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RCRA Facility Investigation Report

On–Site Areas

Former CIBA Site
Cranston, Rhode Island

Volume 3:
Public Health and Environmental Risk Evaluation

Submitted by:

CIBA Corporation

Route 37 West

Toms River, New Jersey 08754

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RCRA Facility Investigation Report

(Production, Waste Water Treatment, and Warwick Areas)

Ciba Site
Cranston, Rhode Island

Chapter 6

Public Health and Environmental Risk Evaluation

Prepared for:
Ciba Corporation
Regional Remediation
P.O. Box 71
Toms River, New Jersey 10502

Prepared by:
PTRL Environmental Services, Inc.
1942 Oak Ridge Turnpike
Oak Ridge, Tennessee 37830

July, 1995

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Acronyms and Abbreviated Terms

Ciba	Ciba-Geigy Corporation
CMS	Corrective Measures Study
COPCs	Chemicals of Potential Concern
CSF	Cancer Slope Factor
CSF_i	Cancer Slope Factor--Inhalation Exposure Route
CSF_o	Cancer Slope Factor--Oral Exposure Route
ETI	Ecological Toxicity Index
HEAST	Health Effects Assessment Summary Tables
HHEM	Human Health Evaluation Manual
HI	Hazard Index
HQ	Hazard Quotient
ILCR	Incremental Lifetime Cancer Risk
IN	Level of Intake
IR_s	Soil Ingestion Rate
IRM	Interim Remedial Measures
IRIS	Integrated Risk Information System
LOAEL	Lowest-Observed-Adverse-Effects Level
MPS	Media Protection Standards
NOAEL	No-Observed-Adverse-Effects Level
PAHs	Polycyclic Aromatic Hydrocarbons
PCBs	Polychlorinated Biphenyls
PCDDs	Polychlorinated Dibenzodioxins
PCDFs	Polychlorinated Dibenzofurans
PHERE	Public Health and Environmental Risk Evaluation
PHRA	Public Health Risk Assessment
QA/QC	Quality Assurance/Quality Control
RCRA	Resource Conservation and Recovery Act
Region I	U.S. Environmental Protection Agency, Region I
RfC	Reference Concentration
RfD	Chronic Reference Dose
RfD_i	Chronic Reference Dose--Inhalation Exposure Route
RFI	RCRA Facility Investigation
RME	Reasonable Maximum Exposure
Site	Ciba-Geigy's Cranston, Rhode Island Site

Acronyms and Abbreviated Terms, continued

SQL ..... Sample Quantitation Limit
SRBCs ..... Sheep Erythrocytes
SWMU ..... Solid Waste Management Unit
SWMU-5 ..... Solid Waste Management Unit No. 5
THI ..... Total Hazard Index
TQ ..... Toxicity Quotient
TRVs ..... Toxicity Reference Values
UCL ..... 95th Percentile Upper Confidence Limit
UR_i ..... Inhalation Unit Risk
USEPA ..... U.S. Environmental Protection Agency
WCC ..... Woodward-Clyde Consultants

Summary

A Public Health and Environmental Risk Evaluation (PHERE) was prepared as part of the RFI, as required in the Order. It separately evaluates the potential human health and ecological risks associated with the Production, Waste Water Treatment, and Warwick Areas. It is consistent with the approach outlined in the USEPA's primary risk assessment guidance documents. The PHERE approach and values for exposure assumptions reflect discussions held with the Region I during several meetings and teleconferences, beginning with the May 17, 1994, meeting with Ciba at the Region I offices. The PHERE is presented in two parts; the public health risk assessment (PHRA) and the ecological risk assessment. The PHERE is a quantitative baseline study which assumes the property will be used without modification regardless of the practicality of this assumption. The risk reduction effectiveness of IRMs conducted in the Production and Warwick Areas is addressed qualitatively.

The purpose of the PHERE is threefold:

- Provide estimates of potential risks posed by site-related chemicals in the Production, Waste Water Treatment, and Warwick Areas of the Site using the conservative guidance specified by Region I.
- Identify the site areas and chemicals that might require corrective action using this risk assessment approach.
- Provide site-specific risk assessment models for human and ecological health to be used in developing risk-based Media Protection Standards (MPS) for soil, if needed.

Public Health Risk Assessment

The PHRA is designed to provide a conservative, quantitative estimate of potential risks associated with residual site-related chemicals in the Production, Waste Water Treatment, and Warwick Areas. It is based on analytical results from soil and groundwater samples collected during Phase I and II of the RFI field activities. It was performed by identifying chemicals of potential concern (COPCs) and carrying them through the risk assessment process. The COPCs were determined based on their toxicities, frequencies of detection, concentrations in site soil, and, for inorganics and polycyclic aromatic hydrocarbons (PAHs), comparison to background concentrations.

Regarding potential future land use, unrestricted residential use was assumed for the Warwick Area and Waste Water Treatment Areas. This is considered a "worst-case" since residential land use may not be the most probable for these areas. Based on a proposal to use the Production Area as a

vehicle parking facility, the PHRA reflects an on-site worker scenario for this area. The PHRA also considered the potential human health effects associated with the migration of site-related chemicals, via groundwater, to the Pawtuxet River. Therefore, potential exposure to a canoeist is estimated for each of the three site areas.

Results of the PHRA are expressed in terms of potential noncancer health effects and potential cancer risks which are summarized in Figures 6-1, 6-2, 6-3, and 6-4. The total hazard index (THI) represents the overall estimated noncancer risks for a given exposure scenario. The potential noncancer risk represented by the THI is considered of no significance if it is equal to or below a value of 1, and is a potential concern if it is greater than a value of 1 (rounded to a whole number). The potential cancer risk posed is expressed in terms of an incremental lifetime cancer risk (ILCR). The ILCR is an increased probability of cancer above that which exists as "background" (3 out of 10 people) for the general population. The USEPA regards an ILCR of between 1×10^{-6} (1 in 1,000,000) and 1×10^{-4} (1 in 10,000) as acceptable. Thus, this may be interpreted as an increase in the United States baseline cancer incidence from 300,000 per million population to a range of 300,001 to 300,100 per million population. If the ILCR exceeds the upper bound of the target risk range (1×10^{-4}), then further evaluation or corrective action may be indicated.

As shown in Figures 6-1 and 6-2, neither the Production nor the Waste Water Treatment Area is predicted to pose an unacceptable potential risk. The potential total ILCR and total PCB ILCR slightly exceed the target risk range for the hypothetical resident in the Warwick Area (Figure 6-3), but only because the PCBs are totaled and treated as if they were the carcinogenic PCB 1260. No PCB 1260 was found in the Warwick Area. The risk numbers presented are highly conservative and may exaggerate actual risks due to a number of factors. For example, the sampling approach was biased in that the field investigation targeted highly localized areas of suspected contamination. Additionally, at Region I's request, the total PCB carcinogenic risk is based on the assumption that all PCBs, including those that are noncarcinogenic (e.g. PCB 1248 and 1254) have a cancer potency factor equal to PCB 1260. These factors are especially significant for the Warwick Area, where contamination (PCB 1248 and 1254) is highly localized and no PCB 1260 was detected. Neither was PCB 1260 detected in the Waste Water Treatment Area. From a land-use standpoint, the likelihood of PCB exposure through surface soil is highly unlikely in the Production Area, since the proposed land use is a paved parking facility. The potential human health risks associated with the canoeist scenario are estimated to be nonexistent, with ILCR values less than 3×10^{-8} and THI values less than 0.003 in each of the three Site areas (Figure 6-4).

Even with the high degree of conservatism, the PHRA showed that corrective actions are not necessary for the three site areas solely on the basis of potential risk to public health, with the possible exception of the Warwick Area. However, Ciba volunteered to conduct some limited remediation in these areas to facilitate their productive use. Based on the concentration and frequency of detection in surface soil (the predominant exposure source), it was determined that PCB hot-spot removal in the Production and Warwick Areas would be the most effective action to facilitate their productive use. IRMs have recently been started in the Production and Warwick Areas.

Ecological Risk Assessment

This ecological risk assessment utilizes the risk assessment process as defined by the *Framework for Ecological Risk Assessment* (USEPA, 1992). The objective of this ecological risk assessment is to evaluate potential risks posed to terrestrial receptors by COPCs contained in surface soils (0-2 feet) at the Production and Warwick Areas, and surface soils and seep sediments at the Waste Water Treatment Area.

A terrestrial/riparian reconnaissance survey was conducted at the Site in March, 1992. The terrestrial survey identified twenty-eight species of upland plants and twenty-six species of riparian/wetland plants at and near the Site. Twenty-six species of birds were identified, including: the great blue heron, mallard duck, and red-tailed hawk. Five mammal species were identified, including the Eastern gray squirrel and the raccoon. A seep area in the Waste Water Treatment Area was electroshocked, but no fish were observed. Tadpoles were present in the seep.

Potential exposure pathways for plants and animals include:

- uptake through roots in contact with surface soils,
- foliar uptake of volatilized contaminants,
- consumption (incidental ingestion) of surface soils,
- dermal uptake, and
- ingestion of contaminants which have bioaccumulated into forage or prey items.

The ecological risk assessment is presented in three steps:

Step I--Problem Formulation

Problem formulation is a process which determines the feasibility, scope, and objective of the

assessment. This step identifies the COPCs, the organisms which are representative of the site, the models to be used to determine potential dose from COPCs, and appropriate assessment and measurement endpoints.

Chemical analysis of the surface soils and the toxicity-background concentration screening process resulted in identification of the following:

	Inorganic COPCs	*Organic COPCs*
Production Area	11	44
Warwick Area	14	61
Waste Water Treatment Area	11	54
Seep in the Waste Water Treatment Area	11	31

Representative species were chosen to represent the major trophic levels: a small omnivorous mammal (deer mouse, *Peromyscus* sp.), a large terrestrial omnivore (raccoon, *Procyon lotor*), an aquatic carnivorous bird (great blue heron, *Ardea herodias)* and a carnivorous bird and protected species (red-tailed hawk, *Buteo jamaicensis*). The site, although highly disturbed, has habitat that could be used by each of these organisms. The great blue heron would only possibly use the groundwater seep in the Waste Water Treatment Area. Measurement endpoints for the organisms were chosen as the no-observable-adverse-effect level (NOAEL) for each COPC.

Step II-Exposure Characterization

Models of exposure pathways were developed to estimate daily dose to the representative organisms. These models include direct exposure, as well as bioaccumulation through the food chain.

Step III--Risk Characterization

The risk characterization step quantifies the likelihood of COPCs to cause adverse effects. The toxicity reference values (TRVs), which are equivalent to the NOAELs, are compared to predicted daily doses consumed by each representative species. A toxicity quotient (TQ) is developed that indicates the potential for adverse effects. Assumptions, strengths, weaknesses,

and uncertainties of the analyses are discussed as well as potential ecological significance of any effects.

A COPC is judged to have potential for adverse effects if the TQ is greater than zero. All TQs were less than zero.

Potential cumulative effects were also estimated by calculating an ecological toxicity index (ETI). Index values below one indicate no potential for adverse effect, values between one and ten have some possibility for effect, and probable adverse impact occurs when the ETI exceeds 10.0. The ETI was greater than 1.0 (1.36) only for the deer mouse in the Waste Water Treatment Area. The ETI was below one for all other species in the three Areas.

Potential risk from COPCs in the three areas at the site is small. The TQ for any single chemical is below zero, indicating no potential for adverse effects. The ETI for the Waste Water Treatment Area is only 1.36 for deer mice. Therefore, the ETI indicates no significant risks are expected to the representative species.

Media Protection Standards

Neither the public health nor ecological risk assessment showed that corrective actions are necessary for the three Site areas, with the possible exception of the Warwick Area. The possible exception is one of perception due to the USEPA-imposed assumption that all PCBs are treated as if they were the carcinogenic PCB 1260. No PCB 1260 was detected in the Warwick Area. The PHERE corroborates that the voluntary PCB hot-spot removals begun during the IRMs in the Production and Warwick Areas are more than sufficient to return the Site to productive uses without unacceptable risks to public health and the environment.

The PHRA models for the scenarios evaluated were used to estimate risk-based MPS values for total PCBs in the hot spots targeted for remediation in the IRMs. These MPSs were developed solely for the purposes of the IRMs, and not because of any overriding potential public health or ecological risks. Using a THI value of 1, MPSs were back-calculated through the risk assessment model to the respective surface soil concentrations. The resulting total PCB MPSs are 50 ppm for the Production Area and 5 ppm for the Warwick Area.

6.1 Introduction

A Public Health and Environmental Risk Evaluation (PHERE) was prepared as part of the RFI, as required in the Order. It separately evaluates the potential human health and ecological risks associated with the Production, Waste Water Treatment, and Warwick Areas (Figure 6-5). The PHERE is presented in two parts: the public health risk assessment (PHRA) and the ecological risk assessment. The PHRA is consistent with the approach outlined in the *U. S. Environmental Protection Agency (USEPA) Risk Assessment Guidance for Superfund, Human Health Evaluation Manual* (HHEM) (USEPA, 1989a). The PHRA approach and values for exposure assumptions include those discussed during several meetings and teleconferences with the USEPA Region I (Region I) beginning with a meeting on May 17, 1994. The ecological risk assessment utilizes the assessment process as defined by the *Framework for Ecological Risk Assessment* (USEPA, 1992). The PHERE is prepared in a baseline approach, as directed by the USEPA. This is, the assumption is made that the property will be used in the future without modification from its present condition, regardless of the practicality of this assumption.

6.1.1 Purpose and Scope

The purpose of the PHERE is threefold:

- Provide estimates of potential risks posed by site-related chemicals in the Production, Waste Water Treatment, and Warwick Areas of the Site using the guidance specified by Region I.
- Identify the site areas and chemicals that might require corrective action.
- Provide site-specific risk assessment models for human and ecological health to develop risk-based media protection standards (MPS) for surface soil.

This PHERE is designed to provide a conservative, quantitative estimate of potential risks associated with residual, site-related chemicals in the Production, Waste Water Treatment, and Warwick Areas. It was performed by selecting chemicals of potential concern (COPCs) and carrying them through the risk assessment process consistent with the principals in the USEPA guidance documents. The COPCs were selected based on their toxicities, frequencies of detection, and the concentrations at which they were detected in site soil. Regarding potential future land use, unrestricted residential use was assumed for the Waste Water Treatment and Warwick Areas. The PHERE reflects an on-site worker scenario for the Production Area, based on a proposal to use the Production Area as a vehicle parking facility. The PHERE also

considers the potential human health effects associated with migration of site-related chemicals, via groundwater, to the Pawtuxet River. Potential impacts to aquatic biota will be evaluated in a separate study to be included with submission of the river RFI in March, 1996.

6.1.2 PHERE Organization

Section 6.2 describes the risk assessment methods and chemical analytical data on which the PHERE is based. Section 6.3 describes background soil and groundwater concentrations of chemicals, compares them to on-site concentrations, and defines under what conditions chemicals were separated for further consideration in the PHERE. Section 6.4 presents the PHRA, and Section 6.5 presents the ecological risk assessment. Both assessments address:

- COPC selection process and lists the COPCs for the three site areas.
- Exposure assessment, which includes a description of the exposure setting, potential exposure pathways, potential receptors, chemical intake assumptions, and potential exposure point concentrations.
- Toxicity of the COPCs.
- Risk characterization estimates potential public health and ecological impacts.
- Uncertainties analysis.

MPSs are proposed in Section 6.6. References follow the body of the text in Section 6.7. Tables and figures follow of the text. Appendices 6-A through 6-H provide back-up for the text.

6.2 Risk Assessment Methods and Analytical Data

6.2.1 Risk Assessment Methods

The PHERE was performed following HHEM guidance for the PHRA and the Framework guidance for the ecological risk assessment . This guidance includes appropriate use of the validated data, selection of COPCs, and methods for exposure assessment, toxicity assessment, risk characterization, and uncertainties analysis. The overall site-specific methods were discussed in many meetings and teleconferences with Region 1. Heavy reliance on guidance documents was stressed in these discussions. A partial list of the primary guidance documents and other key sources of information used in the preparation of the PHERE are listed below. Comprehensive detailed methods are given in the text and appendices of the PHERE.

- Integrated Risk Information System (IRIS), Toxicology Data Network, National Library of Medicine, final on-line search performed January, 1995.

- *Health Effects Assessment Summary Tables*, Office of Solid Waste and Emergency Response, Washington, D.C., (EPA/540/R-94/020), USEPA, 1994.

- *Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons,* Environmental Criteria and Assessment Office, Cincinnati, Ohio (EPA/600/R-93/089), USEPA, 1993.

- *Dermal Exposure Assessment: Principles and Applications*, Interim Report, Office of Research and Development, Washington, D.C., (EPA/600/8-91/011B), USEPA, 1992.

- *Risk Assessment Guidance for Superfund, Volume 1: Human Health Evaluation Manual, Supplemental Guidance, Standard Default Exposure Factors*, Interim Final, Office of Solid Waste and Emergency Response, Washington, D.C., (OSWER Directive 9285.6-03), USEPA, 1991.

- *Human Health Evaluation Manual, Part B: Development of Risk-Based Preliminary Remediation Goals*, Office of Solid Waste and Emergency Response, Washington, D.C., (OSWER Directive 9285.7-01B), USEPA, 1991.

- *Exposure Factors Handbook*, Office of Health and Environmental Assessment, Washington, D.C., (EPA/600/8-89/043), USEPA, 1990.

- *"Corrective Action for Solid Waste Management Units (SWMUs) at Hazardous Waste Management Facilities, Proposed Rule,"* 55 *Federal Register* 30798, July 27, 1990 USEPA, 1990.

- *Risk Assessment Guidance for Superfund, Volume 1: Human Health Evaluation Manual, Part A*, Interim Final, Office of Emergency and Remedial Response, Washington, D.C., (EPA/540/1-89/002), USEPA, 1989.

- Region I guidance for oral absorption and dermal absorption of PCBs, internal memo, based on studies performed by Fries et al., 1989, USEPA, 1995a.

- Region I policy for potential cancer risks related to PCBs, USEPA, 1995b.

- *Framework for Ecological Risk Assessment,* Risk Assessment Forum (EPA/630/R-92/001), USEPA, 1992.

- Ecological Assessment of Superfund Sites: *An Overview, Office of Solid Waste and Emergency Response*, Publication 9345.0-051, USEPA, 1991d.

Other sources of information were used as needed.

6.2.2 Analytical Data

The PHRA is based on analytical results of surface soil, subsurface soil, and groundwater samples provided in electronic database format by Woodward-Clyde Consultants (WCC). These include Phase I and Phase II investigation data. Appendix 6-A provides an evaluation in tabular form of the chemicals detected in soil and groundwater samples from the three on-site areas and the near-site background areas. This evaluation includes their detection frequencies and maximum, minimum, mean, and 95th percentile upper confidence limits (UCLs) of the mean concentrations.

Soil samples were designated by WCC as "surface soil" or "soil boring". The surface soil samples were collected at a depth range of 0.5 to 2.0 feet (or an interval within this range). The

boring samples were collected in 2-foot intervals from the surface using split-spoon samplers. Because the uppermost boring samples were collected at the 0- to 2.0-foot depth, these are included as surface soil samples in the PHRA. The remaining boring samples are referred to in the PHRA as "subsurface soil".

Surface and subsurface soil samples for the Production, Waste Water Treatment, and Warwick Areas were collected in two phases of field investigation, with two rounds of soil sampling in each phase. Phase I-Round 1 soil sampling took place during November and December, 1990; Phase I-Round 2 during March, 1991; Phase II-Round 1 during July and August, 1993; and Phase II-Round 2 during May, 1994. Additional surface soil samples were collected from the Production Area in April 1992; these are included as Phase II-Round 1 samples. Sampling locations and analytical methods are identified and discussed in the *RCRA Facility Investigation Interim Report* (Ciba-Geigy, 1991). The sampling program used a biased approach in that specific locations within Site areas suspected of potential contamination were targeted. This is especially true in the Warwick Area which was not used in the daily operations of the Facility. Therefore, the sampling analytical results are not representative of the entire Warwick Area, but predominantly represent only the highly localized area of SWMU-5 (Figure 6-5). Near-site background soil samples were collected during both rounds of Phase I, and Round 1 of Phase II.

Groundwater samples were collected from background wells (installed hydraulically upgradient of the Site) and from the Production, Waste Water Treatment, and Warwick Areas. Phase I-Round 1 groundwater sampling took place during January 1991; Phase I-Round 2 during April 1991; Phase I-Round 3 during September 1991; Phase II-Round S during August 1992; Phase II-Round 1 during August 1993; and Phase II-Round 2 during April 1994. Samples from each of the three areas were analyzed for USEPA Appendix IX parameters during Phase I-Rounds 1, 2, and 3. Groundwater from the Waste Water Treatment and Warwick Areas was collected and analyzed for Appendix IX parameters during sampling Rounds 1 and 2 of Phase II. Phase II-Round S are groundwater samples collected from the Production Area during investigations to support a stabilization action. These were likewise analyzed for Appendix IX parameters. Phase II-Round 1 and 2 groundwater samples were collected only from the Waste Water Treatment and Warwick Areas.

The acceptable validated data from each of these sampling rounds were used in the PHRA. Data

that were rejected (qualified "R") as a result of the data validation were not included in the PHERE. The data evaluations included in Appendix 6-A are comprised of the following twelve data sets:

- Surface soil for all three Site areas
- Combined surface and subsurface soil ("combined soil") for all three Site areas
- Groundwater for all three Site areas
- Surface water and sediment from the Waste Water Treatment Area seep
- Near-site background surface soil
- Near-site background soil
- Near-site background groundwater

Surface soil and subsurface soil data sets are combined for the soil-to-groundwater transport model used for modeling Pawtuxet River water concentrations, and the soil-to-air transport models used to model volatile emissions in the exposure assessment (Section 6.4.2).

6.2.2.1 Production Area

Production Area soil samples collected during Rounds 1 and 2 of the Phase I investigation were analyzed for the complete list of Appendix IX parameters and indicator compounds, as were some of the samples collected during Round 1 of the Phase II investigation. These came to a total of more than 40 surface soil and 40 subsurface soil samples.

Fifty additional Phase II-Round 1 surface soil samples were collected in April 1992 using a grid sampling pattern. These were analyzed for PCBs only. Only PCB 1248 and PCB 1254 were detected in these samples. The other PCB mixtures were either not detected in these samples or the data were rejected during data validation.

Ten surface soil samples were collected from the Production Area during Phase II-Round 2. Nine of these samples were analyzed for PCBs only; the tenth was analyzed for PCBs and arsenic. The sample analyzed for arsenic was collected from the same sampling location (SF-A13-C27(S)) as was a Phase I-Round 2 sample in which arsenic was detected at a relatively high concentration (125 mg/kg). The Phase II-Round 2 sample collected from this location was analyzed for arsenic to verify the value found in the sample collected during Phase I-Round 2.

Approximately 50 groundwater samples were collected from the Production Area during Phase I-

Rounds 1 through 3, and Phase II-Round S. Most of these were analyzed for the complete list of Appendix IX parameters, although some samples were analyzed for a partial list of these.

6.2.2.2 Waste Water Treatment Area

A total of 21 surface and 13 subsurface soil samples were collected from the Waste Water Treatment Area. Samples collected during Phase I-Rounds 1 and 2, and Phase II-Round 1 were analyzed for the complete list of Appendix IX parameters and indicator compounds. Some of the samples were analyzed for a partial list of these during Phase II. Three additional surface soil samples were collected in Phase II-Round 2 and analyzed for *gamma*-chlordane only.

Twenty-four groundwater samples were collected from the Waste Water Treatment Area during Phase I-Rounds 1 through 3, and Phase II-Rounds 1 and 2. Most of these were analyzed for the complete list of Appendix IX parameters and indicator compounds, although some samples were analyzed for a partial list of these during Phase II.

Sediment and water samples were collected from the groundwater seep in the Wast Water Treatment Area. The analytical results for these samples are summarized in Tables 6-H-9 and 6-H-10.

6.2.2.3 Warwick Area

Surface and subsurface soil samples were collected in the Warwick Area during Phase I and Phase II. Most of these were analyzed for the complete list of Appendix IX parameters, although some samples were limited to a partial list of these. A total of over 30 surface and 20 subsurface soil samples from the Warwick Area were collected for some level of Appendix IX analyses.

Twenty-seven groundwater samples were collected from the Warwick Area during Phase I-Rounds 1 through 3 and Phase II-Rounds 1 and 2. Most of these were analyzed for the complete list of Appendix IX parameters and indicator compounds, although some samples were analyzed for a partial list of these.

6.2.2.4 Background Data

A total of 17 soil samples, 12 surface and 5 subsurface, were collected from background sampling locations. These samples were collected from off-site areas near the Site but not believed to be impacted by the Site. These near-site background soil samples were analyzed for Appendix IX, PCDDs/PCDFs, and indicator compounds. The analytical results of these samples

provide baseline concentrations of the local soils. The background surface soil samples were collected during Rounds 1 and 2 of Phase I, and Round 1 of Phase II. The subsurface background soil samples were collected only during Phase II-Round 1.

Fourteen groundwater samples were collected from upgradient, near-site sampling locations not believed to be impacted by the Site. The analytical results of these samples provide baseline concentrations of chemicals in the local groundwater. These samples were collected during Rounds 1 through 3 of Phase I and Phase II-Round 1, and were analyzed for the complete list of Appendix IX parameters, PCDDs/PCDFs, and indicator compounds.

6.3 On-Site and Background Chemical Concentration Comparison

The purpose of the PHERE is to evaluate the potential risks associated with chemicals related to past Site activities. Although naturally occurring and miscellaneous chemicals originating from human sources not related to Site activities may also pose potential environmental risks, evaluation of risks associated with background soil and groundwater levels of chemicals in this part of Rhode Island is beyond the scope of the PHERE.

Inorganics are ubiquitous in the environment and were found at detectable concentrations in near-site background soils. Therefore, the concentrations of selected inorganics analyzed for in on-site surface and combined soils were compared to those of near-site background surface and combined soils, using a t-test with separate variance. This statistical approach and results are further described in Section 6.3.1

The concentrations of organic compounds in near-site background soils were generally assumed to be zero. However, concentrations of polycyclic aromatic hydrocarbons (PAHs) in near-site background samples were observed to approximate those of the on-site samples. PAHs result from all types of combustion (including that of natural origin) and, like inorganics, are ubiquitous in the environment. Based on site history, PAHs were not used or produced at the Site. Thus, PAH concentrations in on-site soils were compared to near-site background concentrations using the same approach for inorganics. In addition to the statistical t-test approach, on-site and near site detections of PAHs are compared qualitatively in Section 6.3.2.

6.3.1 Statistical Comparisons

Concentration-toxicity screens were performed on each of the on-site media for inorganics and PAHs to determine which are most likely to present the greatest risk to human and ecological health. The approach used for the PHRA was to first list all the inorganics and PAHs separately from the other detected compounds. The screening method described in Appendix 6-B was performed on these inorganics and PAHs. Briefly, this screening involved running *t*-tests on the inorganics and PAHs identified as the greatest contributors to relative ranking scores (relative to all inorganics and PAHs). The *t*-tests were used to determine whether the on-site concentrations were greater than those found in near-site background soils. The subset of inorganics and PAHs shown by the *t*-tests to be at higher concentrations in the on-site soil than in the near-site background soil were assumed to be site-related and the contribution of background to the total

detected concentrations were not subtracted out. These inorganics and PAHs were added to the list of other organic compounds and carried into the next step of the risk assessment process as described in Appendix 6-B. The potential risks of the selected inorganics and PAHs statistically found to be at background concentrations were assessed separately (See Section 6.4.4).

The methods used to assess potential risks posed to ecological receptors differ from those used in human health risk assessment. Therefore, a somewhat different approach was used to select the inorganics and PAHs in the on-site soils that were statistically tested against background. The inorganics and PAHs representing the greatest potential ecological risks on Site, based on toxicity quotients, were analyzed statistically using the *t*-test (See Section 6.5). Those exceeding background were included in the ecological risk assessment. Also, the PAHs and inorganics not selected for statistical comparison were included in the ecological risk assessment without regard to their concentrations relative to background.

The following is the complete list of inorganics and PAHs whose concentrations in the three Site area soils were compared to those of near-site background soils:

Production Area

Arsenic	Barium	Benzo(a)pyrene
Chromium	Dibenz(a,h)anthracene	Nickel
Vanadium		

Waste Water Treatment Area

Arsenic	Benzo(a)anthracene	Benzo(a)pyrene
Benzo(b)fluoranthene	Benzo(g,h,i)perylene	Benzo(k)fluoranthene
Beryllium	Chromium	Chrysene
Dibenz(a,h)anthracene	Indeno(1,2,3-c,d)pyrene	Manganese
Vanadium		

Warwick Area

Antimony	Arsenic	Beryllium
Benzo(a)pyrene	Cadmium	Chromium
Manganese	Zinc	

The *t*-test results are summarized below:

Production Area:

- Nickel is the only selected inorganic or PAH that tested to be significantly above background concentrations. Nickel was found to be above background in combined soils only.

Waste Water Treatment Area:

- No selected inorganic or PAH was found to be present in surface or combined soil at concentrations greater than those found in near-site background soil.

Warwick Area:

- Five of the selected inorganics were determined to be present in surface and combined soils at concentrations significantly above background. These are:
  - -antimony,
  - -beryllium,
  - -cadmium,
  - -chromium, and
  - -zinc.

  Antimony could not be tested statistically because all of the background soil data for antimony were rejected during data validation. Therefore, we assume that it was present in Warwick Area soils at above-background concentrations. None of the PAHs were found to present at concentrations greater than near-site background.

On-site soil represents the source for site-related groundwater contamination. Therefore, if a given inorganic or PAH was not statistically greater in concentration than in background soil, then the presence of that chemical in groundwater was assumed to be unrelated to the Site.

6.3.2 Qualitative Comparison of PAHs

As seen in Section 6.3.1, no PAH was detected significantly above background concentrations in any of the three site areas. A qualitative comparison was also performed to better determine whether PAHs found in on-site soils are likely to be of similar origin as those detected in near-site background soils.

The PAHs were detected at higher concentrations and at greater frequencies in both on-site and near-site background surface soils than in subsurface soils. Since potential exposure at the Site is predominantly associated with surface soil (Sections 6.4.2 and 6.5.3), and PAHs are of low volatility and mobility in soil matrices, only surface soil samples were included in this comparison.

The following subsections describe qualitative comparisons of Production, Waste Water Treatment, and Warwick Areas surface soil concentrations of PAHs to those of near-site background samples. PAHs are discussed with respect to both total PAHs and benzo(a)pyrene. Benzo(a)pyrene was selected for particular discussion because it is regarded (along with dibenz(a,h)anthracene) as being among the most potent PAH animal carcinogens, and it is one of the more commonly detected PAHs in on-site and near-site soils. Data relating to on-site and near-site surface soil concentrations of each detected PAH compound and total PAHs are summarized in Tables 6-1 through 6-3. Concentrations available from literature sources of the individual PAHs found in urban soils are also shown in these tables.

6.3.2.1 Production Area

6.3.2.1.1 Total PAHs

Seventeen PAHs were detected in Production Area surface soil, as well as in the near-site background soil. Generally, the frequencies of detection are slightly greater in the background samples than in the Production Area samples (Table 6-1). Six PAHs were detected in the two sample sets at virtually the same frequency, ten were more frequently detected in the background samples, and only benzo(a)anthracene was more frequently detected in the Production Area samples. The two data sets are strikingly similar with regard to those of the 17 PAHs most frequently and least frequently detected. This is illustrated below (frequencies of detection are shown in parentheses):

Most Frequently Detected:

Frequency Rank	Production Area	Frequency Rank	Background
1	Fluoranthene (81%)	1	Fluroanthene (100%)
2	Pyrene (78%)	1	Pyrene (100%)
3	Benzo(b)fluoranthene (73%)	3	Phenanthrene (92%)
4	Chrysene (68%)	4	Chrysene (75%)
4	Phenanthrene (68%)	4	Benzo(b)fluoranthene (75%)

Least Frequently Detected:

Frequency Rank	Production Area	Frequency Rank	Background
17	2-Methylnaphthalene (9.8%)	17	2-Methylnaphthalene (17%)
16	Acenaphthene (12%)	16	Dibenz(a,h)anthracene (25%)
14	Dibenz(a,h)anthracene (24%)	14	Naphthalene (33%)
14	Acenaphthylene (24%)	14	Acenaphthylene (33%)

The five most commonly detected PAHs in Production Area surface soil are also the five most frequently detected in near-site background surface soil. Likewise, three of the four least frequently detected PAHs in the background surface soil are also three of the four least frequently detected in Production Area surface soil. The nearly identical relative concentrations of PAHs detected in on-site and near-site soils strongly suggest that PAHs detected in the Production Area and near-site areas originate from off-site sources unrelated to Site activities. The analytical results discussed above also indicate that PAHs are found ubiquitously in this urban region of Rhode Island. The mean concentration of total PAHs in the Production Area surface soil samples (22 mg/kg) is less than that of total PAHs in near-site background samples (48 mg/kg), as shown in Table 6-1.

Table 6-1 also lists background concentrations of PAHs in urban soil that are published in the literature. None of the mean or UCL of the mean concentrations exceed these ranges, and are considerably less than the maximum values of the ranges given. This indicates that the PAH concentrations of Production Area and near-site background surface soil may be lower than is typical for an urban setting.

6.3.2.1.2 Benzo(a)pyrene

Benzo(a)pyrene was detected at virtually the same frequencies in Production Area (66%) and near-site background surface soil (67%) (refer to Table 6-1). The mean concentration (1.3 mg/kg) found in Production Area surface soil samples is less than the mean concentration (2.6 mg/kg) detected in the background soil samples. White and Vanderslice (1980) list a typical range of 50 to 75 mg/kg for benzo(a)pyrene in urban soil. The concentrations of benzo(a)pyrene detected in both Production Area and near-site background soil are below this range.

It should be noted that the mean concentration for benzo(a)pyrene found in near-site background soil is skewed higher due to one surface soil sample in which this compound was detected at 22 mg/kg. However, even this value is low in comparison to the typical soil concentration range for benzo(a)pyrene (50 to 75 mg/kg) described in the literature.

6.3.2.2 Waste Water Treatment Area

6.3.2.2.1 Total PAHs

Sixteen PAHs were detected in Waste Water Treatment Area surface soil. These 16 PAHs were likewise detected in the near-site background soil (Table 6-2). The detection frequency of these 16 compounds (including 2-methylnaphthalene) is greater in background than in Waste Water Treatment Area surface soil. Only naphthalene was found to have a greater detection frequency in the Waste Water Treatment Area (50%) than in the near-site background surface soil (33%). Relative detection frequencies of PAHs within the Waste Water Treatment Area soil data set mirror those of the near-site background data set. This observation was also made for the Production Area (Section 6.3.2.1). The individual PAHs detected most and least frequently in the two data sets are listed below in order of rank with respect to frequency of detection (frequencies of detection are shown in parentheses):

Most Frequently Detected:

Frequency Rank	Waste Water Treatment	Frequency Rank	Background
1	Pyrene (89%)	1	Pyrene (100%)
2	Phenanthrene (78%)	1	Fluoranthene (100%)
3	Fluoranthene (67%)	3	Phenanthrene (92%)

Least Frequently Detected:

Frequency Rank	Waste Water Treatment Area	Frequency Rank	Background
17	2-Methylnaphthalene (0%)	17	2-Methylnaphthalene (17%)
14	Dibenz(a,h)anthracene (5.6%)	16	Dibenz(a,h)anthracene (25%)
14	Acenaphthylene (5.6%)	14	Acenaphthylene (33%)
14	Acenaphthane (5.6%)	14	Naphthalene (33%)

The three most frequently detected PAHs in Waste Water Treatment Area surface soil are the same as the three detected most frequently in the near-site background surface soil. Likewise, three of the four least frequently detected PAHs in Waste Water Treatment Area surface soil are the same as those detected in background soil. Just as for the Production Area (Section 6.3.2.1), the nearly identical relative concentrations of PAHs detected in on-site and near-site soils strongly suggest that PAHs detected in the Waste Water Treatment Area and near-site areas originate from off-site sources unrelated to Site activities. The analytical results discussed above also indicate that PAHs are found ubiquitously in this urban region of Rhode Island. The mean concentration of total PAHs in Waste Water Treatment Area surface soil samples (14 mg/kg) is less than that of total PAHs in near-site background surface soil samples (48 mg/kg), as shown in Table 6-2.

Table 6-2 also lists background concentrations of PAHs in urban soil that are published in the literature. None of the mean or UCL of the mean concentrations exceed these ranges, and are considerably less than the maximum values of the ranges given. This indicates that the PAH concentrations of Waste Water Treatment Area and near-site background soil may be lower than is typical for an urban setting.

6.3.2.2.2 Benzo(a)pyrene

Benzo(a)pyrene was detected at a lower frequency in Waste Water Treatment Area surface soil (28%) than in near-site background surface soil (67%) (see Table 6-2). The mean concentration (0.86 mg/kg) found in Waste Water Treatment Area surface soil samples is less than the mean concentration (2.6 mg/kg) detected in the background soil samples. White and Vanderslice (1980) list a typical range of 50 to 75 mg/kg for benzo(a)pyrene in urban soil. The concentrations of benzo(a)pyrene detected in both Waste Water Treatment Area and near-site background soil are below this range.

6.3.2.3 *Warwick Area*

6.3.2.3.1 *Total PAHs*

Seventeen PAHs were detected in Warwick Area surface soil, as well as in the near-site background soil (Table 6-3). The detection frequency for each compound is greater in background than in Warwick Area surface soil. Relative detection frequencies of PAHs within the Warwick Area soil data set mirror those of the near-site background data set. This observation was also made for the Production and Waste Water Treatment Areas (Section 6.3.2.1 and 6.3.2.2). The individual PAHs detected most and least frequently in the two data sets are listed below in order of rank with respect to frequency of detection (frequencies of detection are shown in parentheses):

Most Frequently Detected:

Frequency Rank	Warwick Area	Frequency Rank	Background
1	Pyrene (58%)	1	Pyrene (100%)
2	Fluoranthene (55%)	1	Fluoranthene (100%)
2	Phenanthrene (55%)	2	Phenanthrene (92%)

Least Frequently Detected:

Frequency Rank	Warwick Area	Frequency Rank	Background
15	Acenaphthene (9.7%)	17	2-Methylnaphthalene (17%)
15	Dibenz(a,h)anthracene (9.7%)	16	Dibenz(a,h)anthracene (25%)
15	Acenaphthylene (9.7%)	14	Acenaphthylene (33%)
14	2-Methylnaphthalene (19%)	14	Naphthalene (33%)

The three most frequently detected PAHs in Warwick Area surface soil are the same, in order, as the three detected most frequently in the near-site background surface soil. Likewise, three of the four least frequently detected PAHs in Warwick Area surface soil are the same as those detected in background soil. Just as for the Production and Waste Water Treatment Areas (Section 6.3.2.1 and 6.3.2.2), the nearly identical relative concentrations of PAHs detected in on-site and near-site soils strongly suggest that PAHs detected in the Warwick Area and near-site areas originate from off-site sources unrelated to Site activities. The analytical results discussed above also indicate

that PAHs are found ubiquitously in this urban region of Rhode Island. The mean concentration of total PAHs in Warwick Area surface soil samples (21 mg/kg) is less than that of total PAHs in near-site background samples (48 mg/kg), as shown in Table 6-3.

