excited state of an atom is an unstable or metastable state and will return to ground state by radiation of the excess energy.

Exposure (Chemical)—Contact of an organism with a chemical or physical agent. Exposure is quantified as the amount of the agent available at the exchange boundaries of the organism (e.g., skin, lungs, gut) and available for absorption.

Exposure (Radiation)—Subjection to ionizing radiation or to a radioactive material. For example, exposure in air is a measure of the ionization produced in air by x or gamma radiation; the sum of the electric charges on all ions of one sign produced in air when all electrons liberated by photons in a volume of air are completely stopped in air (dQ), divided by the mass of the air in the volume (dm). The unit of exposure in air is the roentgen, or coulomb per kilogram (SI units). One roentgen is equal to 2.58×10^{-4} coulomb per kilogram (C/kg).

Fission, Nuclear—A nuclear transformation characterized by the splitting of a nucleus into at least two other nuclei with emission of several neutrons, accompanied by the release of a relatively large amount of energy.

Gamma Ray, Penetrating—Short wavelength electromagnetic radiation of nuclear origin.

Genetic Effect of Radiation—Inheritable change, chiefly mutations, produced by the absorption of ionizing radiation by germ cells. Genetic effects have not been observed in any human population exposed at any dose level.

Genotoxicity—A specific adverse effect on the genome of living cells that, upon the duplication of affected cells, can be expressed as a mutagenic, clastogenic or carcinogenic event because of specific alteration of the molecular structure of the genome.

Gray (Gy)—SI unit of absorbed dose, 1 J/kg. One gray equals 100 rad (see Units).

Half-life, Effective—See Half-Time, Effective.

Half-life, Radioactive—Time required for a radioactive substance to lose 50% of its activity by decay. Each radio-nuclide has a unique physical half-life. Known also as physical half-time and symbolized as T_r or T_{rad}.

Half-time, Biological—Time required for an organ, tissue, or the whole body to eliminate one-half of any absorbed substance by regular processes of elimination. This is the same for both stable and radioactive isotopes of a particular element, and is sometimes referred to as half-time, symbolized as t_{biol} or T_b.

Half-time, Effective—Time required for a radioactive element in an organ, tissue, or the whole body to be diminished 50% as a result of the combined action of radioactive decay and biological elimination, symbolized as T_e or T_{eff}.

$$\text{Effective half-time} = \frac{\text{Biological half-time} \times \text{Radioactive half-life}}{\text{Biological half-time} + \text{Radioactive half-life}}$$

Immediately Dangerous to Life or Health (IDLH)—The maximum environmental concentration of a contaminant from which one could escape within 30 minutes without any escape-impairing symptoms or irreversible health effects.

Immunologic Toxicity—The occurrence of adverse effects on the immune system that may result from exposure to environmental agents such as chemicals.

Immunological Effects—Functional changes in the immune response.

In Vitro—Isolated from the living organism and artificially maintained, as in a test tube. Literally, "in glass."

In Vivo—Occurring within the living organism. Literally, "in life."

Intensity—Amount of energy per unit time passing through a unit area perpendicular to the line of propagation at the point in question.

Intermediate Exposure—Exposure to a chemical for a duration of 15–364 days, as specified in the Toxicological Profiles.

Internal Conversion—Process in which a gamma ray knocks an electron out of the same atom from which the gamma ray was emitted. The ratio of the number of internal conversion electrons to the number of gamma quanta emitted in the de-excitation of the nucleus is called the "conversion ratio."

Ion—Atomic particle, atom or chemical radical bearing a net electrical charge, either negative or positive.

Ion Pair—Two particles of opposite charge, usually referring to the electron and positive atomic or molecular residue resulting after the interaction of ionizing radiation with the orbital electrons of atoms.

Ionization—The process by which a neutral atom or molecule acquires a positive or negative charge.

> **Primary Ionization**—(1) In collision theory: the ionization produced by the primary particles as contrasted to the "total ionization" which includes the "secondary ionization" produced by delta

rays. (2) In counter tubes: the total ionization produced by incident radiation without gas amplification.

Specific Ionization—Number of ion pairs per unit length of path of ionizing radiation in a medium; e.g., per centimeter of air or per micrometer of tissue.

Total Ionization—The total electric charge of one sign on the ions produced by radiation in the process of losing its kinetic energy. For a given gas, the total ionization is closely proportional to the initial ionization and is nearly independent of the nature of the ionizing radiation. It is frequently used as a measure of absorption of radiation energy.

Ionization Density—Number of ion pairs per unit volume.

Ionization Path (Track)—The trail of ion pairs produced by an ionizing particle in its passage through matter.

Ionizing Radiation—Any radiation capable of knocking electrons out of atoms and producing ions. Examples: alpha, beta, gamma and x rays, and neutrons.

Isobars—Nuclides having the same mass number but different atomic numbers.

Isomers—Nuclides having the same number of neutrons and protons but capable of existing, for a measurable time, in different quantum states with different energies and radioactive properties. Commonly the isomer of higher energy decays to one with lower energy by the process of isomeric transition.

Isotopes—Nuclides having the same number of protons in their nuclei, and hence the same atomic number, but differing in the number of neutrons, and therefore in the mass number. Identical chemical properties exist in isotopes of a particular element. The term should not be used as a synonym for nuclide because isotopes refer specifically to different nuclei of the same element.

Stable Isotope—A nonradioactive isotope of an element.

Joule—The S.I. unit for work and energy. It is equal to the work done by raising a mass of one newton through a distance of one meter (J = Nm), which corresponds to about 0.7 ft-pound.

Kerma (k)—A measure of the kinetic energy transferred from gamma rays or neutrons to a unit mass of absorbing medium in the initial collision between the radiation and the absorber atoms. The SI unit is J/kg. The special name of this unit is the rad (traditional system of units) or Gray (SI).

Labeled Compound—A compound containing one or more radioactive atoms intentionally added to its structure. By observations of radioactivity or isotopic composition, this compound or its fragments may be followed through physical, chemical, or biological processes.

Late Effects (of radiation exposure)—Effects which appear 60 days or more following an acute exposure.

$LD_{50/30}$—The dose of a chemical or radiation expected to cause 50% mortality in those exposed within 30 days. For radiation, this is about 350 rad (3.5 gray) received by humans over a short period of time.

Lethal Concentration$_{(Lo)}$ (LC_{Lo})—The lowest concentration of a chemical in air that has been reported to have caused death in humans or animals.

Lethal Concentration$_{(50)}$ (LC_{50})—A calculated concentration of a chemical in air to which exposure for a specific length of time is expected to cause death in 50% of a defined experimental animal population within a specified time, usually 30 days.

Lethal Dose$_{(Lo)}$ (LD_{Lo})—The lowest dose of a chemical introduced by a route other than inhalation that is expected to have caused death in humans or animals within a specified time, usually 30 days.

Lethal Dose$_{(50)}$ (LD_{50})—The dose of a chemical which has been calculated to cause death in 50% of a defined experimental animal population.

Lethal Time$_{(50)}$ (LT_{50})—A calculated period of time within which a specific concentration of a chemical is expected to cause death in 50% of a defined experimental animal population.

Linear Energy Transfer (LET)—A measure of the energy that a charged particle transfers to a material per unit path length.

> **Average LET**—The energy of a charged particle divided by the length of the path over which it deposits all its energy in a material. This is averaged over a number of particles.
>
> **High-LET**—Energy transfer characteristic of heavy charged particles such as protons and alpha particles where the distance between ionizing events is small on the scale of a cellular nucleus.
>
> **Low-LET**—Energy transfer characteristic of light charged particles such as electrons produced by x and gamma rays where the distance between ionizing events is large on the scale of a cellular nucleus.

Lowest-Observed-Adverse-Effect Level (LOAEL)—The lowest dose of chemical in a study, or group of studies, that produces statistically or biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control.

Lung Clearance Class (fast, F; medium, M; slow, S)—A classification scheme for inhaled material according to its rate of clearance from the pulmonary region of the lungs to the blood and the gastrointestinal tract.

Lymphoreticular Effects—Represent morphological effects involving lymphatic tissues such as the lymph nodes, spleen, and thymus.

Malformations—Permanent structural changes that may adversely affect survival, development, or function.

Mass Numbers (A)—The number of nucleons (protons and neutrons) in the nucleus of an atom.

Minimal Risk Level—An estimate of daily human exposure to a substance that is likely to be without an appreciable risk of adverse noncancerous effects over a specified duration of exposure.

Morbidity—State of being diseased; morbidity rate is the incidence or prevalence of disease in a specific population.

Mutagen—A substance that causes changes (mutations) in the genetic material in a cell. Mutations can lead to birth defects, miscarriages, or cancer.

Necropsy—The gross examination of the organs and tissues of a dead body to determine the cause of death or pathological conditions.

Neurotoxicity—The occurrence of adverse effects on the nervous system following exposure to a substance.

Neutrino (v)—A neutral particle of infinitesimally small rest mass emitted during beta plus or beta minus decay. This particle accounts for conservation of energy in beta plus and beta minus decays. It plays no role in damage from radiation.

No-Observed-Adverse-Effect Level (NOAEL)—The dose of a substance at which there were no statistically or biologically significant increases in frequency or severity of adverse effects seen between the exposed population and its appropriate control. Effects may be produced at this dose, but they are not considered to be adverse.

Nuclear Reactor—A power plant that heats the medium (typically water) by using the energy released from the nuclear fission of uranium or plutonium isotopes instead of burning coal, oil, or natural gas. All of these sources of energy simply heat water and use the steam which is produced to turn turbines that make electricity or propel a ship.

Nucleon—Common name for a constituent particle of the nucleus. Applied to a proton or neutron.

Nuclide—A species of atom characterized by the constitution of its nucleus. The nuclear constitution is specified by the number of protons (Z), number of neutrons (N), and energy content; or, alternatively, by the atomic number (Z), mass number A(N+Z), and atomic mass. To be regarded as a distinct nuclide, the atom must be capable of existing for a measurable time. Thus, nuclear isomers are separate nuclides, whereas promptly decaying excited nuclear states and unstable intermediates in nuclear reactions are not so considered.

Octanol-Water Partition Coefficient (K_{ow})—The equilibrium ratio of the concentrations of a chemical in n-octanol and water, in dilute solution.

Odds Ratio (OR)—A means of measuring the association between an exposure (such as toxic substances and a disease or condition) which represents the best estimate of relative risk (risk as a ratio of the incidence among subjects exposed to a particular risk factor divided by the incidence among subjects who were not exposed to the risk factor). An odds ratio of greater than 1 is considered to indicate greater risk of disease in the exposed group compared to the unexposed.

Pair Production—An absorption process for x- and gamma radiation in which the incident photon is absorbed in the vicinity of the nucleus of the absorbing atom, with subsequent production of an electron and positron pair (see annihilation). This reaction can only occur for incident photon energies exceeding 1.02 MeV.

Parent—Any radionuclide nuclide which, upon disintegration, yields a new nuclide (termed the progeny or daughter), either directly or as a later member of a radioactive series.

Permissible Exposure Limit (PEL)—A maximum allowable atmospheric level of a substance in workplace air averaged over an 8-hour shift.

Pharmacokinetic Model—A set of equations that can be used to describe the time course of a parent chemical or metabolite in an animal system. There are two types of pharmacokinetic models: data-based and physiologically-based. A data-based model divides the animal system into a series of compartments which, in general, do not represent real, identifiable anatomic regions of the body whereas the physiologically-based model compartments represent real anatomic regions of the body.

Pharmacokinetics—The dynamic behavior of a material in the body, used to predict the fate (disposition) of an exogenous substance in an organism. Utilizing computational techniques, it provides the means of studying the absorption, distribution, metabolism and excretion of chemicals by the body.

Physiologically Based Pharmacodynamic (PBPD) Model—A type of physiologically-based dose-response model which quantitatively describes the relationship between target tissue dose and toxic end points. These models advance the importance of physiologically based models in that they clearly describe the biological effect (response) produced by the system following exposure to an exogenous substance.

Physiologically Based Pharmacokinetic (PBPK) Model—A model comprising a series of compartments representing organs or tissue groups with realistic weights and blood flows. These models require a variety of physiological information: tissue volumes, blood flow rates to tissues, cardiac output, alveolar ventilation rates and, possibly membrane permeabilities. The models also utilize biochemical information such as air/blood partition coefficients, and metabolic parameters. PBPK models are also called biologically based tissue dosimetry models.

Photoelectric Effect—An attenuation process observed for x and gamma radiation in which an incident photon interacts with a tightly bound inner orbital electron of an atom delivering all of its energy to knock the electron out of the atom. The incident photon disappears in the process.

Photon—A quantum of electromagnetic energy (E) whose value is the product of its frequency (v) in hertz and Planck's constant (h). The equation is: $E = hv$.

Population dose—See Collective dose.

Positron—A positively charged electron.

Potential, Ionization—The energy expressed as electron volts (eV) necessary to separate one electron from an atom, resulting in the formation of an ion pair.

Power, Stopping—A measure of the ability of a material to absorb energy from an ionizing particle passing through it; the greater the stopping power, the greater the energy absorbing ability (see Linear Energy Transfer).

Progeny—The decay product or daughter products resulting after a radioactive decay or a series of radioactive decays. The progeny can also be radioactive, and the chain continues until a stable nuclide is formed.

Proton—Elementary nuclear particle with a positive electric charge equal numerically to the charge of the electron and a rest mass of 1.007 mass units.

Quality—A term describing the distribution of the energy deposited by a particle along its track; radiations that produce different densities of ionization per unit intensity are said to have different "qualities."

Quality Factor (Q)—The linear-energy-transfer-dependent factor by which absorbed doses are multiplied to obtain (for radiation protection purposes) a quantity that expresses - on a common scale for all ionizing radiation - the approximate biological effectiveness of the absorbed dose.

Type of radiation	Quality Factor
X, gamma, or beta	1
Alpha particles	20
Neutrons of unknown energy	10
High energy protons	10

Rad—The traditional unit of absorbed dose equal to 100 ergs per gram, or 0.01 joule per kilogram (0.01 Gy) in any medium (see Absorbed Dose).

Radiation—The emission and propagation of energy through space or through a material medium in the form of waves (e.g., the emission and propagation of electromagnetic waves, or of sound and elastic waves). The term radiation or radiant energy, when unqualified, usually refers to electromagnetic radiation. Such radiation commonly is classified according to frequency, as microwaves, infrared, visible (light), ultraviolet, and x and gamma rays (see Photon.) and, by extension, corpuscular emission, such as alpha and beta radiation, neutrons, or rays of mixed or unknown type, as cosmic radiation.

Radiation, Annihilation—Photons produced when an electron and a positron unite and cease to exist. The annihilation of a positron-electron pair results in the production of two photons, each of 0.51 MeV energy.

Radiation, Background—See Background Radiation.

Radiation, Characteristic (Discrete)—Radiation originating from an excited atom after removal of an electron from an atom. The wavelength of the emitted radiation is specific, depending only on the element and particular energy levels involved.

Radiation, External—Radiation from a source outside the body.

Radiation, Internal—Radiation from a source within the body (as a result of deposition of radionuclides in body tissues).

Radiation, Ionizing—Any electromagnetic or particulate radiation capable of producing ions, directly or indirectly, in its passage through matter (see Radiation).

Radiation, Monoenergetic—Radiation of a given type in which all particles or photons originate with and have the same energy.

Radiation, Scattered—Radiation which during its passage through a substance, has been deviated in direction. It may also have been modified by a decrease in energy.

Radiation, Secondary—A particle or ray that is produced when the primary radiation interacts with a material, and which has sufficient energy to produce its own ionization, such as bremsstrahlung or electrons knocked from atomic orbitals with enough energy to then produce ionization (see Delta Rays).

Radiation Weighting Factor (also called Quality Factor)—In radiation protection, a factor (1 for x-rays, gamma rays, beta particles; 20 for alpha particles) weighting the absorbed dose of radiation of a specific type and energy for its effect on tissue.

Radioactive Material—Material containing radioactive atoms.

Radioactivity—Spontaneous nuclear transformations that result in the formation of new elements. These transformations are accomplished by emission of alpha or beta particles from the nucleus or by the capture of an orbital electron. Each of these reactions may or may not be accompanied by a gamma photon.

Radioactivity, Artificial—Man-made radioactivity produced by particle bombardment or nuclear fission, as opposed to naturally occurring radioactivity.

Radioactivity, Induced—Radioactivity produced in a substance after bombardment with neutrons or other particles. The resulting activity is "natural radioactivity" if formed by nuclear reactions occurring in nature and "artificial radioactivity" if the reactions are caused by man.

Radioactivity, Natural—The property of radioactivity exhibited by more than 50 naturally occurring radionuclides.

Radioisotope—An unstable or radioactive isotope of an element that decays or disintegrates spontaneously, emitting radiation.

Radionuclide—Any radioactive isotope of any element. Approximately 5,000 natural and artificial radioisotopes have been identified.

Radiosensitivity—Relative susceptibility of cells, tissues, organs, organisms, or any living substance to the injurious action of radiation. Radiosensitivity and its antonym, radioresistance, are used comparatively, rather than absolutely.

Reference Dose (RfD)—An estimate of the daily exposure of the human population to a potential hazard that is likely to be without risk of deleterious effects during a lifetime. The RfD is operationally derived from the NOAEL (from animal and human studies) by a consistent application of uncertainty factors that reflect various types of data used to estimate RfDs and an additional modifying factor, which is based on a professional judgment of the entire database on the chemical. The RfDs are not applicable to non-threshold effects such as cancer.

Relative Biological Effectiveness (RBE)—The RBE is a factor used to compare the biological effectiveness of absorbed radiation doses (i.e., rad) due to different types of ionizing radiation. More specifically, it is the experimentally determined ratio of an absorbed dose of a radiation in question to the absorbed dose of a reference radiation (typically ^{60}Co gamma rays or 200 kVp x rays) required to produce an identical biological effect in a particular experimental organism or tissue (see Quality Factor).

Rem—The traditional unit of dose equivalent that is used in the regulatory, administrative, and engineering design aspects of radiation safety practice. The dose equivalent in rem is numerically equal to the absorbed dose in rad multiplied by the quality factor (1 rem is equal to 0.01 sievert).

Reportable Quantity (RQ)—The quantity of a hazardous substance that is considered reportable under CERCLA. Reportable quantities are (1) 1 pound or greater or (2) for selected substances, an amount established by regulation either under CERCLA or under Sect. 311 of the Clean Water Act. Quantities are measured over a 24-hour period.

Reproductive Toxicity—The occurrence of adverse effects on the reproductive system that may result from exposure to a chemical. The toxicity may be directed to the reproductive organs and/or the related endocrine system. The manifestation of such toxicity may be noted as alterations in sexual behavior, fertility, pregnancy outcomes, or modifications in other functions that are dependent on the integrity of this system.

Roentgen (R)—A unit of exposure (in air) to ionizing radiation. It is the amount of x or gamma rays required to produce ions carrying 1 electrostatic unit of electrical charge in 1 cubic centimeter of dry air under standard conditions. Named after William Roentgen, a German scientist who discovered x rays in 1895.

Retrospective Study—A type of cohort study based on a group of persons known to have been exposed at some time in the past. Data are collected from routinely recorded events, up to the time the study is undertaken. Retrospective studies are limited to causal factors that can be ascertained from existing records and/or examining survivors of the cohort.

Self-Absorption—Absorption of radiation (emitted by radioactive atoms) by the material in which the atoms are located; in particular, the absorption of radiation within a sample being assayed.

Short-Term Exposure Limit (STEL)—The maximum concentration to which workers can be exposed for up to 15 minutes continually. No more than four excursions are allowed per day, and there must be at least 60 minutes between exposure periods. The daily TLV-TWA may not be exceeded.

SI Units—The International System of Units as defined by the General Conference of Weights and Measures in 1960. These units are generally based on the meter/kilogram/second units, with special quantities for radiation including the becquerel, gray, and sievert.

Sickness, Acute Radiation (Syndrome)—The complex symptoms and signs characterizing the condition resulting from excessive exposure of the whole body (or large part) to ionizing radiation. The earliest of these symptoms are nausea, fatigue, vomiting, and diarrhea, and may be followed by loss of hair (epilation), hemorrhage, inflammation of the mouth and throat, and general loss of energy. In severe cases, where the radiation dose is relatively high (over several hundred rad or several gray), death may occur within two to four weeks. Those who survive six weeks after exposure of a single high dose of radiation may generally be expected to recover.

Sievert (Sv)—The SI unit of any of the quantities expressed as dose equivalent. The dose equivalent in sieverts is equal to the absorbed dose, in gray, multiplied by the quality factor (1 sievert equals 100 rem). The sievert is also the SI unit for effective dose equivalent, which is the sum of the products of the dose equivalent to each organ or tissue and its corresponding tissue weighting factor.

Specific-Activity—Radioactivity per unit mass of a radionuclide, expressed, for example, as Ci/gram or Bq/kilogram.

Specific Energy—The actual energy per unit mass deposited per unit volume in a small target, such as the cell or cell nucleus, as the result of one or more energy-depositing events. This is a stochastic quantity as opposed to the average value over a large number of instance (i.e., the absorbed dose).

Standardized Mortality Ratio (SMR)—A ratio of the observed number of deaths and the expected number of deaths in a specific standard population.

Stochastic Effect—A health effect that occurs randomly and for which the probability of the effect occurring, rather than its severity, is assumed to be a linear function of dose without a threshold (also called a nondeterministic effect).

Stopping Power—The average rate of energy loss of a charged particle per unit thickness of a material or per unit mass of material traversed.

Surface-seeking Radionuclide—A bone-seeking internal emitter that deposits and remains on the bone surface for a long period of time, although it may eventually diffuse into the bone mineral. This contrasts with a volume seeker, which deposits more uniformly throughout the bone volume.

Target Organ Toxicity—This term covers a broad range of adverse effects on target organs or physiological systems (e.g., renal, cardiovascular) extending from those arising through a single limited exposure to those assumed over a lifetime of exposure to a chemical.

Target Theory (Hit Theory)—A theory explaining some biological effects of radiation on the basis that ionization, occurring in a discrete volume (the target) within the cell, directly causes a lesion which subsequently results in a physiological response to the damage at that location. One, two, or more "hits" (ionizing events within the target) may be necessary to elicit the response.

Teratogen—A chemical that causes birth defects.

Threshold Limit Value (TLV®)—The maximum concentration of a substance to which most workers can be exposed without adverse effect. TLV is a term used exclusively by the ACGIH. Other terms used to express similar concepts are the MAC (Maximum Allowable Concentration) and PEL (Permissible Exposure Limits).

Time-Weighted Average (TWA)—An allowable exposure concentration averaged over a normal 8-hour workday or 40-hour workweek.

Tissue Weighting Factor (W_t)—Organ- or tissue-specific factor by which the equivalent dose is multiplied to give the portion of the effective dose for that organ or tissue. Recommended values of tissue weighting factors are:

Tissue/Organ	Tissue Weighting Factor
Gonads	0.70
Bone marrow (red)	0.12
Colon	0.12
Lung	0.12
Stomach	0.12
Bladder	0.05
Breast	0.05
Liver	0.05
Esophagus	0.05
Thyroid	0.05
Skin	0.01
Bone surface	0.01
Remainder (adrenals, brain, upper large intestine, small intestine, pancreas, spleen, thymus, and uterus)	0.05

Toxic Dose (TD_{50})—A calculated dose of a chemical, introduced by a route other than inhalation, which is expected to cause a specific toxic effect in 50% of a defined experimental animal population.

Toxicokinetic—The absorption, distribution and elimination of toxic compounds in the living organism.

Toxicosis—A diseased condition resulting from poisoning.

Transformation, Nuclear—The process of radioactive decay by which a nuclide is transformed into a different nuclide by absorbing or emitting particulate or electromagnetic radiation.

Transition, Isomeric—The process by which a nuclide decays to an isomeric nuclide (i.e., one of the same mass number and atomic number) of lower quantum energy. Isomeric transitions (often abbreviated I.T.) proceed by gamma ray and internal conversion electron emission.

Tritium—The hydrogen isotope with one proton and two neutrons in the nucleus (Symbol: ^{3}H). It is radioactive and has a physical half-life of 12.3 years.

Unattached Fraction—That fraction of the radon daughters, usually ^{218}Po and ^{214}Po, which has not yet attached to a dust particle or to water vapor. As a free atom, it has a high probability of being exhaled and not retained within the lung. It is the attached fraction which is primarily retained.

Uncertainty Factor (UF)—A factor used in operationally deriving the RfD from experimental data. UFs are intended to account for (1) the variation in sensitivity among the members of the human population, (2) the uncertainty in extrapolating animal data to the case of human, (3) the uncertainty in extrapolating from data obtained in a study that is of less than lifetime exposure, and (4) the uncertainty in using LOAEL data rather than NOAEL data. Usually each of these factors is set equal to 10.

Units, Prefixes—Many units of measure are expressed as submultiples or multiples of the primary unit (e.g., 10^{-3} curie is 1 mCi and 10^{3} becquerel is 1 kBq).

Factor	Prefix	Symbol	Factor	Prefix	Symbol
10^{-18}	atto	A	10^{3}	kilo	k
10^{-15}	femto	F	10^{6}	mega	M
10^{-12}	pico	p	10^{9}	giga	G
10^{-9}	nano	N	10^{12}	tera	T
10^{-6}	micro	M	10^{15}	peta	P
10^{-3}	milli	M	10^{18}	exa	E
10^{-2}	centi	C			

Units, Radiological—

Units	Equivalents
Becquerel* (Bq)	1 disintegration per second = 2.7×10^{-11} Ci
Curie (Ci)	3.7×10^{10} disintegrations per second = 3.7×10^{10} Bq
Gray* (Gy)	1 J/kg = 100 rad
Rad (rad)	100 erg/g = 0.01 Gy
Rem (rem)	0.01 sievert
Sievert* (Sv)	100 rem

*International Units, designated (SI)

Working Level (WL)—Any combination of short-lived radon daughters in 1 liter of air that will result in the ultimate emission of 1.3×10^{5} MeV of potential alpha energy.

Working Level Month (WLM)—A unit of exposure to radon daughters corresponding to the product of the radon daughter concentration in Working Level (WL) and the exposure time in nominal months (1 nominal month = 170 hours). Inhalation of air with a concentration of 1 WL of radon daughters for 170 working hours results in an exposure of 1 WLM.

X rays—Penetrating electromagnetic radiations whose wave lengths are very much shorter than those of visible light. They are usually produced by bombarding a metallic target with fast electrons in a high vacuum. X rays (called characteristic x rays) are also produced when an orbital electron falls from a high energy level to a low energy level.

Zero-Threshold Linear Hypothesis (or No-Threshold Linear Hypothesis)—The assumption that a dose-response curve derived from data in the high dose and high dose-rate ranges may be extrapolated through the low dose and low dose range to zero, implying that, theoretically, any amount of radiation will cause some damage.

APPENDIX A. ATSDR MINIMAL RISK LEVELS AND WORKSHEETS

The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) [42 U.S.C. 9601 et seq.], as amended by the Superfund Amendments and Reauthorization Act (SARA) [Pub. L. 99–499], requires that the Agency for Toxic Substances and Disease Registry (ATSDR) develop jointly with the U.S. Environmental Protection Agency (EPA), in order of priority, a list of hazardous substances most commonly found at facilities on the CERCLA National Priorities List (NPL); prepare toxicological profiles for each substance included on the priority list of hazardous substances; and assure the initiation of a research program to fill identified data needs associated with the substances.

The toxicological profiles include an examination, summary, and interpretation of available toxicological information and epidemiologic evaluations of a hazardous substance. During the development of toxicological profiles, Minimal Risk Levels (MRLs) are derived when reliable and sufficient data exist to identify the target organ(s) of effect or the most sensitive health effect(s) for a specific duration for a given route of exposure. An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified duration of exposure. MRLs are based on noncancer health effects only and are not based on a consideration of cancer effects. These substance-specific estimates, which are intended to serve as screening levels, are used by ATSDR health assessors to identify contaminants and potential health effects that may be of concern at hazardous waste sites. It is important to note that MRLs are not intended to define clean-up or action levels.

MRLs are derived for hazardous substances using the no-observed-adverse-effect level/uncertainty factor approach. They are below levels that might cause adverse health effects in the people most sensitive to such chemical-induced effects. MRLs are derived for acute (1–14 days), intermediate (15–364 days), and chronic (365 days and longer) durations and for the oral and inhalation routes of exposure. Currently, MRLs for the dermal route of exposure are not derived because ATSDR has not yet identified a method suitable for this route of exposure. MRLs are generally based on the most sensitive chemical-induced end point considered to be of relevance to humans. Serious health effects (such as irreparable damage to the liver or kidneys, or birth defects) are not used as a basis for establishing MRLs. Exposure to a level above the MRL does not mean that adverse health effects will occur.