Table 6-3 also lists background concentrations of PAHs in urban soil that are published in the literature. None of the mean or UCL of the mean concentrations exceed these ranges, and are considerably less than the maximum values of the ranges given. This indicates that the PAH concentrations of Warwick Area and near-site background soil may be lower than is typical for an urban setting.

6.3.2.3.2 Benzo(a)pyrene

Benzo(a)pyrene was detected at a lower frequency in Warwick Area surface soil (42%) than in near-site background surface soil (67%) (see Table 6-3). The mean concentration (1.2 mg/kg) found in Warwick Area surface soil samples is less than the mean concentration (2.6 mg/kg) detected in the background soil samples. White and Vanderslice (1980) list a typical range of 50 to 75 mg/kg for benzo(a)pyrene in urban soil. The concentrations of benzo(a)pyrene detected in both Warwick Area and near-site background soil are below this range.

6.4 Public Health Risk Assessment

6.4.1 Chemicals of Potential Concern

The COPCs were selected using a screening process based on HHEM guidance and detailed in Appendix 6-B. Detected concentrations, frequencies of detection, and toxicities were considered during screening. Comparisons to background concentrations regarding inorganics and PAHs is described in Section 6.3. PAHs and certain inorganics were previously removed from the selection process discussed here and detailed in Appendix 6-B. The purpose of using the screening process was to limit the PHRA to the COPCs in each Site area which represent the majority of human health risks. Separate COPCs were selected for cancer and noncancer risks. These COPCs were carried through the risk assessment process.

The COPCs for the respective areas and the basis for their selection (*) regarding cancer or noncancer effects are listed below:

PRODUCTION AREA

Cancer Effects	*Noncancer Effects*
PCB 1260*	PCB 1248*
gamma-Chlordane*	PCB 1254*
Vinyl chloride*	*gamma*-Chlordane
Total PCBs	2-Nitroaniline*
	Chlorobenzene*
	Toluene*

WASTE WATER TREATMENT AREA

Cancer Effects	*Noncancer Effects*
2,3,7,8-TCDF*	PCB 1254*
Dieldrin*	*gamma*-Chlordane*
bis(2-Ethylhexyl)phthalate*	Tinuvin 327*
gamma-Chlordane	Dieldrin
Total PCBs	*bis*(2-Ethylhexyl)phthalate*

WARWICK AREA	
Cancer Effects	*Noncancer Effects*
Aldrin*	PCB 1248*
Beryllium*	PCB 1254*
Dieldrin*	2-Nitroaniline*
bis(2-Chloroethyl)ether*	Chlorobenzene*
Total PCBs	Aldrin
	Beryllium
	Dieldrin

Toxicity information for the COPCs is presented in Section 6.4.3.

6.4.2 Exposure Assessment

The exposure assessment is a critical component of the human health risk assessment. Exposure assessment methodologies used in the PHRA and the resulting estimated potential exposures are presented in detail in Appendix 6-C. With respect to chemical hazards, exposure may be defined as the contact of an individual with a chemical agent. Exposure itself does not connote risk, but without exposure or potential exposure a chemical agent poses no risk.

Exposure assessment in human health risk assessment is used to estimate the quantity of a given chemical that could cross the exchange boundaries between the environment and the body. These boundaries are generally at the gastrointestinal tract, the lungs, and the skin. But before an estimation may be made regarding the quantity of exposure, appropriate scenarios must be developed under which exposure could potentially occur.

The basic steps of an exposure assessment are to:

- characterize the exposure setting;
- identify potential exposure pathways;
- identify human receptors;
- develop exposure scenarios;
- develop exposure models; and
- quantify exposure.

The exposure setting consists of the physical environment, including the proximity of the site to current human populations. The identification of potential exposure pathways considers the characterization of the exposure setting, impacted environmental media, and medium-to-medium transport. The identification of human receptors includes both current and future populations identified during the characterization of the exposure setting. Potential future land uses are evaluated to identify potential future human receptors. The associated routes of exposure for an identified receptor population is referred to as an exposure scenario. Exposure scenarios are developed based on the receptors and potential exposure pathways. Determinations are made regarding which routes of exposure are appropriate for inclusion in the exposure assessment for each identified potential human receptor population.

Exposure quantification uses information from the previous exposure assessment steps. Exposure equations are used to quantify exposure associated with each selected pathway in the exposure scenario, and these comprise the exposure models. Variables used in the exposure equations include, among others, measured concentrations of chemical in the media, contact rates with the media, frequency of exposure, exposure duration per exposure event, body weight of the exposed individual, total duration over which an individual is exposed, and the time period over which the exposure is averaged. Most of these input variables are site-, medium-, and receptor-specific and may include measured, modeled, or default values. However, they are also chemical specific, relying on factors that influence concentration.

6.4.2.1 Exposure Setting

This section describes features of the Site and its location as they apply to human health risk assessment. A detailed site description is given in the RFI Report.

The Site is located along the Pawtuxet River in Cranston, Rhode Island. The climate may be characterized as temperate with four well-defined seasons, and is heavily influenced by Narragansett Bay and the Atlantic Ocean. The mean annual temperature is approximately 50°F, with a daily mean during the coldest month (January) of 29°F and the warmest month (July) of 73°F. The mean annual number of freezing days (minimum temperature of 32°F or less) is 114. The average annual rainfall is approximately 42 inches per year. Measurable precipitation (0.01 inches of rain equivalence) averages 124 days annually and is typically distributed evenly throughout the year. The annual snowfall averages approximately 36 inches, over half of which usually falls during January and February. The wind blows most commonly from a northwestern direction and least commonly from an eastern direction. The average annual wind speed is 11

miles per hour. Meteorological data are from the weather station in Providence, Rhode Island and are contained in Volume 1 of the *RCRA Facility Investigation Proposal* (Ciba, 1990). Additional data are from the National Oceanic and Atmospheric Administration (1990).

The Production and Waste Water Treatment Areas are located on the north shore of the Pawtuxet River in Cranston; the Warwick Area is on the south shore of the Pawtuxet River and is in Warwick, Rhode Island (Figure 6-5). Areas surrounding the Site are used for commercial, industrial, or residential purposes. The area west of the Production Area is industrial; areas north and east of the Production Area are residential. Based on different levels of impact and probable future land use, the Production Area, as defined in the Consent Order with the USEPA, is for the purpose of the PHRA divided into two parcels: the Laboratory and Warehouse Building Area and the Production Area. These are identified on Figure 6-1. Virtually no site-related chemicals were found in the Laboratory and Warehouse Building Area, thus it was not evaluated further in the RFI. Therefore, only the risks associated with the Production Area identified on Figure 6-5 are evaluated in the PHRA.

The Warwick Area is bordered by land in commercial use to the east and residential use to the south. The river lies north and west of this area. The areas west and north of the Waste Water Treatment Area are residential, and the land east of this area is currently used for commercial purposes.

6.4.2.2 Potential Exposure Pathways and Human Receptors

An exposure pathway may be defined as a course that a chemical may take from a source of contamination to an individual. The following four elements are necessary for an exposure pathway to be complete:

- Contamination source and release mechanism;
- Retention medium or transport medium;
- Point of potential human contact with the impacted medium; and
- Human exposure route at the contact point.

The sources and release mechanisms involve previous chemical manufacturing, chemical handling, and waste handling and disposal activities which have occurred in the Site areas. Impacted media which may serve to retain and/or release the contamination are surface soils, subsurface soils, and groundwater. Points of human contact with the impacted media are dependent on land use.

One purpose of the PHRA is to provide conservative estimates of risk. On-site residential use would represent a "worst-case" land use. Ciba has assumed for risk assessment purposes that future on-site residential risk ought to be evaluated for the Warwick and Waste Water Treatment Areas. Although residential land use may not be the most probable for this area, to be conservative the PHRA evaluates this land use. On-site occupational exposure is assumed for the Production Area because it is being proposed for use by the City of Cranston as parking for city vehicles, and as a storage and loading area for road salt, sand, and snow removal equipment. An on-site worker will potentially occupy this area full-time, but only during the four coldest months of the year performing activities related to snow and ice management of City streets. Parking and vehicle removal by a wide array of City employees will be the only activity for the other 8 months of the year (City of Cranston, 1995). Vehicle maintenance will be conducted within the buildings. A nonspecific commercial/industrial worker scenario which does not consider the planned use of the Site by the City is also evaluated.

6.4.2.2.1 On-Site Worker Scenario/On-Site Resident Scenario

The on-site worker scenario for the Production Area, and the on-site resident scenario for the Waste Water Treatment and Warwick Areas are evaluated assuming that no modifications are made to the property, such as soil removal or bringing in clean topsoil. The media that may affect a future on-site resident or on-site worker include surface and subsurface soils. Exposure pathways associated with these scenarios are:

- Direct contact with surface soil resulting in incidental ingestion;
- Direct contact with surface soil resulting in dermal absorption;
- Inhalation of airborne chemicals associated with fugitive dust emissions from surface soil; and
- Inhalation of volatilized chemicals associated with surface and subsurface soil.

It is not reasonable to assume that groundwater from the shallow aquifer underlying the vicinity of the Site will be used as drinking water. Groundwater beneath the Site is classified by RIDEM as GB. It is not suitable for public or private drinking water use and is typical of highly urbanized areas with dense concentrations of industrial and commercial activities. This classification is also used for groundwater underlying permanent waste disposal areas (such as landfills). There are no private wells in the vicinity of the site. Because of the limited areal extent of groundwater contamination at the Site, and because the contaminated groundwater discharges directly to the Pawtuxet River, the river pathway for groundwater is the only potential exposure route. In addition, virtually no site-related contamination was found in deeper aquifers.

Site-related chemicals were detected only in shallow groundwater, which follows a strong gradient toward the Pawtuxet River bordering the Production Area. A RCRA Stabilization Action is addressing this groundwater in the Production Area.

Figure 6-6 illustrates the exposure pathways evaluated in the PHRA for these exposure scenarios. Values for the exposure assumptions for these two scenarios are shown in Table 6-4.

6.4.2.2.2 Canoeist Scenario

Because groundwater underlying the Site will not be used for drinking water, a canoeist scenario was developed for each of the Site areas to evaluate the potential risk of impacted groundwater to the human receptor most likely to be in maximum contact with this medium. Potential ingestion of fish will be addressed in the Pawtuxet River RFI. Groundwater contact with regard to the canoeist scenario would be indirect; actual exposure would be to Pawtuxet River water to which groundwater underlying the Site has discharged. Predicted river water concentrations of COPCs were modeled by WCC for each of the three Site areas. The model uses measured groundwater and soil concentrations of the COPCs, as described in Appendix 6-D, to predict Pawtuxet River water concentrations.

Figure 6-6 illustrates the exposure pathways evaluated in the PHRA for this exposure scenario. Values for the exposure assumptions for the canoeist scenario are shown in Table 6-5.

6.4.2.3 Potential Exposure Point Soil Concentrations

The COPC concentrations for the environmental media pertinent to the exposure assessment are shown for the three Site areas in Table 6-6. The concentrations given for surface soils and combined surface and subsurface soils were derived from direct measurements (Appendix 6-A). Air concentrations shown on Table 6-6 are predicted from measured surface soil and combined soil concentrations using the modeling procedures described in Appendix 6-D.

Concentrations of COPCs for these soils are the lesser of either the 95th percentile UCL of the means or the maximum detected concentrations. For compounds detected in one or more surface soil samples from a given Site area, the 95th percentile UCLs of the arithmetic means were calculated using the detected value, or one-half the sample quantitation limit (SQL) for samples in which the chemical was not detected. The methods for determining statistical distribution type and calculating the 95th percentile UCL of the mean are described in Section 6.4.2.3.2. The surface soil values in Table 6-6 were used in the soil ingestion and dermal absorption exposures

estimated for the on-site resident and worker scenarios (Appendix 6-C) . They were also used in the inhalation pathway exposure estimates of fugitive dust (Appendix 6-D). The values shown in Table 6-5 for combined subsurface and surface soil concentrations were used for estimating exposure to chemical vapors (Appendix 6-D), and for modeling soil-to-groundwater concentrations (Appendix 6-E).

6.4.2.3.1 Total PCBs Data Sets

Region I policy is to assume that all PCBs have the same cancer potency as PCB 1260 (see Sections 6.4.3 and 6.4.4) (USEPA, 1995b). However, soil samples are analyzed and concentrations reported for the separate PCB mixtures. Thus, to assess the potential risks of total PCBs as requested by Region I, the analytical results of all the PCBs detected within a medium were combined to form a separate data set for that medium. For example, three PCBs were detected in Production Area surface soil. The concentrations of PCB 1248, PCB 1254, and PCB 1260 were summed for each Production Area surface soil sample. For samples in which a given PCB was not detected, one-half the sample quantitation limit (SQL) was used. This same approach was taken for Warwick Area surface soil, except that only PCB 1248 and PCB 1254 were summed because neither PCB 1260 nor any other PCB was detected in any Warwick Area surface soil sample. The 95th percentile UCL of the mean was used as the exposure point concentration, just as for the data sets of the respective COPCs.

The combined surface soil and subsurface soil data sets are used in the PHRA only for the soil-to-air volatilization model and the soil-to-groundwater-to-surface water model. Because different volatilization rates have been modeled for the individual PCBs, the modeled gaseous concentrations of the separate PCBs were summed to derive an overall exposure to total PCBs with regard to this exposure pathway. The total PCBs surface water concentration is estimated from summing the modeled values of the individual PCBs.

6.4.2.3.2 Statistical Distribution of Chemicals in Soil

Statistical analyses were performed to determine the type of distribution represented by each COPCs detected in soil. The type of statistical distribution of the chemical analytical data should be identified, if possible, for a more meaningful exposure point concentration estimate. If a given analyte is detected in too few soil samples, then the type of statistical distribution of the analyte in the soil cannot be reliably ascertained. As described in Section 6.4.1, if a chemical is not detected in a given sample, then one-half the SQL is the assumed concentration. These one-half SQL values do not accurately portray the actual concentrations, but are used expressly for

exposure assessment purposes. This is particularly true if SQL values of the nondetected samples are high relative to the detected values in a data set. The statistical methods and description of the general procedures used in the PHRA to determine distribution type are described in the following paragraphs.

Each data set was first evaluated for frequency of detection. The PHRA uses a lower limit detection frequency of 75% to determine whether a data set can be statistically tested to evaluate its distribution. Data sets with detection frequencies greater than 75% were evaluated using the Shapiro-Wilk *W*-test for normality. If the data set meets the test criteria for normality at $p<0.05$, then the chemical is assumed to be normally distributed over the given Site area, and the 95th percentile UCL of the arithmetic mean based on a normal distribution was used to estimate the exposure point concentration. If the data set fails the test for normality, then a Shapiro-Wilk *W*-test for lognormality was performed. If the data set meets the criteria for lognormality, then the chemical was assumed to be lognormally distributed over the given Site area, and the 95th percentile UCL of the arithmetic mean concentration based on a lognormal distribution was used to estimate the exposure point concentration. If a data set meets the criteria for neither statistical distribution, then distribution was assumed to be normally distributed for purposes of calculating the 95th percentile UCL of the mean.

6.4.2.4 Potential Exposure Point Pawtuxet River Water Concentrations

The approach used to estimate exposure point concentrations for the Pawtuxet River is similar to that used for the estimation of potential exposure point soil concentrations (Section 6.4.2.3). The lesser of either the highest detected concentration or the 95th percentile UCLs of the groundwater and of combined soil and subsurface soil concentrations were used as input values to predict river water concentrations of the COPCs associated with each of the Site areas (Appendix 6-E). Groundwater concentrations were predicted from the combined soil values using leaching and infiltration assumptions. For each COPC in each Site area, the greater of either the predicted groundwater concentration or the value based on measured groundwater concentrations were used to predict water concentrations in the Pawtuxet River based on groundwater discharge from each Site area. Two modeling scenarios were assumed: a low flow/low distribution coefficient scenario and a high flow/high distribution coefficient scenario. The latter scenario was selected for the PHRA exposure calculations because the modeled chlorobenzene concentration under this scenario (1 ug/l) is similar to those measured over a six-month monitoring period in the Site area of the Pawtuxet River. Chlorobenzene is the only COPC for which this type of monitoring data of the river is available.

6.4.2.5 Exposure Assessment Results

The calculated potential exposures are determined by a number of exposure assumptions and variables for each scenario. Methods for calculating exposure are presented in Appendix 6-C. Input values for calculating exposure are shown in Table 6-4 for the on-site resident and on-site worker scenarios, and in Table 6-6 for the canoeist scenario. The exposure assessment results, given in Appendix 6-C, are carried into the risk characterization.

6.4.3 Toxicity Assessment

Toxicity assessment consists of identifying and evaluating toxicity criteria and health effects information for the chemicals detected in impacted and/or potentially impacted media. In the PHRA, toxicity criteria were identified during the COPC screening process (Appendix 6-B). Attention is given to the relationship between the level of the exposure and the severity of any resultant adverse health effects. Specific adverse health effects are noted for each chemical carried through the risk assessment process, particularly those effects on which the toxicity criteria are based. Information obtained during the toxicity assessment is used in the risk characterization (Section 6.4.4) to estimate risks associated with the exposure levels estimated during the exposure assessment (Section 6.4.2).

Toxicity information for the COPCs and background compounds is shown in Table 6-7 and the full Integrated Risk Information System (IRIS) print-outs are given in Appendix 6-F. This information includes the following:

- Chronic reference doses (RfDs);
- Cancer slope factors (CSFs);
- Target organs for adverse health effects;
- Tumor sites; and
- USEPA weight-of-evidence classification system for cancer effects.

The items listed above are described in the following subsections.

6.4.3.1 Health Effects Classification

Chemicals may exhibit a variety of adverse health effects. For risk assessment purposes, these adverse effects are generally divided into two categories: noncancer and cancer. The reason for this distinction is the opinion that the mechanism for each is different. It is generally believed that the body has protective mechanisms against most noncancer effects. These defenses must be overcome by a given exposure level of a toxicant before any adverse effects occur. Therefore, it

is thought that a range of exposure levels from zero to some finite threshold level can be tolerated with essentially no risk of adverse health effects.

Unlike noncancer effects, cancer is assumed by USEPA in most cases not to have a threshold level (USEPA, 1989a). The hypothesized mechanism of carcinogenesis assumes that there is essentially no level of exposure to a carcinogen that does not pose a finite probability, however small, of generating a carcinogenic response.

The USEPA-preferred and most regularly updated source of toxicity information is the (IRIS) on-line data base. IRIS was the primary source of health effects criteria used in this toxicity assessment, and IRIS toxicity profiles are included as Appendix 6-F. When health effects criteria were not found in IRIS, this information was sought in the Health Effects Assessment Summary Tables (HEAST-USEPA, 1994), the agency's second preference. Other sources of toxicity information were used only when the health effects criteria were not available in IRIS or HEAST. Health effects criteria for noncancer effects and cancer effects are discussed in Sections 6.4.3.2 and 6.4.3.3, respectively. A given chemical may exhibit both noncancer and cancer effects.

6.4.3.2 Health Criteria for Noncancer Effects

The assessment of toxic effects for a noncarcinogenic chemical is based on the RfD approach. An RfD is a daily human oral intake level measured in milligrams of chemical per kilogram of body weight (mg/kg-day), such that a lifetime of exposure to a given toxicant at the RfD level theoretically poses virtually no risk of deleterious effects (USEPA, 1989a). It is developed or verified by USEPA's RfD/RfC Work Group using oral toxicity data. Reference concentrations (RfCs) are developed or verified for inhalation also by USEPA's RfD/RfC Work Group. An RfC is a daily human inhalation intake based on a constant lifetime average concentration of a chemical in air, measured in milligrams of chemical per cubic meter of air (mg/m^3). Likewise, they are derived from toxicity data to be within a tolerable threshold level that poses virtually no risk of deleterious health effects. RfCs may be converted to RfDs using exposure assessment calculations. Note that in Table 6-7, inhalation route RfDs are referred to as "RfD_is". RfDs are also used for the dermal absorption route of exposure. Chemical-specific differences of absorption via the oral and dermal routes are addressed separately in the exposure assessment (Section 6.4.2 and Appendix 6-C).

Even though RfDs are designed to be below threshold health effects levels using conservative

assumptions, it cannot be definitively stated that a given level of exposure below the RfD poses no risk. Neither can it be assumed that a given exposure level above the RfD poses a definite human health risk. The most sensitive subpopulations are considered in establishing RfDs.

An RfD is derived from human studies that provide some quantification of exposure or from animal studies. If available, a no-observed-adverse-effects level (NOAEL) is used. Uncertainty factors, typically of an order of magnitude each, may be used to account for the following:

- Variations in sensitivity among the exposed population;
- Extrapolations from animal studies to human exposures;
- Extrapolations from shorter term studies to chronic exposures; and
- Extrapolations from a lowest-observed-adverse-effects level (LOAEL) to a NOAEL.

An additional uncertainty or modifying factor is used to reflect professional judgement of the uncertainties of the study and the database not explicitly addressed by the above factors. The modifying factor may range from one to less than ten. When combined, these uncertainty factors may result in a nearly 100,000-fold margin of safety with respect to the toxicity criteria. Therefore, an RfD or RfC is biased in overestimating the possibility of toxic effects from exposure to a chemical.

6.4.3.2.1 RfD for PCB 1248

PCB 1248 has no USEPA-established reference dose (RfD), so it was necessary to derive a provisional RfD. PCB 1248 elicits both developmental and immunologic effects, with developmental appearing to be the critical effect. A provisional PCB 1248 RfD of 8×10^{-5} mg/kg-day was derived for developmental effects (Table 6-7), and a provisional RfD of 1×10^{-3} mg/kg-day for immunologic effects was also derived. Because of potential additive toxicity with PCB 1254, immunologic effects of PCB 1248 are relevant to the PHRA. A detailed discussion of how these provisional RfDs were developed is given in the following subsections.

6.4.3.2.1.1 Developmental Effects

In addition to IRIS, the Hazardous Substances Data Bank (HSDB), Registry of Toxic Effects of Chemical Substances (RTECS), Developmental and Reproductive Toxicology Database (DART), and TOXLINE on-line databases were searched for toxicity information on PCB 1248. The journal articles referenced in these databases were reviewed. As mentioned above, the

critical effect that occurs at the lowest dose is a developmental effect. In the key study (Allen et al., 1979) adult female rhesus monkeys (eight per exposure level) were fed PCB 1248 at estimated doses of 0.008 and 0.016 mg/kg-day for 18 months. After seven months of exposure, the primates were bred and the mothers and offspring evaluated for toxic effects. Six of eight conceptions at the lower exposure level, and seven of eight at the higher level resulted in live births, and the infants survived the experimental period. No maternal toxicity was observed at either dose level, but the infants were somewhat smaller than controls at birth. These infants gained less weight than controls during the nursing period, and they developed focal areas of skin hyperpigmentation. These are some of the classic signs of PCB intoxication. A PCB 1248 RfD of 8×10^{-5} mg/kg-day for people is estimated using the following uncertainty factors:

Extrapolation from a lowest-observed adverse effect level to a NOAEL = 10

- The standard default value was used because several of the 16 female rhesus monkeys in the combined 0.008 and 0.016 mg/kg-day dose groups (all of which conceived) had resorptions/abortions (Allen et al., 1979). Unfortunately, the reproductive performance of the control group is not specified. Reproductive performance in rhesus monkeys is highly variable among colonies, but 25 to 35% fetal losses in pregnant females is common. However, other publications by this group of investigators report no fetal losses in control groups for PCB studies conducted in the same time frame as that of Allen et al. (1979). Even though this level of reproductive performance is highly unusual, it can only be assumed from the information given that no fetal losses were experienced in the control group. Otherwise, a LOAEL to NOAEL uncertainty factor of 3 could be justified. The somewhat lower birth weights and weight gain observed in the study relative to controls was not characterized by the authors as statistically significant. Schantz et al. (1989) made similar observations in PCB 1248 rhesus monkey studies conducted at the same laboratory at maternal exposure levels of 0.016 and 0.040 mg/kg-day. They also characterized the hyperpigmentation of infants as mild, and reversible after weaning at these exposure levels. This implies that the 0.008 mg/kg-day exposure level in the Allen et al. (1979) study is close to a NOAEL dose. This justification is similar to that used by the USEPA for using the uncertainty factor of 3 for NOAEL estimation in deriving the RfD for PCB 1254 because of the less severe effects on periocular tissues and nail beds in rhesus monkeys at lower doses (IRIS, 1995; see Appendix 6-F).

- Extrapolation from rhesus monkeys to man = 1
  - Explanation: The vast majority of differences in the severity of toxic effects at similar dose levels of a given chemical among test animal species is related to differences in metabolism and toxicokinetics. Comparative PCB metabolism and toxicokinetic studies in man relative to monkeys, dogs, and rats show that these species handle PCBs in a manner similar to people (Schnellman et al., 1983, 1984, 1985). Monkeys match best with the human data, a conclusion which is corroborated by the Agency for Toxic Substances Disease Registry (ATSDR, 1991) and the USEPA (IRIS, 1995; see Appendix 6-F). This close similarity between monkeys and humans based on data that are rarely available in people, and the fact that rhesus monkeys exhibit adverse PCB health effects at doses ten-fold lower than in other species, justifies direct extrapolation to people.

- Human variability = 10
  - Explanation: Standard default.

6.4.3.2.1.2 Immunologic Effects and Potential Additive Toxicity

The potential for additive toxicity of PCB 1248 and PCB 1254 was evaluated. According to USEPA guidance, additivity is to be considered if two or more compounds affect the same target organ or have the same mechanism of action (USEPA, 1989a). Developmental toxicity, the critical effect of PCB 1248, is not listed in IRIS or any other database searched as a critical effect of PCB 1254. Immunological effects are a critical effect listed in IRIS for PCB 1254. PCB 1248 also elicits immunologic effects. Therefore, potential additive effects of PCB 1248 and PCB 1254 were evaluated with respect to immunologic effects. The application of additive toxicity is discussed in the risk characterization (Section 6.4.4).

The lowest dose at which an immunologic effect was observed for PCB 1248 is 0.2 mg/kg-day (Thomas and Hinsdill, 1978). After eleven months on experimental diets resulting in a dose level of either 0.1 or 0.2 mg/kg-day, two groups of eight rhesus monkeys were injected intravenously with sheep erythrocytes (SRBCs). A third, control group was likewise injected with SRBCs. Compared to the 0.1 mg/kg-day and control groups, the 0.2 mg/kg-day group showed a significantly reduced SRBC antibody titer one week after primary immunization. At a dose of 0.1 mg/kg-day, no immunologic effect was observed. This lower dose is regarded as a NOAEL for immunologic effects. An uncertainty factor of 10 to extrapolate chronic exposure, a factor of 10 to account for human variability, and a factor of 1 to extrapolate from rhesus

monkeys to humans (Section 6.4.3.2.1.1) were used to estimate a PCB 1248 provisional RfD for immunologic effects. If the NOAEL for immunologic effects (0.1 mg/kg-day) is divided by the combined factor of 100, the resulting provisional RfD is 1×10^{-3} mg/kg-day. This value is about 12 times greater than the provisional RfD calculated for developmental effects.

As shown in Table 6-7, the established RfD for PCB 1254 is 2×10^{-5} mg/kg-day. This value is based on ocular exudate, meibomian gland effects, distorted growth of nails, and decreased antibody response to SRBCs in rhesus monkeys dosed at 5×10^{-3} mg/kg-day (IRIS, 1995). The provisional RfD of PCB 1248 with regard to immunologic effects is 50 times higher than the RfD for PCB 1254.

Even though the critical effects of PCB 1248 and PCB 1254 are different, potential additive immunologic effects may affect the estimation of MPS values. As stated above, the RfD for PCB 1254 is 50 times lower than the provisional RfD for PCB 1248 based on immunologic effects. Therefore, 50 mg/kg of PCB 1248 equals 1 mg/kg of "PCB 1254 equivalents" in the use of this relationship to estimate acceptable residual PCB soil concentrations.

Additivity with regard to developmental effects might also be pertinent if PCB 1248 was detected at significantly higher concentrations than PCB 1254 at the Site. However, in the databases that exist for site soil, PCB 1254 is detected with greater frequency and generally at higher concentrations than PCB 1248. Thus, the critical immunologic effects of PCB 1254 and the additive immunologic effects of PCB 1248, from a toxicity viewpoint, "drive" the estimation of MPS values for PCBs.

6.4.3.3 Health Criteria for Cancer Effects

Human carcinogens and potential human carcinogens are categorized into the following groups by USEPA Human Health Assessment Group's weight-of-evidence classification system:

- **Group A**
  Human Carcinogen (sufficient evidence of carcinogenicity in humans).
- **Group B**
  Probable Human Carcinogen (B1--limited evidence of carcinogenicity in humans; B2--

sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans).

- **Group C**
  Possible Human Carcinogen (limited evidence of carcinogenicity in animals and inadequate or lack of human data).
- **Group D**
  Not Classifiable as to Human Carcinogenicity (inadequate or no evidence).
- **Group E**
  Evidence of Noncarcinogenicity for Humans (no evidence of carcinogenicity in adequate studies).

Quantitative cancer risk assessments are performed on chemicals in Groups A and B, and on a case-by-case basis for Group C. The quantification of potential human cancer risks exhibited by a chemical is based on its cancer slope factor (CSF). In practical terms, a CSF is an estimate of the risk associated with a chronic daily intake of one milligram of chemical per kilogram of body weight $(mg/kg\text{-}day)^{-1}$. Separate CSFs are derived for the oral (CSF_o) and inhalation (CSF_i) exposure routes. Typically, IRIS lists no CSF_i value, but instead lists an inhalation unit risk (UR_i). The UR_i is the potential cancer risk associated with an average lifetime exposure to an airborne concentration of one microgram of a chemical per cubic meter of air $(\mu g/m^3)^{-1}$. UR_i values can be converted to provisional CSF_i values using exposure assessment methodologies. Similar to the case of noncancer effects (Section 6.4.3.2), CSF_o values may be used for the dermal absorption exposure route, using chemical-specific factors to adjust for the differences in absorption between the oral and dermal routes.

CSFs are calculated through the use of mathematical extrapolation models. Generally, the USEPA currently limits its extrapolation to the linearized, multistage model, despite heavy criticism from the scientific community. This model incorporates data from studies performed using high doses relative to potential environmental exposure, and estimates the largest possible linear slope within the 95th percentile upper confidence limit, extrapolating the study data to a low dose. Because of the choice of mathematical model and of the 95th percentile upper confidence limit, the CSF represents a conservative, upper-bound estimate of the potential cancer risk of a chemical to humans.

6.4.4 *Risk Characterization*

The objective of risk characterization is to evaluate and quantify the potential risks associated with a site. This is done by combining the exposure levels estimated in the exposure assessment (Section 6.4.2) with the appropriate toxicity criteria identified during the toxicity assessment (Section 6.4.3) to quantitatively estimate potential cancer risk for carcinogens and the potential for noncancer adverse health effects. Because of basic differences in the mechanisms of toxicity, the risks associated with cancer and noncancer adverse health effects of chemicals are characterized separately. Risk characterization methodologies used in the PHRA are consistent with the HHEM and are described in Appendix 6-G. The following provides an overview of the process used in risk characterization.

The total hazard index (THI) represents the overall calculated noncancer risks posed by the COPCs in a given exposure scenario. The calculation of the THI and associated values such as hazard quotients (HQs) and hazard indices (HIs) are described in detail in Appendix 6-G. Briefly the THI is the sum of the separate chemical-specific HQ values for all of the COPCs, via all the relevant routes of exposure for the exposure scenario. The HQ is calculated by dividing the estimated chemical intake level (IN) to a chemical, via one exposure pathway, by the appropriate RfD. Both the IN and the RfD are given in units of milligrams of chemical per kilogram of body weight per day (mg/kg-day). Thus, if the IN is greater than the RfD, the HQ will exceed a threshold value of 1. The chemical-specific HI is the sum of all HQ values (via all exposure pathways) for a particular COPC.

To evaluate noncancer risk, the THI is compared to a target value of 1. The THI is rounded to one significant figure in accordance with the HHEM. If the THI is less than or equal to 1, then it is unlikely, given the exposure assumptions, that the COPCs present a health risk. If the THI (rounded to one significant figure) exceeds 1, then separate THI values should be calculated for the separate target organs. If any of the resultant target organ-specific THI values exceed the target value of 1, then a potential for adverse health effects may be indicated. When exposure to multiple chemicals with the same target organ exist, the combined effect of the chemicals may be additive, synergistic, antagonistic, or they may have no influence on one another at all. Antagonistic relationships result in health effects that are less than those predicted by a chemical given alone; synergistic relationships result in health effects that exceed the results predicted by a chemical given alone and the additive effects of chemicals with similar effects. Combined noncancer health effects on the same target organ are assumed to be additive in the PHRA. It should be noted that the THI value is to be compared to the threshold value of 1, and should not

be used as an independent, quantitative estimator of risk. The reasons for this are related to the assumption discussed in Section 6.4.3.1 of the toxicity assessment that a threshold level of exposure must be exceeded before chemicals elicit adverse noncancer health effects.

The total incremental lifetime cancer risk (ILCR) is the sum of all estimated potential cancer risks associated with all carcinogenic chemicals in a given exposure scenario. Combined cancer risks associated with exposure to multiple carcinogens are assumed to be additive, unless available information suggests otherwise. In weighing exposures to potentially carcinogenic compounds, a reasonable level of risk must be selected. Cancer is of significant occurrence in the United States with an estimated lifetime risk of developing cancer being about three out of every ten people (3×10^{-1}) (American Cancer Society, 1990). Approximately 80 percent of these cases result in death directly attributable to the disease. The USEPA regards an ILCR of between 1×10^{-6} (1 in 1,000,000) and 1×10^{-4} (1 in 10,000) as acceptable. Thus, this may be interpreted as an increase in the United States baseline cancer incidence from 300,000 per million population to a range of 300,001 to 300,100 per million population. Under the Resource Conservation and Recovery Act (RCRA), this is regarded as the protective risk range for media protection standards (USEPA, 1990b). Alternatively, a project-specific target risk range or risk level may be used. If the ILCR exceeds the upper bound of the target risk range, then further evaluation or corrective action may be indicated.

6.4.4.1 Special Considerations of PCBs

6.4.4.1.1 PCB 1248 and PCB 1254

Section 6.4.3.2 discusses the differences in the respective critical effects for PCB 1248 and PCB 1254. PCB 1254 has an RfD of 2×10^{-5} mg/kg-day based on immunologic effects. A provisional RfD of 8×10^{-5} mg/kg-day was derived for PCB 1248, based on developmental effects. This developmental effects RfD was used in the risk characterization. Because the RfD for PCB 1254 and the provisional RfD for the critical effect of PCB 1248 are based on different target organs and mechanisms of toxicity, hazard indices that result from these RfDs are not additive.

A provisional RfD was also derived for PCB 1248 that is specific for immunologic effects; this value is 1×10^{-3} mg/kg-day. This immunologic-based RfD for PCB 1248 was also used in the

risk characterization. The resultant HI is summed with the HI for PCB 1254 to estimate an additive effects THI for immunologic effects, referred to as the "Combined PCB THI".

6.4.4.1.2 Total PCBs

The analytical results of all detected PCBs were summed and referred to as total PCBs. These are PCB 1248, PCB 1254, and PCB 1260 in the Production Area; PCB 1248 and PCB 1254 in the Warwick Area; and PCB 1254 in the Waste Water Treatment Area. The resultant data set was treated as if total PCBs were a different chemical. Total PCBs was used in the risk characterization, using the Region I policy assumption that the combination of all PCBs is equal in cancer potency to PCB 1260 (USEPA 1995b). This practice is not consistent with PCBs toxicity data. A large toxicity database exists for PCB 1254, from which it is concluded that it is not carcinogenic. Also, existing studies suggest that PCB 1248 is not carcinogenic. Since most of the PCBs detected at the Site are PCB 1254 and PCB 1248, to assume that these mixtures are carcinogens with the same cancer potency as PCB 1260 grossly overestimates potential cancer risks.

6.4.4.2 Risk Characterization Results

6.4.4.2.1 Production Area

Production Area noncancer and cancer effects risk characterization results are summarized in Tables 6-8, 6-9, and 6-10 for the workers and the canoeist.

6.4.4.2.1.1 On-Site Worker

The THI for the Production Area on-site worker is estimated as 0.9 (Table 6-8). This value assumes that the noncancer effects of all COPCs are additive. The Combined PCBs THI for the Production Area on-site worker is estimated as 0.4. These values do not exceed the target value of 1. Thus, adverse noncancer health effects associated with Site soils are unlikely to occur in the Production Area. Regarding potential cancer risks, the total ILCR is estimated as 1×10^{-4}, with Total PCBs accounting for virtually 100% of the estimated potential total ILCR. This is within the RCRA protective risk range of 10^{-6} to 10^{-4}.

Overall, both cancer and noncancer potential human health risk estimates are below their respective "action" criteria for the on-site worker in the Production Area. Of the three PCBs detected on Site, toxicological data indicate that PCB 1248 and PCB 1254 are noncarcinogenic; only PCB 1260 appears to be carcinogenic in animals (see Section 6.4.4.1.2). Region I policy is

to assume that all PCBs have a cancer potency equal to that of PCB 1260. PCB 1260 comprises only about 4% of the total PCBs detected in the Production Area. If PCB 1248 and PCB 1254 are considered as noncarcinogens, the estimated potential ILCR for the on-site worker is 6×10^{-6}. This value is also within the RCRA protective cancer risk range.

6.4.4.2.1.2 General Worker

The THI for the general worker is estimated as 0.8 (Table 6-9). This value assumes that the noncancer effects of all COPCs are additive. The Combined PCBs THI for this worker is estimated as 0.3. These values do not exceed the target value of 1, indicating that adverse, noncancer health effects associated with the Production Area soils are unlikely to occur. Regarding potential cancer risks, the total ILCR is estimated as 1×10^{-4}, with total PCBs accounting for virtually 100% of this value. This value is within the RCRA protective risk range of 10^{-6} to 10^{-4}.

Overall, both cancer and noncancer potential human health risk estimates are below their respective "action" criteria for the general worker in the Production Area. Of the three PCBs detected in the Production Area, PCB 1260 is the only one that toxicological data indicate is carcinogenic (see Section 6.4.4.1.2). Region I policy is to assume that all PCBs have a cancer potency equal to that of PCB 1260. PCB 1260 comprises about 4% of the PCBs detected in the Production Area. If PCB 1248 and PCB 1254 are regarded as noncarcinogenic, the estimated potential ILCR for the nonspecific commercial/industrial worker in the Production Area is 5×10^{-6}. This value is also within the RCRA protective risk range.

6.4.4.2.1.3 Canoeist

The THI for the Production Area canoeist is estimated as 0.0006, with over 85 percent of this value being associated with 2-nitroaniline (Table 6-10). This value is far less than the target THI criterion of 1. Thus, noncancer human health effects associated with potential impact of COPCs in the Production Area to the Pawtuxet River are effectively nonexistent.

The ILCR is estimated as 2×10^{-7}. This value is less than the RCRA protective risk range of 10^{-6} to 10^{-4}. Moreover, the ILCR value in the canoeist scenario for total PCBs was calculated by summing the individual contributions of the three PCBs and, then, assuming that PCB 1248 and PCB 1254 are carcinogenic and have the same cancer potency as PCB 1260. Toxicological evidence indicates that neither PCB 1248 nor PCB 1254 are carcinogenic. If these two

compounds are excluded from the evaluation of carcinogens, the estimated ILCR is much lower (7×10^{-10}).