MRLs are intended only to serve as a screening tool to help public health professionals decide where to look more closely. They may also be viewed as a mechanism to identify those hazardous waste sites that

are not expected to cause adverse health effects. Most MRLs contain a degree of uncertainty because of the lack of precise toxicological information on the people who might be most sensitive (e.g., infants, elderly, nutritionally or immunologically compromised) to the effects of hazardous substances. ATSDR uses a conservative (i.e., protective) approach to address this uncertainty consistent with the public health principle of prevention. Although human data are preferred, MRLs often must be based on animal studies because relevant human studies are lacking. In the absence of evidence to the contrary, ATSDR assumes that humans are more sensitive to the effects of hazardous substance than animals and that certain persons may be particularly sensitive. Thus, the resulting MRL may be as much as 100-fold below levels that have been shown to be nontoxic in laboratory animals.

Proposed MRLs undergo a rigorous review process: Health Effects/MRL Workgroup reviews within the Division of Toxicology, expert panel peer reviews, and agency-wide MRL Workgroup reviews, with participation from other federal agencies and comments from the public. They are subject to change as new information becomes available concomitant with updating the toxicological profiles. Thus, MRLs in the most recent toxicological profiles supersede previously published levels. For additional information regarding MRLs, please contact the Division of Toxicology, Agency for Toxic Substances and Disease Registry, 1600 Clifton Road NE, Mailstop F-32, Atlanta, Georgia 30333.

APPENDIX A

MINIMAL RISK LEVEL (MRL) WORKSHEET

Chemical Name: Cobalt
CAS Number: 10026-24-1
Date: March 2004
Profile Status: Final
Route: [x] Inhalation [ ] Oral
Duration: [ ] Acute [ ] Intermediate [x] Chronic
Key to figure: 26
Species: human

Minimal Risk Level: 1×10^{-4} [ ] mg/kg/day [ ] ppm [x] mg/m^3

Reference:

Nemery B, Casier P, Roosels D, et al. 1992. Survey of cobalt exposure and respiratory health in diamond polishers. Am Rev Respir Dis 145:610-616.

Experimental design:

Nemery et al. (1992) conducted a cross-sectional study of cobalt exposure and respiratory effects in diamond polishers. The study group was composed of 194 polishers working in 10 different workshops. In two of these workshops (#1, 2), the workers used cast iron polishing disks almost exclusively, and in the others, they used cobalt-containing disks primarily. The number of subjects from each workshop varied from 6 to 28 and the participation rate varied from 56 to 100%. The low participation in some workshops reflects the fact that only workers who used cobalt disks were initially asked to be in the study, rather than a high refusal rate (only eight refusals were documented). More than a year after the polishing workshops were studied, an additional three workshops with workers engaged in sawing diamonds, cleaving diamonds, or drawing jewelry were studied as an unexposed control group (n=59 workers). Subjects were asked to fill out a questionnaire regarding employment history, working conditions, medical history, respiratory symptoms, and smoking habits, to give a urine sample for cobalt determination, and to undergo a clinical examination and lung function tests. Both area air samples and personal air samples were collected (always on a Thursday). Sampling for area air determinations started 2 hours after work began and continued until 1 hour before the end of the work day. Personal air samples were collected from the breathing zone of a few workers per workshop for four successive 1-hour periods. Air samples were analyzed for cobalt and iron. In addition, personal air samplers were used to sample the air 1 cm above the polishing disks. These samples were analyzed for the entire spectrum of mineral and metallic compounds. Air samples were not obtained at one of the polishing workshops (#4), but this workshop was reported to be almost identical to an adjoining workshop (#3) for which samples were obtained. Urinary cobalt levels were similar between workers in these two workshops, so exposure was considered to be similar as well. It is important to note that the study authors suggested that the available methods used for air sampling may have underestimated the exposure levels.

There was a good correlation (R=0.92) between the results of area and personal air sampling, with area air sampling reporting lower concentrations than personal air samples in all workshops except one (#9) (Nemery et al. 1992). In this workshop, personal air samples appeared to be artificially low in comparison to area air samples and urinary cobalt levels of the workers. When this workshop was excluded, there was a good correlation (R=0.85–0.88) between urinary cobalt and cobalt in the air. Based on urinary cobalt levels, the concentration of cobalt expected in personal air samples from workshop #9 was about 45 μg/m^3 (the mean value actually reported was 6 μg/m^3). The polishing workshops were divided into two groups: those with low exposure to cobalt (#1–5, n=102) and those with high exposure to

cobalt (#6–10, n=91). Mean cobalt exposure concentrations were 0.4, 1.6, and 10.2 μg/m^3 by area air sampling and 0.4, 5.3, and 15.1 μg/m^3 by personal air sampling in the control, low-exposure, and high-exposure groups, respectively. The inclusion of the apparently biased personal air samples from workshop #9 means that the reported mean cobalt exposure in the high-exposure group obtained by personal air sampling (15.1 μg/m^3) may be lower than the true value. Air concentrations of iron were highest in the two polishing workshops that used iron disks and the sawing workshop (highest value =62 μg/m^3), and were not correlated with cobalt levels. Analysis of samples taken near the disks showed the presence of cobalt, with occasional traces of copper, zinc, titanium, manganese, chromium, silicates, and silicon dioxide. No tungsten was detected. There is a possibility that some workers had previously been exposed to asbestos, since pastes containing asbestos had been used in the past to glue the diamonds onto holders. However, the degree of asbestos exposure had apparently been insufficient to produce functional impairment. The researchers considered cobalt to be the only relevant exposure. Smoking habits were similar in workers from the high-exposure, low-exposure, and control groups. Duration of exposure was not discussed.

Effects noted in study and corresponding doses:

Workers in the high-exposure group were more likely than those in the other groups to complain about respiratory symptoms; the prevalences of eye, nose, and throat irritation and cough, and the fraction of these symptoms related to work, were significantly increased in the high-exposure group (Nemery et al. 1992). Workers in the high-exposure group also had significantly reduced lung function compared to controls and low-exposure group workers, as assessed by FVC (forced vital capacity), FEV_1 (forced expiratory volume in 1 second), MMEF (forced expiratory flow between 25 and 75% of the FVC), and mean PEF (peak expiratory flow rate), although the prevalence of abnormal values did not differ significantly between exposure categories. Results in the low-exposure group did not differ from controls. Two-way analysis of variance was used to show that the effect on spirometric parameters in the high exposure group was present in both men and women. Women seemed to be affected more than men, but the interaction between exposure and sex was not significant. Smoking was found to exert a strong effect on lung function, but lung function level remained negatively correlated with exposure to cobalt, independently of smoking.

Dose and end point used for MRL derivation:

[x] NOAEL [ ] LOAEL
Nemery et al. (1992) established a NOAEL of 0.0053 mg cobalt/m^3 for effects on pulmonary function (decreased values upon spirometric examination).

Uncertainty Factors used in MRL derivation:

[x] 1 [ ] 3 [ ] 10 (for use of a NOAEL)
[x] 1 [ ] 3 [ ] 10 (for extrapolation from animals to humans)
[ ] 1 [ ] 3 [x] 10 (for human variability)

The chronic inhalation MRL for cobalt is derived as follows:

$MRL = NOAEL_{[ADJ]} \div UF$
$MRL = 0.0013$ mg cobalt/m$^3 \div 10$
$MRL = 1x10^{-4}$ mg cobalt/m^3

Was a conversion used from ppm in food or water to a mg/body weight dose? No.

Was a conversion used from intermittent to continuous exposure? If so, explain:

0.0053 mg cobalt/m^3 * (8 hours/24 hours) * (5 days/7 days) = 0.0013 mg cobalt/m^3 continuous exposure.

If an inhalation study in animals, list the conversion factors used in determining human equivalent dose: NA.

Other additional studies or pertinent information which lend support to this MRL:

Necrosis and inflammation of the respiratory tract epithelium (larynx, trachea, bronchioles, nasal turbinates) were reported in rats exposed to 19 mg cobalt/m^3 and mice exposed to 1.9 mg cobalt/m^3 (and above) as cobalt sulfate over 16 days (NTP 1991). Exposure of rats and mice to cobalt as cobalt sulfate for 13 weeks resulted in adverse effects on all parts of the respiratory tract, with the larynx being the most sensitive part (NTP 1991). At concentrations of ≥0.11 mg cobalt/m^3, rats and mice had squamous metaplasia of the larynx. Histiocytic infiltrates in the lung were also reported at similar levels in both the rats and mice. In rats, chronic inflammation of the larynx was found at ≥0.38 mg cobalt/m^3, and more severe effects on the larynx, nose, and lung were reported at higher exposures. In mice, acute inflammation of the nose was found at ≥1.14 mg cobalt/m^3, and more severe effects on the larynx, nose, and lung were reported at higher exposures.

Exposure of rats and mice to aerosols of cobalt (as cobalt sulfate) at concentrations from 0.11 to 1.14 mg cobalt/m^3 for 2 years resulted in a spectrum of inflammatory, fibrotic, and proliferative lesions in the respiratory tract of male and female rats and mice (NTP 1998). Squamous metaplasia of the larynx occurred in rats and mice at exposure concentrations of ≥0.11 mg cobalt/m^3, with severity of the lesion increasing with increased exposure concentration. Hyperplastic lesions of the nasal epithelium occurred in rats at concentrations of ≥0.11 mg cobalt/m^3, and in mice at concentrations of ≥0.38 mg cobalt/m^3. Both sexes of rats had greatly increased incidences (>90% incidence) of alveolar lesions at all exposure levels, including inflammatory changes, fibrosis, and metaplasia. Similar changes were seen in mice at all exposure levels, though the changes in mice were less severe.

Both studies by NTP (1991, 1998) failed to define a NOAEL, with the lowest concentration examined (0.11 mg/m^3) a LOAEL for a variety of respiratory effects. If an MRL were to be calculated based upon these studies, it would be as follows:

Duration adjustment: 0.11 mg cobalt/m^3 * (6 h/24 h) * (5 d/7 d) = 0.020 mg cobalt/m^3 continuous exposure.
Calculation of human equivalent concentration:
If fractional depositions in humans and animals are assumed to be equal, then:
$RDDR = V_E(animal)/S_{ET}(animal) \div V_E(human)/S_{ET}(human) = 0.24\ m^3/day\ /\ 15\ cm^2 \div 20\ m^3/day\ /\ 200\ cm^2$
RDDR = 0.16
$LOAEL_{[HEC]} = LOAEL[ADJ] * RDDR$
= 0.020 mg cobalt/m^3 * 0.16 = 0.0032 mg cobalt/m^3

To the $LOAEL_{[HEC]}$, an uncertainty factor of 300 (10 for use of a LOAEL, 3 for animal to human extrapolation, and 10 for human variability) to derive an MRL of $1x10^{-5}$ mg/m^3. This number is an order of magnitude lower than the number derived from the Nemery et al. (1992) data, reflecting the fact that it is derived from animal data, not from a human study, and is based on a LOAEL, not a NOAEL. As the Nemery et al. (1992) study was a well-performed study in humans that defined a NOAEL and LOAEL, it was selected as the basis for derivation of the MRL.

Agency Contact (Chemical Manager): Obaid Faroon D.V.M., Ph.D.

MINIMAL RISK LEVEL (MRL) WORKSHEET

Chemical Name: Cobalt
CAS Number: 10026-24-1
Date: March 2004
Profile Status: Final
Route: [ ] Inhalation [x] Oral
Duration: [ ] Acute [x] Intermediate [ ] Chronic
Key to figure: 30
Species: human

Minimal Risk Level: 1×10^{-4} [x] mg/kg/day [ ] ppm [ ] mg/m^3

Reference:

Davis, J.E. and Fields, J.P. 1958. Experimental production of polycythemia in humans by administration of cobalt chloride. Proc Soc Exp Biol Med 99:493-495.

Experimental design:

Six apparently normal men, ages 20–47, were administered a daily dose of cobalt chloride, administered as a 2% solution diluted in either water or milk, for up to 22 days. Five of the six received 150 mg cobalt chloride per day for the entire exposure period, while the sixth was started on 120 mg/day and later increased to 150 mg/day. Blood samples were obtained daily from free-flowing punctures of fingertips at least 2 hours after eating, and at least 15 hours after the last dosage of cobalt. Blood was analyzed for red blood cell counts, hemoglobin percentage, leukocyte counts, reticulocyte percentages, and thrombocyte counts.

Effects noted in study and corresponding doses:

Exposure to cobalt resulted in the development of polycythemia in all six subjects, with increases in red blood cell numbers ranging from 0.5 to 1.19 million (~16–20% increase above pre-treatment levels). Polycythemic erythrocyte counts returned to normal 9–15 days after cessation of cobalt administration. Hemoglobin levels were also increased by cobalt treatment, though to a lesser extent than the erythrocyte values, with increases of 6–11% over pretreatment values. In five of the six subjects, reticulocyte levels were elevated, reaching at least twice the pre-experiment values. Thrombocyte and total leukocyte counts did not deviate significantly from pretreatment values.

Dose end point used for MRL derivation:

[ ] NOAEL [x] LOAEL

Davis and Fields (1958) identified a LOAEL of 150 mg cobalt chloride per day for increased levels of erythrocytes in volunteers. 150 mg cobalt chloride/day corresponds to ~1 mg Co/kg/day, assuming a reference body weight of 70 kg. Available animal studies, presented below, lend support to this LOAEL, having demonstrated LOAEL values within half an order of magnitude of that identified by Davis and Fields (1958).

Uncertainty factors used in MRL derivation:

[ ] 1 [ ] 3 [x] 10 (for use of a LOAEL)

[x] 1 [ ] 3 [ ] 10 (for extrapolation from animals to humans)
[ ] 1 [ ] 3 [x] 10 (for human variability)

The intermediate oral MRL for cobalt is derived as follows:

MRL = LOAEL ÷ UF
MRL = 1 mg cobalt/kg-day ÷ 100
MRL = 1×10^{-2} mg cobalt/kg-day

Was a conversion factor used from ppm in food or water to a mg/body weight dose? No.

Was a conversion used from intermittent to continuous exposure? If so, explain: No.

If an inhalation study in animals, list conversion factors used in determining human equivalent dose: Not applicable.

Other additional studies or pertinent information that lend support to this MRL:

No other studies of the effect of intermediate oral cobalt exposure on erythrocyte levels in healthy human subjects were identified in a search of the literature. Treatment of pregnant women for 90 days with 0.5–0.6 mg cobalt/kg/day as cobalt chloride did not prevent the reduction in hematocrit and hemoglobin levels often found during pregnancy (Holly 1955). However, treatment of anephric patients (with resulting anemia) with 0.16–1.0 mg cobalt/kg/day daily as cobalt chloride for 3–32 weeks resulted in increased levels of circulating erythrocytes and a decreased need for transfusions (Duckham and Lee 1976b; Taylor et al. 1977). While these studies provide additional evidence that exposure to cobalt can increase erythrocyte levels in humans, the fact that the patients were anephric makes definitive interpretation of the results more difficult.