6.4.4.2.2 Waste Water Treatment Area

Waste Water Treatment Area noncancer and cancer effects risk characterization results are summarized in Tables 6-11 and 6-12 for the on-site resident and canoeist, respectively.

6.4.4.2.2.1 On-Site Resident

The THI for the hypothetical future on-site resident is estimated as 0.4. This value is less than the target THI criterion of 1. Thus, noncancer health effects associated with Site media are unlikely to occur. Regarding potential cancer risks, the total ILCR is estimated as 3×10^{-5}. This value is within the RCRA protective cancer risk range of 10^{-6} to 10^{-4}. Overall, both cancer and noncancer potential human health risk estimates are below their respective "action" criteria for the Waste Water Treatment Area on-site resident.

6.4.4.2.2.2 Canoeist

The THI for the Waste Water Treatment Area canoeist is estimated as 0.000005. This value is far less than the target THI criterion of 1. Thus, noncancer human health effects associated with potential impact of COPCs in the Production Area on the Pawtuxet River are effectively nonexistent.

The ILCR is estimated as 9×10^{-10}. This value is far less than the RCRA protective risk range of 10^{-6} to 10^{-4}. Moreover, the ILCR value in the canoeist scenario for total PCBs was calculated by assuming that PCB 1254 is carcinogenic and has the same cancer potency as PCB 1260. Toxicological evidence indicates that PCB 1254 is not carcinogenic.

6.4.4.2.3 Warwick Area

The Warwick Area noncancer and cancer effects risk characterization results are summarized in Tables 6-13 and 6-14 for the on-site resident and the canoeist, respectively.

6.4.4.2.3.1 On-Site Resident

The THI for the hypothetical Warwick Area on-site resident is estimated as 2 (Table 6-13). This is derived by combining the HI values of all COPCs, and assuming that these effects are additive. This value exceeds the target THI criterion value of 1. Exceeding the criterion value, as mentioned in Section 6.4.3.2, does not necessarily indicate a potential human health risk.

The HHEM recommends that when a THI exceeds 1, the next step in the risk assessment process is to evaluate the potential human health effects by separate target organs. The COPCs contributing to 95% of the noncancer risk are PCB 1254 (HI=0.91), PCB 1248 (HI=0.66), and 2-Nitronaniline (HI=0.63). The target organ is different for each of these compounds, thus the HIs should not be regarded as additive.

PCB 1254 was found to have the highest HI. Its critical effects include a decreased antibody response. Even though toxicity studies report the critical effect of PCB 1248 to be developmental effects, PCB 1248 has also been observed to have immune system effects at higher doses (see Section 6.4.3.2.1). A provisional RfD of 1×10^{-3} mg/kg-day was derived for the immunological effects of PCB 1248, approximately 1/12 that used for the developmental effects of PCB 1248. An HI of approximately 0.06 is estimated for the developmental effects of PCB 1248 by multiplying the HI for the developmental effects of PCB 1248 (0.66) by 1/12. The immunological effect of PCB 1248 can conservatively be considered additive with the critical effect of PCB 1254. This results in a target organ-specific THI of 1 for immunological effects, which equals the target criterion. None of the other target organ-specific THI values would be greater than that for developmental effects (0.7) as none of the other COPCs are additive with either the effects of PCB 1248 or 2-nitroaniline. This indicates that adverse noncancer health effects are unlikely to occur in the Warwick Area.

The total ILCR for the hypothetical future on-site resident is estimated as 3×10^{-4}. This value is the RCRA protective risk range of 10^{-6} to 10^{-4}. However, the USEPA Region I policy is to assume that all PCBs have a cancer potency equal to that of PCB 1260 (USEPA, 1995b). Total PCBs for the Warwick Area is comprised of only PCB 1248 and PCB 1254. As stated in Section 6.4.4.1.2, this practice is not consistent with PCB toxicity data. This is particularly true for the Warwick Area where no PCB 1260 was detected. To assume that these compounds have the same cancer potency as PCB 1260, when they are regarded as noncarcinogenic, greatly exaggerates potential cancer risks. The ILCR of the COPCs excluding total PCBs is 6×10^{-5}. This value also is within the RCRA protective risk range (10^{-6} to 10^{-4}).

Overall, the noncancer health effects have HI values less than the target criterion of 1, a THI of 2, and a maximum target organ-specific THI of 1. These values indicate that the action criterion for the hypothetical on-site resident is met. If noncarcinogenic PCB 1248 and PCB 1254 are assumed to have the same cancer potency as PCB 1260, the result is an ILCR of 3×10^{-4}.

However, if only carcinogens (Classes A, B, and C) are considered, the resultant ILCR is 6×10^{-5}. This value meets the "action" criteria for the hypothetical on-site resident.

6.4.4.2.3.2 Canoeist

The THI for the Warwick Area canoeist is estimated as 0.002, with over 95 percent of this score being associated with 2-nitroaniline. This value is far less than the target THI criterion of 1. Thus, noncancer human health effects associated with potential impact of COPCs in the Warwick to the Pawtuxet River are effectively nonexistent.

The ILCR is estimated as 2×10^{-8}. This value is far less than the RCRA protective risk range of 10^{-6} to 10^{-4}. Approximately 60 percent of the ILCR is associated with beryllium, a naturally occurring component of soils, and about 35 percent of this ILCR is associated with total PCBs. Further, the ILCR value in the canoeist scenario for total PCBs was calculated by summing the individual contributions of PCB 1248 and PCB 1254 and, then, assuming that these two compounds are carcinogenic and have the same cancer potency as PCB 1260. Toxicological evidence indicates that neither PCB 1248 nor PCB 1254 are carcinogenic. If the two PCB compounds are excluded from the evaluation of carcinogens, the estimated ILCR is even lower (1×10^{-8}).

6.4.4.2.4 Background

Cancer and noncancer risks of chemicals determined to present at site-specific background concentrations were also characterized for the Production Area worker scenarios and the Waste Water Treatment and Warwick Area on-site resident scenarios. The major "risk drivers" with respect to background compounds were previously determined for each Site area (see Section 6.3.2). The results of the background risk characterization are presented for the three site areas in the following subsections and are summarized in Tables 6-15 through 6-17.

6.4.4.2.4.1 Production Area

Cancer and noncancer risk results for the site-specific, on-site worker scenario and the nonspecific commercial/industrial on-site worker are summarized in Table 6-15. The background THI for the site-specific, on-site worker scenario was estimated as 0.009. Approximately 87% of this value is associated with arsenic. The background ILCR was estimated as 4×10^{-6}, with about 47% of this associated with benzo(a)pyrene and 34% with arsenic.

The background THI for the nonspecific, commercial/industrial worker was estimated as 0.004, with approximately 83% of this value being associated with arsenic. The background ILCR under this scenario was estimated as 3×10^{-6}, with benzo(a)pyrene accounting for approximately 58% of this value.

6.4.4.2.4.2 Waste Water Treatment Area

The background THI for the Waste Water Treatment Area on-site resident was estimated as 0.09, with about 52% of this value associated with arsenic and 34% with manganese. The background ILCR for under this scenario was estimated as 1×10^{-4}, with dibenz(a,h)anthracene, beryllium, and benzo(a)pyrene contributing approximately 41%, 28%, and 21% of this value, respectively.

6.4.4.2.4.3 Warwick Area

The background THI for the Warwick Area on-site resident was estimated as 0.1, with arsenic accounting for about 58% of this value. The background ILCR was under this scenario was estimated as 4×10^{-5}, benzo(a)pyrene contributing about 64% of this value and arsenic the remaining 36%.

6.4.5 Uncertainties

One of the primary objectives of the PHERE is to characterize and quantify potential risks. The very nature of risk, being comprised of probability statements, connotes that uncertainty is involved. The fact that potential risks in the PHERE are called "potential" accentuates the associated uncertainty because the risks evaluated do not exist at this time. In addition, there are uncertainties associated with the COPC selection process, future land-use scenarios, transport models, exposure input values, toxicity values, and the risk characterization process.

6.4.5.1 COPC Selection Process

The COPCs were selected using a screening process described by the USEPA in the HHEM. While the method is useful for screening, it is based on oral toxicity values and does not address chemical-specific differences to such variables as environmental contaminant transport, dermal absorption rates, and toxicities via exposure routes other than ingestion.

6.4.5.2 Future Land-Use Scenarios

Future land use for the Production Area will be a City of Cranston parking lot and storage facility for road salt, sand, and snow removal equipment. It will include covering the Production Area with approximately one foot of soil and six inches of asphalt. This is based on the plans of the

City of Cranston (1995) and Ciba. This future land use has a high level of certainty. However, because the assumption was made in calculating potential risks that the Production Area would not be paved or in any way covered, the PHERE greatly exaggerates exposure to contaminated soil, and thus, greatly overestimates potential cancer and noncancer risks. A soil and asphalt cover will dramatically reduce soil-to-air and soil-to-groundwater transport, and worker ingestion and dermal contact with the impacted soil will essentially be eliminated. Because of the planned use of the site already described, the general worker is regarded as an unlikely receptor.

Unrestricted residential land use is conservatively assumed for the Waste Water Treatment and Warwick Areas because there is uncertainty as to the future use. Although residential land use is not necessarily the most relevant for these Site areas, it represents a "worst-case" future land use scenario. Future industrial or commercial use of these areas is regarded as very plausible. The assumption of residential land use overestimates the exposure associated with an industrial or commercial land-use scenario.

6.4.5.3 Transport Models

Soil-to-air and soil/groundwater-to-Pawtuxet River transport models were used in the PHRA to estimate COPC exposure point concentrations of air and river water, respectively, attributable to each of the three Site areas (Appendices 6-D and 6-E).

The soil-to-air models were intentionally selected and used in a manner that would tend to overestimate potential exposures of people. For example, a simple event of neutral stability, a mean annual wind speed, and a constant worst-case wind direction were assumed conditions. Also, it was assumed that the soil surface contained no hardened crust. These are unrealistic assumptions which, together, exaggerate wind dispersion of soils and result in overestimates of exposure.

Similarly, the soil/groundwater-to-Pawtuxet River water model is based on conservative assumptions. As an example of a conservative assumption, the model accepts either the greater of the measured groundwater concentrations, or the groundwater concentration based on measured soil concentrations, to estimate Pawtuxet River concentrations. This results in calculating risks on the river water concentrations of several compounds that have not been detected in any groundwater samples collected on-site.

If the Production Area were to be covered with soil and paved, as is planned, exposure to the on-

site worker would be dramatically reduced. The ingestion and dermal absorption pathways would be effectively eliminated, as would airborne particulate emissions. Only the inhalation of volatile emissions would remain as an exposure pathway, and it is estimated that the soil/asphalt cap would reduce volatile emissions by more than 95 percent. This would result in an ILCR and HI to the on-site Production Area worker of approximately 7×10^{-10} and 0.0001, respectively. Likewise, it is expected that the asphalt/soil cap will greatly reduce infiltration of rainwater through the zone of impacted soil. This would decrease mobility of the COPCs in the soil, and prevent leaching to groundwater. As a result, the concentration of COPCs in the Pawtuxet River would be reduced; consequently, exposure to the hypothetical canoeist would also be reduced.

6.4.5.4 Exposure Assumption Values

Exposure assumption values used in the exposure assessment are generally regarded as overestimates of the "true" values. The HHEM advocates a "reasonable maximum exposure" (RME) approach to exposure assessment. The RME does not assume "worst-case" values for each exposure assumption value. However, the RME values recommended by the HHEM, such as contact rates, exposure frequencies, and exposure duration, are decidedly conservative (e.g., 95th percentile UCLs of possible values). The PHRA basically followed the HHEM approach, using assumption values that were reviewed by Region I in the May, 1994, meeting and discussed during subsequent meetings and teleconferences. A few are somewhat less conservative than the default RME values which appear in the HHEM. Although there is uncertainty associated with every selected value, a few of these exposure variables are highlighted in the following paragraphs.

Maximum detected and 95th percentile UCL of the mean concentrations were used as the chemical concentration values. These are overestimates of average values. It is noted that concentrations that were qualified as estimated values during data validation ("J values") were also used in the PHRA to derive the concentration values; nondetected values were assumed to be one-half the sample quantitation limits (SQL). These practices are consistent with the HHEM. The use of "J values" may result in either an overestimate or underestimate of actual average concentrations. Because many of the "J values" are less than one-half their respective SQLs, the assumption that a concentration equal to one-half the SQL is present, tends to overestimate actual average concentrations.

The soil ingestion rates (IR_s) are considered overestimates of actual values. The IR_s used in the exposure assessment for the on-site resident scenario is 200 mg/day for young children and 100

mg/day for older children and adults, as suggested in the HHEM. An ingestion rate of 50 mg/(work)day of soil was assumed for the on-site worker scenario. However, in studies by Calabrese, et al., (1989) using 64 subjects, the median of the range for daily soil ingestion by young children (ages 1 through 4 years old) was found to be 9 to 40 mg per day, depending on the tracer element used for the study. Work cited in the Exposure Factors Handbook (USEPA, 1990a) suggests that individuals 5 years of age and older ingest on average approximately 10 mg of soil per day. Soil ingestion for the resident scenario and contact were assumed to be proportionate to the amount of time spent at the Site. This is an overestimate, especially for adults, since one of the primary sources of ingested soil is associated with food. In addition, if the Production Area were to be paved, the soil ingestion value would be virtually eliminated, making the ingestion of Production Area surface soil an incomplete exposure pathway.

The exposure duration used for the on-site resident scenario and on-site worker are 30 and 25 years, respectively. Few individuals work at the same location with the same job for as long as 25 years. The on-site resident scenario exposure duration is far greater than the median duration time of 9 years that an individual typically lives at a residence as referenced in the HHEM. These conservative exposure values, when combined, may overestimate the potential risk by two orders of magnitude over more realistic exposure assumptions, depending on the exposure scenario and the exposure values selected. This does not include the overestimations of toxicity discussed in Section 6.4.5.5.

6.4.5.5 Toxicity Assessment

Uncertainties pertaining to the toxicity assessment are discussed in Section 6.4.3. These include uncertainties regarding development of the health effects criteria values, the classification of carcinogenicity, the extrapolation of exposure route-specific toxicity values to other routes of exposure, and the extrapolation of toxic effects observed in animal studies to potential adverse health effects in people. A summary of these uncertainties is provided in the following paragraphs.

The development of health effects criteria for noncancer health effects involves professional judgement. Depending on the nature of the toxicity studies, a safety factor of up to nearly four orders of magnitude may be built into the RfD or RfC value.

The USEPA weight-of-evidence classification system for carcinogens is used to examine and classify chemical agents with respect to their human carcinogenic potential. Most compounds

that the USEPA classifies as carcinogens, including the COPCs examined in the PHRA, are B2 carcinogens. The carcinogenicity of these chemicals is based on animal data. There is uncertainty as to the nature of the carcinogenic response in humans, if any. Also, the mathematical models used to extrapolate from relatively high-dose rodent studies to relatively low-dose human exposures are the subject of much controversy. The approach taken by USEPA of almost exclusively using the linearized multistage model, combined with other assumptions, tends to overestimate potential ILCR. The USEPA is currently revising its carcinogen policies. The revised policies may be enacted during 1995 or 1996. These could potentially impact the PHERE.

When a noncancer or cancer health effects criterion was not available for a given route of exposure, the criterion from another route of exposure was adopted for use. This practice adds uncertainty and may either overestimate or underestimate toxicity.

A provisional RfD was derived for PCB 1248 because the USEPA has not established an RfD. An uncertainty factor of 10 was used in the estimation of a NOAEL for a LOAEL observed in a study on rhesus monkeys. An uncertainty factor of 3 may be justifiable, except the investigators of this critical study omitted key information about reproductive performance in the control group and their rhesus colony in general (see Section 6.4.3.2.1).

Total PCBs was used in the risk characterization, using the Region I policy assumption that the combination of all PCBs is equal in cancer potency to PCB 1260 (USEPA 1995b). This practice is not consistent with PCBs toxicity data. A large toxicity database exists for PCB 1254, from which it is concluded that it is not carcinogenic. Also, existing studies suggest that PCB 1248 is not carcinogenic. Since most of the PCBs at the Site are PCB 1254 and PCB 1248, to assume that these mixtures are carcinogens with the same cancer potency as PCB 1260 grossly overestimates potential cancer risks.

6.4.5.6 Risk Characterization

Uncertainty inherent to the risk characterization process involves the additivity assumption of adverse health effects associated with different chemicals. Chemicals in combination may act additively, antagonistically, synergistically, or not influence each other at all. Antagonistic relationships result in health effects that are less than those predicted by a chemical given alone; synergistic relationships result in health effects that exceed the results predicted by a chemical given alone, and additivity is the predicted sum of responses for chemicals with similar effects.

Therefore, the assumption of additivity used in the PHRA may either overestimate or underestimate human health risks.

The conservativeness of health effects criteria are discussed in Section 6.4.4.1. This conservativeness is compounded in the risk characterization process where multiple conservative values are combined together. This tends to exaggerate potential risks. Also, as discussed in Section 6.4.5.5, the Region I assumption that total PCBs are carcinogenic and have the same cancer potency as PCB 1260 grossly overestimates potential cancer risks.

6.5 Terrestrial Ecological Risk Assessment

This baseline ecological risk assessment utilizes the procedures found in the *Framework for Ecological Risk Assessment* (USEPA, 1992b) and *Ecological Assessment of Superfund sites: An Overview* (USEPA 1991d). Detailed descriptions of the Site and Site history are found in Section 1.0 of the Resource Conservation and Recovery Act (RCRA) Facility Investigation (RFI). The Site is divided into three areas: Production, Waste Water Treatment, and Warwick (Figure 6-7). Analysis is presented for each of these areas.

6.5.1 Objectives and Scope

The main objective of this baseline ecological risk assessment is to evaluate the potential risks posed to ecological receptors by COPCs contained in surface soils (0-2 feet) at the Production, Waste Water Treatment, and Warwick Areas and the small groundwater seep in the Waste Water Treatment Area of the Site. Specific objectives are to:

- review ecological data,
- summarize the data into a description of ecological conditions at the Site,
- review data on the chemical contamination of the surface soils and select COPCs based on physiochemical and ecologically relevant criteria,
- develop a conceptual model to identify reasonable exposure pathways and potential ecological receptors, and
- make an assessment of the potential for COPCs to induce adverse ecological effects.

6.5.2 Environmental Setting

A complete site description is found in Sections 1, 2, and 3 of the RFI. The description in this report is focused on the ecological risk assessment.

6.5.2.1 Physiography

The total area of the Site is approximately 25 acres: 8 acres north of the Pawtuxet River in Cranston and 17 acres south of the river in Warwick (Ciba, 1990). The Site is bordered to the north and south by residential areas, to the east by commercial areas, and to the west by a mixed industrial area also owned by Ciba.

The topography of the Site is relatively flat with gentle slopes toward the Pawtuxet River with elevations ranging from about 10 to 25 feet above mean sea level. The Pawtuxet River, a major waterway, flows from west to east through the Site and is about 80 feet wide as it passes the Site.

The entire Warwick Area and about half of the Waste Water Treatment Area are within the 100-year flood plain with about 10 percent of the Production Area along the river being within the 100-year flood plain (FEMA, 1982, 1984).

Two small wetlands and the Pawtuxet River riparian zone are on-site. The Pawtuxet River is designated by the U. S. Fish and Wildlife Service National Wetland Inventory system as R2OW, indicating a free flowing river (open water, lower perennial riverine system). Natural groundwater discharge in the Waste Water Treatment Area appears responsible for a seep wetland (seep) of about 0.25 acre. In the Warwick Area there is a small depressional area of about 0.05 acre located in the northwest corner near the river (Gautreau and Hastings, 1995).

6.5.2.2 Ecological Features

Terrestrial habitats within the Site include upland and riparian wooded and open areas. The upland open areas support plants typical of open fields, roadsides, and areas including both native and exotic plants such as goldenrod, thistle, Queen Anne's lace, milkweed, common mullein, plantain, tickseed, and evening primrose. The edges of wooded upland areas support plants such as staghorn sumac, the exotic Tree-of-Heaven, and multiflora rose which is also found in the wooded areas. Shrubs found in upland wooded areas noted in the Waste Water Treatment Area include lowbush blueberry and rosebay. English ivy and poison ivy serve as both ground plants or cover and vines. Trees include black oak, which prefers drier sites, and black cherry, northern red oak, white oak, Norway maple (introduced), and three species of pine-Austrian (introduced), shortleaf, and white pine-which can tolerate more moisture variations. Trees found in the upland areas but more typical of riparian areas include cottonwood and gray birch with the shrub northern bayberry and herb Japanese knotweed. Table 6-H-1 (Appendix 6-H) provides a complete listing of terrestrial plants and animals noted during the March, 1992 field survey.

The herbaceous layer found in the riparian/wetland areas of the Site include sensitive fern, reed canary grass, soft rush, tussock sedge, cattail, and greenbrier. The riparian shrub/small tree layer includes pepperbush, pussy willow, highbush blueberry, red-osier dogwood, and speckled alder. Riparian area trees include black willow, bear oak, northern red oak, swamp white oak, white oak, black gum, gray birch, mockernut hickory, box elder, red maple, silver maple, American elm, sassafras, and eastern hemlock.

Birds found on site include species which prefer open areas and forest edges such as killdeer,

mourning dove, domestic pigeon, European starling, mockingbird, house sparrow, and American robin. Woodland and edge area birds include Coopers' hawk, red-tailed hawk, great horned owl, blue jay, common crow, black-capped chickadee, cardinal, dark-eyed junco, and song sparrow. Birds found on site preferring a riparian habitat include great blue heron, Canada goose, black duck, American widgeon, mallard, wood duck, hooded merganser, red-winged blackbird, and belted kingfisher.

Mammals on site utilizing both upland and riparian wooded and open areas include the eastern cottontail rabbit, eastern gray squirrel, muskrat, and raccoon with the latter two especially utilizing the riparian area.

6.5.2.3 Protected Species and Habitats

The Ciba Site, located in an industrial park within the urbanized areas of the cities of Cranston and Warwick, has little natural area and is unlikely to provide suitable habitat for endangered plant or animal species. There are no federally designated critical habitats in Rhode Island (Reithel, 1994).

6.5.3 Problem Formulation

This section provides a description of potential ecological problems at each study site and defines objectives for the ecological assessment based on site information. Site history, chemical analyses for soils, potential migration and exposure pathways, potential ecological receptors (habitats and species), and ecologically appropriate assessment and measurement endpoints are discussed in this section.

6.5.3.1 Production Area

6.5.3.1.1 Site Description

This is a narrow, north-south trending rectangular area which parallels Mill Street on the west and broadens where it borders the Pawtuxet River to the southeast (Figure 6-7). Its total areal extent is approximately 2.8 acres (ac), all of which is cleared, open ground. This area fronts the Pawtuxet River for approximately 114 meters (m) (370 feet); however, no riparian vegetation remains along the shoreline. Pedestrian and combined railroad/vehicle bridges cross the river to connect this area with the Warwick Area (Figure 6-7).

6.5.3.1.2 Stressor Selection

Chemical contaminants are the primary stressors evaluated in this report. Chemicals of potential concern (COPCs) are chemicals that were detected in surface soils and which have the potential to adversely impact natural populations or ecosystems.

Analytical results from surface soil samples [0 to 2 feet (0 to 0.75 meters) depth] are used to determine concentrations of chemicals to be used in the risk assessment. Initial screening for identification of analytes as COPCs follows the path shown in Figure 6-8 and is based on the following criteria (USEPA, 1989b):

Blank Contamination: As part of the data validation process, a chemical was not considered further if the maximum sample concentration did not exceed 10 times the highest blank for all common laboratory contaminants (acetone, 2-butanone, methylene chloride, toluene, and phthalates) or five times the highest blank for other chemicals.

Frequency of Detection: Chemicals that are infrequently detected may be artifacts in the data due to sampling, analytical, or other problems. Chemicals were eliminated if they were detected in 5 percent or less of the samples.

Essential Nutrients: Calcium, iron, magnesium, manganese, potassium, sodium, and zinc are considered essential macronutrients and are generally toxic only at very high concentrations. These chemicals, except for zinc, were eliminated as COPCs, since they were not present at excessively high concentrations.

Water Chemistry: Indications of general water chemistry conditions (sulfates, carbonate, bicarbonate, chloride, and total dissolved solids) are also generally toxic only at very high levels. These chemicals were also eliminated as COPCs, since they were not present at excessively high concentrations.

Background: A *t*-test with separate variances was performed for polyaromatic hydrocarbons (a product of fossil fuel-burning) and metals found in background samples and initially selected according to human health risk criteria (See Section 6.3). If the site concentrations were not significantly higher ($\leq .05$) than background concentrations, they were eliminated as a COPC. If site concentrations were significantly higher than background concentrations, or no statistical tests were performed, they were considered a

COPC. If COPCs, that were not initially subjected to a *t*-test, resulted in a risk to animals on the Site, they were subjected to a *t*-test and included or eliminated as a COPCs according to the above criteria.

Regulatory Limits: Limits or regulatory criteria for surface soils for ecological risk assessment have not been promulgated at the state and/or federal level. Therefore, none are available for use.

Persistence: Persistence of chemicals was estimated from their degradation half-life ($t_{0.5}$) in soil and their estimated ability to bioaccumulate in terrestrial food webs. An analyte was identified as a COPC if its octanol-water partition coefficient [an estimator of bioaccumulation potential (Garten and Trabalka, 1983)] was >3.5 (Lyman, et al., 1982), or its half-life in soil was >14 days (336 hours).

Mobility: Mobility of chemicals is a function of water solubility and the soil sorption constant (K_{oc}). Highly water soluble compounds tend to leach from wastes or contaminated soils and are generally mobile in both groundwater or surface water. Soil sorption indicates the tendency for a constituent to be adsorbed to soil particles. Chemicals with $\log(K_{oc})$values <2 are weakly sorbed (and thus more mobile); $\log(K_{oc})$ values between 2 and 4 indicate moderate sorption; $\log(K_{oc})$ values >4 indicate strong adsorption to soils. An analyte was classified as potentially mobile and as a COPC if it exhibited a solubility >1 mg/L or a.$\log(K_{oc})$ < 3 (BEIA, 1989).

Analytes selected as COPCs are shown in Table 6-18. Additional detail is found in Tables 6-H-2 and 6-H-3.

6.5.3.1.3 Exposure Pathway Identification

For exposures to occur, complete exposure pathways must exist, which require:

- a source and mechanism for COPC release,
- a transport medium,
- a point of environmental contact, and
- an exposure route at the exposure point (USEPA, 1989c).

If any of these four components is absent, a pathway is generally considered incomplete. However, the transport medium may be missing and the pathway may still be complete if the

contact point is directly at the contaminant release point. Specific potential exposure pathways include:

Dermal Contact (soil): Significant exposure via dermal contact would be limited to organic contaminants which are lipophilic and can cross epidermal barriers. Fur-bearing mammals and birds are less susceptible to exposure via dermal contact with soils because their fur or feathers prevents much of their skin surface from coming into full contact with soil. Calculations showed risk to deer mice and raccoons from dermal contact to be two to three orders of magnitude lower than risk from soil ingestion or ingestion of contaminated plants and animals. Young mammals or birds, which are normally born hairless or featherless, would be much more susceptible to transdermal exposure. Quantification of dermal exposure was therefore performed assuming young (newborn) rather than adult animals. Newborn shrews were used as representative newborn animals, since shrews are representative burrowing animals and have a large skin surface to body weight ratio. Calculations were performed to determine doses to newborn shrews from dermal contact (Table 6-H-4). These calculations were done using equations developed for dermal exposure (Hope, 1995) and all resulted in potential risks that were several orders of magnitude lower than risks from soil ingestion and from eating contaminated plants and animals. Thus, dermal contact was not considered to be a significant contributor to risk.

Root Contact (soil): Contaminants that can partition into soil interstitial waters may be taken-up via roots in contact with soil and translocated into edible foliage or reproductive structures (seeds). Terrestrial plants were assumed to be exposed primarily through contact with contaminated soils rather than with contaminated surface water.

Consumption (soil): Incidental soil ingestion may inadvertently occur while grooming, burrowing or consuming plants, insects or burrowing invertebrates resident in soil. Some organisms, such as deer, deliberately ingest soil as a source of minerals.

Inhalation (vapors): Inhalation of organic vapors would be limited to those contaminants with relatively high vapor pressures (Henry's Law constant > 10^{-4}). Animals that have the greatest potential for exposure would be those in burrows, such as shrews. Quantification of exposure due to inhalation was performed for compounds with inhalation toxicity information available and with the greatest concentrations (Table 6-H-

5). These calculations were done using equations developed for inhalation exposure for burrowing animals (Hope, 1995) and all resulted in potential risks that were several orders of magnitude lower than risks from soil ingestion and from eating contaminated plants and animals. Thus, inhalation was not considered to be a significant contributor to risk, and this pathway is not considered.

Food Web Interactions: Indirect exposure pathways involve contaminants that biomagnify within the food chain. Contaminants bound to soil or sediment are assumed to be bioavailable only after they partition into the water phase. Water-borne contaminants may bioaccumulate into plant tissues in contact with soil, sediment, or surface water or into terrestrial or aquatic species ingesting soil, sediment, or surface water. As these plants and/or animals are consumed, contaminants may be passed up the food chain to impact organisms within higher trophic levels.

Exposure pathways that were determined to be relevant include root contact for plants, consumption of soil, and biomagnification within the food chain.

6.5.3.1.4 Ecological Receptor Identification

In March, 1992, a terrestrial/riparian reconnaissance survey was conducted at the Site (IT, 1992). The terrestrial survey identified twenty-eight species of upland plants and twenty-six species of riparian/wetland plants at and near the Site. Twenty-six species of birds were identified as well. These included the great blue heron (*Ardea herodias*), mallard duck (*Anas platyrhynchos*), and red-tailed hawk (*Buteo jamaicensis*). Five mammal species were identified, including the Eastern gray squirrel (*Sciurus carolinensis*) and the raccoon (*Procyon lotor*). A terrestrial wildlife and plant species inventory is provided in Table 6-H-1. Three representative species were selected for evaluations:

- a small mammal (deer mouse, *Peromyscus* sp.), not found in survey, but probably present,
- a large terrestrial omnivore and protected species (red-tailed hawk), and
- a large terrestrial omnivore (raccoon).

The Production Area is a former industrial operations center, surrounded on all sides by barren ground, roads, industrial buildings, and residential areas. Its historic use as an industrial site and parking lot, highly compacted soils, sparse cover, various man-made structures, and chain-link fences has eliminated habitats and natural communities that might attract or shelter a variety of

species. This flat site harbors many exotic weed and grass species typical of disturbed habitats and is largely devoid of native vegetation. Potential receptors in the cleared and paved portions of the Production Area include exotic plant species, small mammals (rats, mice, voles, etc.), and terrestrial invertebrates (insects, earthworms) that can tolerate disturbed, human-dominated habitats. Other potential receptors include a few higher trophic level species (such as the red-tailed hawk or raccoon) for which these species are prey. Therefore, representative species for this Area are the deer mouse, raccoon, and red-tailed hawk.

6.5.3.1.5 Potential Adverse Ecological Effects

For adverse ecological effects to be possible, a study site must:

- contain COPCs in abiotic media at detectable and biologically significant concentrations,
- provide exposure pathways linking contaminants to receptors, and
- have ecological receptors that either utilize the site, are present nearby, or are in range of COPCs migrating from the site.

If these three fundamental conditions cannot be met, the probability of adverse effects due to site-related contaminants is minimal.

It has been shown that the Production Area contains COPCs in surface soils and provides exposure pathways potentially linking these COPCs to both on-site and off-site ecological receptors. Onsite bioaccumulation may be providing an exposure pathway to off-site receptors. Figure 6-9 illustrates a simplified food web pathway.

6.5.3.1.6 Endpoints

The potential for adverse effects was addressed in this assessment through comparison of observed exposure point concentrations to the No Observed Adverse Effects Level (NOAEL) for terrestrial animals (see Section 6.5.5.1). A NOAEL is the dose or concentration at or below which no adverse effects have been observed in exposed animals and is one to which a population of organisms may be exposed with no expected adverse impacts on any individuals. A NOAEL is an acceptable level for this assessment.

6.5.3.2 Waste Water Treatment Area

6.5.3.2.1 Site Description

This is a roughly square area which borders the Pawtuxet River to the south and is directly across

the river from the eastern end of the Warwick Area. It has no direct connection to either the Production or Warwick Areas. Its total areal extent is approximately 5.18 ac, of which 0.79 ac is a woodland, 0.54 ac is grassland, 0.25 ac is a seep area, 0.10 ac is paved, and the remainder 3.54 ac is cleared, open ground. Land use as a percentage of total area is woodland, 15.3%; pavement, 1.2%; grassland, 10.4%; seep area, 4.8%; and cleared ground, 68.3%. Much of the cleared land has experienced revegetation by a variety of grass and shrub species; several large trees remain in cleared areas along the river shoreline. The Pawtuxet River fronts the Area for approximately 179 m (590 feet).

6.5.3.2.2 Stressor Selection

COPCs for the Waste Water Treatment Area surface soils, identified using the process described in Section 6.5.5.3.1, are shown in Tables 6-18, 6-H-6 and 6-H-7. COPCs for the seep sediments and surface water are shown in Tables 6-19, 6-H-8, 6-H-9, and 6-H-10.

6.5.3.2.3 Exposure Pathway Identification

Primary and secondary exposure pathways for the Warwick Area are similar to those identified in Section 6.5.3.1.3. However, the seep area also provides a semi-terrestrial exposure pathway that is possible.

6.5.3.2.4 Ecological Receptor Identification

Although the Waste Water Treatment Area is a former industrial operations center, all significant structures have been removed and the Area now encompasses several habitats and natural communities that might attract or shelter a variety of terrestrial species. The woodland edge habitat along the northern boundary may be capable of providing shelter to a number of species; potential receptors in this wooded habitat include terrestrial invertebrates, birds, squirrels, and raccoons. Potential receptors in the grassland and revegetated (cleared) portions of the Area include exotic plant species, small mammals (rats, mice, voles, etc.) and terrestrial invertebrates (insects, earthworms) that can tolerate disturbed, human-dominated habitats. Other potential receptors also include a few higher trophic level species (such as the red-tailed hawk or raccoon) for which these species are prey. In the small seep area (0.25 ac), the amount of standing water appears to vary. As the water recedes, the exposed sediments become part of the terrestrial components. As the standing water increases, those sediments become part of the aquatic pathway. There are no fish in the seep but tadpoles were found. Therefore, the food pathway would be frogs. The great blue heron was chosen as the primary receptor for the seep because it

is at a high tropic level and occurs on-site. The representative species for this Area are the deer mouse, raccoon, red-tailed hawk, and great blue heron.

6.5.3.2.5 Potential Adverse Ecological Effects

It has been shown that the Waste Water Treatment Area contains COPCs in surface soils and in the seep area sediments and water at detectable concentrations and provides exposure pathways linking these COPCs to both on-site and off-site ecological receptors. Onsite bioaccumulation may be providing an exposure pathway to off-site receptors.

6.5.3.2.6 Endpoints

Endpoints for risk assessment at this Area are the same as those described in Section 6.5.3.1.6.

6.5.3.3 Warwick Area

6.5.3.3.1 Site Description

This is an east-west trending roughly rectangular area which parallels Vine Street on the south and borders the Pawtuxet River to the north. The Area protrudes southward across Vine Street for approximately 81m (270 feet). Its total areal extent is approximately 17.17 ac, of which 3.94 ac) is a woodland, 10.17 ac is paved, and the remainder 3.06 ac is cleared, open ground. Land use as a percentage of total area is woodland, 23.0%; pavement, 59.2%, and cleared ground, 17.8%. Much of the cleared land has experienced revegetation by a variety of herbaceous and shrub species; several large trees remain in cleared areas along the river shoreline. The Pawtuxet River fronts the Area for approximately 390 m (1300 feet). Pedestrian and combined railroad/vehicle bridges cross the river to connect this Area with the Production Area.

6.5.3.3.2 Stressor Selection

COPCs for the Warwick Area, identified using the process described in Section 6.5.5.3.1, are shown in Tables 6-18, 6-H-11, and 6-H-12.

6.5.3.3.3 Exposure Pathway Identification

Primary and secondary exposure pathways for the Warwick Area are identical to those identified in Section 6.5.3.1.3. However, this site contains a woodland and supports more animals than the Production Area. Within the woodland is a solid waste management unit (SWMU-5), which contains most of the COPCs found in the Warwick Area. SWMU-5 and an adjacent zinc oxide

soil storage pile (SWMU-6) will be removed by interim remedial actions, thus removing this exposure pathway. However, the risk assessment is performed assuming that the current levels of contamination will continue to exist.

6.5.3.3.4 Ecological Receptor Identification

The Warwick Area is a former administration and engineering operations center, now largely covered by abandoned parking lots, roads, highly disturbed and compacted soils, and various chain-link fences. Over approximately two-thirds of this Area, these historic actions have eliminated habitats and natural communities that might attract or shelter a variety of terrestrial species. It is unlikely that significant numbers of potential receptors would be found in the paved portions due to the lack of habitat. However, natural revegetation in some of the cleared portions has created habitat for some species. The eastern third of the Area is covered with woodland habitat capable of providing shelter to a number of species; potential receptors in this wooded habitat include terrestrial invertebrates, birds, squirrels, and raccoons. Potential receptors in the revegetated (cleared) and paved portions of the Area may include exotic plant species, small mammals (rats, mice, voles, etc.) and terrestrial invertebrates (insects, earthworms) that can tolerate disturbed, human-dominated habitats. Receptors might also include a few higher trophic level species (such as the red-tailed hawk or raccoon) for which these species are prey. Therefore, the representative species are the deer mouse, raccoon, and red-tailed hawk.

6.5.3.3.5 Potential Adverse Ecological Effects

It has been shown that the Warwick Area contains COPCs in surface soils at detectable concentrations and provides exposure pathways linking these COPCs to both on-site and off-site ecological receptors. Onsite bioaccumulation may be providing an exposure pathway to off-site receptors.

6.5.3.3.6 Endpoints

Endpoints for risk assessment at this Area are the same as those described in Section 6.5.3.1.6.

6.5.3.4 Problem Formulation Summary

A summary of problem formulation results for all Areas appears in Table 6.20. Conceptual modeling suggests that COPCs in surface soils in each Area of the Site could be contributing to the potential for adverse effects in ecological receptors utilizing or traversing these Areas. Lower trophic level terrestrial plant and wildlife species are directly exposed to COPCs in surface soils, while higher trophic level receptors (e.g., raccoon, red-tailed hawk, great blue heron) may be

exposed to COPCs bioconcentrated in their prey species (terrestrial invertebrates, small mammals, aquatic vertebrates).

6.5.4 Exposure Characterization

The COPCs identified in Table 6-18 and 6-19 are evaluated as to their potential to affect the site-representative organisms: deer mouse, raccoon, red-tailed hawk, and great blue heron. Potential doses to these organisms are calculated.