Roche and Layrisse (1956) exposed volunteers to similar levels (150 mg $CoCl_2$/day) of cobalt, and reported a reversible decrease in uptake of ^{131}I by the thyroid. The decreased uptake is believed to result from cobalt blocking the organic binding of iodine (Paley et al. 1958). This observation adds support to the choice of effect level, as a similar exposure resulted in measurable effects in volunteers, though whether the changes in iodine uptake operate through the same mechanisms as the changes in erythrocyte numbers has not been determined.

Stanley et al. (1947) exposed groups (n=4, 6 for controls) of 6 Sprague-Dawley rats to 0, 0.62, 2.5, or 10 mg cobalt/kg/day (0, 2.5, 10, or 40 mg/kg-day of $CoCl_2 \cdot 6H_2O$) in gelatin capsules for 8 weeks. Blood counts and hemoglobin levels were examined at the beginning of the experiment and at 2-week intervals. Rats exposed to 0.62 mg cobalt/kg-day showed no change in erythrocyte number. At 2.5 mg cobalt/kg-day, a progressive increase in erythrocyte number was seen, increasing up to a maximum of 17% above pretreatment values on week 6. At the highest exposure level, a progressive increase in erythrocyte numbers was seen, reaching 29% above pretreatment values at 8 weeks of exposure. Statistical analyses of the group means were not provided, and the study provided only mean values of the measurements, precluding statistical analysis. However, if a 10% change is assumed to be an effect level, exposure to 2.5 mg cobalt/kg-day was the LOAEL for this study, with a NOAEL of 0.62 mg cobalt/kg-day.

Krasovskii and Fridyland (1971) exposed groups of rats to 0, 0.05, 0.5, or 2.5 mg Co/kg/day for up to 7 months. In the 2.5 mg/kg-day group, a persistent increase in erythrocyte levels was seen. The increase was transient in the 0.5 mg/kg/day rats, and was not present in rats exposed to 0.05 mg/kg/day. However, numerical data were not presented and statistical significance was not reported.

A number of other studies in animals have reported increases in erythrocyte levels following intermediate oral administration of cobalt compounds (see the LSE table for further details of these studies). However, the majority of them have considerable methodological limitations, including examination of either very high exposure levels or only one exposure level, limited reporting of results, or limited or no statistical analysis.

Whether or not polycythemia, a condition wherein an excess of erythrocytes is produced, constitutes an adverse effect is open to interpretation. At the levels seen in the available studies, and in particular in the Davis and Fields (1958) study, the subjects would be expected to be asymptomatic. However, data on the long-term effects of elevated erythrocyte levels are not available. As such, this end point was considered an adverse effect as a health-protective assumption, and was utilized as a critical end point for MRL derivation.

Agency Contact (Chemical Manager): Obaid Faroon D.V.M., Ph.D.

APPENDIX A

MINIMAL RISK LEVEL (MRL) WORKSHEET

Chemical name: Radioactive Cobalt
CAS number: Multiple
Date:March 2004
Profile status: Final
Route: [ ] Inhalation [ ] Oral [x] External
Duration: [x] Acute [ ] Intermediate [ ] Chronic
Species: Human

Minimal Risk Level: 4 [ ] mg/kg/day [ ] ppm [ ] mg/m^3 [x] mSv (400 mrem)

References:

Schull WJ, Otake M, Yoshimaru H. 1988. Effect on intelligence test score of prenatal exposure to ionizing radiation in Hiroshima and Nagasaki: A comparison of the T65DR and DS86 dosimetry systems. Radiation Effects Research Foundation (RERF) Technical Report No. 3-88. Hirroshima, Japan. NTIS Report Number: DE89-906462.

Burt C. 1966. The genetic determination of differences in intelligence: A study of monozygotic twins reared together and apart. Brit J Psychol 57(1&2):137-153.

Experimental design:

Schull et al. (1988) study: Schull et al. (1988) evaluated the quantitative effect of exposure to ionizing radiation on the developing fetal and embryonic human brain. The end point measured was changes in intelligence test scores. The effects on individuals exposed *in utero* to the atomic bombing of Hiroshima and Nagasaki were based on the original PE86 samples (n=1,759; data on available intelligence testing) and a clinical sample (n=1,598). The original PE86 sample included virtually all prenatally exposed individuals who received tissue-absorbed doses of 0.50 Gy or more. There were many more individuals in the dose range 0–0.49 Gy in the PE86 sample than in the clinical sample. The clinical sample does not include children prenatally exposed at distances between 2,000 and 2,999 m in Hiroshima and Nagasaki. Children exposed at greater distances or not present in the city were selected as controls. In 1955–1956, Tanaka-B (emphasis on word-sense, arithmetic abilities, and the like, which were associated with the more subtle processing of visual clues than their simple recognition and depended more on connectedness) and the Koga (emphasis on perception of spatial relationships) intelligence tests were conducted in Nagasaki and the Koga test in Hiroshima.

Burt (1966) study: This study determined differences in intelligence in monozygotic twins reared together (n=95) and apart (n=53). All tests conducted in school consisted of (1) a group test of intelligence containing both non-verbal and verbal items, (2) an individual test (the London Revision of the Terman-Binet Scale) used primarily for standardization and for doubtful cases, and (3) a set of performance tests, based on the Pitner-Paterson tests and standardization. The methods and standard remained much the same throughout the study. Some of the reasons for separation of the twins were given as follows: death of the mother (n=9), unable to bring them up properly, mother's poor health (n=12), unmarried (n=6), and economic difficulties. The children were brought up by parents or foster parents (occupation ranged from unskilled to professional). IQ scores in the study group ranged from 66 to 137. The standard deviation of the group of separated monozygotic twins was reported at 15.3 as compared to 15.0 of ordinary siblings. Twins brought up in different environments were compared with those brought up in similar circumstances.

Effects noted in study and corresponding doses:

Schull et al. (1986) study: No evidence of radiation-related effect on intelligence was observed among individuals exposed within 0–7 weeks after fertilization or in the 26th or subsequent weeks. The highest risk of radiation damage to the embryonic and fetal brain occurs 8–15-weeks after fertilization under both dosimetric systems. The regression of intelligence score on estimated DS86 uterine absorbed dose is linear with dose, and the diminution in intelligence score is 21–29 points per Gy for the 8–15-week group and 10–26 points per Gy for the 16–25-week group. The results for 8–15 weeks applies regardless whether or not the mentally retarded individuals were included. The cumulative distribution of test scores suggested a progressive shift downwards in individual scores with increasing exposure. The mean IQ scores decrease significantly and systematically with uterine or fetal tissue dose within the 8–15- and 16–25-week groups.

In summary, analysis of intelligence test scores at 10–11 years of age of individuals exposed prenatally showed that:

- There is no evidence of a radiation-related effect on intelligence scores among those individuals exposed within 0–7 weeks of fertilization or in the 26th week of gestation and beyond;
- The cumulative distribution of test scores suggests a progressive shift downwards in intelligence scores with increasing exposure to ionizing radiation (dose-response relationship).
- The most sensitive group was the 8–15 weeks exposure group. The regression in intelligence scores was found to be linear, with 1 Gy dose resulting in a 21–29 point decline in intelligence scores.
- There was no indication of groups of individuals with differing sensitivities to radiation.

Burt (1966) study: The average intelligence of the twins measured on a conventional IQ scale (SD=15) was 97.8 for the separated monozygotes, 98.1 for monozygotes brought up together, 99.3 for the dizygotes as compared with 100.2 for the siblings, and 100.0 for the population as a whole. The difference of 0.3 IQ point between the separated and unseparated identical twins is considered a NOAEL for this study.

Dose endpoint used for MRL derivation:

[x] NOAEL [ ] LOAEL 0.3 IQ point reduction in twins, between those raised together and those raised apart.

Uncertainty factors (UF) used in MRL derivation:

[x] 1 [ ] 3 [ ] 10 (for use of a NOAEL)
[x] 1 [ ] 3 [ ] 10 (for extrapolation from animals to humans)
[ ] 1 [x] 3 [ ] 10 (for human variability/sensitive population)

Was a conversion factor used from ppm in food or water to a mg/body weight dose? If so, explain: No.

If an inhalation study in animals, list conversion factors used in determining human equivalent dose: NA

Was a conversion used from intermittent to continuous exposure? No.

Other additional studies or pertinent information that lend support to this MRL:

Husen (1959) reported a study involving 269 pairs of Swedish monozygotic (identical) twins where the intrapair IQ difference was 4 IQ points for a combination of twins raised together and apart. This is somewhat lower than the value of 7 IQ points for identical twins raised apart, and just larger than the range of IQ scores for Washington, DC children repetitively tested (Jacobi and Glauberman 1995).

Supporting evidence for the acute MRL is provided by Jacobi and Glauberman (1995). Children in the 1st, 3rd, and 5th grades born in Washington, DC were tested, and average IQ levels of 94.2, 97.6, and 94.6, respectively, were reported. The range of 3.4 IQ points is considered to be a LOAEL for this study, which, if used for MRL derivation, would yield an MRL of 0.004 Sv (3.4 IQ points x 1 Sv/25 IQ points ÷ 30 [10 for use of a LOAEL and 3 for a sensitive population]).

Additional supporting evidence for the acute MRL is provided by Berger et al. 1997, in a case study of accidental radiation injury to the hand. A Mexican engineer suffered an accidental injury to the hand while repairing an x-ray spectrometer. The day after the accident, his symptoms included a tingling sensation and itching in the index and middle fingers. On days 4 and 7, a "pinching" sensation, swelling, and slight erythema were observed. By day 7, the tip of his index fingers was erythematous and a large blister developed with swelling on other fingers. On day 10, examination by a physician showed that the lesions had worsened and the fingers and palms were discolored. On day 10, he was admitted to the hospital where hyperbaric oxygen therapy was administered without success. One month after the accident, the patient entered the hospital again with pain, discoloration, and desquamation of his hand. Clinical examination showed decreased circulation in the entire hand, most notably in the index and middle finger. Total white blood count decreased to 3,000/μL (normal range 4,300–10,800/μL). Cytogenic studies of peripheral blood lymphocytes revealed four dicentrics, two rings, and eight chromosomal fragments in the 300 metaphases studied. The estimated whole body dose was reported to be 0.382 Gy (38.2 rad). This dose is a potential LOAEL for acute ionizing radiation and would yield an MRL of 0.004 Sv (0.38 Sv ÷100 [10 for use of LOAEL and 10 for sensitive human population]).

The USNRC set a radiation exposure limit of 0.5 rem (50 mSv) for pregnant working women over the full gestational period (USNRC 1991). For the critical gestational period of 8–15 weeks, ATSDR believes that the conservative acute MRL of 4 mSv is consistent with the USNRC limit and could be applied to either acute (0–14-day) or intermediate (15–365-day) exposure periods.

Calculations

Given: 0.3 IQ point is a NOAEL. A 1 Sv dose results in a 25 IQ point reduction (range=21–29 points; mean=25) and provides a conversion factor from IQ prediction to radiation dose. Assume that the radiation dose and the subsequent reduction in IQ is a linear relationship.
MRL = NOAEL x CF ÷ UF
MRL = 0.3 x 1/25 ÷ 3
MRL = 0.004 Sv = 4 mSv (400 mrem)

Agency Contact (Chemical Manager): Obaid Faroon D.V.M., Ph.D.

APPENDIX A

MINIMAL RISK LEVEL (MRL) WORKSHEET

Chemical Name: Radioactive Cobalt
CAS Number: Multiple
Date: March 2004
Profile Status: Final
Route: [ ] Inhalation [ ] Oral [X] External
Duration: [ ] Acute [ ] Intermediate [X] Chronic
Species: Human

Minimal Risk Level: 1 [ ] mg/kg/day [ ] ppm [ ] mg/m^3 [X] mSv/year (100 mrem/year)

Reference: BEIR V. 1990. Health effects of exposure to low levels of ionizing radiation. Committee on the Biological Effects of Ionizing Radiations, National Research Council. National Academy Press. Washington, DC.

Experimental design: Not applicable

Effects noted in study and corresponding doses: No individual studies were identified that could be used to base a chronic-duration external exposure MRL that did not result in a cancer-producing end point. However, two sources of information were identified that did provide doses of ionizing radiation that have not been reported to be associated with detrimental effects (NOAELs). These sources provide estimates of background levels of primarily natural sources of ionizing radiation that have not been implicated in producing cancerous or noncancerous toxicological endpoints. BEIR V states that the average annual effective dose to the U.S. population is 3.6 mSv/year. A total annual effective dose equivalent of 3.6 mSv (360 mrem)/year to members of the U.S. population is obtained mainly by naturally occurring radiation from external sources, medical uses of radiation, and radiation from consumer products. The largest contribution (82%) is from natural sources, two-thirds of which is from naturally occurring radon and its decay products. Specific sources of this radiation are demonstrated in Table A-1.

The annual dose of 3.6 mSv per year has not been associated with adverse health effects or increases in the incidences of any type of cancers in humans or other animals.

Dose and end point used for MRL derivation: 3.6 mSv/year

[X] NOAEL [ ] LOAEL 3.6 mSv/year

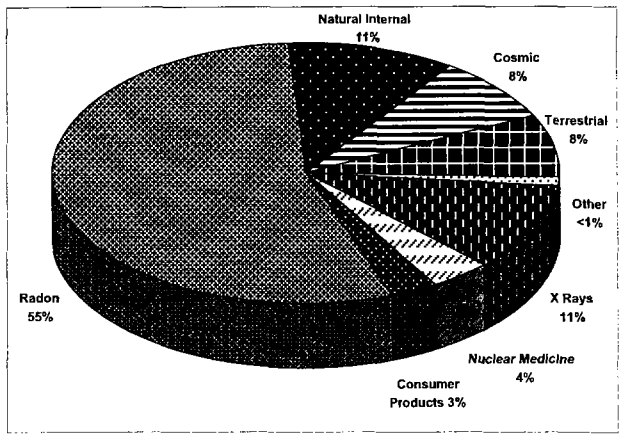
Uncertainty Factors used in MRL derivation:

[X] 1 [ ] 3 [ ] 10 (for use of a NOAEL)
[X] 1 [ ] 3 [ ] 10 (for extrapolation from animals to humans)
[ ] 1 [X] 3 [ ] 10 (for human variability)

Was a conversion used from ppm in food or water to a mg/body weight dose? No.

Table A-1. Average Annual Effective Dose Equivalent from Ionizing Radiation to a Member of the U.S. Population[a]

	Effective dose equivalent	
Source	mSv	Percent of total dose
Natural		
Radon[b]	2.0	55
Cosmic	0.27	8.0
Terrestrial	0.28	8.0
Internal	0.39	11
Total natural	3.0	82
Artificial		
Medical		
X-ray	0.39	11
Nuclear	0.14	4.0
Consumer products	0.10	3.0
Other		
Occupational	<0.01	<0.3
Nuclear fuel cycle	<0.01	<0.03
Fallout	<0.01	<0.03
Miscellaneous[c]	<0.01	<0.03
Total artificial	0.63	18
Total natural and artificial	3.6	100



[a]Adapted from BEIR V, Table 1-3 , page 18.
[b]Dose equivalent to bronchi from radon daughter products
[c]DOE facilities, smelter, transportation, etc.

If an inhalation study in animals, list the conversion factors used in determining human equivalent dose: Not applicable.

Was a conversion used from intermittent to continuous exposure? No.

Other additional studies or pertinent information which lend support to this MRL: ICRP has developed recommended dose limits for occupational and public exposure to ionizing radiation sources. The ICRP recommends limiting public exposure to 1 mSv/year (100 mrem/year), but does note that values at high altitudes above sea level and in some geological areas can sometimes be twice that value (≥2 mSv). In Annex C of ICRP 60, the commission provides data that suggests increasing the dose from 1 mSv to 5 mSv results in a very small, but detectable, increase in age-specific human mortality rate. ICRP states that the value of 1 mSv/year was chosen over the 5 mSv value because 5 mSv/year (500 mrem/year) causes this increase in age specific mortality rate, and 1 mSv/year (100 mrem/year) is typical of the annual effective dose from background, less radon (ICRP 1991). The 1 mSv estimate may underestimate the annual exposure to external sources of ionizing radiation to the U.S. population, as it does not include radiation from radon. Conversely, the 5 mSv estimate may be high, in that increases in mortality rate been reported. The most useful estimate appears to be the BEIR V estimate of 3.6 mSv, in that it accounts for an annual exposure to radon, is specific to the U.S. population, has not been associated with increases mortality, and it falls short of the 5 mSv value associated with small increases in human mortality.

Calculations:

$MRL = NOAEL_{(ADJ)} \div UF$
$MRL = 3.6$ mSv/year $\div 3$
$MRL = 1.20$ mSv/year
$MRL = 1.0$ mSv/year $=100$ mrem/year above background

Agency Contact (Chemical Manager): Obaid Faroon D.V.M., Ph.D.

APPENDIX B. USER'S GUIDE

Chapter 1

Public Health Statement

This chapter of the profile is a health effects summary written in non-technical language. Its intended audience is the general public, especially people living in the vicinity of a hazardous waste site or chemical release. If the Public Health Statement were removed from the rest of the document, it would still communicate to the lay public essential information about the chemical.

The major headings in the Public Health Statement are useful to find specific topics of concern. The topics are written in a question and answer format. The answer to each question includes a sentence that will direct the reader to chapters in the profile that will provide more information on the given topic.

Chapter 2

Relevance to Public Health

This chapter provides a health effects summary based on evaluations of existing toxicologic, epidemiologic, and toxicokinetic information. This summary is designed to present interpretive, weight-of-evidence discussions for human health end points by addressing the following questions.

1. What effects are known to occur in humans?

2. What effects observed in animals are likely to be of concern to humans?

3. What exposure conditions are likely to be of concern to humans, especially around hazardous waste sites?

The chapter covers end points in the same order that they appear within the Discussion of Health Effects by Route of Exposure section, by route (inhalation, oral, and dermal) and within route by effect. Human data are presented first, then animal data. Both are organized by duration (acute, intermediate, chronic). *In vitro* data and data from parenteral routes (intramuscular, intravenous, subcutaneous, etc.) are also considered in this chapter.

The carcinogenic potential of the profiled substance is qualitatively evaluated, when appropriate, using existing toxicokinetic, genotoxic, and carcinogenic data. ATSDR does not currently assess cancer potency or perform cancer risk assessments. Minimal Risk Levels (MRLs) for noncancer end points (if derived) and the end points from which they were derived are indicated and discussed.

Limitations to existing scientific literature that prevent a satisfactory evaluation of the relevance to public health are identified in the Chapter 3 Data Needs section.

Interpretation of Minimal Risk Levels

Where sufficient toxicologic information is available, ATSDR has derived MRLs for inhalation and oral routes of entry at each duration of exposure (acute, intermediate, and chronic). These MRLs are not meant to support regulatory action, but to acquaint health professionals with exposure levels at which adverse health effects are not expected to occur in humans.

MRLs should help physicians and public health officials determine the safety of a community living near a chemical emission, given the concentration of a contaminant in air or the estimated daily dose in water. MRLs are based largely on toxicological studies in animals and on reports of human occupational exposure.

MRL users should be familiar with the toxicologic information on which the number is based. Chapter 2, "Relevance to Public Health," contains basic information known about the substance. Other sections such as Chapter 3 Section 3.10, "Interactions with Other Substances," and Section 3.11, "Populations that are Unusually Susceptible" provide important supplemental information.

MRL users should also understand the MRL derivation methodology. MRLs are derived using a modified version of the risk assessment methodology that the Environmental Protection Agency (EPA) provides (Barnes and Dourson 1988) to determine reference doses (RfDs) for lifetime exposure.

To derive an MRL, ATSDR generally selects the most sensitive end point which, in its best judgement, represents the most sensitive human health effect for a given exposure route and duration. ATSDR cannot make this judgement or derive an MRL unless information (quantitative or qualitative) is available for all potential systemic, neurological, and developmental effects. If this information and reliable quantitative data on the chosen end point are available, ATSDR derives an MRL using the most sensitive species (when information from multiple species is available) with the highest no-observed-adverse-effect level (NOAEL) that does not exceed any adverse effect levels. When a NOAEL is not available, a lowest-observed-adverse-effect level (LOAEL) can be used to derive an MRL, and an uncertainty factor (UF) of 10 must be employed. Additional uncertainty factors of 10 must be used both for human variability to protect sensitive subpopulations (people who are most susceptible to the health effects caused by the substance) and for interspecies variability (extrapolation from animals to humans). In deriving an MRL, these individual uncertainty factors are multiplied together. The product is then divided into the inhalation concentration or oral dosage selected from the study. Uncertainty factors used in developing a substance-specific MRL are provided in the footnotes of the levels of significant exposure (LSE) Tables.

Chapter 3

Health Effects

Tables and Figures for Levels of Significant Exposure (LSE)

Tables and figures are used to summarize health effects and illustrate graphically levels of exposure associated with those effects. These levels cover health effects observed at increasing dose concentrations and durations, differences in response by species, MRLs to humans for noncancer end points, and EPA's estimated range associated with an upper- bound individual lifetime cancer risk of 1 in 10,000 to 1 in 10,000,000. Use the LSE tables and figures for a quick review of the health effects and to locate data for a specific exposure scenario. The LSE tables and figures should always be used in conjunction with the text. All entries in these tables and figures represent studies that provide reliable, quantitative estimates of NOAELs, LOAELs, or Cancer Effect Levels (CELs).

The legends presented below demonstrate the application of these tables and figures. Representative examples of LSE Table 3-1 and Figure 3-1 are shown. The numbers in the left column of the legends correspond to the numbers in the example table and figure.

LEGEND

See Sample LSE Table 3-1 (page B-6)

(1) Route of Exposure. One of the first considerations when reviewing the toxicity of a substance using these tables and figures should be the relevant and appropriate route of exposure. Typically when sufficient data exists, three LSE tables and two LSE figures are presented in the document. The three LSE tables present data on the three principal routes of exposure, i.e., inhalation, oral, and dermal (LSE Table 3-1, 3-2, and 3-3, respectively). LSE figures are limited to the inhalation (LSE Figure 3-1) and oral (LSE Figure 3-2) routes. Not all substances will have data on each route of exposure and will not, therefore, have all five of the tables and figures.

(2) Exposure Period. Three exposure periods—acute (less than 15 days), intermediate (15–364 days), and chronic (365 days or more)—are presented within each relevant route of exposure. In this example, an inhalation study of intermediate exposure duration is reported. For quick reference to health effects occurring from a known length of exposure, locate the applicable exposure period within the LSE table and figure.

(3) Health Effect. The major categories of health effects included in LSE tables and figures are death, systemic, immunological, neurological, developmental, reproductive, and cancer. NOAELs and LOAELs can be reported in the tables and figures for all effects but cancer. Systemic effects are further defined in the "System" column of the LSE table (see key number 18).

(4) Key to Figure. Each key number in the LSE table links study information to one or more data points using the same key number in the corresponding LSE figure. In this example, the study represented by key number 18 has been used to derive a NOAEL and a Less Serious LOAEL (also see the two "18r" data points in sample Figure 3-1).

(5) Species. The test species, whether animal or human, are identified in this column. Chapter 2, "Relevance to Public Health," covers the relevance of animal data to human toxicity and Section 3.5, "Toxicokinetics," contains any available information on comparative toxicokinetics. Although NOAELs and LOAELs are species specific, the levels are extrapolated to equivalent human doses to derive an MRL.

(6) Exposure Frequency/Duration. The duration of the study and the weekly and daily exposure regimen are provided in this column. This permits comparison of NOAELs and LOAELs from different studies. In this case (key number 18), rats were exposed to "Chemical x" via inhalation for 6 hours/day, 5 days/week, for 13 weeks. For a more complete review of the dosing regimen refer to the appropriate sections of the text or the original reference paper (i.e., Nitschke et al. 1981).

(7) System. This column further defines the systemic effects. These systems include respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, hepatic, renal, and dermal/ocular. "Other" refers to any systemic effect (e.g., a decrease in body weight) not covered in these systems. In the example of key number 18, one systemic effect (respiratory) was investigated.

(8) NOAEL. A NOAEL is the highest exposure level at which no harmful effects were seen in the organ system studied. Key number 18 reports a NOAEL of 3 ppm for the respiratory system, which was used to derive an intermediate exposure, inhalation MRL of 0.005 ppm (see footnote "b").

(9) LOAEL. A LOAEL is the lowest dose used in the study that caused a harmful health effect. LOAELs have been classified into "Less Serious" and "Serious" effects. These distinctions help readers identify the levels of exposure at which adverse health effects first appear and the gradation of effects with increasing dose. A brief description of the specific end point used to quantify the adverse effect accompanies the LOAEL. The respiratory effect reported in key number 18 (hyperplasia) is a Less Serious LOAEL of 10 ppm. MRLs are not derived from Serious LOAELs.

(10) Reference. The complete reference citation is given in Chapter 9 of the profile.

(11) CEL. A CEL is the lowest exposure level associated with the onset of carcinogenesis in experimental or epidemiologic studies. CELs are always considered serious effects. The LSE tables and figures do not contain NOAELs for cancer, but the text may report doses not causing measurable cancer increases.

(12) Footnotes. Explanations of abbreviations or reference notes for data in the LSE tables are found in the footnotes. Footnote "b" indicates that the NOAEL of 3 ppm in key number 18 was used to derive an MRL of 0.005 ppm.

LEGEND

See Sample Figure 3-1 (page B-7)

LSE figures graphically illustrate the data presented in the corresponding LSE tables. Figures help the reader quickly compare health effects according to exposure concentrations for particular exposure periods.

(13) Exposure Period. The same exposure periods appear as in the LSE table. In this example, health effects observed within the acute and intermediate exposure periods are illustrated.

(14) Health Effect. These are the categories of health effects for which reliable quantitative data exists. The same health effects appear in the LSE table.

(15) Levels of Exposure. Concentrations or doses for each health effect in the LSE tables are graphically displayed in the LSE figures. Exposure concentration or dose is measured on the log scale "y" axis. Inhalation exposure is reported in mg/m^3 or ppm and oral exposure is reported in mg/kg/day.

(16) NOAEL. In this example, the open circle designated 18r identifies a NOAEL critical end point in the rat upon which an intermediate inhalation exposure MRL is based. The key number 18 corresponds to the entry in the LSE table. The dashed descending arrow indicates the extrapolation from the exposure level of 3 ppm (see entry 18 in the Table) to the MRL of 0.005 ppm (see footnote "b" in the LSE table).

(17) CEL. Key number 38r is one of three studies for which CELs were derived. The diamond symbol refers to a CEL for the test species-mouse. The number 38 corresponds to the entry in the LSE table.

(18) Estimated Upper-Bound Human Cancer Risk Levels. This is the range associated with the upper-bound for lifetime cancer risk of 1 in 10,000 to 1 in 10,000,000. These risk levels are derived from the EPA's Human Health Assessment Group's upper-bound estimates of the slope of the cancer dose response curve at low dose levels (q_1*).

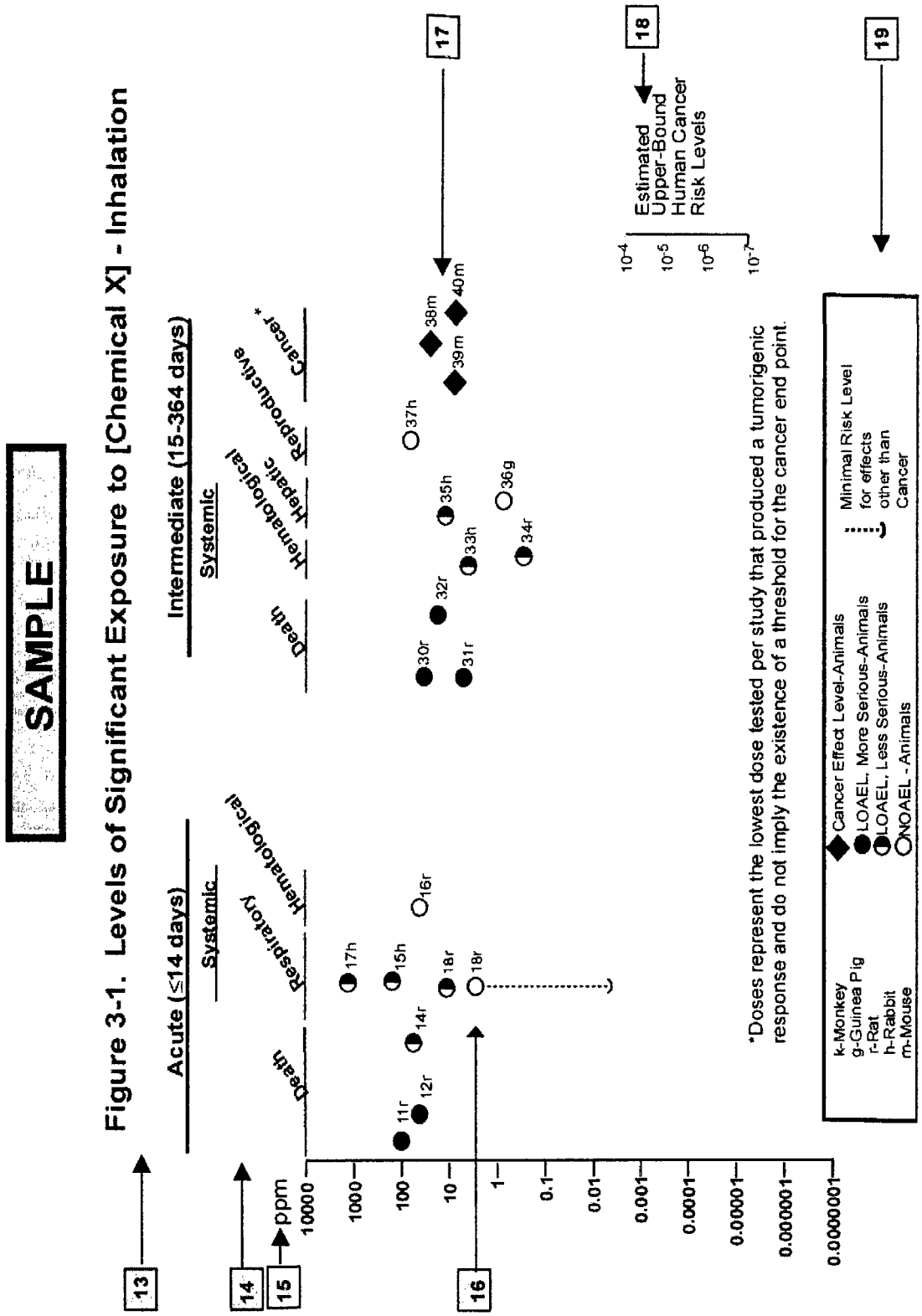
(19) Key to LSE Figure. The Key explains the abbreviations and symbols used in the figure.