6.5.4.1 Production Area

For the purposes of this assessment, tissue residue levels were estimated for terrestrial plants and invertebrates and applied daily doses were estimated for deer mice, red-tailed hawks, and raccoons. Figure 6-9 indicates the feeding relationships for which quantitative exposure estimates were made. Vegetation and insects were assumed to be exposed primarily through root uptake and direct contact with COPCs in surface soils and these COPCs were bioaccumulated when these organisms were eaten by higher trophic-level species. Higher trophic level species in the terrestrial food web (e.g., red-tailed hawk and great blue heron), not necessarily in direct contact with contaminated media, were assumed to be exposed primarily through consumption of contaminated prey. Intake from direct consumption of, or contact with, contaminated surface water, or from inhalation of contaminated dust or vapors, was assumed to be negligible.

Life history parameters required for model calculations are shown in Table 6-21. Species-specific models are described below.

Terrestrial Plants: Toxicant concentrations in reproductive tissues of terrestrial vegetation were calculated using an equilibrium partitioning model (Landrum et al., 1992), as follows:

$$C_{pl} = C_{soil} \times B_r$$

where:

C_{pl} = concentration of toxicant in terrestrial vegetation, mg/kg

C_{soil} = concentration of toxicant in surface soil, mg/kg

B_r = toxicant-specific soil to reproductive vegetation transfer coefficient, unitless (see Table 6-H-13)

Soil to vegetation transfer coefficients for inorganic chemicals (B_r) were obtained from Baes et al. (1984). Organic chemicals were calculated using Travis and Arms (1988):

$$log\ (B_r) = 1.588 - 0.578 \times log\ (K_{ow})$$

where:

K_{ow} = octanol/water partition coefficient for toxicant, unitless (see Table 6-H-13).

Terrestrial Invertebrates: Insect species (terrestrial macroinvertebrates) were assumed to be primarily exposed though direct consumption of terrestrial vegetation; pathways involving surface water consumption, inhalation, or incidental consumption of soil were assumed to be of negligible significance, so that:

$$C_i = \left(\frac{\alpha \times \left(C_{pl} \times F_{pl}\right) \times \frac{R_i}{W_i}}{K_e} \right) \Theta_i \times \Psi_i$$

and

$$R_i = 0.136 \times W_i$$

where:

C_i = terrestrial insect tissue contaminant concentration (absorbed dose), mg/kg

C_{pl} = concentration of toxicant in terrestrial vegetation, mg/kg

F_{pl} = dietary fraction of terrestrial vegetation, unitless

Θ_i = area use factor for terrestrial insects, unitless

Ψ_i = seasonality factor for terrestrial insects, unitless

k_e = toxicant-specific loss rate, day^{-1}

α = species-specific assimilation factor, unitless

R_i = food intake rate, wet diet, kg/d

W_i = body weight of terrestrial insect, kg

The calculation of R_i was performed by assuming that the herbivorous insect ingestion rate is equivalent to approximately 13.6% of insect body weight per day (Crossley and Howden, 1961).

Assimilation efficiency (α) is an estimate of the ratio of the quantity of contaminated material consumed (in food or water) by an organism to the quantity of contaminant actually incorporated into its tissues. Species-specific values for these factors are scarce in the literature. For lipophilic organic COPCs, α was assigned a conservative default values of 0.9 to provide a reasonable maximum estimate of contaminant uptake; this assumption is likely to produce an overestimate of contaminant loading. Inorganic COPCs (those other than organometallic or methylated forms) were assigned assimilation coefficients as follows:

Ba	=	0.10
Be	=	0.001
Cd	=	0.06
CN^-	=	0
Cr^{+3}	=	0.01
Cu	=	0.5
Hg	=	0.15
Ni	=	0.05
Zn	=	0.5

(after Owen, 1989)

The k_e parameter represents the rate at which assimilated COPCs are released from tissues and is included to show recognition of such a process. However, because estimation of this parameter for wildlife species is highly problematic, it was assigned a default value of 1.0 in all calculations and is not shown in the following equations.

When a terrestrial receptor's foraging area exceeds the area of contamination, an area use factor (Θ) is included to account for the effect of receptor mobility on frequency and duration of contact with contaminated media or prey. This factor is defined as the ratio of contaminated area to foraging area for a given indicator species, so that $1 \geq \Theta > 0$ (DeSesso and Price, 1990). An animal whose home/foraging range is very small in comparison to an Area or which is assumed to be a permanent resident of an Area will have an assigned area use factor of 1.0. Because several indicator species are migratory and not continuously present at the Site or are not active throughout the year, an indicator species seasonality factor (Ψ) is defined to account for effects

of receptor seasonality on frequency and duration of contact with contaminated media or prey. This factor is defined as the ratio of the time spent at the Site (in months) to 12, so that $1 \geq \Psi > 0$.

Deer Mouse: Tissue residue concentrations in, and doses received by, deer mice through consumption of contaminated prey items and incidental ingestion of contaminated soils were determined as follows (USEPA, 1993b):

$$C_{dm} = \left(\alpha \times \left(C_i \times F_i + C_{pl} \times F_{pl} + C_{soil} \times F_{soil} \right) \left(\frac{R_{dm}}{W_{dm}} \right) \right) \Theta_{dm} \times \Psi_{dm}$$

and:

$$D_{dm} = \left(\left(C_i \times F_i + C_{pl} \times F_{pl} + C_{soil} \times F_{soil} \right) \left(\frac{R_{dm}}{W_{dm}} \right) \right) \Theta_{dm} \times \Psi_{dm}$$

where:

C_i = terrestrial insect tissue contaminant concentration (absorbed dose), mg/kg
C_{dm} = concentration of toxicant in deer mouse tissue, mg/kg
D_{dm} = applied daily dose for deer mouse, mg/kg-d
F_i = dietary fraction of terrestrial insect, unitless
C_{soil} = concentration of toxicant found in soil, mg/kg
C_{pl} = concentration of toxicant in terrestrial vegetation, mg/kg
F_{pl} = dietary fraction of terrestrial vegetation, unitless
F_{soil} = dietary fraction of soil, unitless
Θ_{dm} = area use factor for deer mouse, unitless
Ψ_{dm} = seasonality factor for deer mouse, unitless
R_{dm} = deer mouse food intake rate, wet diet, $= 0.0687 \times W_{dm}^{0.822}$ kg/d (USEPA, 1993b)
W_{dm} = body weight of deer mouse, kg

Red-Tailed Hawk: Doses received by red-tailed hawks through consumption of contaminated prey items were determined as follows (USEPA, 1993):

$$D_{hk} = C_{dm} \times F_{dm} \left(\frac{R_{hk}}{W_{hk}} \right) \Theta_{hk} \times \Psi_{hk}$$

where:

D_{hk} = applied daily dose for red-tailed hawk, mg/kg-d
F_{dm} = dietary fraction of rodents, unitless
Θ_{hk} = area use factor for hawk, unitless
Ψ_{hk} = seasonality factor for hawk, unitless
R_{hk} = hawk food intake rate, wet diet, = $0.0582 \times W_{hk}^{0.651}$ kg/d
W_{hk} = body weight of hawk, kg

Raccoon: Doses received by, raccoons through consumption of contaminated prey items and incidental ingestion of contaminated soils were determined as follows:

$$D_{rc} = \left(C_{dm} \times F_{dm} + C_i \times F_i + C_{pl} \times F_{pl} + C_{soil} \times F_{soil}\right) \left(\frac{R_{rc}}{W_{rc}} \right) \Theta_{rc} \times \Psi_{rc}$$

where:

D_{rc} = applied daily dose for raccoon, mg/kg-d
C_{dm} = concentration of toxicant in deer mouse tissue, mg/kg
F_{dm} = dietary fraction of rodents, unitless
C_i = terrestrial insect tissue contaminant concentration (absorbed dose), mg/kg
F_i = dietary fraction of terrestrial insect, unitless
C_{soil} = concentration of toxicant found in soil, mg/kg
F_{soil} = dietary fraction of soil, unitless
Θ_{rc} = area use factor for raccoon, unitless
Ψ_{rc} = seasonality factor for raccoon, unitless
R_{rc} = raccoon food intake rate, wet diet, = $0.0687 \times W_{rc}^{0.822}$ kg/d (USEPA, 1993b)
W_{rc} = body weight of raccoon, kg

Tissue concentrations in plants and insects, and potential applied daily doses to mice, hawks, and raccoons are shown in Table 6-22.

6.5.4.2 Waste Water Treatment Area

Models and assumptions used in the Production Area are also used in the Waste Water Treatment Area. In addition, an exposure pathway to the great blue heron is used because this aquatic bird could possibly use the seep area as a forage area.

The estimated daily dose received by the great blue heron through consumption of contaminated prey was determined as follows:

$$D_{gbh} = \left(\frac{\left(C_{pw} \times BCF\right) R_h \times \quad \alpha \quad \times F_{aq}}{BW_h} \right) \Theta$$

where:

C_{pw} = Concentration in pore water, mg/l

BCF = COPC-specific bioconcentration factor

R_h = great blue heron food ingestion rate = $0.648 \times BW^{0.651} = 0.118$ kg/day (USEPA, 1993b)

α = COPC-specific assimilation efficiency; assigned as described in Section 6.5.4.1

F_{aq} = fraction of aquatic/semi-terrestrial species in diet = 1.0 for great blue heron

BW_h = median adult great blue heron body weight = 3.0 kg (USEPA, 1993)

Θ = area use factor for heron, unitless

The bioconcentration factor values for inorganic COPCs were obtained from USEPA (1986), and were calculated for organic COPCs as follows (Lyman, et al., 1982):

$$Log\ (BCF) = 0.76 \times Log\ (K_{ow}) - 0.23$$

where:

K_{ow} = COPC-specified octanol-water partitioning coefficient (see Table 6-H-13). The pore water concentration (C_{pw}) was estimated as follows:

$$C_{pw} = \frac{C_{sed}}{K_{oc} \times f_{oc}}$$

where:

K_{oc} = soil/water partitioning coefficient normalized for organic carbon (unitless)

f_{oc} = fractional organic melts content of the sediment (estimated from total organic carbon results for surface soil in the Waste Water Treatment Area)

C_{sed} = concentration of toxicant in the sediment, mg/kg

Soil/water partition coefficients normalized for organic carbon for metals were calculated using the relationship:

$$K_{oc} = K_d/f_{oc}$$

where:

K_d = soil sorption coefficient (from Baes, et al., 1984)

Partition coefficient values for organic COPCs were calculated as follows (USEPA, 1993c):

$$\log (K_{oc}) = 0.00028 - 0.983 \times \log (K_{ow})$$

The estimated daily dose received by the great blue heron through consumption of drinking water was determined as follows:

$$D_{dw}=C_{sw} \times WR_h \times F_w$$

where:

D_{dw} = dose received from drinking water, mg/day

C_{sw} = concentration in surface water, mg/l

WR_h = water intake rate = $0.059 \times BW_h^{0.67}$ = 0.123 liters/day

F_w = fraction of water intake supplied by seep, 0.001

Tissue concentrations in plants and insects, and potential applied daily doses to mice, raccoons, and hawks are shown in Table 6-23. This evaluation assumes the seep area sediment is part of the terrestrial surface soils, since this seep area probably shrinks in size during dry seasons.

Potential applied daily doses to the great blue heron are shown in Tables 6-24 and 6-25. This evaluation assumes the seep area sediment is part of the seep, and the COPCs that it contains are available for bioaccumulation in the semi-terrestrial pathway.

6.5.4.3 Warwick Area

Models and assumptions used in the Production area are also used in the Warwick Area. SWMU-5 and SWMU-6 contain the majority of COPCs. These SWMUs represent about 10,000 ft^2, or about 1.3 percent of the Warwick Area, and about 5.8 percent of the habitat available to the biota in the Area. Tissue concentrations in plants and insects, and potential applied daily doses to mice, hawks, and raccoons are shown in Table 6-26.

6.5.5 Risk Characterization

Risk characterization quantitatively defines the magnitude of potential risks to ecological receptors under a specific set of circumstances. It is the process of applying numerical methods and professional judgement to determine whether adverse effects are occurring or are likely to occur due to the presence of COPCs at a given study site. This section addresses the following questions:

- Are ecological receptors currently exposed to site-related stressors at levels capable of causing harm, or is future exposure likely?
- If adverse ecological effects are observed or predicted, what are the types, extent, and severity of effects?
- What are the principle uncertainties associated with the risk characterization?

6.5.5.1 Risk Estimation Methodology

For all Areas at the Site, risk estimation involved a quantitative comparison of estimated potential applied daily doses with toxicity reference values (TRVs) for selected receptors to identify the potential for occurrence of adverse effects due to direct (dermal exposure) and secondary (consumption of contaminated prey or forage) exposures. Applied daily doses are calculated in Section 6.5. TRVs are equivalent to NOAELs. When data were available concerning toxicity of a COPC to avian or mammalian indicator species, the NOAEL derived from a chronic study using an indicator species or a taxonomically similar species was the preferred test endpoint and was used as the TRV. When literature data (particularly NOAEL values) were not available for a given COPC-indicator species combination, acceptable TRVs were extrapolated from other test endpoints–usually median lethal dose (LD50), median lethal concentration (LC50), lowest observed effect level (LOEL) or lowest observed adverse effect

(LOAEL) values–and from toxicological studies on other, more common, test species. NOAEL values were used directly as TRVs, while any acute effect level (LD50, LC50) was divided by an uncertainty factor of 100 to derive a TRV, and any LOEL or LOAEL was divided by an uncertainty factor of 10 to estimate a TRV (Sloof et al., 1986; Suter, 1993; Urban and Cook, 1986). The TRV was taken to represent an applied daily dose (ug/g-d) at or below which no adverse effects are expected. TRVs derived for avian and mammalian receptors are summarized in Table 6-H-14.

A toxicity quotient (TQ) was calculated to facilitate this comparison, as follows:

$$TQ_{km} = \log\left(\frac{D_k}{TRV_m}\right)$$

where:

TQ_{km} = toxicity quotient for the *k*th indicator species relative to the *m*th COPC

D_k = applied daily dose–from Tables 6-22, 6-23, 6-24, 6-25 or 6-26 for the *k*th indicator species,

TRV_m = toxicity reference value (from Table 6-H-14) for the *m*th COPC.

A TQ does not define dose-response relationships and its numerical value should not be construed as a direct or probablistic measure of risk. It is merely a convenient method for indicating whether dose exceeds acceptable toxicity values. Positive TQ values indicate that exposure is greater than acceptable levels, negative values indicate that it is not, and zero indicates that exposure equals acceptable levels. Each whole integer above zero indicates an order of magnitude increase in the potential for toxic effects. For example, a TQ of 2 shows that the applied daily dose is approximately equal to the LD50 and that severe effects are possible in 50% of the population. A TQ > 0 does not automatically imply potential risk, but values >0 should be used to identify COPCs that require further evaluation.

Cumulative effects of COPCs were evaluated by taking the antilog of each TQ, then summing all of the values to produce an Ecological Toxicity Index (ETI). This approach assumes that the toxic action of all chemicals is the same; thus, toxicity potential is additive. If the ETI is less than 1.0, the probability of an adverse impact is negligible; if the index is between 1.0 and 10.0, adverse impact is possible; if the index is over 10.0, adverse impact is likely (Barnthouse, et al, 1986).

6.5.5.2 Risk Description

Risk description for all Areas involves summarizing and interpreting the ecological significance of any observed or predicted effects and the degree of risk they pose to ecological receptors. Interpretation of ecological significance must take into account such factors as nature and magnitude of effects, spatial and temporal distribution of effects, and the potential for study site recovery. A weight-of-evidence approach is used wherein several qualitative and quantitative lines of evidence are integrated to describe ecological risks posed by each Area of the Site.

6.5.5.2.1 Production Area

Based on modeling of potential applied daily doses (Table 6-22) and a toxicity quotient analysis (Table 6-27), risk estimation identified no COPCs that have potential for adverse effects in the Production Area. No TQ values were above zero.

The ETI for the Production Area is 0.344, 0.002, and 0.003 for the deer mouse, hawk, and raccoon, respectively (Table 6-27). Thus, no cumulative impacts are expected. Planned IRMs will reduce risk even further.

6.5.5.2.2 Waste Water Treatment Area

Based on modeling of applied daily doses (Table 6-23) and a toxicity quotient analysis (Tables 6-28 and 6-29), risk estimation identified no COPCs that have potential for adverse effects. As shown in Tables 6-18 and 6-29, no TQ values were greater than zero. The ETIs for this Area are 1.36, 0.006, 0.004, and 0.6 for the deer mouse, hawk, raccoon, and heron, respectively. Therefore, it is concluded that no significant risks are expected in the representative species.

6.5.5.2.3 Warwick Area

Based on modeling of potential applied daily doses (Table 6-26) and a toxicity quotient analysis (Table 6-30), no COPCs were identified as being a potential risk to receptors. As shown in Table 6-30, no TQ values were greater than zero. The ETIs for this Area are 0.262, 0.001, and 0.016 for the deer mouse, hawk, and raccoon, respectively. Since all ETIs are less than one, no cumulative impacts are expected.

6.5.5.3 Uncertainty Analysis

The primary objective of the Ecological Risk Assessment is to characterize and quantify potential risk to ecological receptors. Estimates of the potential for adverse effects from

exposure to COPCs are often made with incomplete and imperfect data. To ensure protection of ecological receptors, assumptions are made that tend to overestimate risk rather than underestimate risk. The principal sources of uncertainty are described below and should be considered when evaluating the risk assessment results and when formulating risk management decisions.

6.5.5.3.1 COPC Selection Process

The COPC selection process:

- eliminated all compounds that were not detected at a certain level above the maximum blank concentration;
- eliminated all compounds detected less than 5% of the time; and
- eliminated all essential nutrients (except zinc) and water chemistry parameters (e.g.; total alkalinity).

Selected inorganics and PAHs in the three areas were compared to the background concentrations in soils in the nearby area using a *t*-test with separate variances (see Appendix 6-B). If the values for the Site were lower ($p<.05$) than the background values, these were eliminated as COPCs. If site concentrations were higher than background concentrations, or there were no statistical test performed, then the chemicals were considered COPCs. There are no regulatory standards for soil, so ARARs could not be used to screen COPCs. Chemical and physical parameters that are indicators of persistence or mobility were reviewed, but didn't eliminate any compounds.

Uncertainty is introduced in the selection and quantification of the COPCs due to quality and limitations of background data and the impact of nondetects and sample quantitation limits. Inclusion of all chemicals as COPCs, if not treated with the *t*-test statistic, probably results in an overestimate of the number of COPCs. Maximum detected and 95th percentile UCL of the mean concentrations were used as the selected concentration values for dose calculation. These are overestimates of average values. Concentrations that were qualified as estimated values during data validation (J values) were used to derive the selected concentration values; nondetected values were assumed to be one-half the sample quantitation limit (SQL). The use of J values may result in either an overestimate or an underestimate of the actual average concentration. Because many of the J values are less than one half the SQL, the assumption for nondetects that a concentration equal to one-half the SQL is present tends to overestimate the actual average concentration.

6.5.5.3.2 Receptor Selection

The potential suite of receptors was surveyed on a Site-wide basis and not on an Area-specific basis and thus species uniquely associated with a given Area may have been missed. Knowledge of potential ecological receptors is based on a relatively short period of observation during only one season. Rare, sensitive species or protected migratory species may be under represented in the terrestrial species inventory. Indicator species selected for inclusion in the food web model, as well as the structure of the food web itself, are also sources of uncertainty in that they do not necessarily completely represent ecological relationships at the Site. However, the contaminated areas in question are highly disturbed areas and do not include any unique habitat. As such, they are unlikely to harbor any rare, sensitive, or endangered species.

6.5.5.3.3 Exposure Estimation

Exposure estimation process: Using methods developed and accepted by USEPA, the concentration from uptake and ingestion of each compound in plants, insects, and deer mice was estimated. Once concentration was estimated, the estimated daily dose of each compound in the selected ecological receptors (deer mice, red-tailed hawks, raccoons, and great blue herons) was calculated.

Dermal contact was calculated for several highly toxic compounds, including PCBs, that had dermal toxicity data available. The calculation was done for baby shrews since they live underneath the ground and are hairless when born. None of the compounds presented a significant risk when compared to the risk from ingestion, so dermal contact was not considered further.

Inhalation was calculated for compounds for which data were available. The calculation was done for shrews, since they live in burrows in contact with toxicants. None of the compounds presented a significant risk; thus, inhalation was not considered further.

Concentration was calculated for plants, insects, and deer mice, and these values were used to establish a daily dose for deer mice (based on eating plants and insects and ingesting soil); for red-tailed hawks (based on eating deer mice); and for raccoons (based on eating plants, insects, deer mice and ingesting soil). In the Waste Water Treatment Area, the sediment values in the seep area and the biomagnification to aquatic receptors (frogs) were used to establish a daily dose for the great blue heron. If fish had been present it would have resulted in the same bioconcentration factors as frogs.

For concentration, assimilation of the compound was assumed to be 90%, with the exception of some inorganic compounds for which an actual assimilation factor was available. The toxicant specific loss rate (i.e., the rate at which assimilated COPCs are released from tissues) was assigned a default value of 1 since estimation of this parameter is problematic. Thus, concentration in tissues would tend to be over-estimated, resulting in an overestimation of risk.

Other factors that would also reduce exposure values include:

- bioavailability from soil,
- degradation rates in soil,
- metabolic transformation in vegetation or invertebrates,
- receptor avoidance of contaminated soils,
- dilution over distance, and
- frequency of receptor exposure to contaminated media.

These factors were not included in the risk assessment process; thus, risk would tend to be overestimated.

Home ranges for the red-tailed hawk and the raccoon assumed the amount of foraging done in the contaminated areas was 10%. This estimate is high, since the home range for a red-tailed hawk is about one square mile; and for a raccoon approximately 100 to 6000 acres. Areal photograph analysis indicates that the Site is much less than 10 percent of the potential available habitat for these two animals, assuming the Site is in the center of their home ranges. Therefore, any risk to these animals may be overestimated. Forage area for the heron is between 5-7 km for coastal birds. Several hundred acres of heron habitat is found within 5.7 km of the Cranston Site. Since the seep area is only 0.25 acres and has no fish, the heron's primary food, the wetland seep area in the Warwick Area is assumed to represent less than 0.1 percent of available foraging habitat. This value was used when estimating a risk.

Estimates of COPC concentrations in the plants and terrestrial invertebrates do not account for absorption or elimination factors. This is likely to result in an overestimate of tissue residual levels and thus of risk.

6.5.5.3.4 Effects Characterization

Oral NOAEL data for the mouse were the first choice for a mammalian TRV, with the rat and the rabbit being the second choice. If no NOAEL data were available, then LOAEL data were the

next choice and LD-50 data were used if no LOAEL or NOAEL data were found. If no mammal data were available, then data on birds were selected next. If more than one value was found for the selected species, the most conservative value was used. LD-50 data were divided by 100 to estimate an NOAEL. LOAEL data were divided by 10 to estimate an NOAEL. Oral NOAEL data for birds were the first choice for an avian TRV. If no bird data were available, mammalian data were used as described above.

The NOAEL represents the ecotoxicological chronic effect. NOAELs were not available for all compounds and acute effects data was used to establish a TRV. Once a TRV was established, the daily dose was divided by the TRV and the log value was derived. Anything greater than 0 would be considered a potential risk.

Uncertainty is introduced in this calculation in several ways. The TRV's are based on data obtained for single species under laboratory conditions, as opposed to field conditions. Using similar species lowers the uncertainty, but data for similar species is not always available. Extrapolating chronic effects (NOAEL) from acute effects data (LOAEL, LD-50) is another source of uncertainty. High doses that induce acute effects do not always produce chronic effects at lower levels. Uncertainty arises from the practice of extrapolating an acceptable TRV from acute effects data (LOAEL, LD50, LC-50) by use of an uncertainty factor. Estimating NOAEL tends to overestimate risk. Chemicals for which toxicity data is unavailable further increases the uncertainty. Additionally, comparison of TRVs to the upper 95% confidence interval of the mean concentration is inherently conservative and will tend to overestimate both exposure levels and risk.

As an additional evaluation, the daily doses divided by the TRVs were added together to determine cumulative effects from exposure to many different chemicals. This scenario assumes that the effects caused by the chemicals are additive. There is also the possibility that the effects of exposure to multiple chemicals could be antagonistic or synergistic without additional information, assuming additive effects are probably appropriate.

Due to the availability of data, impacts to individual organisms are considered in this ecological risk assessment, rather than impacts to populations. For the deer mouse, population effects in the Warwick Area were approximated because there is enough habitat to support a population. The home range of a deer mouse is only 1/3 acre. Since the contaminated area in the Warwick Area is only 1/4 acre and represents only about 3.5 percent of deer mouse habitat, most of the

individual deer mice will not come in contact with contamination. Therefore, risks to deer mice in the Warwick area are calculated for the individual then translated into a population effect. Risk to deer mice in the other areas are calculated for the individual only, since contamination is not as isolated and each mouse could be affected. Generally, except for threatened and endangered species, assessments need only to evaluate population effects. Evaluating risks to individuals tends to overestimate risk to both populations and communities (USEPA, 1989b).

Regulatory standards were unavailable for all the compounds and toxicological data was unavailable for a total of six compounds. These data gaps may cause an underestimate of risk, since unevaluated COPCs could be unrecognized sources of risk. The toxicological data could over- or underestimate risk. Other toxicological factors, such as interactions between chemicals, synergism, etc. may also influence effect, but these factors are extremely difficult to assess for wildlife receptors.

6.5.5.3.5 Risk Characterization

Uncertainty arises in comparing the exposure (daily dose) to the effect (TRV). Both of these represent a population with a unique set of statistical characteristics, strongly influencing the assessment of risk and the quantification of uncertainty. Uncertainty also arises when the uncertainty of the exposure assessment is combined with the uncertainty of the effects assessment, producing a multiplicative bias toward a conservative outcome.

Uncertainty inherent to the risk characterization process for assessment of cumulative effects involves the additive assumption of adverse health effects associated with different chemicals. Chemicals in combination may act additively, antagonistically, synergistically, or not influence each other at all. Therefore, the assumptions of additivity used to predict cumulative effects may either overestimate or underestimate ecological risk.

The COPCs identified in this report are not the only source of stress to the Areas investigated. Mechanical disturbance and destruction of habitat, close proximity of human activities, and atmospheric deposition of non site-specific contaminants (particularly polycyclic aromatic hydrocarbons (PAHs) may all contribute to the stresses endured by the ecological receptors in each area of the Site.

6.5.5.4 Conclusions and Recommendations

6.5.5.4.1 Production Area

COPCs are not present in surface soils at concentrations potentially capable of inducing adverse effects; i.e., their toxicity quotient values are less than 0, and thus the Production Area meets the assessment endpoint for terrestrial wildlife. The ETIs do not exceed 1.0.; thus, potential additive adverse effects are negligible. In other words, the presence of site-related chemicals does not appear to pose an unacceptable risk to terrestrial receptors.

6.5.5.4.2 Waste Water Treatment Area

COPCs are not present in surface soils or seep area sediments at concentrations potentially capable of inducing adverse effects; i.e. their toxicity quotient values are less than 0, thus the Waste Water Treatment Area meets the assessment end point for terrestrial wildlife. One ETI for deer mice barely exceeds 1.0 (1.26) and is less than 1.0 for the other representative species. Therefore, potential cumulative effects are not expected. Cumulative effects are likely only if the ETI exceeds 10.0.

6.5.5.4.3 Warwick Area

COPCs are not present in surface soils or seep area sediments at concentrations potentially capable of inducing adverse effects; i.e. their toxicity quotient values are less than 0, thus the Warwick Area meets the assessment end point for terrestrial wildlife. One ETI for deer mice does not exceed 1.0 for the other representative species. Therefore, potential cumulative effects are not expected. Cumulative effects are likely only if the ETI exceeds 10.0.

6.6 Proposed Media Protection Standards

The PHERE provides estimates of potential risks for the Cranston Site using the conservative guidance provided during the May, 1994, meeting with Region I and other meetings and teleconferences. The approach taken is biased towards overestimating risk. For example, all of the risk estimates are based on calculations using the 95 percent UCL of mean chemical concentrations instead of the actual mean. Even with this conservative approach, neither the Production, Waste Water Treatment, nor Warwick Area is predicted to pose an unacceptable potential risk. This was found in spite of the biased sampling approach used in the field investigations that targeted highly localized areas of suspected contamination. The Warwick Area could be an exception but only under the USEPA-imposed assumption that all PCBs are treated as if they were the carcinogenic PCB 1260. No PCB 1260 was found in the Warwick Area.

PCBs are widespread in the Production Area as evidenced by the 89 percent frequency of detection for PCB 1254 and 39 percent for the PCB 1248 in surface soil samples (Appendix 6-A). The risk assessment model for the on-site worker scenario (that is a combination of all the exposure assumption values, environmental transport models, and toxicity criteria used in the risk assessment) can be used to estimate risk-based MPS values for PCB 1248 and PCB 1254. Under these circumstances, the MPSs would be used to identify "hot spots" and their removal considered, even though there are no unacceptable risks. Estimating an MPS is accomplished by beginning with the target THI value of 1 and "back-calculating" through the risk assessment model to the respective surface soil concentrations (Appendix 6-G). Estimated MPS values can be used to compare with the highest concentrations in the Production Area to determine if there are specific zones where PCB concentrations are higher than the MPSs. The estimated MPS value is 50 mg/kg (ppm) for both PCB 1248 and 1254 using the approach described above. Potential additivity of PCB health effects are taken into account in these proposed MPSs, so these values are applicable to all PCBs detected on-site.

A similar approach to estimating MPS values can be taken for the Warwick Area even though PCBs are obviously not as widespread there as in the Production Area with a frequency of detection for PCB 1248 in surface soil of 9 percent and for PCB 1254 of 47 percent (Appendix 6-A). Also, most of the attention in the field investigation was concentrated on SWMU-5. It contains a highly localized remnant of dredge materials from Pawtuxet River sediments taken from a waste water outfall and Cofferdam area immediately adjacent to the Ciba Facility.

Therefore, surface soil MPS values specific to the resident scenario for the Warwick Area would be useful to compare to PCB concentrations found in SWMU-5. Furthermore, residential may not be the most likely land use for this area. The risk assessment model for the resident scenario is used to "back-calculate" MPSs for the PCBs. The estimated surface soil MPS is 5 ppm for PCBs. Potential additive toxicity of PCB 1248 and PCB 1254 is accounted for in these proposals (actually, since PCB 1248 is less toxic than PCB 1254 the MPS value for PCB 1248 alone is 24 ppm).

This PHERE shows that corrective actions are not necessary for the three site areas solely on the basis of unacceptable potential risk to public health or the site ecology. However, Ciba began some limited remediation during the IRMs in the Production and Warwick Areas for reasons other than potential risk; specifically, to facilitate productive use of the areas. Therefore, the risk-based total PCB surface soil MPS proposed for hot spot removal in the Production Area is 50 ppm, and for the Warwick Area is 5 ppm.

PCBs are not even a remote concern in the Waste Water Treatment Area, since they were detected in only two of 31 samples and at concentrations below 1 ppm.

Ciba identified a zone where soil concentrations of PCBs were consistently above 50 ppm in the production area. The presence of this zone of PCB contamination was not a realistic public exposure concern because of the proposed use of this property as a paved vehicle parking facility. However, Ciba volunteered to remove PCB-contaminated surface soil to facilitate its productive use (IRM Work Plan submitted in March, 1995, to Region I).

Similarly, SWMU-5 (Figure 6-1) in the Warwick Area had soil PCB concentrations that were consistently above the 5 ppm MPS. This area is not a realistic exposure concern for the resident scenario because of the highly localized nature of this PCB contamination. However, Ciba volunteered to remove PCB-contaminated soil from the SWMU-5 area (IRM Work Plan submitted to March, 1995, to Region I).

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Table 6-1
Comparison of Production Area Polycyclic Aromatic Hydrocarbon (PAH) Concentrations in Surface Soil with those of Near-Site Background Surface Soil

Chemical	Production Area						Near-Site Background						Urban Background
	Detection/ Total Samples	Frequency of Detection (%)	Minimum Det. Conc. (mg/kg)	Maximum Det. Conc. (mg/kg)	Mean Conc. (mg/kg)	95th UCL[a] (mg/kg)	Detection/ Total Samples	Frequency of Detection (%)	Minimum Det. Conc. (mg/kg)	Maximum Det. Conc. (mg/kg)	Mean Conc. (mg/kg)	95th UCL[a] (mg/kg)	Typical Concentration (mg/kg)
2- Methylnaphthalene	4/41	9.8	0.038	0.38	1.3	1.7	2/12	17	0.57	4.5	0.66	1.3	NA[b]
Acenaphthene	10/41	24	0.057	0.21	1.2	1.7	5/12	42	0.031	5.4	0.69	1.5	NA
Acenaphthylene	5/41	12	0.043	0.18	1.3	1.7	4/12	33	0.044	0.61	0.30	0.4	NA
Anthracene	24/41	59	0.034	1.6	0.88	1.3	7/12	58	0.041	20	2.2	5.1	NA
Benzo(a)anthracene	28/41	68	0.15	3.1	1.1	1.5	7/12	58	0.28	28	3.2	7.4	20[c]
Benzo(a)pyrene	27/41	66	0.024	3.1	1.3	1.7	8/12	67	0.13	22	2.6	5.8	50-75[d]
Benzo(b)fluoranthene	30/41	73	0.027	4.3	1.6	2.0	9/12	75	0.026	36	4.2	9.5	NA
Benzo(g,h,i)perylene	21/41	51	0.13	2.9	1.4	1.8	8/12	67	0.080	12	1.6	3.3	100[e]
Benzo(k)fluoranthene	27/41	66	0.074	5.5	1.5	1.9	8/12	67	0.079	43	4.7	11	NA
Chrysene	28/41	68	0.15	3.3	1.2	1.6	9/12	75	0.14	30	3.4	7.9	20[d]
Dibenz(a,h)anthracene	10/41	24	0.046	0.68	1.2	1.7	3/12	25	0.12	3.7	0.61	1.1	NA
Fluoranthene	33/41	80	0.051	8.4	1.6	2.1	12/12	100	0.043	57	6.6	15	5-120[d]
Fluorene	12/41	29	0.048	0.18	1.2	1.7	5/12	42	0.053	9.4	1.1	2.5	NA
Indeno(1,2,3-cd)pyrene	21/41	51	0.045	2.3	1.3	1.8	7/12	58	0.23	14	1.8	3.9	NA
Naphthalene	14/41	34	0.033	0.68	0.83	1.3	4/12	33	0.023	7.3	0.86	1.9	NA
Phenanthrene	28/41	68	0.093	5.0	1.1	1.5	11/12	92	0.052	69	7.2	17	NA
Pyrene	32/41	78	0.061	6.7	1.8	2.3	12/12	100	0.038	56	6.3	15	5-120[d]

Table 6-1
Comparison of Production Area Polycyclic Aromatic Hydrocarbon (PAH) Concentrations in Surface Soil with those of Near-Site Background Surface Soil

Chemical	Production Area						Near-Site Background						Urban Background
	Detection/ Total Samples	Frequency of Detection (%)	Minimum Det. Conc. (mg/kg)	Maximum Det. Conc. (mg/kg)	Mean Conc. (mg/kg)	95th UCL[a] (mg/kg)	Detection/ Total Samples	Frequency of Detection (%)	Minimum Det. Conc. (mg/kg)	Maximum Det. Conc. (mg/kg)	Mean Conc. (mg/kg)	95th UCL[a] (mg/kg)	Typical Concentration (mg/kg)
Total PAHs[f]					22						48		

a. 95th percentile upper confidence limit of the mean concentration.

b. "NA" = Information not available.

c. Source: IRAC, 1973.

d. Source: White and Vanderslice, 1980.

e. Source: Radian Corporation, 1983.

f. The mean concentration for Total PAHs was derived by adding the mean concentration of each individual PAH in the data set.

Table 6-2
Comparison of Waste Water Treatment Area Polycyclic Aromatic Hydrocarbon (PAH) Concentrations in Surface Soil with those of Near-Site Background Surface Soil

Chemical	Waste Water Treatment Area						Near-Site Background						Urban Background
	Detection/ Total Samples	Frequency of Detection (%)	Minimum Det. Conc. (mg/kg)	Maximum Det. Conc. (mg/kg)	Mean Conc. (mg/kg)	95th UCL[a] (mg/kg)	Detection/ Total Samples	Frequency of Detection (%)	Minimum Det. Conc. (mg/kg)	Maximum Det. Conc. (mg/kg)	Mean Conc. (mg/kg)	95th UCL[a] (mg/kg)	Typical Concentration (mg/kg)
2- Methylnaphthalene	0/181	0	ND	ND	ND	ND	2/12	17	0.57	4.5	0.66	1.3	NA[b]
Acenaphthene	1/18	5.6	0.60	0.60	0.77	1.3	5/12	42	0.031	5.4	0.69	1.5	NA
Acenaphthylene	1/18	5.6	0.045	0.045	0.79	1.3	4/12	33	0.044	0.61	0.30	0.4	NA
Anthracene	2/18	11	0.38	0.38	0.73	1.2	7/12	58	0.041	20	2.2	5.1	NA
Benzo(a)anthracene	6/18	33	0.16	3.90	0.86	1.5	7/12	58	0.28	28	3.2	7.4	20[c]
Benzo(a)pyrene	5/18	28	0.046	3.60	0.86	1.5	8/12	67	0.13	22	2.6	5.8	50-75[d]
Benzo(b)fluoranthene	11/18	61	0.081	3.20	1.0	1.6	9/12	75	0.026	36	4.2	9.5	NA
Benzo(g,h,i)perylene	3/18	17	0.30	2.50	0.90	1.4	8/12	67	0.080	12	1.6	3.3	100[e]
Benzo(k)fluoranthene	8/18	44	0.16	3.80	0.95	1.5	8/12	67	0.079	43	4.7	11	NA
Chrysene	9/18	50	0.10	4.10	0.86	1.5	9/12	75	0.14	30	3.4	7.9	20[d]
Dibenz(a,h)anthracene	1/18	5.6	17.00	17.00	1.4	3.0	3/12	25	0.12	3.7	0.61	1.1	NA
Fluoranthene	12/18	67	0.046	6.90	0.81	1.5	12/12	100	0.043	57	6.6	15	5-120[d]
Fluorene	2/18	11	0.11	0.51	0.77	1.3	5/12	42	0.053	9.4	1.1	2.5	NA
Indeno(1,2,3-cd)pyrene	4/18	22	0.12	2.10	0.85	1.4	7/12	58	0.23	14	1.8	3.9	NA

Table 6-2
Comparison of Waste Water Treatment Area Polycyclic Aromatic Hydrocarbon (PAH) Concentrations in Surface Soil with those of Near-Site Background Surface Soil

Chemical	Waste Water Treatment Area						Near-Site Background						Urban Background
	Detection/ Total Samples	Frequency of Detection (%)	Minimum Det. Conc. (mg/kg)	Maximum Det. Conc. (mg/kg)	Mean Conc. (mg/kg)	95th UCL[a] (mg/kg)	Detection/ Total Samples	Frequency of Detection (%)	Minimum Det. Conc. (mg/kg)	Maximum Det. Conc. (mg/kg)	Mean Conc. (mg/kg)	95th UCL[a] (mg/kg)	Typical Concentration (mg/kg)
Naphthalene	9/18	50	0.066	2.50	0.57	0.9	4/12	33	0.023	7.3	0.86	1.9	NA
Phenanthrene	14/18	78	0.049	5.10	0.82	1.4	11/12	92	0.052	69	7.2	17	NA
Pyrene	16/18	89	0049	5.1	1.5	2.4	12/12	100	0.038	56	6.3	15	5-120[d]
Total PAHs[f]					14						48		

a. 95th percentile upper confidence limit of the mean concentration.

b. "NA" = Information not available.

c. Source: IRAC, 1973.

d. Source: White and Vanderslice, 1980.

e. Source: Radian Corporation, 1983.

f. The mean concentration for Total PAHs was derived by adding the mean concentration of each individual PAH in the data set.