SAMPLE

1 → **TABLE 3-1. Levels of Significant Exposure to [Chemical x] – Inhalation**

Key to figure[a]	Species	Exposure frequency/ duration	System	NOAEL (ppm)	LOAEL (effect) Less serious (ppm)	LOAEL (effect) Serious (ppm)	Reference
2 → INTERMEDIATE EXPOSURE							
	5	6	7	8	9		10
3 → Systemic	↓	↓	↓	↓	↓		↓
18	Rat	13 wk 5 d/wk 6 hr/d	Resp	3[b]	10 (hyperplasia)		Nitschke et al. 1981
4 → CHRONIC EXPOSURE							
Cancer					11		
					↓		
38	Rat	18 mo 5 d/wk 7 hr/d			20	(CEL, multiple organs)	Wong et al. 1982
39	Rat	89-104 wk 5 d/wk 6 hr/d			10	(CEL, lung tumors, nasal tumors)	NTP 1982
40	Mouse	79-103 wk 5 d/wk 6 hr/d			10	(CEL, lung tumors, hemangiosarcomas)	NTP 1982

12 → a The number corresponds to entries in Figure 3-1.
b Used to derive an intermediate inhalation Minimal Risk Level (MRL) of 5x10-3 ppm; dose adjusted for intermittent exposure and divided by an uncertainty factor of 100 (10 for extrapolation from animal to humans, 10 for human variability).


SAMPLE
Figure 3-1. Levels of Significant Exposure to [Chemical X] - Inhalation
Acute (≤14 days)
Systemic
Death
Respiratory
Hematological
Intermediate (15-364 days)
Systemic
Death
Hematological
Hepatic
Reproductive
Cancer *
ppm
10000
1000
100
10
1
0.1
0.01
0.001
0.0001
0.00001
0.000001
0.0000001
11r
12r
14r
15h
16r
17h
18r
18r
30r
31r
32r
33h
34r
35h
36g
37h
38m
39m
40m
10^{-4}
10^{-5}
10^{-6}
10^{-7}
Estimated Upper-Bound Human Cancer Risk Levels
*Doses represent the lowest dose tested per study that produced a tumorigenic response and do not imply the existence of a threshold for the cancer end point.
k-Monkey
g-Guinea Pig
r-Rat
h-Rabbit
m-Mouse
Cancer Effect Level-Animals
LOAEL, More Serious-Animals
LOAEL, Less Serious-Animals
NOAEL - Animals
Minimal Risk Level for effects other than Cancer
13
14
15
16
17
18
19

APPENDIX C. ACRONYMS, ABBREVIATIONS, AND SYMBOLS

Some terms are generic and may not be used in this profile.

ACGIH	American Conference of Governmental Industrial Hygienists
ACOEM	American College of Occupational and Environmental Medicine
ADI	acceptable daily intake
ADME	absorption, distribution, metabolism, and excretion
AED	atomic emission detection
AFID	alkali flame ionization detector
AFOSH	Air Force Office of Safety and Health
ALI	annual limit on intake
ALT	alanine aminotransferase
AML	acute myeloid leukemia
AOAC	Association of Official Analytical Chemists
AOEC	Association of Occupational and Environmental Clinics
AP	alkaline phosphatase
APHA	American Public Health Association
AST	aspartate aminotransferase
atm	atmosphere
ATSDR	Agency for Toxic Substances and Disease Registry
AWQC	Ambient Water Quality Criteria
BAT	best available technology
BCF	bioconcentration factor
BEI	Biological Exposure Index
BMD	benchmark dose
BMR	benchmark response
BSC	Board of Scientific Counselors
C	centigrade
CAA	Clean Air Act
CAG	Cancer Assessment Group of the U.S. Environmental Protection Agency
CAS	Chemical Abstract Services
CDC	Centers for Disease Control and Prevention
CEL	cancer effect level
CELDS	Computer-Environmental Legislative Data System
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CFR	Code of Federal Regulations
Ci	curie
CI	confidence interval
CL	ceiling limit value
CLP	Contract Laboratory Program
cm	centimeter
CML	chronic myeloid leukemia
CPSC	Consumer Products Safety Commission
CWA	Clean Water Act
DAC	derived air concentration
DHEW	Department of Health, Education, and Welfare
DHHS	Department of Health and Human Services
DNA	deoxyribonucleic acid
DOD	Department of Defense

DOE	Department of Energy
DOL	Department of Labor
DOT	Department of Transportation
DOT/UN/ NA/IMCO	Department of Transportation/United Nations/ North America/International Maritime Dangerous Goods Code
DWEL	drinking water exposure level
ECD	electron capture detection
ECG/EKG	electrocardiogram
EEG	electroencephalogram
EEGL	Emergency Exposure Guidance Level
EPA	Environmental Protection Agency
F	Fahrenheit
F_1	first-filial generation
FAO	Food and Agricultural Organization of the United Nations
FDA	Food and Drug Administration
FEMA	Federal Emergency Management Agency
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
FPD	flame photometric detection
fpm	feet per minute
FR	Federal Register
FSH	follicle stimulating hormone
g	gram
GC	gas chromatography
gd	gestational day
GLC	gas liquid chromatography
GPC	gel permeation chromatography
HPLC	high-performance liquid chromatography
HRGC	high resolution gas chromatography
HSDB	Hazardous Substance Data Bank
IARC	International Agency for Research on Cancer
IDLH	immediately dangerous to life and health
ILO	International Labor Organization
IRIS	Integrated Risk Information System
Kd	adsorption ratio
kg	kilogram
kkg	metric ton
K_{oc}	organic carbon partition coefficient
K_{ow}	octanol-water partition coefficient
L	liter
LC	liquid chromatography
LC_{50}	lethal concentration, 50% kill
LC_{Lo}	lethal concentration, low
LD_{50}	lethal dose, 50% kill
LD_{Lo}	lethal dose, low
LDH	lactic dehydrogenase
LH	luteinizing hormone
LOAEL	lowest-observed-adverse-effect level
LSE	Levels of Significant Exposure
LT_{50}	lethal time, 50% kill
m	meter
MA	*trans,trans*-muconic acid

MAL	maximum allowable level
mCi	millicurie
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
MFO	mixed function oxidase
mg	milligram
mL	milliliter
mm	millimeter
mmHg	millimeters of mercury
mmol	millimole
mppcf	millions of particles per cubic foot
MRL	Minimal Risk Level
MS	mass spectrometry
NAAQS	National Ambient Air Quality Standard
NAS	National Academy of Science
NATICH	National Air Toxics Information Clearinghouse
NATO	North Atlantic Treaty Organization
NCE	normochromatic erythrocytes
NCEH	National Center for Environmental Health
NCI	National Cancer Institute
ND	not detected
NFPA	National Fire Protection Association
ng	nanogram
NHANES	National Health and Nutrition Examination Survey
NIEHS	National Institute of Environmental Health Sciences
NIOSH	National Institute for Occupational Safety and Health
NIOSHTIC	NIOSH's Computerized Information Retrieval System
NLM	National Library of Medicine
nm	nanometer
nmol	nanomole
NOAEL	no-observed-adverse-effect level
NOES	National Occupational Exposure Survey
NOHS	National Occupational Hazard Survey
NPD	nitrogen phosphorus detection
NPDES	National Pollutant Discharge Elimination System
NPL	National Priorities List
NR	not reported
NRC	National Research Council
NS	not specified
NSPS	New Source Performance Standards
NTIS	National Technical Information Service
NTP	National Toxicology Program
ODW	Office of Drinking Water, EPA
OERR	Office of Emergency and Remedial Response, EPA
OHM/TADS	Oil and Hazardous Materials/Technical Assistance Data System
OPP	Office of Pesticide Programs, EPA
OPPT	Office of Pollution Prevention and Toxics, EPA
OPPTS	Office of Prevention, Pesticides and Toxic Substances, EPA
OR	odds ratio
OSHA	Occupational Safety and Health Administration

OSW	Office of Solid Waste, EPA
OTS	Office of Toxic Substances
OW	Office of Water
OWRS	Office of Water Regulations and Standards, EPA
PAH	polycyclic aromatic hydrocarbon
PBPD	physiologically based pharmacodynamic
PBPK	physiologically based pharmacokinetic
PCE	polychromatic erythrocytes
PEL	permissible exposure limit
pg	picogram
PHS	Public Health Service
PID	photo ionization detector
pmol	picomole
PMR	proportionate mortality ratio
ppb	parts per billion
ppm	parts per million
ppt	parts per trillion
PSNS	pretreatment standards for new sources
RBC	red blood cell
REL	recommended exposure level/limit
RfC	reference concentration
RfD	reference dose
RNA	ribonucleic acid
RQ	reportable quantity
RTECS	Registry of Toxic Effects of Chemical Substances
SARA	Superfund Amendments and Reauthorization Act
SCE	sister chromatid exchange
SGOT	serum glutamic oxaloacetic transaminase
SGPT	serum glutamic pyruvic transaminase
SIC	standard industrial classification
SIM	selected ion monitoring
SMCL	secondary maximum contaminant level
SMR	standardized mortality ratio
SNARL	suggested no adverse response level
SPEGL	Short-Term Public Emergency Guidance Level
STEL	short term exposure limit
STORET	Storage and Retrieval
TD_{50}	toxic dose, 50% specific toxic effect
TLV	threshold limit value
TOC	total organic carbon
TPQ	threshold planning quantity
TRI	Toxics Release Inventory
TSCA	Toxic Substances Control Act
TWA	time-weighted average
UF	uncertainty factor
U.S.	United States
USDA	United States Department of Agriculture
USGS	United States Geological Survey
USNRC	United States Nuclear Regulatory Commission
VOC	volatile organic compound

WBC	white blood cell
WHO	World Health Organization

>	greater than
≥	greater than or equal to
=	equal to
<	less than
≤	less than or equal to
%	percent
α	alpha
β	beta
γ	gamma
δ	delta
μm	micrometer
μg	microgram
q_1^*	cancer slope factor
–	negative
+	positive
(+)	weakly positive result
(–)	weakly negative result

APPENDIX D. OVERVIEW OF BASIC RADIATION PHYSICS, CHEMISTRY, AND BIOLOGY

Understanding the basic concepts in radiation physics, chemistry, and biology is important to the evaluation and interpretation of radiation-induced adverse health effects and to the derivation of radiation protection principles. This appendix presents a brief overview of the areas of radiation physics, chemistry, and biology and is based to a large extent on the reviews of Mettler and Moseley (1985), Hobbs and McClellan (1986), Eichholz (1982), Hendee (1973), Cember (1996), and Early et al. (1979).

D.1 RADIONUCLIDES AND RADIOACTIVITY

The substances we call elements are composed of atoms. Atoms in turn are made up of neutrons, protons and electrons: neutrons and protons in the nucleus and electrons in a cloud of orbits around the nucleus. Nuclide is the general term referring to any nucleus along with its orbital electrons. The nuclide is characterized by the composition of its nucleus and hence by the number of protons and neutrons in the nucleus. All atoms of an element have the same number of protons (this is given by the atomic number) but may have different numbers of neutrons (this is reflected by the atomic mass numbers or atomic weight of the element). Atoms with different atomic mass but the same atomic numbers are referred to as isotopes of an element.

The numerical combination of protons and neutrons in most nuclides is such that the nucleus is quantum mechanically stable and the atom is said to be stable, i.e., not radioactive; however, if there are too few or too many neutrons, the nucleus is unstable and the atom is said to be radioactive. Unstable nuclides undergo radioactive transformation, a process in which a neutron or proton converts into the other and a beta particle is emitted, or else an alpha particle is emitted. Each type of decay is typically accompanied by the emission of gamma rays. These unstable atoms are called radionuclides; their emissions are called ionizing radiation; and the whole property is called radioactivity. Transformation or decay results in the formation of new nuclides some of which may themselves be radionuclides, while others are stable nuclides. This series of transformations is called the decay chain of the radionuclide. The first radionuclide in the chain is called the parent; the subsequent products of the transformation are called progeny, daughters, or decay products.

In general there are two classifications of radioactivity and radionuclides: natural and artificial (man-made). Naturally-occurring radioactive materials (NORMs) exist in nature and no additional energy is necessary to place them in an unstable state. Natural radioactivity is the property of some naturally occurring, usually heavy elements, that are heavier than lead. Radionuclides, such as radium and uranium, primarily emit alpha particles. Some lighter elements such as carbon-14 and tritium (hydrogen-3) primarily emit beta particles as they transform to a more stable atom. Natural radioactive atoms heavier than lead cannot attain a stable nucleus heavier than lead. Everyone is exposed to background radiation from naturally-occurring radionuclides throughout life. This background radiation is the major source of radiation exposure to man and arises from several sources. The natural background exposures are frequently used as a standard of comparison for exposures to various artificial sources of ionizing radiation.

Artificial radioactive atoms are produced either as a by-product of fission of uranium or plutonium atoms in a nuclear reactor or by bombarding stable atoms with particles, such as neutrons or protons, directed at the stable atoms with high velocity. These artificially produced radioactive elements usually decay by emission of particles, such as positive or negative beta particles and one or more high energy photons (gamma rays). Unstable (radioactive) atoms of any element can be produced.

Both naturally occurring and artificial radioisotopes find application in medicine, industrial products, and consumer products. Some specific radioisotopes, called fall-out, are still found in the environment as a result of nuclear weapons use or testing.

D.2 RADIOACTIVE DECAY

D.2.1 Principles of Radioactive Decay

The stability of an atom is the result of the balance of the forces of the various components of the nucleus. An atom that is unstable (radionuclide) will release energy (decay) in various ways and transform to stable atoms or to other radioactive species called daughters, often with the release of ionizing radiation. If there are either too many or too few neutrons for a given number of protons, the resulting nucleus may undergo transformation. For some elements, a chain of daughter decay products may be produced until stable atoms are formed. Radionuclides can be characterized by the type and energy of the radiation emitted, the rate of decay, and the mode of decay. The mode of decay indicates how a parent compound undergoes transformation. Radiations considered here are primarily of nuclear origin, i.e., they arise from nuclear excitation, usually caused by the capture of charged or uncharged nucleons by a nucleus, or by the radioactive decay or transformation of an unstable nuclide. The type of radiation may be categorized as charged or uncharged particles, protons, and fission products) or electromagnetic radiation (gamma rays and x rays). Table D-1 summarizes the basic characteristics of the more common types of radiation encountered.

D.2.2 Half-Life and Activity

For any given radionuclide, the rate of decay is a first-order process that is constant, regardless of the radioactive atoms present and is characteristic for each radionuclide. The process of decay is a series of random events; temperature, pressure, or chemical combinations do not effect the rate of decay. While it may not be possible to predict exactly which atom is going to undergo transformation at any given time, it is possible to predict, on average, the fraction of the radioactive atoms that will transform during any interval of time.

The *activity* is a measure of the quantity of radioactive material. For these radioactive materials it is customary to describe the activity as the number of disintegrations (transformations) per unit time. The unit of activity is the curie (Ci), which was originally related to the activity of one gram of radium, but is now defined as that quantity of radioactive material in which there are:

1 curie (Ci) = 3.7×10^{10} disintegrations (transformations)/second (dps) or 2.22×10^{12} disintegrations (transformations)/minute (dpm).

The SI unit of activity is the becquerel (Bq); 1 Bq = that quantity of radioactive material in which there is 1 transformation/second. Since activity is proportional to the number of atoms of the radioactive material, the quantity of any radioactive material is usually expressed in curies, regardless of its purity or concentration. The transformation of radioactive nuclei is a random process, and the number of transformations is directly proportional to the number of radioactive atoms present. For any pure radioactive substance, the rate of decay is usually described by its radiological half-life, T_R, i.e., the time it takes for a specified source material to decay to half its initial activity. The specific activity is the activity of a radionuclide per mass of that radionuclide. If properly qualified, it can refer to activity per unit mass of related materials, such as the element itself or a chemical compound labeled with the radionuclide. The higher the specific activity of a radioisotope, the faster it is decaying.

The activity of a radionuclide at time t may be calculated by:

$$A = A_0 e^{-0.693t/Trad}$$

where A is the activity in dps or curies or becquerels, A_o is the activity at time zero, t is the time at which measured, and T_{rad} is the radiological half-life of the radionuclide (T_{rad} and t must be in the same units of time). The time when the activity of a sample of radioactivity becomes one-half its original value is the radioactive half-life and is expressed in any suitable unit of time.

Table D-1. Characteristics of Nuclear Radiations

Radiation	Rest mass[a]	Charge	Typical energy range	Path length[b] Air	Path length[b] Solid	Comments
Alpha (α)	4.00 amu	+2	4–10 MeV	5–10 cm	25–80 μm	Identical to ionized He nucleus
Negatron (β^-)	5.48×10^{-4} amu; 0.51 MeV	–1	0–4 MeV	0–10 m	0–1 cm	Identical to electron
Positron (β^+)	5.48×10^{-4} amu; 0.51 MeV	+1	0-4 MeV	0–10 m	0–1 cm	Identical to electron except for sign of charge
Neutron	1.0086 amu; 939.55 MeV	0	0–15 MeV	b	b	Free half-life: 16 min
X ray (e.m. photon)	–	0	5 keV–100 keV	b	b	Photon from transition of an electron between atomic orbits
Gamma (γ) (e.m. photon)	–	0	10 keV–3 MeV	b	b	Photon from nuclear transformation

[a] The rest mass (in amu) has an energy equivalent in MeV that is obtained using the equation $E=mc^2$, where 1 amu = 932 MeV.
[b] Path lengths are not applicable to x- and gamma rays since their intensities decrease exponentially; path lengths in solid tissue are variable, depending on particle energy, electron density of material, and other factors.

amu = atomic mass unit; e.m. = electromagnetic; MeV = Megaelectron Volts

The specific activity is a measure of activity, and is defined as the activity of a radionuclide per mass of that radionuclide. This activity is usually expressed in curies per gram and may be calculated by

$$\text{curies/gram} = 1.3 \times 10^8 / (T_{rad})\ (\text{atomic weight}) \quad \text{or}$$

$$[3.577 \times 10^5 \times \text{mass(g)}] / [T_{rad} \times \text{atomic weight}]$$

where T_{rad} is the radiological half-life in days.

In the case of radioactive materials contained in living organisms, an additional consideration is made for the reduction in observed activity due to regular processes of elimination of the respective chemical or biochemical substance from the organism. This introduces a rate constant called the biological half-life (T_{biol}) which is the time required for biological processes to eliminate one-half of the activity. This time is virtually the same for both stable and radioactive isotopes of any given element.

Under such conditions the time required for a radioactive element to be halved as a result of the combined action of radioactive decay and biological elimination is the effective clearance half-time:

$$T_{eff} = (T_{biol} \times T_{rad}) / (T_{biol} + T_{rad}).$$

Table D-2 presents representative effective half-lives of particular interest.

Table D-2. Half-Lives of Some Radionuclides in Adult Body Organs

		Half-life[a]		
Radionuclide	Critical organ	Physical	Biological	Effective
Uranium 238	Kidney	4,460,000,000 y	4 d	4 d
Hydrogen 3[b] (Tritium)	Whole body	12.3 y	10 d	10 d
Iodine 131	Thyroid	8 d	80 d	7.3 d
Strontium 90	Bone	28 y	50 y	18 y
Plutonium 239	Bone surface	24,400 y	50 y	50 y
	Lung	24,400 y	500 d	474 d
Cobalt 60	Whole body	5.3 y	99.5 d	95 d
Iron 55	Spleen	2.7 y	600 d	388 d
Iron 59	Spleen	45.1 d	600 d	42 d
Manganese 54	Liver	303 d	25 d	23 d
Cesium 137	Whole body	30 y	70 d	70 d

[a]d = days, y = years
[b]Mixed in body water as tritiated water

D.2.3 Interaction of Radiation with Matter

Both ionizing and nonionizing radiation will interact with materials; that is, radiation will lose kinetic energy to any solid, liquid or gas through which it passes by a variety of mechanisms. The transfer of energy to a medium by either electromagnetic or particulate radiation may be sufficient to cause formation of ions. This process is called ionization. Compared to other types of radiation that may be absorbed, such as ultraviolet radiation, ionizing radiation deposits a relatively large amount of energy into a small volume.

The method by which incident radiation interacts with the medium to cause ionization may be direct or indirect. Electromagnetic radiations (x rays and gamma photons) are indirectly ionizing; that is, they give up their energy in various interactions with cellular molecules, and the energy is then utilized to produce a fast-moving charged particle such as an electron. It is the electron that then may react with a target molecule. This particle is called a "primary ionizing particle. Charged particles, in contrast, strike the tissue or medium and directly react with target molecules, such as oxygen or water. These particulate radiations are directly ionizing radiations. Examples of directly ionizing particles include alpha and beta particles. Indirectly ionizing radiations are always more penetrating than directly ionizing particulate radiations.

Mass, charge, and velocity of a particle, as well as the electron density of the material with which it interacts, all affect the rate at which ionization occurs. The higher the charge of the particle and the lower the velocity, the greater the propensity to cause ionization. Heavy, highly charged particles, such as alpha particles, lose energy rapidly with distance and, therefore, do not penetrate deeply. The result of these

interaction processes is a gradual slowing down of any incident particle until it is brought to rest or "stopped" at the end of its range.

D.2.4 Characteristics of Emitted Radiation

D.2.4.1 Alpha Emission. In alpha emission, an alpha particle consisting of two protons and two neutrons is emitted with a resulting decrease in the atomic mass number by four and reduction of the atomic number of two, thereby changing the parent to a different element. The alpha particle is identical to a helium nucleus consisting of two neutrons and two protons. It results from the radioactive decay of some heavy elements such as uranium, plutonium, radium, thorium, and radon. The alpha particles emitted by a given radionuclide have the same energy and intensity combination. Most of the alpha particles that are likely to be found have energies in the range of about 4 to 8 MeV, depending on the isotope from which they came.

The alpha particle has an electrical charge of +2. Because of this double positive charge and their size, alpha particles have great ionizing power and, thus, lose their kinetic energy quickly. This results in very little penetrating power. In fact, an alpha particle cannot penetrate a sheet of paper. The range of an alpha particle (the distance the charged particle travels from the point of origin to its resting point) is about 4 cm in air, which decreases considerably to a few micrometers in tissue. These properties cause alpha emitters to be hazardous only if there is internal contamination (i.e., if the radionuclide is inside the body).

D.2.4.2 Beta Emission. A beta particle (β) is a high-velocity electron ejected from a disintegrating nucleus. The particle may be either a negatively charged electron, termed a negatron (β-) or a positively charged electron, termed a positron (β+). Although the precise definition of "beta emission" refers to both β- and β+, common usage of the term generally applies only to the negative particle, as distinguished from the positron emission, which refers to the β+ particle.

D.2.4.2.1 Beta Negative Emission. Beta particle (β-) emission is another process by which a radionuclide, with a neutron excess achieves stability. Beta particle emission decreases the number of neutrons by one and increases the number of protons by one, while the atomic mass number remains unchanged.[1] This transformation results in the formation of a different element. The energy spectrum of beta particle emission ranges from a certain maximum down to zero with the mean energy of the spectrum being about one-third of the maximum. The range of betas is much less in tissue than in air. Beta negative emitting radionuclides can cause injury to the skin and superficial body tissues, but mostly present an internal contamination hazard.

D.2.4.2.2 Positron Emission. In cases in which there are too many protons in the nucleus, positron emission may occur. In this case a proton may be thought of as being converted into a neutron, and a positron (β+) is emitted.1 This increases the number of neutrons by one, decreases the number of protons by one, and again leaves the atomic mass number unchanged. The gamma radiation resulting from the annihilation (see glossary) of the positron makes all positron emitting isotopes more of an external radiation hazard than pure β emitters of equal energy.

D.2.4.2.3 Gamma Emission. Radioactive decay by alpha, beta, or positron emission, or electron capture often leaves some of the energy resulting from these changes in the nucleus. As a result, the nucleus is raised to an excited level. None of these excited nuclei can remain in this high-energy state. Nuclei release this energy returning to ground state or to the lowest possible stable energy level. The energy released is in the form of gamma radiation (high energy photons) and has an energy equal to the change in the energy state of the nucleus. Gamma and x rays behave similarly but differ in their origin;

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[1] Neutrinos also accompany negative beta particles and positron emissions

gamma emissions originate in the nucleus while x rays originate in the orbital electron structure or from rapidly changing the velocity of an electron (e.g., as occurs when shielding high energy beta particles or stopping the electron beam in an x ray tube).

D.3 ESTIMATION OF ENERGY DEPOSITION IN HUMAN TISSUES

Two forms of potential radiation exposures can result: internal and external. The term exposure denotes physical interaction of the radiation emitted from the radioactive material with cells and tissues of the human body. An exposure can be "acute" or "chronic" depending on how long an individual or organ is exposed to the radiation. Internal exposures occur when radionuclides, which have entered the body (e.g., through the inhalation, ingestion, or dermal pathways), undergo radioactive decay resulting in the deposition of energy to internal organs. External exposures occur when radiation enters the body directly from sources located outside the body, such as radiation emitters from radionuclides on ground surfaces, dissolved in water, or dispersed in the air. In general, external exposures are from material emitting gamma radiation, which readily penetrate the skin and internal organs. Beta and alpha radiation from external sources are far less penetrating and deposit their energy primarily on the skin's outer layer. Consequently, their contribution to the absorbed dose of the total body dose, compared to that deposited by gamma rays, may be negligible.

Characterizing the radiation dose to persons as a result of exposure to radiation is a complex issue. It is difficult to: (1) measure internally the amount of energy actually transferred to an organic material and to correlate any observed effects with this energy deposition; and (2) account for and predict secondary processes, such as collision effects or biologically triggered effects, that are an indirect consequence of the primary interaction event.

D.3.1 Dose/Exposure Units

D.3.1.1 Roentgen. The roentgen (R) is a unit of x or gamma-ray exposure and is a measured by the amount of ionization caused in air by gamma or x radiation. One roentgen produces 2.58×10^{-4} coulomb per kilogram of air. In the case of gamma radiation, over the commonly encountered range of photon energy, the energy deposition in tissue for a dose of 1 R is about 0.0096 joules (J) /kg of tissue.

D.3.1.2 Absorbed Dose and Absorbed Dose Rate. The absorbed dose is defined as the energy imparted by radiation to a unit mass of the tissue or organ. The unit of absorbed dose is the rad; 1 rad = 100 erg/gram = 0.01 J/kg in any medium. An exposure of 1 R results in a dose to soft tissue of approximately 0.01 J/kg. The SI unit is the gray which is equivalent to 100 rad or 1 J/kg. Internal and external exposures from radiation sources are not usually instantaneous but are distributed over extended periods of time. The resulting rate of change of the absorbed dose to a small volume of mass is referred to as the absorbed dose rate in units of rad/unit time.