Table 6-3
Comparison of Warwick Area Polycyclic Aromatic Hydrocarbon (PAH) Concentrations in Surface Soil with those of Near-Site Background

Chemical	Warwick Area						Near-Site Background						Urban Background
	Detection/ Total Samples	Frequency of Detection (%)	Minimum Det. Conc. (mg/kg)	Maximum Det. Conc. (mg/kg)	Mean Conc. (mg/kg)	95th UCL[a] (mg/kg)	Detection/ Total Samples	Frequency of Detection (%)	Minimum Det. Conc. (mg/kg)	Maximum Det. Conc. (mg/kg)	Mean Conc. (mg/kg)	95th UCL[a] (mg/kg)	Typical Concentration (mg/kg)
2- Methylnaphthalene	6/31	19	0.014	0.36	1.3	2.0	2/12	17	0.57	4.5	0.66	1.3	NA[b]
Acenaphthene	3/31	9.7	0.016	0.16	1.4	2.0	5/12	42	0.031	5.4	0.69	1.5	NA
Acenaphthylene	3/31	9.7	0.061	0.11	1.4	2.0	4/12	33	0.044	0.61	0.30	0.40	NA
Anthracene	10/31	32	0.031	0.32	1.3	2.0	7/12	58	0.041	20	2.2	5.1	NA
Benzo(a)anthracene	15/31	48	0.14	1.6	0.97	1.4	7/12	58	0.28	28	3.2	7.4	20[c]
Benzo(a)pyrene	13/31	42	0.025	1.7	1.2	1.6	8/12	67	0.13	22	2.6	5.8	50-75[d]
Benzo(b)fluoranthene	14/31	45	0.042	2.8	1.2	1.6	9/12	75	0.026	36	4.2	9.5	NA
Benzo(g,h,i)perylene	9/31	29	0.064	1.2	1.4	2.1	8/12	67	0.08	12	1.6	3.3	100[e]
Benzo(k)fluoranthene	13/31	42	0.062	3.6	1.3	1.8	8/12	67	0.079	43	4.7	11	NA
Chrysene	14/31	45	0.12	2.3	1.07	1.5	9/12	75	0.14	30	3.4	7.9	20[d]
Dibenz(a,h)anthracene	3/31	9.7	0.083	0.13	1.4	2.0	3/12	25	0.12	3.7	0.61	1.1	NA
Fluoranthene	17/31	55	0.038	3.7	1.2	1.6	12/12	100	0.043	57	6.6	15	5-120[d]
Fluorene	7/31	23	0.035	0.23	1.3	2.0	5/12	42	0.053	9.4	1.1	2.5	NA

Table 6-3
Comparison of Warwick Area Polycyclic Aromatic Hydrocarbon (PAH) Concentrations in Surface Soil with those of Near-Site Background

Chemical	Warwick Area						Near-Site Background						Urban Background
	Detection/ Total Samples	Frequency of Detection (%)	Minimum Det. Conc. (mg/kg)	Maximum Det. Conc. (mg/kg)	Mean Conc. (mg/kg)	95th UCL[a] (mg/kg)	Detection/ Total Samples	Frequency of Detection (%)	Minimum Det. Conc. (mg/kg)	Maximum Det. Conc. (mg/kg)	Mean Conc. (mg/kg)	95th UCL[a] (mg/kg)	Typical Concentration (mg/kg)
Indeno(1,2,3-cd)pyrene	8/31	26	0.07	0.86	1.4	2.1	7/12	58	0.23	14	1.8	3.9	NA
Naphthalene	16/31	52	0.036	3.5	1.1	1.6	4/12	33	0.023	7.3	0.86	1.9	NA
Phenanthrene	17/31	55	0.18	1.7	0.87	1.1	11/12	92	0.052	69	7.2	17	NA
Pyrene	18/31	58	0.053	3	1.3	1.6	12/12	100	0.038	56	6.3	15	5-120[d]
Total PAHs[f]					21						47.9		

a. 95th percentile upper confidence limit of the mean concentration.
b. "NA" = Information not available.
c. Source: IARC, 1973.
d. Source: White and Vanderslice, 1980.
e. Source: USEPA, 1983.
f. The mean concentration for Total PAHs was derived by adding the mean concentration of each individual PAH in the data set.

Table 6-4
Exposure Assumption Values for Worker and Resident Scenarios

Parameter	Units	Exposure Scenario Values		
		On-Site Worker	General Worker	On-Site Resident
Body Weight (BW)	kg	70[a]	70[a]	70/15[b]
Averaging Time - Noncarcinogenic (AT_n)	days	9,125[c]	9,125[c]	10,950[c]
Averaging Time - Carcinogenic (AT_c)	days	25,550[d]	25,550[d]	25,550[d]
Exposure Frequency (EF)	(events/yr)	80[e]	250[f]	350[g]
Conversion Factor (CF)	(kg/mg)	1×10^{-6}	1×10^{-6}	1×10^{-6}
Exposure Duration (ED)	yrs	25[h]	25[h]	30[i]
Soil Ingestion Rate (IR_s)	(mg/day)	50[j]	50[k]	100/200[l]
Fraction of Soil Originating from Source (FS)	(none)	1.0[m]	1.0[m]	1.0[m]
Inhalation Rate (I_hR)	(m^3/hr)	1.4[n]	1.4[n]	0.6/0.3[o]
Exposure Time (ET)	(hr/day)	8[p]	1.5[q]	17[r]
Body Surface Area Exposed to Soils (SA_s)	(cm^2/event)	5,000[s]	5,000[s]	5,000/2,000[s]
Soil Adherence Factor (AF)	(mg/cm^2)	1[t]	1[u]	1[t]

a. Default value for an adult (USEPA, 1991a).
b. Adult/child default values (USEPA, 1991a).
c. Equals ED x 365 days/yr.
d. Equals a lifetime (70 years x 365 days/yr).
e. 85 winter work days/year (17 weeks or about 4 months), *minus* 5 days vacation and holidays.
f. Five working days per week, 52 weeks per year, *minus* 2 weeks for vacation days, holidays, and other time off work *equals* 250 exposure days for indoor and outdoor inhalation exposure (USEPA, 1991a). 250 working days per year, *minus* 4 months of working days (85 days) during the winter when exposure would be minimal due to less outdoor activity and frozen/covered ground, *equals* 165 days for the ingestion and dermal exposure pathways.
g. 365 days/year *minus* 15 vacation days, holidays, weekend trips *equals* 350 exposure days/year for indoor and outdoor inhalation exposure (USEPA, 1991a). 365 days/year *minus* 120 winter days/year *minus* 15 vacation days, holidays, weekend trips *equals* 230 outdoor exposure days/year for ingestion and dermal exposures.
h. Upper-bound estimate for time at one place of employment (USEPA, 1991a).
i. Upper-bound estimate for time at one residence (USEPA, 1991b).
j. Default value for industrial/commercial occupations (USEPA, 1991a).
k. Default value for industrial/commercial occupations (USEPA, 1991a), prorated to the portion of the working day (1.5 hours) spent doing outdoor activities.
l. Default value for adult/child residents (USEPA, 1991a). Prorated to portion of the day spent at home (17 hours).

Table 6-4
Exposure Assumption Values for Worker and Resident Scenarios

m. Maximum default value (USEPA, 1991a).
n. Value for moderate activity (USEPA, 1991b).
o. Adult (USEPA, 1990a) child (International Commission on Radiation Protection, 1976).
p. Standard workday.
q. Average daily outdoor activity assumed.
r. Mean hours per day spent at home by men and women is 15.4 (USEPA, 1990a).
s. Default value equaling approximately 25% of the body surface area assumed for the summer months. 5% for the winter months, and 10% for the remainder of the year as recommended by the USEPA's dermal exposure guidance (USEPA, 1992).
t. Default value based on adherence of soil to the hands (USEPA, 1992).
u. Default value based on adherence of soil to the hands (USEPA, 1992), prorated to the portion of the working day spent doing outdoor activities.

Table 6-5
Exposure Assumption Values for Canoeist Scenario

Parameter	Units	Value
Body Weight (BW)	kg	70[a]
Averaging Time - Noncarcinogenic (AT_n)	days	10,950[a]
Averaging Time - Carcinogenic (AT_c)	days	25,550[b]
Exposure Frequency (EF)	(events/yr)	8[c]
Exposure Duration (ED)	yrs	30[a]
Exposure Time (ET)	(hr/event)	2[d]
Water Ingestion Rate (IR_w)	(L/event)	0.005[e]
Body Surface Area Exposed to Water (SA_w)	cm^2	2,300[f]

a. Default value for an adult (USEPA, 1991a).

b. Represents a life expectancy of 75 years.

c. Assumes one exposure event per month, excluding four winter months. Winter months are defined as those with a daily mean temperature less than 40°F. Based on data from the National Oceanic and Atmospheric Administration (1990).

d. Assumes reasonable duration for a canoeing event.

e. In a canoeist scenario, little or virtually no water would be ingested (5 ml = 1 teaspoon).

f. Surface area of hands and feet (USEPA, 1990a).

Table 6-6
Potential Upper-Bound Exposure Point Concentrations for Chemicals of Potential Concern and Background Chemicals

PRODUCTION AREA

Chemical	Frequency of Detection (No. of detects/No. of samples)		Concentration			
	Surface Soil	Combined Soil	Surface Soil[a] (mg/kg)	Combined Soil[b] (mg/kg)	Airborne Emissions[c] (mg/m^3)	River Water (mg/l)
COPCs						
PCB-1248	30/101	39/144	127	88	1.6×10^{-5}	7.6×10^{-6}
PCB-1254	94/104	127/149	20	15	7.0×10^{-7}	1.4×10^{-6}
PCB-1260	7/83	15/126	6.1	4.8	1.5×10^{-7}	1.3×10^{-8}
gamma-Chlordane	7/43	13/86	0.13	0.070	5.0×10^{-9}	1.3×10^{-7}
Vinyl chloride	0/40	0/80	ND[d]	ND	NA[e]	2.6×10^{-5}
2-Nitroaniline	8/41	9/84	0.89	4.2	2.4×10^{-6}	4.8×10^{-3}
Toluene	18/40	31/80	0.71	62	1.6×10^{-3}	9.9×10^{-3}
Chlorobenzene	7/40	9/80	0.28	0.28	4.9×19^{-6}	1.2×10^{-3}
BACKGROUND						
Arsenic	29/32	52/55	15	12	5.9×10^{-8}	--[f]
Benzo(a)pyrene	27/41	44/84	1.7	1.1	6.7×10^{-9}	--[f]
Chromium	29/31	52/54	14	12	5.5×10^{-8}	--[f]
Dibenz(a,h)anthracene	10/41	12/84	0.68	0.68	2.7×10^{-9}	--[f]
Vanadium	28/31	45/52	21	16	8.2×10^{-8}	--[f]

Table 6-6
Potential Upper-Bound Exposure Point Concentrations for Chemicals of Potential Concern and Background Chemicals

WASTE WATER TREATMENT AREA

Chemical	Frequency of Detection (No. of detects/No. of samples)		Concentration			
	Surface Soil	Combined Soil	Surface Soil[a] (mg/kg)	Combined Soil[b] (mg/kg)	Airborne Emissions[c] (mg/m^3)	River Water (mg/l)
COPCs						
2,3,7,8 TCDF	2/6	3/7	6.2×10^{-4}	5.4×10^{-4}	7.7×10^{-11}	7.7×10^{-12}
bis(2-ethylhexyl)phthalate	5/18	11/31	2.1	1.4	2.2×10^{-8}	8.6×10^{-6}
Dieldrin	4/18	6/31	0.17	0.13	9.7×10^{-9}	9.1×10^{-8}
gamma-Chlordane	7/21	14/34	3.2	2.2	1.6×10^{-7}	8.6×10^{-8}
PCB 1254	1/18	2/31	0.21	0.21	9.9×10^{-8}	2.8×10^{-9}
Tinuvin 327	2/9	2/15	11	8.2	8.2×10^{-8}	4.9×10^{-7}
BACKGROUND						
Arsenic	15/15	23/23	7.7	6.4	5.8×10^{-8}	--[f]
Benzo(a)pyrene	5/18	8/31	1.5	1.1	1.1×10^{-8}	--[f]
Beryllium	14/15	20/23	0.52	0.56	3.9×10^{-9}	--[f]
Chromium	15/15	23/23	12	10	9.0×10^{-8}	--[f]
Dibenz(a,h)anthracene	1/18	3/31	3.0	1.9	2.2×10^{-8}	--[f]
Manganese	9/9	15/15	273	228	2.0×10^{-6}	--[f]
Vanadium	15/15	23/23	12	10	9.0×10^{-8}	--[f]

Table 6-6
Potential Upper-Bound Exposure Point Concentrations for Chemicals of Potential Concern and Background Chemicals

WARWICK AREA

Chemical	Frequency of Detection (No. of detects/No. of samples)		Concentration			
	Surface Soil	Combined Soil	Surface Soil[a] (mg/kg)	Combined Soil[b] (mg/kg)	Airborne Emissions[c] (mg/m^3)	River Water (mg/l)
COPCs						
PCB-1248	3/34	3/54	15	9.7	1.6×10^{-6}	2.1×10^{-7}
PCB-1254	15/32	17/52	5.2	3.3	1.4×10^{-7}	9.7×10^{-8}
2-Nitroaniline	2/31	3/41	7.0	7.0	3.7×10^{-6}	1.7×10^{-2}
Chlorobenzene	9/32	12/52	0.67	2.7	4.3×10^{-4}	6.1×10^{-4}
Aldrin	3/33	3/53	0.21	0.14	7.4×10^{-9}	3.9×10^{-6}
Beryllium	3/31	43/44	0.82	0.83	2.9×10^{-9}	4.8×10^{-6}
Dieldrin	5/32	6/52	0.16	0.11	7.4×10^{-9}	4.6×10^{-8}
bis(2-Chloroethyl)ether	2/31	2/41	0.43	0.43	1.5×10^{-9}	2.7×10^{-6}
BACKGROUND						
Antimony	6/23	7/30	5.4	4.2	$1.\times 9\ 10^{-8}$	--[f]
Arsenic	27/27	34/34	10	8.9	3.6×10^{-8}	--[f]
Benzo(a)pyrene	13/31	20/41	1.6	1.3	5.8×10^{-9}	--[f]
Manganese	25/25	26/26	245	254	8.7×10^{-7}	--[f]

a. Lesser of the 95th percent upper confidence limit (UCL) of the mean concentration and the maximum detected concentration in surface soil samples.

b. Lesser of the 95th percent UCL of the mean concentration and the maximum detected concentration in combined surface and subsurface samples.

Table 6-6
Potential Upper-Bound Exposure Point Concentrations for Chemicals of Potential Concern and Background Chemicals

c. Modeled from soil concentrations. Includes fugitive dust emissions predicted using surface soil and gaseous emissions using combined surface and subsurface soil. Refer to Section 5.3.1 of text and Appendix D.

d. "ND"-- Not detected in this medium.

e. "NA"-- Not applicable because this compound was not detected in Production Area soil, from which airborne emissions are modeled.

f. River water concentrations were not modeled for background compounds.

Table 6-7
Toxicity Summary for Chemicals of Potential Concern and Background Chemicals

Chemical	RfD[a] Oral (mg/kg-day)	RfD_i[b] Inhalation (mg/kg-day)	Target Organ[c]	Cancer WOE[d]	CSF_o[e] Oral (/mg/kg-day)	CSF_i[f] Inhalation (/mg/kg-day)	Tumor Site(s)[g]
COPCs							
Aldrin	3×10^{-5}	(see RfD)[h]	Liver	B2	1.7×10^{1}	1.7×10^{1}	Liver carcinoma
Beryllium	5×10^{-3}	(see RfD)[h]	(None)	B2	4.3	8.4 [i]	Lung cancer, osteosarcomas
gamma-Chlordane	6×10^{-5}	(see RfD)[h]	Liver	B2	1.3	1.3	Liver (hepatocellular carcinomas)
Chlorobenzene	2×10^{-2}	5.71×10^{-3} [j]	Liver	D			
bis(2-Chloroethyl)ether				B2	1.1	(See CSF_o)[k]	Liver (hepatocellular carcinomas)
Dieldrin	5×10^{-5}	(see RfD)[h]	Liver	B2	1.6×10^{1}	1.6×10^{1}	Liver carcinoma
bis(2-Ethylhexyl)phthalate	2×10^{-2}	(see RfD)[h]	Liver	B2	1.4×10^{-2}	(See CSF_o)[k]	Liver (hepatocellular carcinomas)
2-Nitroaniline	(see RfD_i)[l]	5.71×10^{-5} [m]	Blood				
PCB 1248	8×10^{-5} [n]	(see RfD)[h]	Developing offspring				
PCB 1254	2×10^{-5}	(see RfD)[h]	Decreased antibody response eyes; nail beds				
PCB 1260				B2	7.7	(see CSF_o) [k]	Liver (hepatocellular carcinoma; neoplastic liver nodules)
2,3,7,8-TCDF[o]				B2[o]	1.56×10^{4} [o]	(see CSF_o) [k]	Liver, respiratory [o]
Tinuvin 327	2.5×10^{-3} [p]		Liver, kidney [p]				
Toluene	2×10^{-1}	1.14×10^{-1} [q]	Ingestion: liver, kidney Inhalation: nervous system				

Table 6-7
Toxicity Summary for Chemicals of Potential Concern and Background Chemicals

Chemical	RfD[a] Oral (mg/kg-day)	RfD_i[b] Inhalation (mg/kg-day)	Target Organ[c]	Cancer WOE[d]	CSF_o[e] Oral (/mg/kg-day)	CSF_i[f] Inhalation (/mg/kg-day)	Tumor Site(s)[g]
Vinyl chloride				A	1.9[j]	3.0×10^{-1}[j]	Liver and lung tumors
BACKGROUND							
Arsenic	3×10^{-4}	(see RfD)[h]	Skin, blood vessels	A	1.75	1.51×10^{1}	Lungs
Benz(a)pyrene				B2	7.3	(see CSF_o)[k]	Oral: Upper gastrointestinal Dermal: Skin Inhalation: Upper respiratory
Beryllium	5×10^{-3}	(see RfD)[h]	Heart, kidney, liver, spleen	B2	4.3	8.4	Lungs, Osteosarcomas
Chromium[r]	5×10^{-3}	(see RfD)[h]	None observed	A	Not reported	4.2×10^{1}	
Dibenz(a,h)anthracene				B2	7.3[s]	(see CSF_o)[k]	Lungs; mammae; skin; subcutaneous injection sites
Manganese[t]	1.4×10^{-1}	1.4×10^{-5}[u]	Central nervous system, muscle				
Vanadium	7×10^{-3}[j]	(See RfD)[h]	Not reported[h]				

a. Chronic reference dose, oral route. Source: Integrated Risk Information System database (IRIS), unless otherwise noted.

b. Chronic reference dose, inhalation exposure route. Calculated from reference concentrations (RfCs).

c. Source: Same as for the RfD value(s).

d. USEPA weight-of-evidence (WOE) classification system regarding carcinogenic effects. Source: IRIS, unless otherwise noted.

e. Cancer slope factor, oral exposure route. Source: IRIS, unless otherwise noted.

f. Cancer slope factor, inhalation route. Source: IRIS, unless otherwise noted.

g. Source: Same as for the CSF value.

Table 6-7
Toxicity Summary for Chemicals of Potential Concern and Background Chemicals

h. No RfD_i available in IRIS or *Health Effects Assessment Summary Tables* (HEAST); RfD value was substituted in the PHRA.

i. Value was estimated from the inhalation unit risk (UR_i) value of 2.4×10^{-3} $(ug/m^{-3})^{-1}$. Source of UR_i: IRIS.

j. Source: HEAST.

k. No CSF_i available in IRIS or HEAST; CSF_O was substituted in the PHERE.

l. No RfD available in IRIS or HEAST; RfD_i value was substituted in the PHERE.

m. RfC value of 2×10^{-4} (mg/m^{-3}) was used to calculate the RfD_i; Source of RfC: HEAST.

n. No toxicity data available in IRIS or the HEAST (USEPA, 1994). Values were derived from a rhesus monkey toxicity study (Allen et al., 1979). The monkeys were given a dose equivalent to 8×10^{-3} mg/kg-day for approximately 1.5 years. This dose was divided by an uncertainty factor of 100. This includes a factor of 10 to account for human variability and a factor of 10 to extrapolate from a lowest-observed-adverse-effects level to a no-observed-adverse-effects level. A provisional PCB 1248 RfD of 1×10^{-3} mg/kg-day was estimated for immunologic effects, the critical effect of PCB 1254. Refer to Section 6.2 for discussion of the immunologic effects RfD for PCB 1248 and potential additive toxicity of PCB 1248 and PCB 1254.

o. Information on 2,3,7,8-tetrachlorodibenzofuran (2,3,7,8-TCDF) is from listing of 2,3,7,8-tetrachlorodibenzodioxin (TCDD). The CSFs for TCDD ($1.5 \times 10^{+5}$) $(mg/kg\text{-}day)^{-1}$ have been multiplied by a toxicity equivalency factor (TEF) of 0.1 to derive the CSF values for 2,3,7,8-TCDF. Source of TEF: USEPA, 1989b.

p. Derived from toxicity data provided by Ciba-Geigy.

q. Derived from the RfC of 4×10^{-1} mg/m^3. Source of RfC: IRIS.

r. Toxicity values are for Chromium VI.

s. Based on the CSF of benzo(a)pyrene using a toxicity equivalence factor of 1. Source: USEPA, 1993.

t. Value for manganese in food. Source: IRIS.

u. RfC value of 5×10^{-5} mg/m^3 was used to calculate the RfD_i.

Table 6-8
Risk Summary for Production Area
On-Site Worker Scenario

Noncancer Risks

CHEMICAL	HAZARD QUOTIENT			HAZARD INDEX
	Ingestion	Dermal	Inhalation	
PCB 1248 [a]	0.25	0.30	0.0069	0.55
PCB 1254	0.16	0.19	0.0012	0.35
gamma-Chlordane	0.00034	0.00080	0.0000029	0.0011
2-Nitroaniline	0.0024	0.0054	0.0015	0.0094
Chlorobenzene	0.0000022	0.0000097	0.000030	0.000042
Toluene	0.00000056	0.0000022	0.00050	0.00050
TOTAL HAZARD INDEX [b]				0.9

Cancer Risks

CHEMICAL	CANCER RISK			
	Ingestion	Dermal	Inhalation	Combined Routes
PCB 1260	2.6×10^{-6}	3.2×10^{-6}	1.4×10^{-8}	5.8×10^{-6}
gamma-Chlordane	9.5×10^{-9}	2.2×10^{-8}	8.1×10^{-11}	3.2×10^{-8}
Vinyl Chloride [c]	0	0	0	0
Total PCBs [d]	6.2×10^{-5}	7.5×10^{-5}	1.6×10^{-6}	1.4×10^{-4}
TOTAL LIFETIME INCREMENTAL CANCER RISK[e]				1×10^{-4}

a. Noncancer risks based on developmental effects.

b Assumes additivity for the effects of all COPCs . The THI for combined immunologic effects of PCB 1248 and PCB 1254 and PCBis estimated as 0.4.

c. Vinyl chloride was not detected in Production Area soils. It was selected as a COPC based on its detection in Production Area groundwater.

d. In accordance with USEPA Region I policy, risk of total PCBs was estimated assuming that all PCBs have the same cancer potency as PCB 1260. This policy contradicts toxicological data which indicate that PCB 1248 and PCB 1254 are noncarcinogenic. The total PCBs data set was created from the combined eoncentrations fo PCB 1248, PCB 1254, and PCB 1260 in surface soil (refer to Section 6.4.2.3.1.)

Table 6-9
Risk Summary for Production Area
General Worker Scenario

Noncancer Risks

CHEMICAL	HAZARD QUOTIENT			HAZARD INDEX
	Ingestion	Dermal	Inhalation	
PCB 1248 [a]	0.097	0.35	0.0041	0.45
PCB 1254	0.061	0.22	0.00072	0.28
gamma-Chlordane	0.00013	0.00094	0.0000017	0.0011
2-Nitroaniline	0.00096	0.0064	0.00087	0.0082
Chlorobenzene	0.00000086	0.000012	0.000018	0.000030
Toluene	0.00000022	0.0000026	0.00029	0.00029
TOTAL HAZARD INDEX [b]				0.8

Cancer Risks

CHEMICAL	CANCER RISK			
	Ingestion	Dermal	Inhalation	Combined Routes
PCB 1260	1.0×10^{-6}	3.7×10^{-6}	8.2×10^{-9}	4.7×10^{-6}
gamma-Chlordane	3.7×10^{-9}	2.6×10^{-8}	4.8×10^{-11}	3.0×10^{-8}
Vinyl Chloride [c]	0	0	0	0
Total PCBs [d]	2.5×10^{-5}	8.8×10^{-5}	9.4×10^{-7}	1.1×10^{-4}
TOTAL LIFETIME INCREMENTAL CANCER RISK[e]				1×10^{-4}

a. Noncancer risks based on developmental effects.

b. Assumes additivity for the effects of all COPCs. The THI for combined immunologic effects of PCB 1248 and PCB 1254 is estimated as 0.3.

c. Vinyl chloride was not detected in Production Area soils. It was selected as a COPC based on its detection in Production Area groundwater.

d. In accordance with USEPA Region I policy, risk of total PCBs was estimated assuming that all PCBs have the same cancer potency as PCB 1260. This policy contradicts toxicological data which indicate that PCB 1248 and PCB 1254 are noncarcinogenic. The total PCBs data set was created from the combined concentrations fo PCB 1248, PCB 1254, and PCB 1260 in surface soil (refer to Section 6.4.2.3.1.)

Table 6-10
Risk Summary for Production Area
Canoeist Scenario

Noncancer Risks

CHEMICAL	HAZARD QUOTIENT		HAZARD INDEX
	Ingestion	Dermal	
PCB 1248[a]	0.00000015	0.00071	0.00071
PCB 1254	0.00000011	0.00053	0.00053
gamma-Chlordane	0.0000000034	0.0000020	0.0000020
2-Nitroaniline	0.00013	0.00039	0.00052
Chlorobenzene	0.000000091	0.0000056	0.0000056
Toluene	0.000000077	0.0000043	0.0000044
TOTAL HAZARD INDEX[b]			0.0006

Cancer Risks

CHEMICAL	CANCER RISK		
	Ingestion	Dermal	Combined Routes
PCB 1260	6.9×10^{-14}	3.3×10^{-10}	3.3×10^{-10}
gamma-Chlordane	1.2×10^{-13}	6.6×10^{-11}	6.6×10^{-11}
Vinyl Chloride	3.4×10^{-11}	3.0×10^{-10}	3.3×10^{-10}
Total PCBs[c]	4.6×10^{-11}	2.2×10^{-7}	2.2×10^{-7}
TOTAL LIFETIME INCREMENTAL CANCER RISK[e]			2×10^{-7}

a. Noncancer risks based on developmental effects.

b. Assumes additivity for the effects of all COPCs. The THI for combined immunologic effects of PCB 1248 and PCB 1254 is estimated as 0.0006.

c. Vinyl chloride was not detected in Production Area soils. It was selected as a COPC based on its detection in Production Area groundwater.

d. In accordance with USEPA Region I policy, risk of total PCBs was estimated assuming that all PCBs have the same cancer potency as PCB 1260. This policy contradicts toxicological data which indicate that PCB 1248 and PCB 1254 are noncarcinogenic. The total PCBs data set was created from the combined concentrations of PCB 1248, PCB 1254, and PCB 1260 in surface soil (refer to Section 6.4.2.3.1.

Table 6-11
Risk Summary for Waste Water Treatment Area
On-Site Resident Scenario

Noncancer Risks

CHEMICAL	HAZARD QUOTIENT			HAZARD INDEX
	Ingestion	Dermal	Inhalation	
gamma-Chlordane	0.091	0.19	0.00047	0.28
PCB 1254	0.018	0.019	0.000087	0.037
Tinuvin 327	0.0075	0.016	0.0000058	0.023
Dieldrin	0.0058	0.012	0.000034	0.018
bis(2-Ethylhexyl)phthalate	0.00018	0.00037	0.00000019	0.00055
TOTAL HAZARD INDEX[a]				0.4

Cancer Risks

CHEMICAL	CANCER RISK			
	Ingestion	Dermal	Inhalation	Combined Routes
2,3,7,8 TCDF	7.0×10^{-6}	4.4×10^{-6}	9.1×10^{-8}	1.2×10^{-5}
gamma-Chlordane	3.0×10^{-6}	6.3×10^{-6}	1.6×10^{-8}	9.3×10^{-6}
Dieldrin	2.0×10^{-6}	4.1×10^{-6}	1.2×10^{-8}	6.1×10^{-6}
bis(2-Ethylhexyl)phthalate	2.1×10^{-8}	4.5×10^{-8}	2.3×10^{-11}	6.6×10^{-8}
Total PCBs[b]	1.2×10^{-6}	1.3×10^{-6}	5.8×10^{-9}	2.4×10^{-6}
TOTAL LIFETIME INCREMENTAL CANCER RISK				3×10^{-5}

a. Assumes additivity for the effects of all COPCs.

b. In accordance with USEPA Region I policy, risk of total PCBs was estimated assuming that all PCBs have the same cancer potency as PCB 1260. This policy is not consistent with toxicological data which indicate that PCB 1254 is noncarcinogenic. PCB 1254 is the only PCB detected in Waste Water Treatment Area soil or groundwater.

Table 6-12
Risk Summary for Waste Water Treatment Area
Canoeist Scenario

Noncancer Risks

CHEMICAL	HAZARD QUOTIENT		HAZARD INDEX
	Ingestion	Dermal	
gamma-Chlordane	0.0000000022	0.0000013	0.0000013
PCB 1254	0.00000000022	0.0000010	0.0000010
Tinuvin 327	0.00000000031	0.0000026	0.0000026
Dieldrin	0.0000000029	0.00000041	0.00000041
bis(2-Ethylhexyl)phthalate	0.00000000068	0.0000000097	0.000000010
TOTAL HAZARD INDEX[a]			0.000005

Cancer Risks

CHEMICAL	CANCER RISK		
	Ingestion	Dermal	Combined Routes
2,3,7,8 TCDF[b]	8.0×10^{-14}	6.8×10^{-10}	6.8×10^{-10}
gamma-Chlordane	7.5×10^{-14}	4.4×10^{-11}	4.4×10^{-11}
Dieldrin	9.8×10^{-13}	1.4×10^{-10}	1.4×10^{-10}
bis(2-Ethylhexyl)phthalate	8.1×10^{-14}	1.2×10^{-12}	1.3×10^{-12}
Total PCBs[c]	1.5×10^{-14}	6.9×10^{-11}	6.9×10^{-11}
TOTAL LIFETIME INCREMENTAL CANCER RISK			9×10^{-10}

a. Assumes additivity for the effect of all COPCs.

b. The transport of 2,3,7,8-TCDF in soil, groundwater, and surface water could not be modeled. Refer to Appendix E.

c. In accordance with USEPA Region I policy, risk of total PCBs was estimated assuming that all PCBs have the same cancer potency as PCB 1260. This policy is not consistent with toxicological data which indicate that PCB 1254 is noncarcinogenic. PCB 1254 is the only PCB detected in Waste Water Treatment Area soil.

Table 6-13
Risk Summary for Warwick Area
On-Site Resident Scenario

Noncancer Risks

CHEMICAL	HAZARD QUOTIENT			HAZARD INDEX
	Ingestion	Dermal	Inhalation	
PCB 1248 [a]	0.32	0.34	0.0035	0.66[a]
PCB 1254	0.44	0.47	0.0013	0.91
2-Nitroaniline	0.21	0.41	0.012	0.63
Chlorobenzene	0.000057	0.000022	0.013	0.013
Aldrin	0.012	0.025	0.000044	0.037
Beryllium	0.00028	0.0049	0.00000010	0.0052
Dieldrin	0.0054	0.011	0.000026	0.017
TOTAL HAZARD INDEX[b]				1[b]

Cancer Risks

CHEMICAL	CANCER RISK			
	Ingestion	Dermal	Inhalation	Combined Routes
Aldrin	2.6×10^{-6}	5.4×10^{-6}	9.7×10^{-9}	8.0×10^{-6}
Beryllium	2.6×10^{-6}	4.5×10^{-5}	1.9×10^{-9}	4.8×10^{-5}
Dieldrin	1.9×10^{-6}	3.9×10^{-6}	8.9×10^{-9}	5.7×10^{-6}
bis(2-Chloroethyl)ether	3.5×10^{-7}	7.1×10^{-7}	1.3×10^{-10}	1.1×10^{-6}
Total PCBs[c]	1.0×10^{-4}	1.1×10^{-4}	1.0×10^{-6}	2.1×10^{-4}
TOTAL LIFETIME INCREMENTAL CANCER RISK				3×10^{-4}

a. Noncancer risks for PCB 1248 based on developmental effects. Hazard Index based on immunologic effects is 0.06, which is additive with PCB 1254.

b. Assumes additivity of the critical effects for chemicals with similar target organs. The target organ-specific THI for combined immunologic effects of PCB 1248 and PCB 1254 results in the highest target organ-specific THI, i.e., 1.

c. In accordance with USEPA Region I policy, risk of total PCBs was estimated assuming that all PCBs have the same cancer potency as PCB 1260. This policy is not consistent with toxicological data which indicate that PCB 1248 and PCB 1254 are noncarcinogenic. PCB 1248 and PCB 1254 are the only PCBs detected in Warwick Area soil.

Table 6-14
Risk Summary for Warwick Area
Canoeist Scenario

Noncancer Risks

CHEMICAL	HAZARD QUOTIENT		HAZARD INDEX
	Ingestion	Dermal	
PCB 1248[a]	0.0000000042	0.000020	0.000020
PCB 1254	0.0000000076	0.000036	0.000036
2-Nitroaniline	0.00046	0.0014	0.0018
Chlorobenzene	0.000000048	0.0000020	0.00000020
Aldrin	0.00000020	0.0000027	0.0000029
Beryllium	0.0000000015	0.0000014	0.0000014
Dieldrin	0.0000000014	0.00000020	0.00000020
TOTAL HAZARD INDEX[b]			0.002

Cancer Risks

CHEMICAL	CANCER RISK		
	Ingestion	Dermal	Combined Routes
Aldrin	4.4×10^{-11}	5.8×10^{-10}	6.2×10^{-10}
Beryllium	1.4×10^{-11}	1.3×10^{-8}	1.3×10^{-8}
Dieldrin	5.0×10^{-13}	7.0×10^{-11}	7.0×10^{-11}
bis(2-Chloroethyl)ether	2.0×10^{-12}	7.6×10^{-12}	9.6×10^{-12}
Total PCBs[c]	1.6×10^{-12}	7.6×10^{-9}	7.6×10^{-9}
TOTAL LIFETIME INCREMENTAL CANCER RISK			2×10^{-8}

a. Noncancer risks based on developmental effects.

b. Assumes additivity for the effects of all COPCs. The THI for the combined immunologic effects of PCB 1248 and 1254 is estimated as 0.00004

c. In accordance with USEPA Region I policy, risk of total PCBs was estimated assuming that all PCBs have the same cancer potency as PCB 1260. This policy is not consistent with toxicological data which indicate that PCB 1248 and PCB 1254 are noncarcinogenic. PCB 1248 and PCB 1254 are the only PCBs detected in Warwick Area soil or groundwater.

Table 6-15
Background Risk Summary for Production Area

On-Site Worker Scenario--Noncancer Risks

CHEMICAL	HAZARD QUOTIENT			HAZARD INDEX
	Ingestion	Dermal	Inhalation	
Arsenic	0.0078	0.00016	0.0000068	0.0080
Chromium	0.00044	0.00018	0.00000038	0.00061
Vanadium	0.00047	0.000094	0.00000041	0.00056
TOTAL HAZARD INDEX[a]				0.009

On-Site Worker Scenario--Cancer Risks

CHEMICAL	CANCER RISK			
	Ingestion	Dermal	Inhalation	Combined Routes
Arsenic	1.5×10^{-6}	3.0×10^{-8}	1.1×10^{-8}	1.5×10^{-6}
Benzo(a)pyrene	6.9×10^{-7}	1.4×10^{-6}	6.1×10^{-10}	2.1×10^{-6}
Chromium[b]	--	--	2.9×10^{-8}	2.9×10^{-8}
Dibenz(a,h) anthracene	2.8×10^{-7}	5.6×10^{-7}	2.4×10^{-10}	8.3×10^{-7}
TOTAL LIFETIME INCREMENTAL CANCER RISK				4×10^{-6}

General Worker Scenario--Noncancer Risks

CHEMICAL	HAZARD QUOTIENT			HAZARD INDEX
	Ingestion	Dermal	Inhalation	
Arsenic	0.00031	0.00019	0.0000040	0.0033
Chromium	0.00017	0.00021	0.00000022	0.00038
Vanadium	0.00018	0.00011	0.00000024	0.00030
TOTAL HAZARD INDEX[a]				0.004

Table 6-15
Background Risk Summary for Production Area

General Worker Scenario--Cancer Risks

CHEMICAL	CANCER RISK			
	Ingestion	Dermal	Inhalation	Combined Routes
Arsenic	5.8×10^{-7}	3.5×10^{-8}	6.5×10^{-9}	6.2×10^{-7}
Benzo(a)pyrene	2.7×10^{-7}	1.6×10^{-6}	3.6×10^{-10}	1.9×10^{-6}
Chromium[b]	--	--	1.7×10^{-8}	1.7×10^{-8}
Dibenz(a,h) anthracene	1.1×10^{-7}	6.5×10^{-7}	1.4×10^{-10}	7.6×10^{-7}
TOTAL LIFETIME INCREMENTAL CANCER RISK				3×10^{-6}

a. Assumes additivity for the effects of all background compounds.

b. Chromium is considered a carcinogen by the inhalation pathway only.

Table 6-16
Background Risk Summary for Waste Water Treatment Area
On-Site Resident Scenario

Noncancer Risks

CHEMICAL	HAZARD QUOTIENT			HAZARD INDEX
	Ingestion	Dermal	Inhalation	
Arsenic	0.044	0.00079	0.000034	0.045
Beryllium	0.00018	0.0031	0.00000014	0.0033
Chromium	0.0041	0.0014	0.0000032	0.0055
Manganese	0.0033	0.00059	0.026	0.030
Vanadium	0.0029	0.00051	0.0000023	0.0034
TOTAL HAZARD INDEX [a]				0.09

Cancer Risks

CHEMICAL	CANCER RISK			
	Ingestion	Dermal	Inhalation	Combined Routes
Arsenic	9.8×10^{-6}	1.8×10^{-8}	6.6×10^{-8}	1.0×10^{-5}
benzo(a)Pyrene	8.0×10^{-6}	1.4×10^{-5}	6.3×10^{-9}	2.2×10^{-5}
Beryllium	1.6×10^{-6}	2.9×10^{-5}	2.5×10^{-9}	3.0×10^{-5}
Chromium[b]	–	–	2.9×10^{-7}	2.9×10^{-7}
Dibenz(a,h)anthracene	1.6×10^{-5}	2.8×10^{-5}	1.2×10^{-8}	4.4×10^{-5}
TOTAL LIFETIME INCREMENTAL CANCER RISK				1×10^{-4}

a. Assumes additivity for the critical effects of all background compounds.

Table 6-17
Background Risk Summary for Warwick Area
On-Site Resident Scenario

Noncancer Risks

CHEMICAL	HAZARD QUOTIENT			HAZARD INDEX
	Ingestion	Dermal	Inhalation	
Antimony	0.023	0.0041	0.0000085	0.027
Arsenic	0.057	0.0010	0.000021	0.058
Manganese	0.0030	0.00053	0.0011	0.015
TOTAL HAZARD INDEX [a]				0.1

Cancer Risks

CHEMICAL	CANCER RISK			
	Ingestion	Dermal	Inhalation	Combined Routes
Arsenic	1.3×10^{-5}	2.3×10^{-7}	4.1×10^{-8}	1.3×10^{-5}
Benzo(a)pyrene	8.5×10^{-6}	1.5×10^{-5}	3.2×10^{-9}	2.4×10^{-5}
TOTAL LIFETIME INCREMENTAL CANCER RISK				4×10^{-5}

a. Assumes additivity for the critical effects of all background compounds.

Table 6-18
Chemicals Of Potential Concern

PRODUCTION AREA	WARWICK AREA	WASTEWATER TREATMENT AREA
INORGANICS	**INORGANICS**	**INORGANICS**
BERYLLIUM	ANTIMONY	BARIUM
CADMIUM	BARIUM	CADMIUM
COBALT	BERYLLIUM	COBALT
COPPER	CADMIUM	COPPER
LEAD	COBALT	LEAD
MERCURY	CHROMIUM	MERCURY
NICKEL	COPPER	NICKEL
THALLIUM	LEAD	SILVER
TIN	MERCURY	THALLIUM
ZINC	NICKEL	ZINC
CYANIDE	TIN	CYANIDE
	VANADIUM	
	ZINC	
	CYANIDE	
ORGANICS	**ORGANICS**	**ORGANICS**
2-METHYLNAPHTHALENE	1,1-BIPHENYL	2-METHYLNAPHTHALENE
2-NITROANILINE	1,4-DICHLOROBENZENE	2-NITROANILINE
3&4-METHYLPHENOL	2-BUTANONE	2,3,7,8-TCDF
4-CHLOROANILINE	2-METHYLNAPHTHALENE	2,4,5-T
ACENAPHTHENE	2-NITROANILINE	2,4-DICHLOROPHENOL
ACENAPHTHYLENE	2,4,5-TP (SILVEX)	3&4-METHYLPHENOL
ANTHRACENE	3&4-METHYLPHENOL	4-CHLOROANILINE
BENZO(A)ANTHRACENE	4-CHLOROANILINE	4-CHLOROPHENYL-PHENYLETHER
BENZO(B)FLUORANTHENE	4,4'-DDD	4-METHYLPHENOL
BENZO(G,H,I)PERYLENE	4,4'-DDE	4,4'-DDD
BENZO(K)FLUORANTHENE	4,4'-DDT	4,4'-DDE
BIS(2-ETHYLHEXYL)PHTHALATE	ALDRIN	4,4'-DDT
BUTYLBENZYLPHTHALATE	ALPHA-BHC	ACENAPHTHENE

Table 6-18
Chemicals Of Potential Concern

PRODUCTION AREA	WARWICK AREA	WASTEWATER TREATMENT AREA
CHLOROBENZENE	ALPHA-CHLORDANE	ACENAPHTHYLENE
CHRYSENE	ACENAPHTHENE	ACETONE
DI-N-BUTYLPHTHALATE	ACENAPHTHYLENE	ACETOPHENONE
DIBENZOFURAN	ANTHRACENE	ALPHA-BHC
DINOSEB	BENZO(A)ANTHRACENE	ANILINE
ETHYLBENZENE	BENZO(B)FLUORANTHENE	ANTHRACENE
FLUORENE	BENZO(G,H,I)PERYLENE	BIS(2-ETHYLHEXYL)PHTHALATE
FLUORANTHENE	BENZO(K)FLUORANTHENE	BUTYLBENZYLPHTHALATE
GAMMA-CHLORDANE	BETA-BHC	CHLOROBENZENE
INDENO(1,2,3-CD)PYRENE	BIS(2-CHLOROETHYL)ETHER	DCDF
IRGASAN DP-300	BIS(2-ETHYLHEXYL)PHTHALATE	DIBENZOFURAN
M&P-XYLENE	BUTYLBENZYLPHTHALATE	DIELDRIN
METHOXYCHLOR	CHLOROBENZENE	DIETHYLPHTHALATE
METHYLENE CHLORIDE	CHRYSENE	ENDRIN
NAPHTHALENE	DELTA-BHC	ENDRIN ALDEHYDE
NITROBENZENE	DIBENZ(A,H)ANTHRACENE	ETHYL PARATHION
O-XYLENE	DI-N-OCTYLPHTHALATE	ETHYLBENZENE
OCDD	DIBENZOFURAN	FAMPHUR
ORTHOPHOSPHATE	DIELDRIN	FLUORANTHENE
PCB-1248	DISULFOTON	FLUORENE
PCB-1254	ENDOSULFAN I	GAMMA-CHLORDANE
PCB-1260	ENDOSULFAN SULFATE	HEPTACHLOR
PHENANTHRENE	ENDRIN	IRGASAN DP-300
PYRENE	ENDRIN ALDEHYDE	M&P-XYLENE
TCDF	ETHYL PARATHION	METHYL PARATHION
TOLUENE	FLUORANTHENE	METHYLENE CHLORIDE
TRCDF	FLUORENE	NAPHTHALENE
TRICHLOROFLUOROMETHANE	GAMMA-BHC	O-XYLENE
	GAMMA-CHLORDANE	OCDD
	HEPTACHLOR EPOXIDE	ORTHOPHOSPHATE

Table 6-18
Chemicals Of Potential Concern

PRODUCTION AREA	WARWICK AREA	WASTEWATER TREATMENT AREA
	INDENO(1,2,3-CD)PYRENE	PHENANTHRENE
	ISODRIN	PHENOL
	M&P-XYLENE	PHORATE
	METHOXYCHLOR	PROPAZINE
	METHYLENE CHLORIDE	PYRENE
	NAPHTHALENE	SULFOTEPP
	NITROBENZENE	TCDF
	O-XYLENE	TINUVIN 327
	ORTHOPHOSPHATE	TOLUENE
	PCB-1248	TRCDF
	PCB-1254	
	PHENANTHRENE	
	PHENOL	
	PYRENE	
	SAFROLE	
	TETRACHLOROETHENE	
	TINUVIN 327	
	TOLUENE	
	TRICHLOROETHENE	

Table 6-19
Chemicals Of Potential Concern Seep Sediment And Surface Water
Waste Water Treatment Area

SEDIMENT	SURFACE WATER
INORGANICS	**INORGANICS**
BARIUM	
CADMIUM	BARIUM
COBALT	LEAD
COPPER	ZINC
LEAD	
NICKEL	
THALLIUM	
ZINC	
ORGANICS	**ORGANICS**
2-METHYLNAPHTHALENE	2-HEXANONE
2,3,7,8-TCDF	4,4'-DDE
4-CHLOROPHENYL-PHENYLETHER	ALDRIN
4,4'-DDD	KEPONE
4,4'-DDE	
4,4'-DDT	
ACENAPHTHENE	
ACETONE	
ALPHA-CHLORDANE	
ANTHRACENE	
BIS(2-ETHYLHEXYL)PHTHALATE	
BUTYLBENZYLPHTHALATE	
CHLOROBENZENE	
DCDF	
DIBENZOFURAN	
ENDRIN	
FLUORANTHENE	
GAMMA-CHLORDANE	
KEPONE	
M&P-XYLENE	
NAPHTHALENE	
OCDD	
PHENANTHRENE	
PYRENE	
TCDF	
TINUVIN 327	
TOLUENE	
TRCDF	

Table 6-20
Summary of Problem Formulation Results

Study Site	COPCs Present?	Receptors Available?	Complete Exposure Pathways?	Comments/ Recommendations
Production Area	yes	yes	limited	COPCs are present; poor habitat quality suggests that potential receptors are likely to be few in number; poor habitat and lack of forage/cover suggests infrequent site use (and hence exposure); site has potential to pose risk to receptors; interim remedial measures will greatly lower any potential risk.
Waste Water Treatment Area	yes	yes	yes	COPCs are present; majority of Area contains a variety of habitats (grassland, woodland, seeps, shrubs) that could harbor potential receptors; suitable habitat with good cover/forage suggests resident receptors with frequent use and exposure; site has potential to pose risk to receptors.
Warwick Area	yes	yes	yes	COPCs are present; revegetation in cleared areas and remaining woodlands offer habitat for potential receptors; suitable habitat with good cover/forage suggests residential receptors with frequent use and exposure; site has potential to pose risk to receptors; interim remedial measures will greatly lower any potential risk.

Table 6-21
Summary of Model Parameters

Parameter	Insect	Deer Mouse	Red-tailed Hawk	Raccoon	Great Blue Heron[d]
dietary fraction (F_{xx})[a]	1.0, terrestrial plants	0.25, terrestrial insect 0.73, terrestrial plants 0.02, incidentally ingested soil	0.75, deer mice 0.25, birds	0.51, terrestrial plant 0.10, terrestrial insect 0.30, deer mouse 0.09, incidentally ingested soil	1.0, aquatic species
area use factor (Θ_{xx})	1.0, assumed to be permanent resident of a given Area	Production:1.0 Warwick: 0.035 Wastewater: 1.0	0.1, any Area is assumed to comprise only 10% of total foraging area	0.01, any Area is assumed to comprise only 1% of total foraging area	0.001, Waste Water Treatment Area is assumed to comprise only 0.1% of total foraging area
seasonality factor (Ψ_{xx})	0.58, assumed to be actively feeding in each Area from April to September	Production: 0.58 assumed to be actively feeding mid-June to mid-December (due to lack of cover vegetation). Waste Water Treatment Area: 1.0 Warwick Area: 1.0 (USEPA, 1993)	0.75, assumed to be actively feeding in each Area from March to October (USEPA, 1993b)	0.75, assumed to be actively feeding in each Area March to October (USEPA, 1993b)	
mean body weight (W_{xx})	0.000135 kg[b]	0.0215 kg[b]	1.0 kg[c]	7.6 kg[c]	3 kg[c]
food intake rate (R_{xx})[d]	0.0000184 kg/d	0.0029 kg/d	0.0582 kg/d	0.3639 kg/d	0.119 kg/d

a. Estimated using data from USEPA (1993b) and MacIntosh et al. (1992)
b. From MacIntosh et al. (1992)
c. From USEPA, 1993
d. Waste Water Treatment Area only

Table 6-22
Estimated Receptor Exposures from Surface Soil
Production Area

Chemical Name	Selected Concentration	Plant Tissue Concentration	Insect Tissue Concentration	Deer Mouse Concentration	Deer Mouse Daily Dose	Red-Tailed Hawk Daily Dose	Raccoon Daily Dose
INORGANICS							
BERYLLIUM	4.54E-01	6.80E-04	4.83E-05	6.75E-04	7.50E-04	2.21E-06	1.48E-05
CADMIUM	8.74E-01	1.31E-01	6.21E-04	5.32E-04	8.87E-03	1.74E-06	5.23E-05
COBALT	3.44E+00	2.41E-02	1.71E-03	6.11E-03	6.79E-03	2.00E-05	1.16E-04
COPPER[a]	3.05E+01	7.63E+00	3.01E-01	2.45E-01	4.90E-01	8.01E-04	2.42E-03
LEAD	7.95E+01	7.15E-01	5.64E-03	1.65E-02	1.65E-01	5.41E-05	2.70E-03
MERCURY	6.11E-01	1.22E-01	1.44E-03	1.19E-03	7.96E-03	3.90E-06	4.22E-05
NICKEL[a]	1.16E+01	6.97E-01	2.75E-03	2.90E-03	5.81E-02	9.50E-06	5.03E-04
THALLIUM	2.14E-01	8.57E-05	6.09E-06	3.07E-04	3.41E-04	1.00E-06	6.97E-06
TIN	6.60E+00	3.96E-02	2.81E-03	1.14E-02	1.26E-02	3.72E-05	2.22E-04
ZINC[a]	3.68E+02	3.31E+02	1.31E+01	9.87E+00	1.97E+01	3.23E-02	7.40E-02
CYANIDE	1.98E+00	0.00E+00	0.00E+00	0.00E+00	3.10E-03	0.00E+00	6.40E-05
ORGANICS							
2-METHYLNAPHTHALENE	3.80E-01	8.64E-02	6.82E-03	5.10E-03	5.66E-03	1.67E-05	2.89E-05
2-NITROANILINE	8.90E-01	3.18E+00	2.26E-01	1.69E-01	1.88E-01	5.52E-04	6.37E-04
3&4-METHYLPHENOL	2.52E-01	7.10E-01	5.04E-02	3.77E-02	4.19E-02	1.23E-04	1.44E-04
4-CHLOROANILINE	6.40E-01	2.17E+00	1.54E-01	1.15E-01	1.28E-01	3.77E-04	4.36E-04
ACENAPHTHENE	2.10E-01	3.16E-02	2.49E-03	1.97E-03	2.18E-03	6.43E-06	1.29E-05
ACENAPHTHYLENE	1.80E-01	3.78E-02	2.98E-03	2.25E-03	2.50E-03	7.36E-06	1.31E-05
ANTHRACENE	1.29E+00	1.43E-01	1.13E-02	9.39E-03	1.04E-02	3.07E-05	6.94E-05
BENZO(A)ANTHRACENE	1.52E+00	3.40E-02	2.68E-03	3.93E-03	4.37E-03	1.29E-05	5.57E-05
BENZO(B)FLUORANTHENE	1.98E+00	1.22E-02	9.64E-04	3.43E-03	3.82E-03	1.12E-05	6.65E-05
BENZO(G,H,I)PERYLENE	1.81E+00	4.64E-03	3.66E-04	2.80E-03	3.11E-03	9.14E-06	5.96E-05
BENZO(K)FLUORANTHENE	1.95E+00	8.40E-03	6.63E-04	3.19E-03	3.54E-03	1.04E-05	6.49E-05
BIS(2-ETHYLHEXYL)PHTHALATE	1.49E+00	3.29E-02	2.34E-03	3.83E-03	4.25E-03	1.25E-05	5.45E-05
BUTYLBENZYLPHTHALATE	3.22E+00	3.26E-01	2.31E-02	2.17E-02	2.41E-02	7.09E-05	1.67E-04
CHLOROBENZENE	2.80E-01	2.48E-01	1.76E-02	1.34E-02	1.49E-02	4.39E-05	5.64E-05
CHRYSENE	1.62E+00	3.63E-02	2.86E-03	4.19E-03	4.66E-03	1.37E-05	5.94E-05
DI-N-BUTYLPHTHALATE	1.30E+00	2.92E-02	2.07E-03	3.37E-03	3.74E-03	1.10E-05	4.78E-05

Table 6-22
Estimated Receptor Exposures from Surface Soil
Production Area

Chemical Name	Selected Concentration	Plant Tissue Concentration	Insect Tissue Concentration	Deer Mouse Concentration	Deer Mouse Daily Dose	Red-Tailed Hawk Daily Dose	Raccoon Daily Dose
DIBENZOFURAN	1.30E-01	2.09E-02	1.49E-03	1.29E-03	1.43E-03	4.20E-06	8.22E-06
DINOSEB	8.70E-03	2.58E-03	1.83E-04	1.48E-04	1.65E-04	4.85E-07	7.76E-07
ETHYLBENZENE	3.41E+00	1.99E+00	1.42E-01	1.10E-01	1.22E-01	3.59E-04	4.92E-04
FLUORANTHENE	2.05E+00	6.61E-02	5.21E-03	6.38E-03	7.09E-03	2.09E-05	7.93E-05
FLUORENE	1.80E-01	2.67E-02	2.11E-03	1.67E-03	1.85E-03	5.45E-06	1.10E-05
GAMMA-CHLORDANE	1.32E-01	3.04E-03	2.16E-04	3.46E-04	3.85E-04	1.13E-06	4.86E-06
INDENO(1,2,3-CD)PYRENE	1.79E+00	2.59E-03	2.04E-04	2.66E-03	2.95E-03	8.69E-06	5.85E-05
IRGASAN DP-300	4.20E+00	data gap	data gap	data gap	data gap	data gap	data gap
M&P-XYLENE	2.70E+01	1.48E+01	1.05E+00	8.18E-01	9.09E-01	2.68E-03	3.71E-03
METHOXYCHLOR	5.55E-01	7.03E-02	4.99E-03	4.48E-03	4.98E-03	1.47E-05	3.15E-05
METHYLENE CHLORIDE	9.50E-03	6.52E-02	4.63E-03	3.45E-03	3.83E-03	1.13E-05	1.28E-05
NAPHTHALENE	6.80E-01	3.01E-01	2.37E-02	1.68E-02	1.87E-02	5.51E-05	7.97E-05
NITROBENZENE	1.40E-01	4.27E-01	3.03E-02	2.27E-02	2.52E-02	7.41E-05	8.61E-05
O-XYLENE	8.12E+00	6.20E+00	4.40E-01	3.38E-01	3.76E-01	1.11E-03	1.45E-03
OCDD	4.60E-04	3.71E-06	2.63E-07	8.44E-07	9.37E-07	2.76E-09	1.56E-08
ORTHOPHOSPHATE	8.13E+00	data gap	data gap	data gap	data gap	data gap	data gap
PCB-1248	1.27E+02	2.33E-02	1.65E-03	1.80E-01	2.00E-01	5.88E-04	4.11E-03
PCB-1254	2.03E+01	3.73E-03	2.64E-04	2.87E-02	3.19E-02	9.40E-05	6.58E-04
PCB-1260	6.10E+00	1.12E-03	7.96E-05	8.66E-03	9.62E-03	2.83E-05	1.98E-04
Total PCB's	1.45E+02	2.67E-02	1.89E-03	2.06E-01	2.29E-01	6.73E-04	4.71E-03
PHENANTHRENE	1.54E+00	1.57E-01	1.24E-02	1.05E-02	1.16E-02	3.43E-05	8.01E-05
PYRENE	2.30E+00	9.04E-02	7.13E-03	8.02E-03	8.91E-03	2.62E-05	9.20E-05
TCDF	1.77E-04	1.42E-06	1.01E-07	3.24E-07	3.60E-07	1.06E-09	6.01E-09
TOLUENE	7.14E-01	7.71E-01	5.47E-02	4.16E-02	4.62E-02	1.36E-04	1.71E-04
TRCDF	5.41E-01	4.36E-03	3.10E-04	9.93E-04	1.10E-03	3.25E-06	1.84E-05
TRICHLOROFLUOROMETHANE	3.30E-01	4.41E-01	3.13E-02	2.37E-02	2.63E-02	7.74E-05	9.50E-05

a- compound is log normally distributed. Mean concentration is the geometric mean.

Table 6-23

Estimated Receptor Exposure from Surface Soil[a]
Waste Water Treatment Area

Chemical Name	Selected Concentration (mg/kg)	Plant Tissue Concentration (mg/kg)	Insect Tissue Concentration (mg/kg)	Deer Mouse Concentration (mg/kg)	Deer Mouse Daily Dose (mg/kg)	Red-Tailed Hawk Daily Dose (mg/kg)	Raccoon Daily Dose (mg/kg)
INORGANICS							
BARIUM	3.52E+01	5.27E-01	4.16E-02	1.48E-02	1.48E-01	4.85E-05	1.24E-03
CADMIUM	3.86E-01	5.79E-02	4.57E-03	4.14E-04	6.90E-03	1.35E-06	2.33E-05
COBALT	3.81E+00	2.67E-02	2.10E-03	1.17E-02	1.30E-02	3.82E-05	1.29E-04
COPPER	4.53E+01	1.13E+01	8.94E-01	6.34E-01	1.27E+00	2.07E-03	3.64E-03
LEAD	5.36E+01	4.82E-01	3.80E-02	1.93E-02	1.93E-01	6.33E-05	1.82E-03
MERCURY	1.93E-01	3.85E-02	3.04E-03	6.63E-04	4.42E-03	2.17E-06	1.35E-05
NICKEL	8.55E+00	5.13E-01	4.04E-02	3.75E-03	7.49E-02	1.23E-05	3.72E-04
SILVER	1.19E+00	1.19E-01	9.38E-03	1.37E-02	1.52E-02	4.49E-05	6.20E-05
THALLIUM	2.76E-01	1.11E-04	8.72E-06	6.82E-04	7.58E-04	2.23E-06	9.02E-06
ZINC	3.59E+02	3.23E+02	2.55E+01	1.68E+01	3.37E+01	5.50E-02	7.35E-02
CYANIDE	1.14E+00	0.00E+00	0.00E+00	0.00E+00	3.07E-03	0.00E+00	3.68E-05
ORGANICS							
2-METHYLNAPHTHALENE	5.00E-01	1.14E-01	8.97E-03	1.16E-02	1.29E-02	3.78E-05	3.85E-05
2-NITROANILINE	7.30E-02	2.61E-01	1.85E-02	2.39E-02	2.65E-02	7.81E-05	5.34E-05
2,3,7,8-TCDF	4.58E-04	3.69E-06	2.62E-07	1.45E-06	1.61E-06	4.73E-09	1.56E-08
2,4,5-T	3.11E-02	1.87E-02	1.32E-03	1.77E-03	1.97E-03	5.79E-06	4.66E-06
2,4-DICHLOROPHENOL	8.39E-01	4.41E-01	3.13E-02	4.21E-02	4.68E-02	1.38E-04	1.14E-04
3&4-METHYLPHENOL	2.51E-01	7.17E-01	5.08E-02	6.57E-02	7.30E-02	2.15E-04	1.48E-04
4-CHLOROANILINE	1.71E+00	5.81E+00	4.12E-01	5.32E-01	5.91E-01	1.74E-03	1.19E-03
4-CHLOROPHENYL-PHENYLETHER	2.80E-01						
4-METHYLPHENOL	2.90E-01	8.61E-01	6.10E-02	7.89E-02	8.76E-02	2.58E-04	1.78E-04
4,4'-DDD	1.73E-01	2.57E-03	1.82E-04	6.53E-04	7.26E-04	2.14E-06	6.13E-06
4,4'-DDE	1.55E-01	3.09E-03	2.19E-04	6.58E-04	7.32E-04	2.15E-06	5.66E-06
4,4'-DDT	9.70E-03	1.31E-04	9.31E-06	3.55E-05	3.94E-05	1.16E-07	3.41E-07
ACENAPHTHENE	1.00E+00	1.51E-01	1.19E-02	1.61E-02	1.79E-02	5.28E-05	6.20E-05
ACENAPHTHYLENE	4.50E-02	9.45E-03	7.45E-04	9.70E-04	1.08E-03	3.17E-06	3.31E-06

Table 6-23

Estimated Receptor Exposure from Surface Soil[a]
Waste Water Treatment Area

Chemical Name	Selected Concentration (mg/kg)	Plant Tissue Concentration (mg/kg)	Insect Tissue Concentration (mg/kg)	Deer Mouse Concentration (mg/kg)	Deer Mouse Daily Dose (mg/kg)	Red-Tailed Hawk Daily Dose (mg/kg)	Raccoon Daily Dose (mg/kg)
ACETONE	1.34E-01	7.15E+00	5.07E-01	6.50E-01	7.22E-01	2.12E-03	1.40E-03
ACETOPHENONE	2.60E-02	1.08E-01	7.63E-03	9.84E-03	1.09E-02	3.22E-05	2.19E-05
ALPHA-BHC	2.10E-03	4.53E-04	3.21E-05	4.62E-05	5.14E-05	1.51E-07	1.57E-07
ANILINE	1.23E+00	1.43E+01	1.02E+00	1.30E+00	1.45E+00	4.27E-03	2.84E-03
ANTHRACENE	1.00E+00	1.11E-01	8.75E-03	1.25E-02	1.39E-02	4.10E-05	5.43E-05
BIS(2-ETHYLHEXYL)PHTHALATE	1.78E+00	3.95E-02	2.80E-03	7.91E-03	8.79E-03	2.59E-05	6.57E-05
BUTYLBENZYLPHTHALATE	1.50E-01	1.52E-02	1.07E-03	1.74E-03	1.93E-03	5.69E-06	7.84E-06
CHLOROBENZENE	2.62E+00	2.31E+00	1.64E-01	2.17E-01	2.41E-01	7.08E-04	5.37E-04
DCDF	2.02E+00	1.63E-02	1.16E-03	6.40E-03	7.11E-03	2.09E-05	6.91E-05
DIBENZOFURAN	7.03E-01	1.13E-01	8.03E-03	1.20E-02	1.33E-02	3.92E-05	4.50E-05
DIELDRIN	1.50E-01	5.52E-02	3.92E-03	5.38E-03	5.98E-03	1.76E-05	1.57E-05
DIETHYLPHTHALATE	7.36E-01	1.08E+00	7.65E-02	9.97E-02	1.11E-01	3.26E-04	2.35E-04
ENDRIN	7.00E-03	1.57E-04	1.11E-05	3.13E-05	3.48E-05	1.02E-07	2.59E-07
ENDRIN ALDEHYDE	2.09E-01	4.70E-03	3.33E-04	9.35E-04	1.04E-03	3.06E-06	7.73E-06
ETHYL PARATHION	5.40E-02	1.40E-02	9.95E-04	1.41E-03	1.56E-03	4.60E-06	4.50E-06
ETHYLBENZENE	4.41E-01	2.58E-01	1.83E-02	2.45E-02	2.72E-02	8.01E-05	6.48E-05
FAMPHUR	7.70E-02	1.42E-01	1.01E-02	1.31E-02	1.45E-02	4.27E-05	3.02E-05
FLUORANTHENE	1.56E+00	5.03E-02	3.97E-03	8.38E-03	9.31E-03	2.74E-05	6.08E-05
FLUORENE	5.10E-01	7.58E-02	5.98E-03	8.14E-03	9.04E-03	2.66E-05	3.14E-05
GAMMA-CHLORDANE	2.58E+00	5.95E-02	4.22E-03	1.17E-02	1.30E-02	3.81E-05	9.56E-05
HEPTACHLOR	7.79E-02	3.02E-03	2.14E-04	4.63E-04	5.15E-04	1.51E-06	3.13E-06
IRGASAN DP-300	8.61E+01						
M&P-XYLENE	1.65E+00	9.02E-01	6.40E-02	8.59E-02	9.55E-02	2.81E-04	2.30E-04
METHYL PARATHION	2.13E-02	6.50E-02	4.61E-03	5.95E-03	6.61E-03	1.95E-05	1.34E-05
METHYLENE CHLORIDE	1.10E-02	7.55E-02	5.36E-03	6.88E-03	7.65E-03	2.25E-05	1.51E-05
NAPHTHALENE	8.27E-01	3.66E-01	2.89E-02	3.53E-02	3.92E-02	1.16E-04	9.85E-05
O-XYLENE	6.35E-01	4.85E-01	3.44E-02	4.56E-02	5.06E-02	1.49E-04	1.15E-04

Table 6-23

Estimated Receptor Exposure from Surface Soil[a]
Waste Water Treatment Area

Chemical Name	Selected Concentration (mg/kg)	Plant Tissue Concentration (mg/kg)	Insect Tissue Concentration (mg/kg)	Deer Mouse Concentration (mg/kg)	Deer Mouse Daily Dose (mg/kg)	Red-Tailed Hawk Daily Dose (mg/kg)	Raccoon Daily Dose (mg/kg)
OCDD	3.00E-04	2.42E-06	1.71E-07	9.48E-07	1.05E-06	3.10E-09	1.02E-08
ORTHOPHOSPHATE	6.15E+01						
PHENANTHRENE	1.44E+00	1.47E-01	1.16E-02	1.69E-02	1.88E-02	5.52E-05	7.56E-05
PHENOL	7.54E-01	4.18E+00	2.97E-01	3.82E-01	4.24E-01	1.25E-03	8.42E-04
PHORATE	1.70E-02	1.35E-02	9.58E-04	1.27E-03	1.41E-03	4.15E-06	3.19E-06
PROPAZINE	9.10E+00	7.14E+00	5.06E-01	6.70E-01	7.44E-01	2.19E-03	1.69E-03
PYRENE	2.25E+00	8.82E-02	6.96E-03	1.35E-02	1.50E-02	4.41E-05	9.04E-05
SULFOTEPP	6.31E-02	1.49E-02	1.06E-03	1.51E-03	1.68E-03	4.94E-06	4.98E-06
TCDF	3.30E-03	2.66E-05	1.89E-06	1.04E-05	1.16E-05	3.42E-08	1.13E-07
TINUVIN 327	8.67E+00	1.14E-01	8.11E-03	3.15E-02	3.49E-02	1.03E-04	3.05E-04
TOLUENE	1.81E+00	1.96E+00	1.39E-01	1.82E-01	2.02E-01	5.95E-04	4.41E-04
TRCDF	4.31E+00	3.47E-02	2.46E-03	1.36E-02	1.51E-02	4.45E-05	1.47E-04

a. Includes results from sediment sampling in seep.

Table 6-24
Estimated Receptor Exposure From Seep Sediments
Waste Water Treatment Area

Chemical Name	Selected Concentration	Pore Water Concentration	Daily Dose Heron
INORGANICS			
BARIUM	3.54E+01	5.90E-01	
CADMIUM	5.70E-01	8.77E-02	1.10E-04
COBALT	4.47E+00	9.93E-02	
COPPER	2.62E+01	7.50E-01	1.04E-01
LEAD	5.29E+01	5.87E-02	1.03E-02
NICKEL	8.80E+00	5.87E-02	8.20E-04
THALLIUM	4.28E-01	2.85E-04	
ZINC	2.21E+02	5.53E+00	2.06E-01
ORGANICS			
2-METHYLNAPHTHALENE	2.80E-01	1.32E-02	5.05E-03
2,3,7,8-TCDF	5.00E-05	8.04E-09	7.29E-05
4-CHLOROPHENYL-PHENYLETHER	2.80E-01	8.03E-03	7.42E-03
4,4'-DDD	1.34E-02	6.09E-06	8.72E-03
4,4'-DDE	1.71E-02	1.28E-05	7.58E-03
4,4'-DDT	9.70E-03	3.77E-06	7.15E-03
ACETONE	1.90E-01	9.61E+01	2.62E-06
ALPHA-CHLORDANE	4.50E-03	2.65E-05	4.06E-04
ACENAPHTHENE	1.00E+00	2.34E-02	3.10E-02
ANTHRACENE	1.43E+00	1.99E-02	6.64E-02
BIS(2-ETHYLHEXYL)PHTHALATE	1.00E+00	9.03E-04	3.87E-01
BUTYLBENZYLPHTHALATE	1.50E-01	1.78E-03	7.87E-03
CHLOROBENZENE	1.05E-01	5.00E-02	3.19E-04
DCDF	2.45E+00	3.93E-04	3.57E+00
DIBENZOFURAN	5.60E-01	1.47E-02	1.59E-02
ENDRIN	7.00E-03	6.43E-06	2.65E-03
FLUORANTHENE	3.64E+00	6.17E-03	8.61E-01
GAMMA-CHLORDANE	1.31E-02	1.26E-05	4.80E-03
KEPONE	3.97E-02	1.26E-01	2.76E-05
NAPHTHALENE	8.20E-01	1.20E-01	6.17E-03
M&P-XYLENE	1.00E-02	2.10E-03	5.68E-05
OCDD	3.00E-04	4.83E-08	4.38E-04
PHENANTHRENE	3.32E+00	4.03E-02	1.71E-01
PYRENE	3.57E+00	8.49E-03	6.49E-01
TCDF	4.00E-04	6.44E-08	5.83E-04
TINUVIN 327	5.10E-01	1.90E-04	3.89E-01
TOLUENE	1.73E-01	1.15E-01	4.02E-04
TRCDF	5.09E+00	6.16E-04	7.42E+00

Table 6-25
Estimated Receptor Exposure From Seep Surface Water
Waste Water Treatment Area

Chemical Name	Selected Concentration	Daily Dose Water
INORGANICS		
BARIUM	6.69E+01	8.23E-03
LEAD	2.55E+01	3.14E-03
ZINC	6.35E+01	7.81E-03
ORGANICS		
2-HEXANONE	7.00E+00	8.61E-04
4,4'-DDE	1.20E-02	1.48E-06
ALDRIN	1.60E-02	1.97E-06
KEPONE	7.90E-02	9.72E-06

Table 6-26
Estimated Receptor Exposure from Surface Soil
Warwick Area

Chemical Name	Selected Concentration	Plant Tissue Concentration	Insect Tissue Concentration	Deer Mouse Concentration	Deer Mouse Daily Dose	Red-Tailed Hawk Daily Dose	Raccoon Daily Dose
INORGANICS							
ANTIMONY	5.35E+00	1.61E-01	1.14E-02	9.66E-04	1.07E-03	3.16E-06	2.03E-04
BARIUM	1.83E+02	2.75E+00	1.95E-01	2.70E-03	2.70E-02	8.83E-06	6.42E-03
BERYLLIUM[a]	8.18E-01	1.23E-03	8.71E-05	8.16E-08	8.16E-05	2.67E-10	2.66E-05
CADMIUM	2.22E+00	3.33E-01	1.58E-03	8.16E-05	1.36E-03	2.67E-07	1.33E-04
CHROMIUM	1.01E+02	4.55E-01	3.59E-04	1.11E-04	1.11E-02	3.64E-07	3.35E-03
COBALT	5.01E+00	3.51E-02	2.49E-03	5.38E-04	5.97E-04	1.76E-06	1.68E-04
COPPER	2.25E+02	5.63E+01	2.22E+00	1.09E-01	2.18E-01	3.56E-04	1.77E-02
LEAD	1.18E+02	1.06E+00	8.36E-03	2.45E-02	2.45E-01	8.01E-05	4.00E-03
MERCURY	3.11E-01	6.22E-02	2.45E-03	3.70E-05	2.47E-04	1.21E-07	2.15E-05
NICKEL[a]	2.63E+01	1.58E+00	1.12E-01	4.02E-04	8.05E-03	1.32E-06	1.14E-03
TIN	9.75E+00	5.85E-02	4.15E-03	1.68E-02	1.87E-02	5.50E-05	3.28E-04
VANADIUM	1.22E+01	3.67E-02	2.61E-03	1.92E-02	2.13E-02	6.27E-05	4.04E-04
ZINC	3.91E+03	3.52E+03	1.39E+02	6.34E+00	1.27E+01	2.07E-02	7.76E-01
CYANIDE	2.70E+00	0.00E+00	0.00E+00	0.00E+00	2.55E-04	0.00E+00	8.73E-05
ORGANICS							
1,1-BIPHENYL	1.90E+00	3.83E-01	2.72E-02	1.38E-03	1.53E-03	4.51E-06	1.33E-04
1,4-DICHLOROBENZENE	3.90E-02	1.66E-02	1.18E-03	5.60E-05	6.22E-05	1.83E-07	4.34E-06
2-BUTANONE	1.22E-01	4.67E-01	3.32E-02	1.49E-03	1.66E-03	4.89E-06	9.08E-05
2-METHYLNAPHTHALENE	3.60E-01	8.19E-02	6.46E-03	2.92E-04	5.37E-03	9.53E-07	2.69E-05
2-NITROANILINE	7.00E+00	2.50E+01	1.78E+00	8.01E-02	8.91E-02	2.62E-04	4.88E-03
2,4,5-TP (SILVEX)	5.27E-02	1.09E-02	7.76E-04	3.92E-05	4.36E-05	1.28E-07	3.73E-06
3&4-METHYLPHENOL	2.20E-02	6.27E-02	4.45E-03	2.01E-04	2.24E-04	6.58E-07	1.24E-05
4-CHLOROANILINE	2.01E+00	6.81E+00	4.84E-01	2.18E-02	2.42E-02	7.13E-05	1.33E-03
4,4'-DDD	1.34E-01	1.99E-03	1.42E-04	1.77E-05	1.97E-05	5.80E-08	4.70E-06
4,4'-DDE	1.49E-01	2.96E-03	2.10E-04	2.21E-05	2.45E-05	7.21E-08	5.35E-06
4,4'-DDT	2.52E-01	3.42E-03	2.43E-04	3.23E-05	3.59E-05	1.06E-07	8.79E-06
ALDRIN	2.08E-01	1.47E-01	1.04E-02	4.84E-04	5.38E-04	1.58E-06	3.40E-05
ALPHA-BHC	1.83E-01	3.94E-02	2.79E-03	1.41E-04	1.56E-04	4.60E-07	1.32E-05
ALPHA-CHLORDANE	7.70E-02	5.15E-03	3.65E-04	2.29E-05	2.55E-05	7.49E-08	3.44E-06

Table 6-26
Estimated Receptor Exposure from Surface Soil
Warwick Area

Chemical Name	Selected Concentration	Plant Tissue Concentration	Insect Tissue Concentration	Deer Mouse Concentration	Deer Mouse Daily Dose	Red-Tailed Hawk Daily Dose	Raccoon Daily Dose
ACENAPHTHENE	1.60E-01	2.41E-02	1.90E-03	9.04E-05	1.66E-03	2.96E-07	9.65E-06
ACENAPHTHYLENE	1.10E-01	2.31E-02	1.82E-03	8.30E-05	1.53E-03	2.71E-07	7.85E-06
ANTHRACENE	3.20E-01	3.55E-02	2.80E-03	1.40E-04	2.58E-03	4.59E-07	1.69E-05
BENZO(A)ANTHRACENE	1.36E+00	3.04E-02	2.40E-03	2.12E-04	3.91E-03	6.94E-07	4.95E-05
BENZO(B)FLUORANTHENE	1.65E+00	1.02E-02	8.02E-04	1.73E-04	3.18E-03	5.64E-07	5.51E-05
BENZO(G,H,I)PERYLENE	1.20E+00	3.08E-03	2.43E-04	1.12E-04	2.06E-03	3.66E-07	3.93E-05
BENZO(K)FLUORANTHENE	1.76E+00	7.60E-03	5.99E-04	1.74E-04	3.21E-03	5.70E-07	5.84E-05
BETA-BHC	9.60E-03	2.07E-03	1.47E-04	7.40E-06	8.22E-06	2.42E-08	6.95E-07
BIS(2-CHLOROETHYL)ETHER	4.30E-01	2.26E+00	1.61E-01	7.23E-03	8.03E-03	2.36E-05	4.34E-04
BIS(2-ETHYLHEXYL)PHTHALATE	2.00E+01	4.44E-01	3.15E-02	3.12E-03	3.46E-03	1.02E-05	7.30E-04
BUTYLBENZYLPHTHALATE	7.80E-01	7.88E-02	5.59E-03	3.17E-04	3.52E-04	1.04E-06	3.98E-05
CHLOROBENZENE	6.68E-01	5.91E-01	4.20E-02	1.93E-03	2.15E-03	6.33E-06	1.31E-04
CHRYSENE	1.47E+00	3.30E-02	2.60E-03	2.30E-04	4.23E-03	7.52E-07	5.36E-05
DELTA-BHC	1.23E-01	2.03E-02	1.44E-03	7.51E-05	8.35E-05	2.46E-07	7.76E-06
DIBENZ(A,H)ANTHRACENE	1.30E-01	8.81E-04	6.95E-05	1.39E-05	2.55E-04	4.53E-08	4.36E-06
DI-N-OCTYLPHTHALATE	4.12E+00	1.54E-01	1.09E-02	8.39E-04	9.32E-04	2.74E-06	1.62E-04
DIBENZOFURAN	2.00E-01	3.22E-02	2.28E-03	1.19E-04	1.33E-04	3.90E-07	1.24E-05
DIELDRIN	1.61E-01	5.90E-02	4.19E-03	2.01E-04	2.23E-04	6.58E-07	1.62E-05
DISULFOTON	7.70E-03	1.42E-03	1.00E-04	5.15E-06	5.73E-06	1.69E-08	5.12E-07
ENDOSULFAN I	1.80E-02	6.19E-03	4.39E-04	2.12E-05	2.35E-05	6.93E-08	1.73E-06
ENDOSULFAN SULFATE	2.90E-01	6.34E-02	4.50E-03	2.26E-04	2.51E-04	7.39E-07	2.12E-05
ENDRIN	1.41E-01	3.17E-03	2.25E-04	2.21E-05	2.45E-05	7.22E-08	5.15E-06
ENDRIN ALDEHYDE	4.44E-01	9.97E-03	7.08E-04	6.95E-05	7.72E-05	2.27E-07	1.62E-05
ETHYL PARATHION	6.40E-03	1.66E-03	1.18E-04	5.83E-06	6.48E-06	1.91E-08	5.16E-07
FLUORANTHENE	1.58E+00	5.07E-02	4.00E-03	2.96E-04	5.45E-03	9.67E-07	6.04E-05
FLUORENE	2.30E-01	3.42E-02	2.70E-03	1.28E-04	2.37E-03	4.20E-07	1.38E-05
GAMMA-BHC	1.70E-03	4.72E-04	3.35E-05	1.65E-06	1.83E-06	5.38E-09	1.43E-07
GAMMA-CHLORDANE	1.49E-01	3.43E-03	2.43E-04	2.35E-05	2.62E-05	7.70E-08	5.44E-06
HEPTACHLOR EPOXIDE	1.92E-01	2.04E-01	1.45E-02	6.65E-04	7.39E-04	2.17E-06	4.41E-05
INDENO(1,2,3-CD)PYRENE	8.60E-01	1.24E-03	9.82E-05	7.71E-05	1.42E-03	2.52E-07	2.80E-05

Table 6-26
Estimated Receptor Exposure from Surface Soil
Warwick Area

Chemical Name	Selected Concentration	Plant Tissue Concentration	Insect Tissue Concentration	Deer Mouse Concentration	Deer Mouse Daily Dose	Red-Tailed Hawk Daily Dose	Raccoon Daily Dose
ISODRIN	1.55E-01	6.87E-03	4.88E-04	3.50E-05	3.89E-05	1.15E-07	6.29E-06
M&P-XYLENE	5.46E-02	2.99E-02	2.12E-03	9.97E-05	1.11E-04	3.26E-07	7.32E-06
METHOXYCHLOR	2.32E+02	2.94E+01	2.08E+00	1.13E-01	1.26E-01	3.70E-04	1.30E-02
METHYLENE CHLORIDE	2.50E-02	1.72E-01	1.22E-02	5.48E-04	6.08E-04	1.79E-06	3.27E-05
NAPHTHALENE	1.58E+00	6.99E-01	5.51E-02	2.36E-03	4.35E-02	7.72E-06	1.81E-04
NITROBENZENE	2.13E+00	6.48E+00	4.60E-01	2.08E-02	2.31E-02	6.80E-05	1.27E-03
O-XYLENE	4.60E-02	3.51E-02	2.49E-03	1.16E-04	1.28E-04	3.78E-07	8.02E-06
ORTHOPHOSPHATE	6.88E+00						
PCB-1248	1.52E+01	2.79E-03	1.98E-04	1.30E-03	1.44E-03	4.24E-06	4.90E-04
PCB-1254	5.19E+00	9.55E-04	6.78E-05	4.45E-04	4.94E-04	1.45E-06	1.68E-04
Total PCB's	1.80E+01	3.31E-03	2.35E-04	1.54E-03	1.71E-03	5.04E-06	5.82E-04
PHENANTHRENE	1.13E+00	1.16E-01	9.11E-03	4.64E-04	8.55E-03	1.52E-06	5.80E-05
PHENOL	8.90E-01	4.94E+00	3.51E-01	1.58E-02	1.75E-02	5.16E-05	9.47E-04
PYRENE	1.60E+00	6.30E-02	4.97E-03	3.37E-04	6.21E-03	1.10E-06	6.36E-05
SAFROLE	4.05E+00	5.41E+00	3.84E-01	1.75E-02	1.95E-02	5.74E-05	1.14E-03
TETRACHLOROETHENE	2.52E-01	3.36E-01	2.39E-02	1.09E-03	1.21E-03	3.57E-06	7.07E-05
TINUVIN 327	9.27E+00	1.22E-01	8.67E-03	1.18E-03	1.31E-03	3.85E-06	3.22E-04
TOLUENE	3.55E-01	3.84E-01	2.72E-02	1.25E-03	1.39E-03	4.09E-06	8.28E-05
TRICHLOROETHENE	5.97E-02	9.74E-02	6.92E-03	3.15E-04	3.50E-04	1.03E-06	2.00E-05

[a]Compound is log normally distributed. Concentration is the geometric mean.