D.3.1.3 Working Levels and Working Level Months. Working level (WL) is a measure of the atmospheric concentration of radon and its short-lived progeny. One WL is defined as any combination of short-lived radon daughters (through polonium-214), per liter of air, that will result in the emission of 1.3×10^5 MeV of alpha energy. An activity concentration of 100 pCi radon-222/L of air, in equilibrium with its daughters, corresponds approximately to a potential alpha-energy concentration of 1 WL. The WL unit can also be used for thoron daughters. In this case, 1.3×10^5 MeV of alpha energy (1 WL) is released by the thoron daughters in equilibrium with 7.5 pCi thoron/L. The potential alpha energy exposure of miners is commonly expressed in the unit Working Level Month (WLM). One WLM corresponds to exposure to a concentration of 1 WL for the reference period of 170 hours, or more generally

WLM = concentration (WL) x exposure time (months) (one "month" = 170 working hours).

D.3.2 Dosimetry Models

Dosimetry models are used to estimate the dose from internally deposited to radioactive substances. The models for internal dosimetry consider the amount of radionuclides entering the body, the factors affecting their movement or transport through the body, distribution and retention of radionuclides in the body, and the energy deposited in organs and tissues from the radiation that is emitted during spontaneous decay processes. The dose pattern for radioactive materials in the body may be strongly influenced by the route of entry of the material. For industrial workers, inhalation of radioactive particles with pulmonary deposition and puncture wounds with subcutaneous deposition have been the most frequent. The general population has been exposed via ingestion and inhalation of low levels of naturally occurring radionuclides as well as radionuclides from nuclear weapons testing.

The models for external dosimetry consider only the photon doses (and neutron doses, where applicable) to organs of individuals who are immersed in air or are exposed to a contaminated object.

D.3.2.1 Ingestion. Ingestion of radioactive materials is most likely to occur from contaminated foodstuffs or water or eventual ingestion of inhaled compounds initially deposited in the lung. Ingestion of radioactive material may result in toxic effects as a result of either absorption of the radionuclide or irradiation of the gastrointestinal tract during passage through the tract, or a combination of both. The fraction of a radioactive material absorbed from the gastrointestinal tract is variable, depending on the specific element, the physical and chemical form of the material ingested, and the diet, as well as some other metabolic and physiological factors. The absorption of some elements is influenced by age, usually with higher absorption in the very young.

D.3.2.2 Inhalation. The inhalation route of exposure has long been recognized as being a major portal of entry for both nonradioactive and radioactive materials. The deposition of particles within the lung is largely dependent upon the size of the particles being inhaled. After the particle is deposited, the retention will depend upon the physical and chemical properties of the dust and the physiological status of the lung. The retention of the particle in the lung depends on the location of deposition, in addition to the physical and chemical properties of the particles. The converse of pulmonary retention is pulmonary clearance. There are three distinct mechanisms of clearance which operate simultaneously. Ciliary clearance acts only in the upper respiratory tract. The second and third mechanisms act mainly in the deep respiratory tract. These are phagocytosis and absorption. Phagocytosis is the engulfing of foreign bodies by alveolar macrophages and their subsequent removal either up the ciliary "escalator" or by entrance into the lymphatic system. Some inhaled soluble particles are absorbed into the blood and translocated to other organs and tissues.

D.3.3 Internal Emitters

An internal emitter is a radionuclide that is inside the body. The absorbed dose from internally deposited radionuclide depends on the energy absorbed per unit mass by the irradiated tissue. For a radionuclide distributed uniformly throughout an infinitely large medium, the concentration of absorbed energy must be equal to the concentration of energy emitted by the radionuclide. An infinitely large medium may be approximated by a tissue mass whose dimensions exceed the range of the particle. All alpha and most beta radiation will be absorbed in the organ (or tissue) of reference. Gamma-emitting radionuclide emissions are penetrating radiation, and a substantial fraction of gamma energy may be absorbed in tissue. The dose to an organ or tissue is a function of the effective retention half-time, the energy released in the tissue, the amount of radioactivity initially introduced, and the mass of the organ or tissue.

D.4 BIOLOGICAL EFFECTS OF RADIATION

When biological material is exposed to ionizing radiation, a chain of cellular events occurs as the ionizing particle passes through the biological material. A number of theories have been proposed to describe the interaction of radiation with biologically important molecules in cells and to explain the resulting damage to biological systems from those interactions. Many factors may modify the response of a living organism to a given dose of radiation. Factors related to the exposure include the dose rate, the energy of the radiation, and the temporal pattern of the exposure. Biological considerations include factors such as species, age, sex, and the portion of the body exposed. Several excellent reviews of the biological effects of radiation have been published, and the reader is referred to these for a more in-depth discussion (Brodsky 1996; Hobbs and McClellan 1986; ICRP 1984; Mettler and Moseley 1985; Rubin and Casarett 1968).

D.4.1 Radiation Effects at the Cellular Level

According to Mettler and Moseley (1985), at acute doses up to 10 rad (100 mGy), single strand breaks in DNA may be produced. These single strand breaks may be repaired rapidly. With doses in the range of 50–500 rad (0.5–5 Gy), irreparable double-stranded DNA breaks are likely, resulting in cellular reproductive death after one or more divisions of the irradiated parent cell. At large doses of radiation, usually greater than 500 rad (5 Gy), direct cell death before division (interphase death) may occur from the direct interaction of free-radicals with essential cellular macromolecules. Morphological changes at the cellular level, the severity of which are dose-dependent, may also be observed.

The sensitivity of various cell types varies. According to the Bergonie-Tribondeau law, the sensitivity of cell lines is directly proportional to their mitotic rate and inversely proportional to the degree of differentiation (Mettler and Moseley 1985). Rubin and Casarett (1968) devised a classification system that categorized cells according to type, function, and mitotic activity. The categories range from the most sensitive type, "vegetative intermitotic cells", found in the stem cells of the bone marrow and the gastrointestinal tract, to the least sensitive cell type, "fixed postmitotic cells," found in striated muscles or long-lived neural tissues.

Cellular changes may result in cell death, which if extensive, may produce irreversible damage to an organ or tissue or may result in the death of the individual. If the cell recovers, altered metabolism and function may still occur, which may be repaired or may result in the manifestation of clinical symptoms. These changes may also be expressed at a later time as tumors or cellular mutations, which may result in abnormal tissue.

D.4.2 Radiation Effects at the Organ Level

In most organs and tissues the injury and the underlying mechanism for that injury are complex and may involve a combination of events. The extent and severity of this tissue injury are dependent upon the radiosensitivity of the various cell types in that organ system. Rubin and Casarett (1968) describe and schematically display the events following radiation in several organ system types. These include: a rapid renewal system, such as the gastrointestinal mucosa; a slow renewal system, such as the pulmonary epithelium; and a nonrenewal system, such as neural or muscle tissue. In the rapid renewal system, organ injury results from the direct destruction of highly radiosensitive cells, such as the stem cells in the bone marrow. Injury may also result from constriction of the microcirculation and from edema and inflammation of the basement membrane, designated as the histohematic barrier, which may progress to fibrosis. In slow renewal and nonrenewal systems, the radiation may have little effect on the parenchymal cells, but ultimate parenchymal atrophy and death over several months result from fibrosis and occlusion of the microcirculation.

D.4.3 Low Level Radiation Effects

Cancer is the major latent harmful effect produced by ionizing radiation and the one that most people exposed to radiation are concerned about. The ability of alpha, beta, and gamma radiation to produce cancer in virtually every tissue and organ in laboratory animals has been well-demonstrated. The development of cancer is not an immediate effect. Radiation-induced leukemia has the shortest latent period at about 2 years, while other radiation induced cancers, such as osteosarcoma, have latent periods greater than 20 years. The mechanism by which cancer is induced in living cells is complex and is a topic of intense study. Exposure to ionizing radiation can produce cancer at any site within the body; however, some sites appear to be more common than others, such as the breast, lung, stomach, and thyroid.

DNA is the major target molecule during exposure to ionizing radiation. Other macromolecules, such as lipids and proteins, are also at risk of damage when exposed to ionizing radiation. The genotoxicity of ionizing radiation is an area of intense study, as damage to the DNA is ultimately responsible for many of the adverse toxicological effects ascribed to ionizing radiation, including cancer. Damage to genetic material is basic to developmental or teratogenic effects, as well. However, for effects other than cancer, there is little evidence of human effects at low levels of exposure.

D.5 UNITS IN RADIATION PROTECTION AND REGULATION

D.5.1 Dose Equivalent (or Equivalent Dose)

Dose equivalent (as measured in rem or sievert) is a special radiation protection quantity that is used for administrative and radiation safety purposes to express the absorbed dose in a manner which considers the difference in biological effectiveness of various kinds of ionizing radiation. ICRP (1990) changed this term to equivalent dose, but it has not yet been adopted by the USNRC or DOE.

The USNRC defines the dose equivalent, H, as the product of the absorbed dose, D, and the quality factor, Q, at the point of interest in biological tissue. This relationship is expressed as $H = D \times Q$. The dose equivalent concept is applicable only to doses that are not great enough to produce biomedical effects.

The quality factor or radiation weighting factor is a dimensionless quantity that depends in part on the stopping power for charged particles, and it accounts for the differences in biological effectiveness found among the types of radiation. Originally relative biological effectiveness (RBE) was used rather than Q to define the quantity, rem, which was of use in risk assessment. The generally accepted values for quality factors and radiation weighting factors for various radiation types are provided in Table D-3. The dose equivalent rate is the time rate of change of the dose equivalent to organs and tissues and is expressed as rem/unit time or sievert/unit time.

Table D-3. Quality Factors (Q) and Absorbed Dose Equivalencies

Type of radiation	Quality factor (Q)	Radiation weighting factor (w_r)*
X, gamma, or beta radiation	1	1
Alpha particles, multiple-charged particles, fission fragments and heavy particles of unknown charge	20	0.05
Neutrons (other than thermal >> 100 keV to 2 MeV), protons, alpha particles, charged particles of unknown energy	10	20
Neutrons of unknown energy	10	
High-energy protons	10	0.1
Thermal neutrons		5

*Absorbed dose in rad equal to 1 rem or the absorbed dose in gray equal to 1 sievert.

Source: USNRC. 2004. Standards for the protection against radiation, table 1004(b).1. 10 CFR 20.1004. U.S. Nuclear Regulatory Commission, Washington, D.C. NCRP 1993

D.5.2 Relative Biological Effectiveness

RBE is used to denote the experimentally determined ratio of the absorbed dose from one radiation type to the absorbed dose of a reference radiation required to produce an identical biologic effect under the same conditions. Gamma rays from cobalt-60 and 200–250 kVp x-rays have been used as reference standards. The term RBE has been widely used in experimental radiobiology, and the term quality factor (or radiation weighting factor) used in calculations of dose equivalents for radiation safety purposes (ICRP 1977; NCRP 1971; UNSCEAR 1982). Any RBE value applies only to a specific biological end point, in a specific exposure, under specific conditions to a specific species. There are no generally applicable values of RBE since RBEs are specific to a given exposure scenario.

D.5.3 Effective Dose Equivalent (or Effective Dose)

The absorbed dose is usually defined as the mean energy imparted per unit mass to an organ or tissue. This represents a simplification of the actual problem. Normally when an individual ingests or inhales a radionuclide or is exposed to external radiation that enters the body (gamma), the dose is not uniform throughout the whole body. The simplifying assumption is that the detriment will be the same whether the body is uniformly or non-uniformly irradiated. In an attempt to compare detriment from absorbed dose of a limited portion of the body with the detriment from total body dose, the ICRP (1977) has derived a concept of effective dose equivalent. ICRP (1990) changed this term to effective dose, but it has not yet been adopted by the USNRC or DOE.

The effective dose equivalent, H_E, is

$$H_E = (\text{the sum of})\ W_t\, H_t$$

where H_t is the dose equivalent (or equivalent dose) in the tissue t, W_t is the tissue weighting factor in that tissue, which represents the estimated proportion of the stochastic risk resulting from tissue, t, to the stochastic risk when the whole body is uniformly irradiated for occupational exposures under certain conditions (ICRP 1977). Tissue weighting factors for selected tissues are listed in Table D-4.

D.5.4 SI Units

The ICRU (1980), ICRP (1984), and NCRP (1985) now recommend that the rad, roentgen, curie, and rem be replaced by the SI units: gray (Gy), Coulomb per kilogram (C/kg), Becquerel (Bq), and sievert (Sv), respectively. The relationship between the customary units and the international system of units (SI) for radiological quantities is shown in Table D-5.

Table D-4. Tissue Weighting Factors for Calculating Effective Dose Equivalent and Effective Dose for Selected Tissues

	Tissue weighting factor	
Tissue	NCRP115/ ICRP60	USNRC/ICRP26
Bladder	0.05	–
Bone marrow	0.12	0.12
Bone surface	0.01	0.03
Breast	0.05	0.15
Colon	0.12	–
Esophagus	0.05	–
Gonads	0.20	0.25
Liver	0.05	–
Lung	0.12	0.12
Skin	0.01	–
Stomach	0.12	–
Thyroid	0.05	0.03
Remainder	0.05	0.30
Total	1.00	1.00

ICRP60 = International Commission on Radiological Protection, 1990 Recommendations of the ICRP
NCRP115 = National Council on Radiation Protection and Measurements. 1993. Risk Estimates for Radiation Protection, Report 115. Bethesda, Maryland
USNRC = Nuclear Regulatory Commission, Title 10, Code of Federal Regulations, Part 20

Table D-5. Comparison of Common and SI Units for Radiation Quantities

Quantity	Customary units	Definition	SI units	Definition
Activity (A)	curie (Ci)	3.7×10^{10} transformations s^{-1}	becquerel (Bq)	s^{-1}
Absorbed dose (D)	rad	10^{-2} Jkg^{-1}	gray (Gy)	Jkg^{-1}
Absorbed dose rate ($\dot{D}$)	rad per second (rad s^{-1})	10^{-2} $Jkg^{-1}s^{-1}$	gray per second (Gy s^{-1})	Jkg^{-1} s^{-1}
Dose equivalent (H)	rem	10^{-2} Jkg^{-1}	sievert (Sv)	Jkg^{-1}
Dose equivalent rate ($\dot{H}$)	rem per second (rem s^{-1})	10^{-2} $Jkg^{-1}s^{-1}$	sievert per second (Sv s^{-1})	Jkg^{-1} s^{-1}
Effective dose	rem	10^{-2} Jkg^{-1}	Sievert (Sv)	Jkg^{-1}
Equivalent dose (H)	rem	10^{-2} Jkg^{-1}	Sievert (Sv)	Jkg^{-1}
Linear energy transfer (LET)	kiloelectron volts per micrometer (keV μm^{-1})	1.602×10^{-10} Jm^{-1}	kiloelectron volts per micrometer (keV μm^{-1})	1.602×10^{-10} Jm^{-1}

Jkg^{-1} = Joules per kilogram; $Jkg^{-1}s^{-1}$ = Joules per kilogram per second; Jm^{-1} = Joules per meter; s^{-1} = per second

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