Table 6-27
Toxicity Quotients and Cumulative Effect Totals
Production Area

Chemical Name	TQ Deer Mouse	TQ Hawk	TQ Raccoon	Cumulative Effect Totals		
				TQ Antilog Deer Mouse	TQ Antilog Hawk	TQ Antilog Raccoon
INORGANICS						
BERYLLIUM	-2.86E+00	-5.39E+00	-4.56E+00	1.39E-03	4.09E-06	2.75E-05
CADMIUM	-3.00E+00	-5.36E+00	-5.23E+00	9.97E-04	4.35E-06	5.88E-06
COBALT	-3.34E+00	-5.88E+00	-5.11E+00	4.53E-04	1.33E-06	7.74E-06
COPPER[a]	-2.31E+00	-5.10E+00	-4.62E+00	4.90E-03	8.01E-06	2.42E-05
LEAD	-1.48E+00	-4.97E+00	-3.27E+00	3.31E-02	1.08E-05	5.40E-04
MERCURY	-1.21E+00	-5.11E+00	-3.49E+00	6.12E-02	7.81E-06	3.25E-04
NICKEL[a]	-1.78E+00	-5.57E+00	-3.84E+00	1.66E-02	2.71E-06	1.44E-04
THALLIUM						
TIN	-1.90E+00	-4.43E+00	-3.65E+00	1.26E-02	3.72E-05	2.22E-04
ZINC[a]	-1.37E+00	-2.97E+00	-3.79E+00	4.31E-02	1.08E-03	1.62E-04
CYANIDE	-1.44E+00		-3.12E+00	3.65E-02		7.53E-04
ORGANICS						
2-METHYLNAPHTHALENE	-1.95E+00	-4.48E+00	-4.24E+00	1.13E-02	3.33E-05	5.78E-05
2-NITROANILINE	-2.28E+00	-4.13E+00	-4.75E+00	5.27E-03	7.36E-05	1.79E-05
3&4-METHYLPHENOL	-3.08E+00	-5.61E+00	-5.54E+00	8.39E-04	2.47E-06	2.88E-06
4-CHLOROANILINE	-9.90E-01	-3.52E+00	-3.46E+00	1.02E-01	3.01E-04	3.49E-04
ACENAPHTHENE	-2.36E+00	-4.89E+00	-4.59E+00	4.37E-03	1.29E-05	2.57E-05
ACENAPHTHYLENE	-2.30E+00	-4.83E+00	-4.58E+00	5.00E-03	1.47E-05	2.62E-05
ANTHRACENE	-4.28E+00	-6.81E+00	-6.46E+00	5.22E-05	1.53E-07	3.47E-07
BENZO(A)ANTHRACENE	-2.06E+00	-4.59E+00	-3.95E+00	8.74E-03	2.57E-05	1.11E-04
BENZO(B)FLUORANTHENE	-2.12E+00	-4.65E+00	-3.88E+00	7.63E-03	2.25E-05	1.33E-04
BENZO(G,H,I)PERYLENE	-2.21E+00	-4.74E+00	-3.92E+00	6.21E-03	1.83E-05	1.19E-04
BENZO(K)FLUORANTHENE	-2.15E+00	-4.68E+00	-3.89E+00	7.09E-03	2.09E-05	1.30E-04
BIS(2-ETHYLHEXYL)PHTHALATE	-4.85E+00	-7.38E+00	-6.74E+00	1.42E-05	4.17E-08	1.82E-07
BUTYLBENZYLPHTHALATE	-3.24E+00	-5.77E+00	-5.40E+00	5.78E-04	1.70E-06	4.00E-06
CHLOROBENZENE	-3.19E+00	-5.72E+00	-5.61E+00	6.49E-04	1.91E-06	2.45E-06
CHRYSENE	-2.03E+00	-4.56E+00	-3.93E+00	9.32E-03	2.74E-05	1.19E-04
DI-N-BUTYLPHTHALATE	-4.15E+00	-6.68E+00	-6.04E+00	7.08E-05	2.08E-07	9.03E-07
DIBENZOFURAN	-2.85E+00	-5.39E+00	-5.09E+00	1.40E-03	4.12E-06	8.06E-06

Table 6-27
Toxicity Quotients and Cumulative Effect Totals
Production Area

Chemical Name	TQ Deer Mouse	TQ Hawk	TQ Raccoon	Cumulative Effect Totals: TQ Antilog Deer Mouse	TQ Antilog Hawk	TQ Antilog Raccoon
DINOSEB	-3.55E+00	-6.01E+00	-5.87E+00	2.84E-04	9.70E-07	1.34E-06
ETHYLBENZENE	-2.46E+00	-4.99E+00	-4.85E+00	3.49E-03	1.03E-05	1.41E-05
FLUORANTHENE	-3.45E+00	-5.98E+00	-5.40E+00	3.55E-04	1.04E-06	3.97E-06
FLUORENE	-2.43E+00	-4.96E+00	-4.66E+00	3.70E-03	1.09E-05	2.19E-05
GAMMA-CHLORDANE	-4.07E+00	-5.82E+00	-5.97E+00	8.42E-05	1.51E-06	1.06E-06
INDENO(1,2,3-CD)PYRENE	-2.23E+00	-4.76E+00	-3.93E+00	5.91E-03	1.74E-05	1.17E-04
IRGASAN DP-300						
M&P-XYLENE	-1.67E+00	-4.21E+00	-4.06E+00	2.11E-02	6.22E-05	8.63E-05
METHOXYCHLOR	-3.00E+00	-6.53E+00	-5.20E+00	9.94E-04	2.93E-07	6.28E-06
METHYLENE CHLORIDE	-3.18E+00	-5.72E+00	-5.66E+00	6.55E-04	1.93E-06	2.18E-06
NAPHTHALENE	-2.45E+00	-4.99E+00	-4.83E+00	3.51E-03	1.03E-05	1.50E-05
NITROBENZENE	-2.37E+00	-4.90E+00	-4.84E+00	4.27E-03	1.26E-05	1.46E-05
O-XYLENE	-2.06E+00	-4.59E+00	-4.47E+00	8.74E-03	2.57E-05	3.37E-05
OCDD	-3.09E+00	-5.62E+00	-4.86E+00	8.22E-04	2.42E-06	1.37E-05
ORTHOPHOSPHATE						
PCB-1248	-2.74E+00	-4.35E+00	-4.43E+00	1.81E-03	4.49E-05	3.74E-05
PCB-1254	-2.50E+00	-5.06E+00	-4.19E+00	3.16E-03	8.62E-06	6.52E-05
PCB-1260	-3.13E+00	-5.65E+00	-4.82E+00	7.40E-04	2.25E-06	1.52E-05
Total PCB's	-1.65E+00	-4.21E+00	-3.33E+00			
PHENANTHRENE	-2.78E+00	-5.31E+00	-4.94E+00	1.66E-03	4.90E-06	1.14E-05
PYRENE	-2.95E+00	-5.48E+00	-4.94E+00	1.11E-03	3.28E-06	1.15E-05
TCDF	-3.50E+00	-6.03E+00	-5.28E+00	3.16E-04	9.30E-07	5.27E-06
TOLUENE	-3.03E+00	-5.57E+00	-5.47E+00	9.25E-04	2.72E-06	3.41E-06
TRCDF	-4.66E+00	-7.19E+00	-6.43E+00	2.21E-05	6.49E-08	3.68E-07
TRICHLOROFLUOROMETHANE	-3.12E+00	-5.65E+00	-5.57E+00	7.54E-04	2.22E-06	2.72E-06
	Ecological Toxicity Index			3.44E-01	1.90E-03	2.87E-03

[a]Compound is log normally distributed. Mean concentration is the geometric mean.

Table 6-28

Toxicity Quotients and Cumulative Effect Totals[a]
Waste Water Treatment Area

Chemical Name	TQ Deer Mouse	TQ Hawk	TQ Raccoon	Cumulative Effect Totals		
				TQ Antilog Deer Mouse	TQ Antilog Hawk	TQ Antilog Raccoon
INORGANICS						
BARIUM	-1.53E+00	-5.01E+00	-3.61E+00	2.97E-02	9.70E-06	2.47E-04
CADMIUM	-3.11E+00	-5.47E+00	-5.58E+00	7.75E-04	3.38E-06	2.61E-06
COBALT	-3.06E+00	-5.59E+00	-5.06E+00	8.65E-04	2.55E-06	8.61E-06
COPPER	-1.90E+00	-4.68E+00	-4.44E+00	1.27E-02	2.07E-05	3.64E-05
LEAD	-1.41E+00	-4.90E+00	-3.44E+00	3.87E-02	1.27E-05	3.64E-04
MERCURY	-1.49E+00	-5.40E+00	-4.00E+00	3.27E-02	3.94E-06	9.97E-05
NICKEL	-1.67E+00	-5.46E+00	-3.97E+00	2.14E-02	3.50E-06	1.06E-04
SILVER	-3.82E+00	-6.35E+00	-6.21E+00	1.52E-04	4.49E-07	6.20E-07
THALLIUM						
ZINC	-1.13E+00	-2.74E+00	-3.79E+00	7.35E-02	1.83E-03	1.60E-04
CYANIDE	-1.44E+00		-3.36E+00	3.62E-02		4.33E-04
ORGANICS						
2-METHYLNAPHTHALENE	-1.59E+00	-4.12E+00	-4.11E+00	2.57E-02	7.57E-05	7.71E-05
2-NITROANILINE	-3.13E+00	-4.98E+00	-5.82E+00	7.45E-04	1.04E-05	1.50E-06
2,3,7,8-TCDF	-2.85E+00	-5.38E+00	-4.86E+00	1.41E-03	4.15E-06	1.37E-05
2,4,5-T	-3.09E+00	-5.73E+00	-5.72E+00	8.13E-04	1.86E-06	1.93E-06
2,4-DICHLOROPHENOL	-2.38E+00	-4.91E+00	-5.00E+00	4.14E-03	1.22E-05	1.00E-05
3&4-METHYLPHENOL	-2.84E+00	-5.37E+00	-5.53E+00	1.46E-03	4.30E-06	2.96E-06
4-CHLOROANILINE	-3.25E-01	-2.86E+00	-3.02E+00	4.73E-01	1.39E-03	9.53E-04
4-CHLOROPHENYL-PHENYLETHER						
4-METHYLPHENOL	-2.76E+00	-5.29E+00	-5.45E+00	1.75E-03	5.16E-06	3.55E-06
4,4'-DDD	-4.67E+00	-6.32E+00	-6.74E+00	2.13E-05	4.80E-07	1.80E-07
4,4'-DDE	-4.08E+00	-5.11E+00	-6.19E+00	8.31E-05	7.69E-06	6.44E-07
4,4'-DDT	-4.71E+00	-7.43E+00	-6.77E+00	1.97E-05	3.73E-08	1.71E-07
ACENAPHTHENE	-1.45E+00	-3.98E+00	-3.91E+00	3.59E-02	1.06E-04	1.24E-04
ACENAPHTHYLENE	-2.67E+00	-5.20E+00	-5.18E+00	2.16E-03	6.34E-06	6.63E-06

Table 6-28

Toxicity Quotients and Cumulative Effect Totals[a]
Waste Water Treatment Area

Chemical Name	TQ Deer Mouse	TQ Hawk	TQ Raccoon	Cumulative Effect Totals		
				TQ Antilog Deer Mouse	TQ Antilog Hawk	TQ Antilog Raccoon
ACETONE	-1.62E+00	-4.15E+00	-4.33E+00	2.41E-02	7.08E-05	4.67E-05
ACETOPHENONE	-4.59E+00	-7.12E+00	-7.29E+00	2.58E-05	7.60E-08	5.17E-08
ALPHA-BHC	-4.54E+00	-6.70E+00	-7.05E+00	2.90E-05	2.02E-07	8.86E-08
ANILINE						
ANTHRACENE	-4.16E+00	-6.69E+00	-6.57E+00	6.96E-05	2.05E-07	2.71E-07
BIS(2-ETHYLHEXYL)PHTHALATE	-4.53E+00	-7.06E+00	-6.66E+00	2.93E-05	8.63E-08	2.19E-07
BUTYLBENZYLPHTHALATE	-4.33E+00	-6.86E+00	-6.73E+00	4.64E-05	1.36E-07	1.88E-07
CHLOROBENZENE	-1.98E+00	-4.51E+00	-4.63E+00	1.05E-02	3.08E-05	2.34E-05
DCDF	-3.85E+00	-6.38E+00	-5.86E+00	1.42E-04	4.18E-07	1.38E-06
DIBENZOFURAN	-1.88E+00	-4.42E+00	-4.36E+00	1.31E-02	3.84E-05	4.41E-05
DIELDRIN	-2.02E+00	-4.45E+00	-4.60E+00	9.49E-03	3.52E-05	2.49E-05
DIETHYLPHTHALATE	-2.75E+00	-5.28E+00	-5.42E+00	1.80E-03	5.29E-06	3.80E-06
ENDRIN	-2.60E+00	-5.13E+00	-4.72E+00	2.54E-03	7.47E-06	1.89E-05
ENDRIN ALDEHYDE	-1.12E+00	-5.27E+00	-3.25E+00	7.59E-02	5.37E-06	5.65E-04
ETHYL PARATHION	-1.51E+00	-4.04E+00	-4.05E+00	3.12E-02	9.19E-05	9.00E-05
ETHYLBENZENE	-3.11E+00	-5.64E+00	-5.73E+00	7.78E-04	2.29E-06	1.85E-06
FAMPHUR	-1.27E+00	-2.62E+00	-3.95E+00	5.37E-02	2.40E-03	1.12E-04
FLUORANTHENE	-3.33E+00	-5.86E+00	-5.52E+00	4.66E-04	1.37E-06	3.04E-06
FLUORENE	-1.74E+00	-4.27E+00	-4.20E+00	1.81E-02	5.32E-05	6.29E-05
GAMMA-CHLORDANE	-2.55E+00	-4.29E+00	-4.68E+00	2.84E-03	5.09E-05	2.09E-05
HEPTACHLOR	-3.12E+00	-5.12E+00	-5.34E+00	7.57E-04	7.57E-06	4.60E-06
IRGASAN DP-300						
M&P-XYLENE	-2.65E+00	-5.18E+00	-5.27E+00	2.22E-03	6.53E-06	5.35E-06
METHYL PARATHION	-8.79E-01	-3.41E+00	-3.57E+00	1.32E-01	3.89E-04	2.68E-04
METHYLENE CHLORIDE	-2.88E+00	-5.41E+00	-5.59E+00	1.31E-03	3.85E-06	2.58E-06
NAPHTHALENE	-2.13E+00	-4.66E+00	-4.73E+00	7.36E-03	2.17E-05	1.85E-05
O-XYLENE	-2.93E+00	-5.46E+00	-5.57E+00	1.18E-03	3.46E-06	2.68E-06

Table 6-28
Toxicity Quotients and Cumulative Effect Totals[a]
Waste Water Treatment Area

Chemical Name	TQ Deer Mouse	TQ Hawk	TQ Raccoon	Cumulative Effect Totals		
				TQ Antilog Deer Mouse	TQ Antilog Hawk	TQ Antilog Raccoon
OCDD	-3.98E+00	-6.51E+00	-5.99E+00	1.05E-04	3.10E-07	1.02E-06
ORTHOPHOSPHATE						
PHENANTHRENE	-2.57E+00	-5.10E+00	-4.97E+00	2.68E-03	7.89E-06	1.08E-05
PHENOL	-8.04E-01	-2.96E+00	-3.51E+00	1.57E-01	1.10E-03	3.12E-04
PHORATE	-1.20E+00	-4.23E+00	-3.85E+00	6.26E-02	5.82E-05	1.42E-04
PROPAZINE	-8.27E-01	-4.66E+00	-3.47E+00	1.49E-01	2.19E-05	3.38E-04
PYRENE	-2.73E+00	-5.26E+00	-4.95E+00	1.87E-03	5.52E-06	1.13E-05
SULFOTEPP	-2.47E+00	-5.01E+00	-5.00E+00	3.36E-03	9.88E-06	9.95E-06
TCDF	-2.94E+00	-5.47E+00	-4.95E+00	1.16E-03	3.42E-06	1.13E-05
TINUVIN 327	-3.30E+00	-5.83E+00	-5.36E+00	4.99E-04	1.47E-06	4.35E-06
TOLUENE	-2.39E+00	-4.92E+00	-5.05E+00	4.04E-03	1.19E-05	8.82E-06
TRCDF	-3.52E+00	-6.05E+00	-5.53E+00	3.03E-04	8.91E-07	2.94E-06
	Ecological Toxicity Index			1.36E+00	6.08E-03	3.80E-03

[a]Includes results from sediment sampling in seep.

Table 6-29
Toxicity Quotients and Cumulative Effects Totals for Seep Waste Water Treatment Area

SEDIMENT		
Chemical Name	TQ Heron	Cumulative Effects Antilog TQ
INORGANICS		
BARIUM		
CADMIUM	-3.56E+00	2.75E-04
COBALT		
COPPER	-2.98E+00	1.04E-03
LEAD	-2.69E+00	2.06E-03
NICKEL	-3.63E+00	2.34E-04
THALLIUM		
ZINC	-2.16E+00	6.87E-03
ORGANICS		
2-METHYLNAPHTHALENE	-2.00E+00	1.01E-02
2,3,7,8-TCDF	-1.19E+00	6.40E-02
4-CHLOROPHENYL-PHENYLETHER		
4,4'-DDD	-2.71E+00	1.96E-03
4,4'-DDE	-1.57E+00	2.71E-02
4,4'-DDT	-2.64E+00	2.30E-03
ACENAPHTHENE	-1.21E+00	6.21E-02
ACETONE	-7.06E+00	8.75E-08
ALPHA-CHLORDANE	-3.27E+00	5.42E-04
ANTHRACENE	-3.48E+00	3.32E-04
BIS(2-ETHYLHEXYL)PHTHALATE	-2.89E+00	1.29E-03
BUTYLBENZYLPHTHALATE	-3.72E+00	1.89E-04
CHLOROBENZENE	-4.86E+00	1.39E-05
DCDF	-1.15E+00	7.13E-02
DIBENZOFURAN	-1.81E+00	1.56E-02
ENDRIN	-2.33E+00	4.65E-03
FLUORANTHENE	-1.37E+00	4.30E-02
GAMMA-CHLORDANE	-2.19E+00	6.40E-03
KEPONE	-4.26E+00	5.52E-05
M&P-XYLENE	-5.88E+00	1.32E-06
NAPHTHALENE	-2.94E+00	1.16E-03
OCDD	-1.36E+00	4.38E-02
PHENANTHRENE	-1.61E+00	2.45E-02
PYRENE	-1.09E+00	8.11E-02
TCDF	-1.23E+00	5.83E-02
TINUVIN 327	-2.25E+00	5.56E-03
TOLUENE	-5.10E+00	8.03E-06
TRCDF	-8.29E-01	1.48E-01
Total		6.00E-01

Table 6-29
Toxicity Quotients and Cumulative Effects Totals for Seep Waste Water Treatment Area

SURFACE WATER		
Chemical Name	TQ Heron	Cumulative Effects Antilog TQ
INORGANICS		
BARIUM	-2.78E+00	1.65E-03
LEAD	-3.20E+00	6.28E-04
ZINC	-3.58E+00	2.60E-04
ORGANICS		
2-HEXANONE	-4.45E+00	3.54E-05
4,4'-DDE	-5.28E+00	5.27E-06
ALDRIN	-5.46E+00	3.45E-06
KEPONE	-4.71E+00	1.94E-05
Total		2.60E-03
Ecological Toxicity Index		6.02E-01

Table 6-30
Toxicity Quotients and Cumulative Effect Totals
Warwick Area

Chemical Name	TQ Deer Mouse	TQ Hawk	TQ Raccoon	Cumulative Effect Totals		
				TQ Antilog Deer Mouse	TQ Antilog Hawk	TQ Antilog Raccoon
INORGANICS						
ANTIMONY	-4.81E+00	-7.35E+00	-5.54E+00	1.53E-05	4.51E-08	2.90E-06
BARIUM	-2.27E+00	-5.75E+00	-2.89E+00	5.40E-03	1.77E-06	1.28E-03
BERYLLIUM[a]	-3.82E+00	-9.31E+00	-4.31E+00	1.51E-04	4.94E-10	4.94E-05
CADMIUM	-3.82E+00	-7.18E+00	-4.83E+00	1.53E-04	6.67E-08	1.49E-05
CHROMIUM	-2.06E+00	-6.44E+00	-2.58E+00	8.75E-03	3.64E-07	2.64E-03
COBALT	-4.40E+00	-6.93E+00	-4.95E+00	3.98E-05	1.17E-07	1.12E-05
COPPER	-2.66E+00	-5.45E+00	-3.75E+00	2.18E-03	3.56E-06	1.77E-04
LEAD	-1.31E+00	-4.80E+00	-3.10E+00	4.90E-02	1.60E-05	8.00E-04
MERCURY	-2.72E+00	-6.62E+00	-3.78E+00	1.90E-03	2.42E-07	1.66E-04
NICKEL[a]	-2.64E+00	-6.42E+00	-3.49E+00	2.30E-03	3.76E-07	3.26E-04
TIN	-1.73E+00	-4.26E+00	-3.48E+00	1.87E-02	5.50E-05	3.28E-04
VANADIUM						
ZINC	-1.56E+00	-3.16E+00	-2.77E+00	2.77E-02	6.91E-04	1.70E-03
CYANIDE	-2.52E+00		-2.99E+00			1.03E-03
ORGANICS						
1,1-BIPHENYL	-4.42E+00	-6.95E+00	-5.48E+00	3.83E-05	1.13E-07	3.32E-06
1,4-DICHLOROBENZENE	-5.68E+00	-8.21E+00	-6.83E+00	2.11E-06	6.21E-09	1.47E-07
2-BUTANONE	-4.39E+00	-6.92E+00	-5.65E+00	4.10E-05	1.21E-07	2.24E-06
2-METHYLNAPHTHALENE	-1.97E+00	-5.72E+00	-4.27E+00	1.07E-02	1.91E-06	5.38E-05
2-NITROANILINE	-2.60E+00	-4.46E+00	-3.86E+00	2.50E-03	3.49E-05	1.37E-04
2,4,5-TP (SILVEX)	-4.80E+00	-7.38E+00	-5.87E+00	1.58E-05	4.12E-08	1.35E-06
3&4-METHYLPHENOL	-5.35E+00	-7.88E+00	-6.61E+00	4.47E-06	1.32E-08	2.47E-07
4-CHLOROANILINE	-1.71E+00	-4.24E+00	-2.97E+00	1.94E-02	5.71E-05	1.06E-03
4,4'-DDD	-6.24E+00	-7.88E+00	-6.86E+00	5.80E-07	1.30E-08	1.38E-07
4,4'-DDE	-5.56E+00	-6.59E+00	-6.22E+00	2.79E-06	2.58E-07	6.09E-07
4,4'-DDT	-4.75E+00	-7.47E+00	-5.36E+00	1.80E-05	3.40E-08	4.39E-06
ALDRIN	-2.92E+00	-5.56E+00	-4.12E+00	1.19E-03	2.78E-06	7.55E-05
ALPHA-BHC	-4.05E+00	-6.21E+00	-5.13E+00	8.83E-05	6.13E-07	7.47E-06

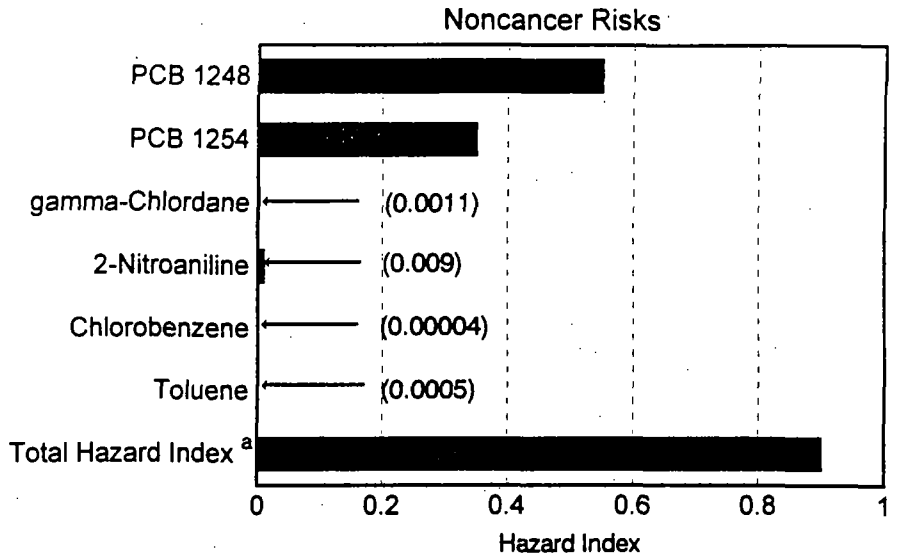
Table 6-30
Toxicity Quotients and Cumulative Effect Totals
Warwick Area

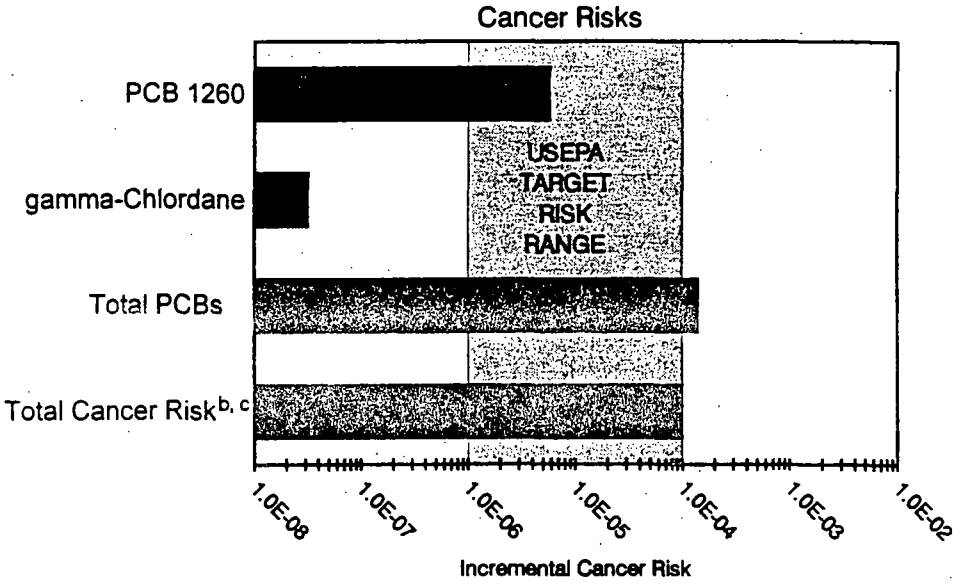
Chemical Name	TQ Deer Mouse	TQ Hawk	TQ Raccoon	Cumulative Effect Totals		
				TQ Antilog Deer Mouse	TQ Antilog Hawk	TQ Antilog Raccoon
ALPHA-CHLORDANE	-5.25E+00	-7.00E+00	-6.12E+00	5.57E-06	9.99E-08	7.54E-07
ACENAPHTHENE	-2.48E+00	-6.23E+00	-4.71E+00	3.33E-03	5.91E-07	1.93E-05
ACENAPHTHYLENE	-2.51E+00	-6.27E+00	-4.80E+00	3.06E-03	5.43E-07	1.57E-05
ANTHRACENE	-4.89E+00	-8.64E+00	-7.07E+00	1.29E-05	2.29E-09	8.47E-08
BENZO(A)ANTHRACENE	-2.11E+00	-5.86E+00	-4.00E+00	7.82E-03	1.39E-06	9.89E-05
BENZO(B)FLUORANTHENE	-2.20E+00	-5.95E+00	-3.96E+00	6.35E-03	1.13E-06	1.10E-04
BENZO(G,H,I)PERYLENE	-2.39E+00	-6.14E+00	-4.10E+00	4.12E-03	7.32E-07	7.87E-05
BENZO(K)FLUORANTHENE	-2.19E+00	-5.94E+00	-3.93E+00	6.41E-03	1.14E-06	1.17E-04
BETA-BHC	-6.10E+00	-7.49E+00	-7.18E+00	7.90E-07	3.23E-08	6.68E-08
BIS(2-CHLOROETHYL)ETHER	-2.23E+00	-4.76E+00	-3.50E+00	5.90E-03	1.74E-05	3.19E-04
BIS(2-ETHYLHEXYL)PHTHALATE	-4.94E+00	-7.47E+00	-5.61E+00	1.15E-05	3.40E-08	2.43E-06
BUTYLBENZYLPHTHALATE	-5.07E+00	-7.60E+00	-6.02E+00	8.44E-06	2.48E-08	9.56E-07
CHLOROBENZENE	-4.03E+00	-6.56E+00	-5.24E+00	9.35E-05	2.75E-07	5.71E-06
CHRYSENE	-2.07E+00	-5.82E+00	-3.97E+00	8.47E-03	1.50E-06	1.07E-04
DELTA-BHC	-5.10E+00	-6.48E+00	-6.13E+00	8.03E-06	3.28E-07	7.46E-07
DIBENZ(A,H)ANTHRACENE	-3.29E+00	-7.04E+00	-5.06E+00	5.11E-04	9.07E-08	8.73E-06
DI-N-OCTYLPHTHALATE	-4.84E+00	-7.38E+00	-5.60E+00	1.43E-05	4.21E-08	2.49E-06
DIBENZOFURAN	-3.89E+00	-6.42E+00	-4.91E+00	1.30E-04	3.82E-07	1.22E-05
DIELDRIN	-3.45E+00	-5.88E+00	-4.59E+00	3.55E-04	1.32E-06	2.56E-05
DISULFOTON	-4.34E+00	-7.47E+00	-5.39E+00	4.58E-05	3.37E-08	4.10E-06
ENDOSULFAN I	-3.87E+00	-6.86E+00	-5.00E+00	1.35E-04	1.39E-07	9.89E-06
ENDOSULFAN SULFATE	-2.84E+00	-5.83E+00	-3.92E+00	1.44E-03	1.48E-06	1.21E-04
ENDRIN	-2.75E+00	-6.90E+00	-3.43E+00	1.79E-03	1.27E-07	3.76E-04
ENDRIN ALDEHYDE	-2.25E+00	-6.40E+00	-2.93E+00	5.63E-03	3.98E-07	1.18E-03
ETHYL PARATHION	-3.89E+00	-6.42E+00	-4.99E+00	1.30E-04	3.81E-07	1.03E-05
FLUORANTHENE	-3.57E+00	-7.32E+00	-5.52E+00	2.72E-04	4.84E-08	3.02E-06
FLUORENE	-2.33E+00	-6.08E+00	-4.56E+00	4.73E-03	8.40E-07	2.76E-05
GAMMA-BHC	-5.38E+00	-8.14E+00	-6.49E+00	4.15E-06	7.17E-09	3.24E-07
GAMMA-CHLORDANE	-5.24E+00	-6.99E+00	-5.92E+00	5.72E-06	1.03E-07	1.19E-06

Table 6-30
Toxicity Quotients and Cumulative Effect Totals
Warwick Area

Chemical Name	TQ Deer Mouse	TQ Hawk	TQ Raccoon	Cumulative Effect Totals		
				TQ Antilog Deer Mouse	TQ Antilog Hawk	TQ Antilog Raccoon
HEPTACHLOR EPOXIDE	-2.96E+00	-4.96E+00	-4.19E+00	1.09E-03	1.09E-05	6.49E-05
INDENO(1,2,3-CD)PYRENE	-2.55E+00	-6.30E+00	-4.25E+00	2.84E-03	5.04E-07	5.60E-05
ISODRIN	-4.11E+00	-6.64E+00	-4.90E+00	7.79E-05	2.29E-07	1.26E-05
M&P-XYLENE	-5.59E+00	-8.12E+00	-6.77E+00	2.58E-06	7.58E-09	1.70E-07
METHOXYCHLOR	-1.60E+00	-5.13E+00	-2.59E+00	2.51E-02	7.39E-06	2.58E-03
METHYLENE CHLORIDE	-3.98E+00	-6.51E+00	-5.25E+00	1.04E-04	3.06E-07	5.59E-06
NAPHTHALENE	-2.09E+00	-5.84E+00	-4.47E+00	8.16E-03	1.45E-06	3.40E-05
NITROBENZENE	-2.41E+00	-4.94E+00	-3.67E+00	3.91E-03	1.15E-05	2.16E-04
O-XYLENE	-5.52E+00	-8.06E+00	-6.73E+00	2.99E-06	8.79E-09	1.86E-07
ORTHOPHOSPHATE		#VALUE!				
PCB-1248	-4.88E+00	-6.49E+00	-5.35E+00	1.31E-05	3.24E-07	4.46E-06
PCB-1254	-4.31E+00	-6.87E+00	-4.78E+00	4.89E-05	1.33E-07	1.66E-05
Total PCB's	-3.77E+00	-6.33E+00	-4.24E+00			
PHENANTHRENE	-2.91E+00	-6.66E+00	-5.08E+00	1.22E-03	2.17E-07	8.28E-06
PHENOL	-2.19E+00	-4.34E+00	-3.46E+00	6.49E-03	4.56E-05	3.51E-04
PYRENE	-3.11E+00	-6.86E+00	-5.10E+00	7.76E-04	1.38E-07	7.95E-06
SAFROLE	-3.08E+00	-5.61E+00	-4.32E+00	8.29E-04	2.44E-06	4.84E-05
TETRACHLOROETHENE	-4.82E+00	-7.35E+00	-6.05E+00	1.51E-05	4.46E-08	8.83E-07
TINUVIN 327	-4.73E+00	-7.26E+00	-5.34E+00	1.87E-05	5.50E-08	4.60E-06
TOLUENE	-4.56E+00	-7.09E+00	-5.78E+00	2.78E-05	8.17E-08	1.66E-06
TRICHLOROETHENE	-4.84E+00	-7.37E+00	-6.08E+00	1.46E-05	4.29E-08	8.35E-07
	Cumulative Effect Totals			2.62E-01	9.78E-04	1.59E-02

[a]Compound is log normally distributed. Concentration is the geometric mean.



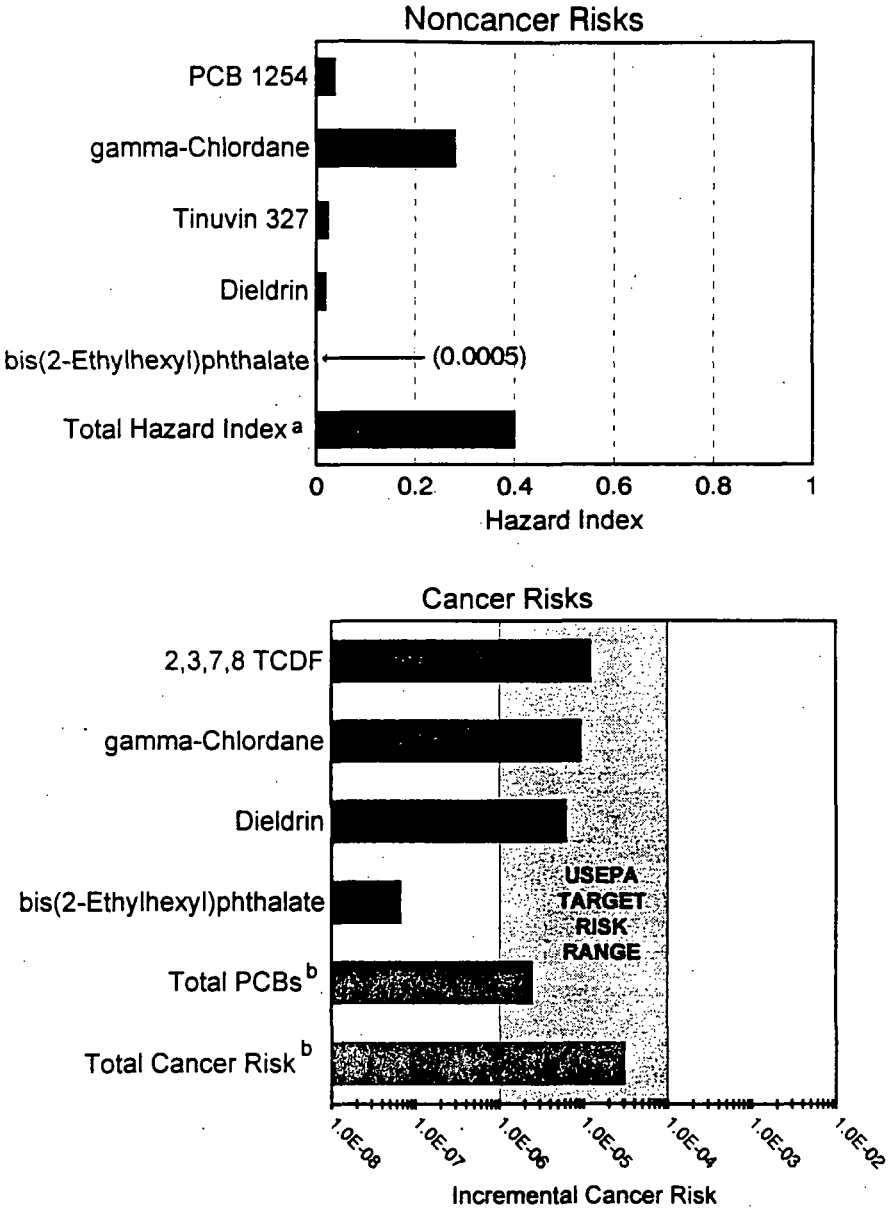


a. All hazards are summed regardless of target organ. Refer to Section 6.4.3.2 and Appendix 6-G.

b. According to USEPA policy, all PCBs were totaled, even though only PCB 1260 is carcinogenic.

c. Rounded to one significant figure, as described in the Human Health Evaluation Manual (USEPA, 1989), making total risk slightly lower than that for total PCBs.

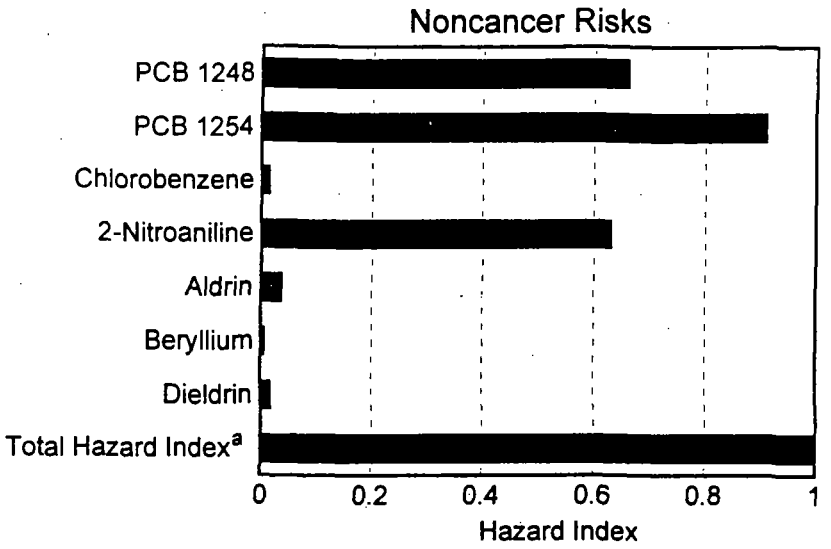
Figure 6-1. Risk Summary for Production Area On-Site Worker Scenario

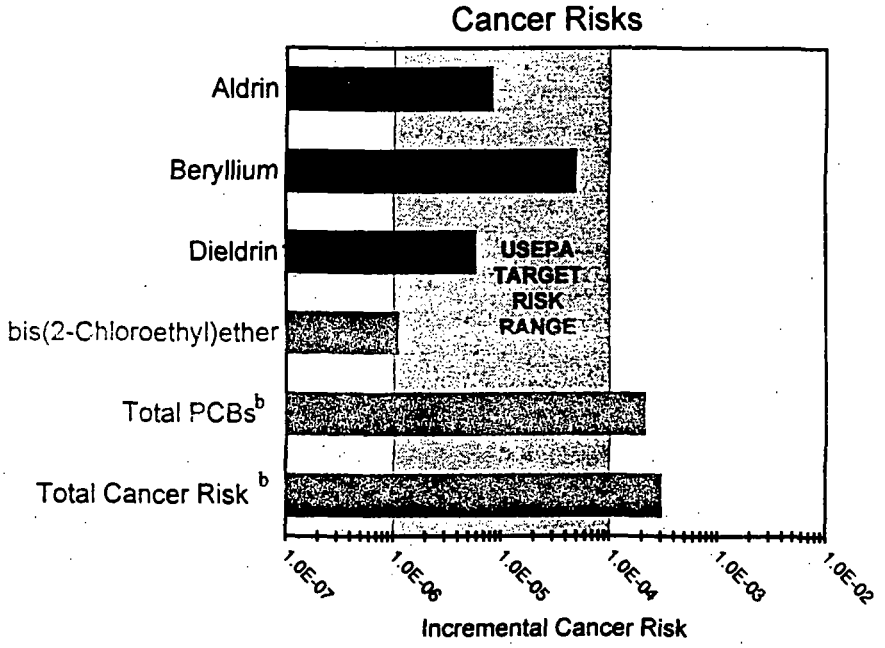


a. All hazards are summed regardless of target organ. Refer to Section 6.4.3.2 and Appendix 6-G.

b. According to USEPA policy, all PCBs were totaled, even though only PCB 1260 is carcinogenic and no PCB 1260 was detected.

Figure 6-2. Risk Summary for Waste Water Treatment Area On-Site Resident Scenario

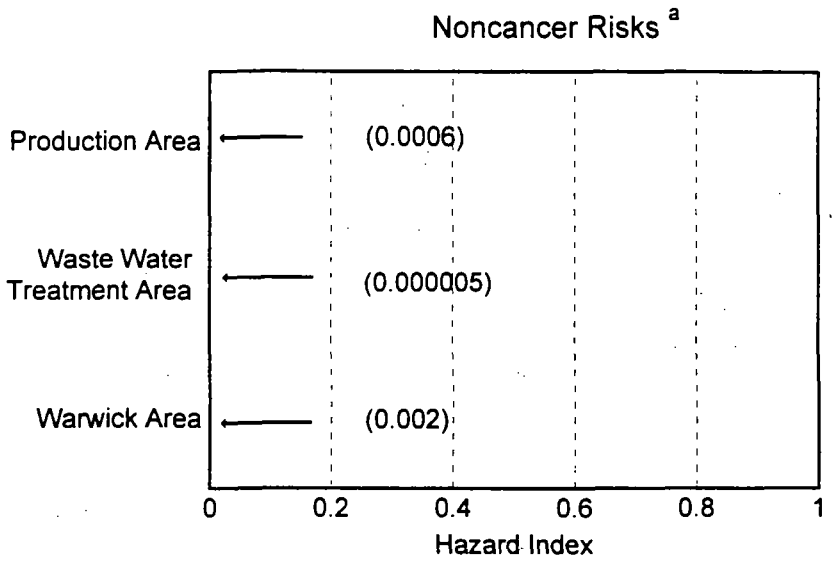


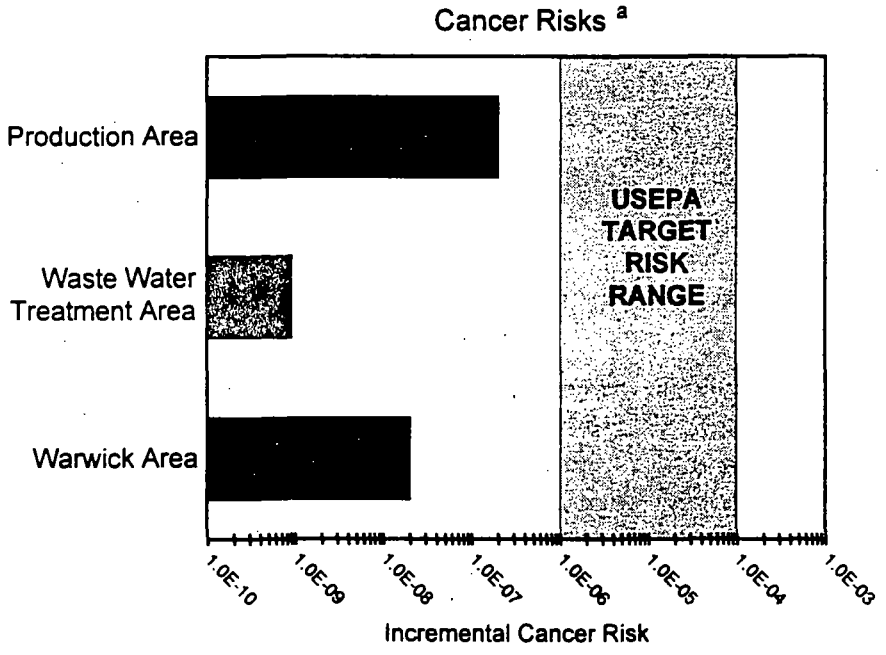


a. Only similar hazards are summed. Refer to Section 6.4.3.2 and Appendix 6-G.

b. According to USEPA policy, all PCBs were totaled, even though only PCB 1260 is carcinogenic and no PCB 1260 was detected.

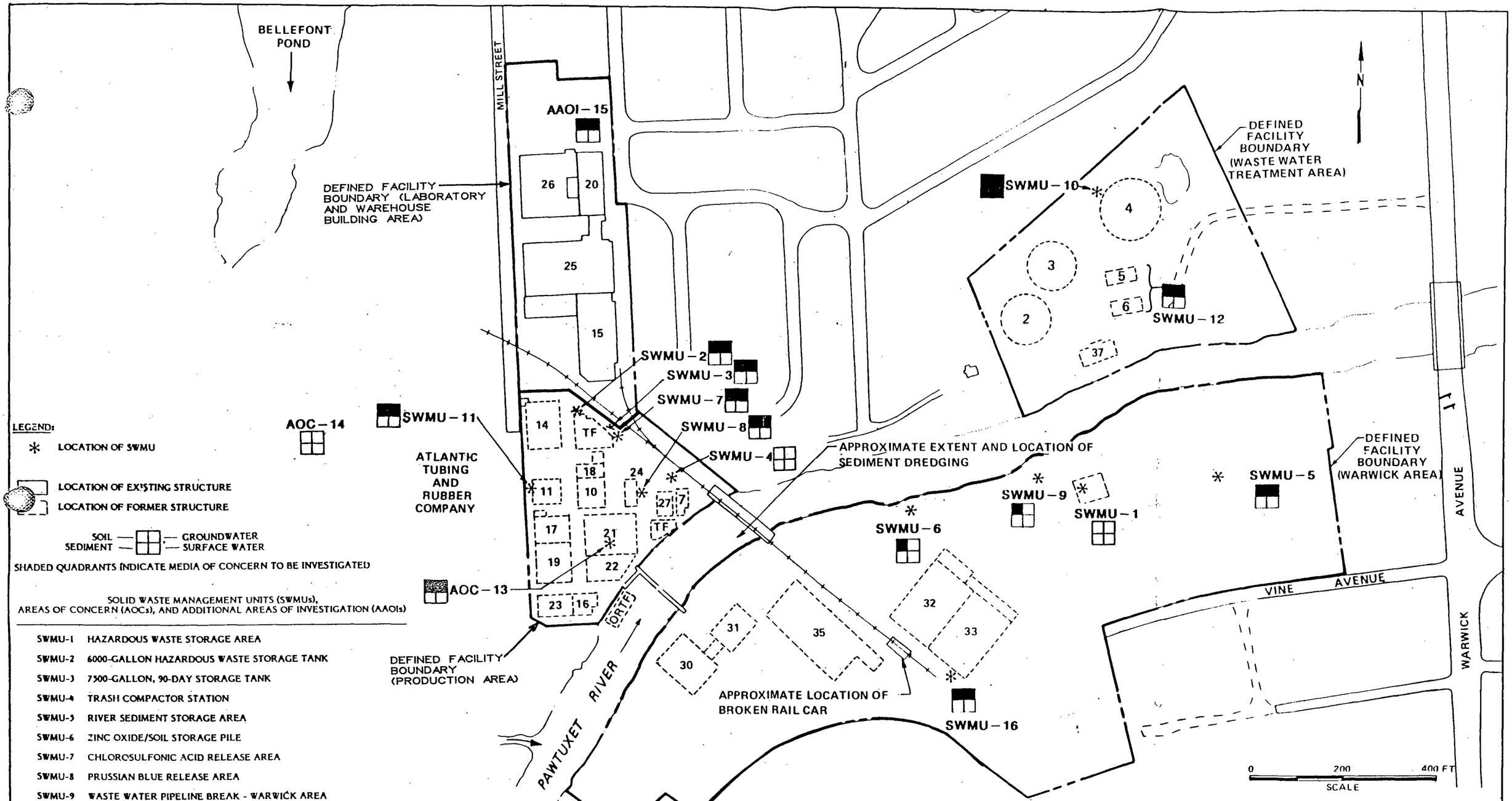
Figure 6-3. Risk Summary for Warwick Area On-Site Resident Scenario





a. Same COPCs evaluated as for other scenarios associated with Site areas. Values assume additivity for the effects of all COPCs.

Figure 6-4. Risk Summary for Canoeist Scenario



LEGEND:

* LOCATION OF SWMU

LOCATION OF EXISTING STRUCTURE

LOCATION OF FORMER STRUCTURE

SOIL — GROUNDWATER
SEDIMENT — SURFACE WATER

SHADED QUADRANTS INDICATE MEDIA OF CONCERN TO BE INVESTIGATED

SOLID WASTE MANAGEMENT UNITS (SWMUs), AREAS OF CONCERN (AOCs), AND ADDITIONAL AREAS OF INVESTIGATION (AAOIs)

SWMU-1	HAZARDOUS WASTE STORAGE AREA
SWMU-2	6000-GALLON HAZARDOUS WASTE STORAGE TANK
SWMU-3	7500-GALLON, 90-DAY STORAGE TANK
SWMU-4	TRASH COMPACTOR STATION
SWMU-5	RIVER SEDIMENT STORAGE AREA
SWMU-6	ZINC OXIDE/SOIL STORAGE PILE
SWMU-7	CHLOROSULFONIC ACID RELEASE AREA
SWMU-8	PRUSSIAN BLUE RELEASE AREA
SWMU-9	WASTE WATER PIPELINE BREAK - WARWICK AREA
SWMU-10	WASTE WATER PIPELINE BREAK - WASTE WATER TREATMENT AREA
SWMU-11	TOLUENE RELEASE AREA
SWMU-12	WASTE WATER TREATMENT PLANT
AOC-13	PROCESS BUILDING AREA
AOC-14	ATLANTIC TUBING AND RUBBER COMPANY PROPERTY
AAOI-15	LABORATORY BUILDING WASTE WATER SUMP
SWMU-16	MAINTENANCE DEPARTMENT CLEANING AREA

NOTES:

1. STRUCTURE LEGEND IS INCLUDED IN THE BCBA FACILITY INVESTIGATION PROPOSAL, CHAPTER 1, FIGURE 3 – 1.
2. CIBA – GEIGY HAS IDENTIFIED ONE ADDITIONAL AREA OF INVESTIGATION (AAOI). THIS AAOI HAS BEEN DESIGNATED AS AAOI – 15.

BASE MAP SOURCE: GEOD CORPORATION OF NEWFOUNDLAND, NEW JERSEY, MAPPED BY PHOTOGRAMMETRIC METHODS FROM AERIAL PHOTOGRAPHS. DATE FLOWN: 2 APRIL 1989

MODIFIED BY: ptrl environmental services

Project Number: 1.003.03
Date: July 25, 1995

Figure 6-5
Four Site Subdivisions/Areas and Locations of SWMUs, AOCs, and AAOIs

WOODWARD – CLYDE CONSULTANTS
CONSULTING ENGINEERS, GEOLOGISTS AND ENVIRONMENTAL SCIENTISTS
WAYNE, NEW JERSEY

DR. BY	KF	SCALE	1:2400	PROJ NO	87X4660
CK'D BY	MH	DATE	20 JAN 1992	FIG NO	1-1

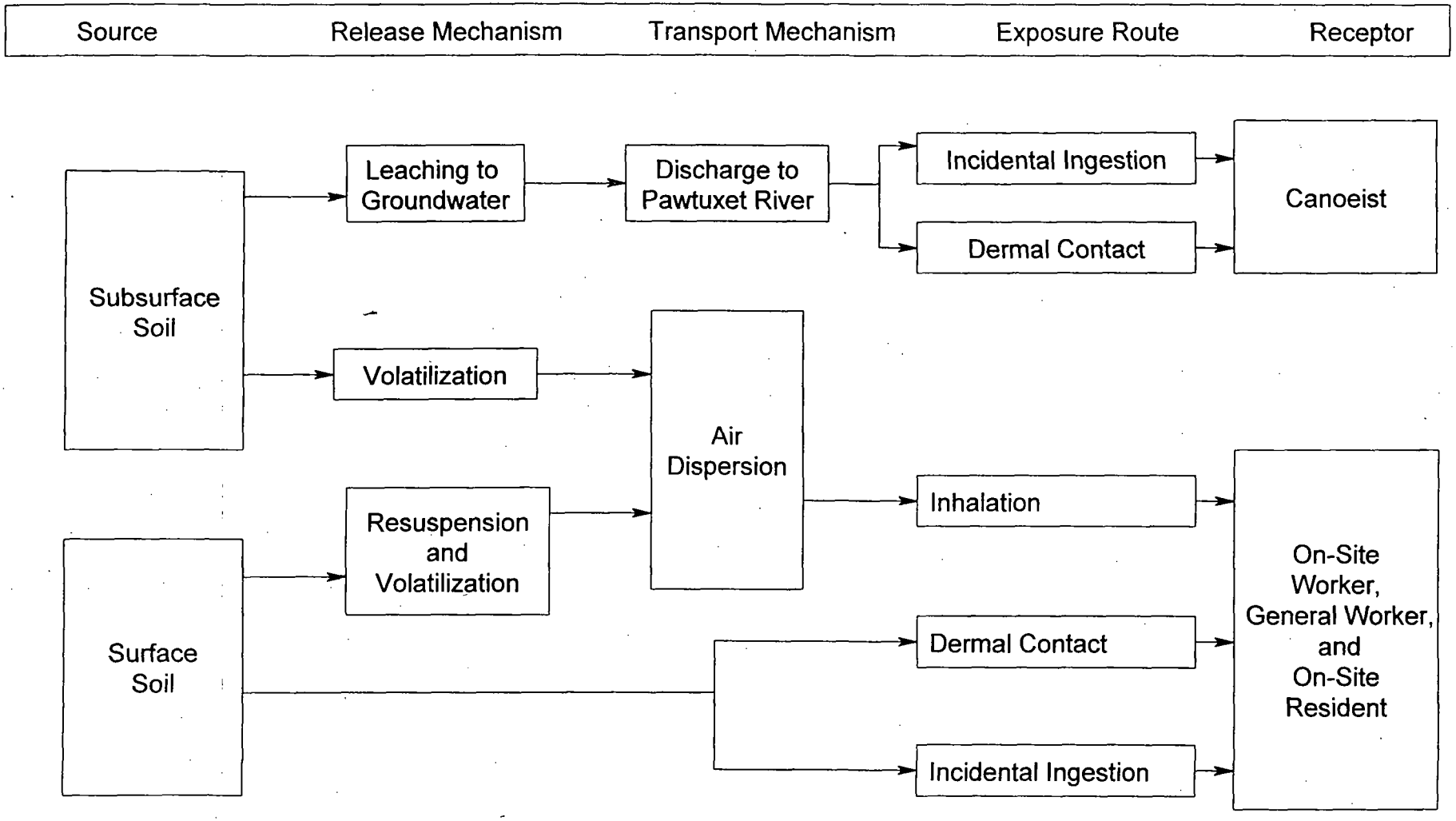


Figure 6-6
Exposure Assessment Schematic

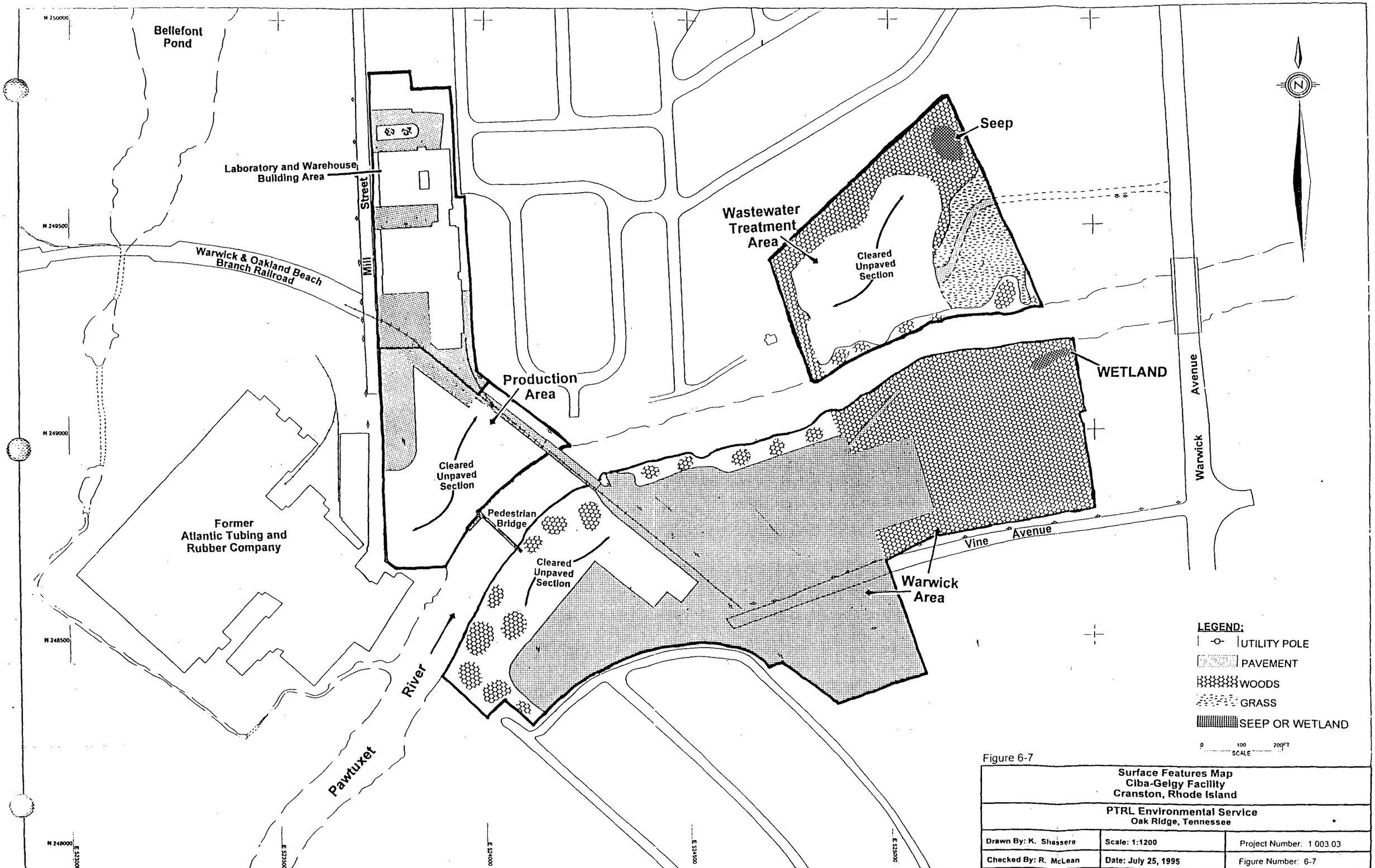


Figure 6-7

Surface Features Map Ciba-Geigy Facility Cranston, Rhode Island		
PTRL Environmental Service Oak Ridge, Tennessee		
Drawn By: K. Shassere	Scale: 1:1200	Project Number: 1 003.03
Checked By: R. McLean	Date: July 25, 1995	Figure Number: 6-7

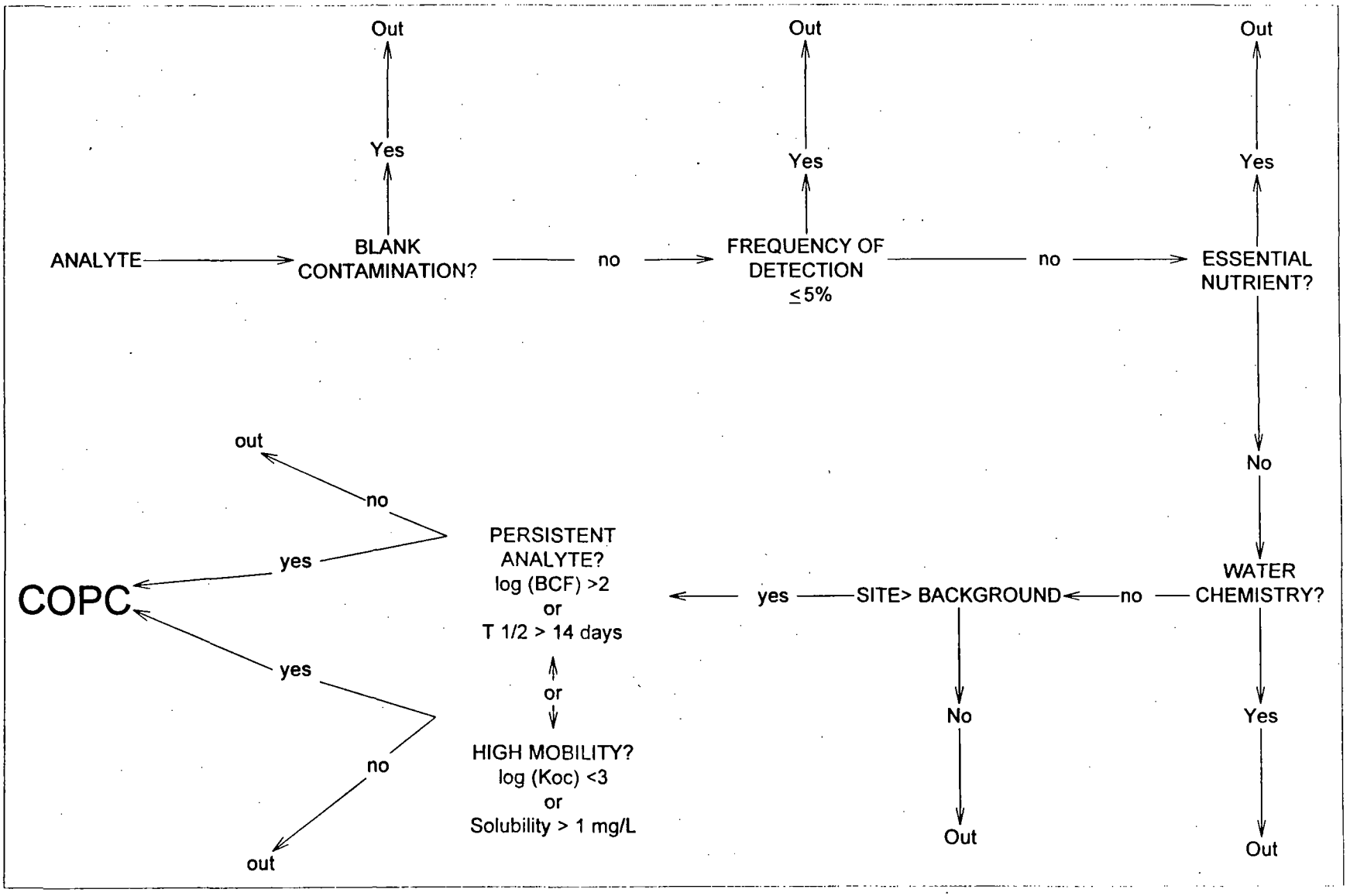


Figure 6-8. Chemicals of Potential Concern Selection Process Flowchart

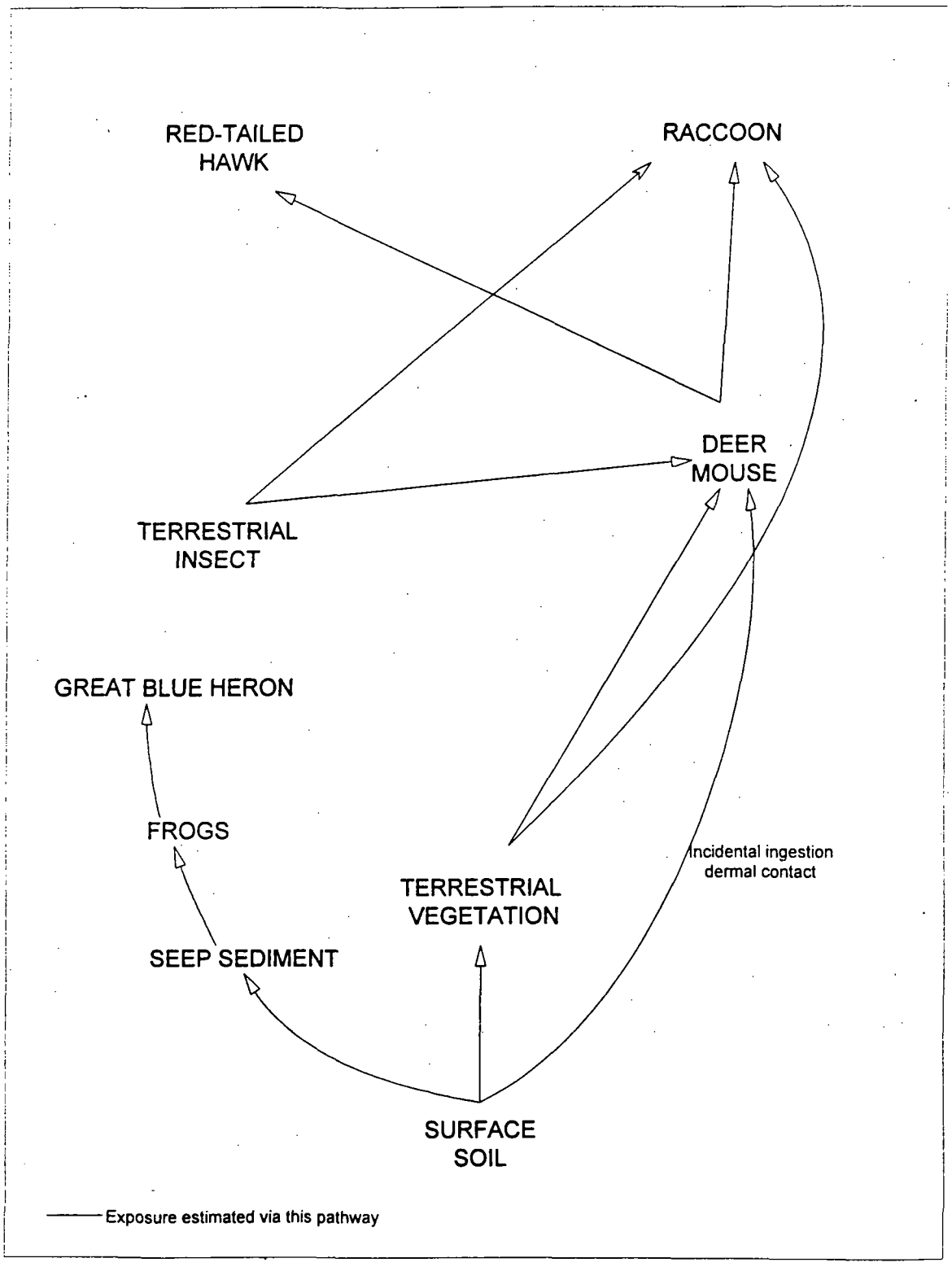


Figure 6-9. Cranston Facility Terrestrial and Semi-Terrestrial Food Web Used to Estimate Potential Dose

Appendix 6-A

Analytical Summary and Statistical Analyses of Detected Chemicals

Table 6-A-1
Analytical Summary of Detected Compounds
Production Area
Surface Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
1,2-DICHLOROBENZENE	2	41	4.9	1.254	1.832	1.736	0.120	0.120	0.120
1,4-DICHLOROBENZENE	1	41	2.4	1.268	1.825	1.748	0.240	0.240	0.240
2,4,5-TP (SILVEX)	1	28	3.6	0.009	0.001	0.0093	0.0060	0.0060	0.0060
2,4-DIMETHYLPHENOL	1	41	2.4	1.274	1.822	1.753	0.110	0.110	0.110
2-METHYLNAPHTHALENE	4	41	9.8	1.263	1.828	1.744	0.380	0.038	0.380
2-NITROANILINE (d)	8	41	19.5	6.249	9.061	8.632	0.890	0.044	0.890
3&4-METHYLPHENOL	5	16	31.3	0.179	0.166	0.252	0.770	0.023	0.252
4,4'-DDD	1	43	2.3	0.040	0.107	0.067	0.003	0.003	0.003
4-CHLOROANILINE	5	41	12.2	1.324	1.794	1.796	0.640	0.045	0.640
4-METHYLPHENOL	1	25	4.0	1.970	2.058	2.674	0.240	0.240	0.240
ACENAPHTHENE	10	41	24.4	1.235	1.843	1.720	0.210	0.057	0.210
ACENAPHTHYLENE	5	41	12.2	1.257	1.831	1.739	0.180	0.043	0.180
ACETOPHENONE	1	41	2.4	1.272	1.823	1.752	0.048	0.048	0.048
ALDRIN	1	43	2.3	0.030	0.061	0.0459	0.0035	0.0035	0.0035
ALPHA-BHC	2	43	4.7	0.030	0.061	0.0460	0.0099	0.0016	0.0099
ALPHA-CHLORDANE	2	43	4.7	0.030	0.061	0.0461	0.0097	0.0025	0.0097
AMMONIA AS N	9	25	36.0	0.761	1.059	1.123	5.300	0.290	1.123
ANILINE	1	41	2.4	1.277	1.820	1.755	0.230	0.230	0.230
ANTHRACENE	24	41	58.5	0.881	1.564	1.292	1.600	0.034	1.292
ARSENIC	29	32	90.6	9.011	21.409	15.434	125.000	0.520	15.434
BARIUM	31	31	100.0	46.535	31.698	56.197	106.000	4.600	56.197
BENZO(A)ANTHRACENE	28	41	68.3	1.144	1.413	1.516	3.100	0.150	1.516
BENZO(A)PYRENE	27	41	65.9	1.292	1.524	1.693	3.100	0.024	1.693
BENZO(B)FLUORANTHENE	30	41	73.2	1.568	1.562	1.979	4.300	0.027	1.979
BENZO(G,H,I)PERYLENE	21	41	51.2	1.351	1.745	1.810	2.900	0.130	1.810
BENZO(K)FLUORANTHENE	27	41	65.9	1.504	1.693	1.949	5.500	0.074	1.949
BERYLLIUM	30	31	96.8	0.400	0.176	0.454	0.730	0.090	0.454
BICARBONATE ALKALINITY	20	25	80.0	1382.000	1679.016	1956.559	7200.000	150.000	1956.559
BIS(2-CHLOROETHYL)ETHER	1	41	2.4	1.279	1.820	1.757	0.680	0.680	0.680

Table 6-A-1
Analytical Summary of Detected Compounds
Production Area
Surface Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
BIS(2-ETHYLHEXYL)PHTHALATE	16	41	39.0	1.094	1.490	1.486	4.100	0.061	1.486
BUTAZOLIDIN	1	25	4.0	9.978	10.017	13.406	5.200	5.200	5.200
BUTYLBENZYLPHTHALATE	13	41	31.7	1.834	5.285	3.224	33.000	0.042	3.224
CADMIUM	18	31	58.1	0.648	0.742	0.874	3.900	0.280	0.874
CALCIUM	26	26	100.0	20713.962	19653.293	27297.153	58500.00	207.000	27297.153
CARBONATE ALKALINITY	14	25	56.0	1248.680	1800.492	1864.809	7300.000	392.000	1864.809
CATION EXCHANGE CAPACITY	25	25	100.0	6.252	3.034	7.290	13.000	1.200	7.290
CHLORIDE	11	25	44.0	35.320	39.613	48.876	140.000	10.000	48.876
CHLOROBENZENE (d)	7	40	17.5	0.203	1.022	0.477	0.280	0.032	0.280
CHLOROFORM	1	40	2.5	0.196	1.023	0.470	0.034	0.034	0.034
CHROMIUM	29	31	93.5	11.130	8.312	13.664	30.700	0.900	13.664
CHRYSENE	28	41	68.3	1.243	1.417	1.616	3.300	0.150	1.616
COBALT	29	31	93.5	3.039	1.307	3.437	6.000	0.380	3.437
COPPER	29	31	93.5	17.618	16.708	30.53[e]	76.100	3.700	30.530
CYANIDE	11	34	32.4	1.323	2.261	1.981	12.600	0.560	1.981
DELTA-BHC	1	43	2.3	0.030	0.061	0.0459	0.0024	0.0024	0.0024
DI-N-BUTYLPHTHALATE	8	41	19.5	0.999	1.640	1.430	1.300	0.045	1.300
DIBENZ(A,H)ANTHRACENE	10	41	24.4	1.232	1.845	1.717	0.680	0.046	0.680
DIBENZOFURAN	12	41	29.3	1.200	1.862	1.689	0.130	0.035	0.130
DIMETHYLPHTHALATE	1	41	2.4	1.213	1.811	1.689	0.250	0.250	0.250
DINOSEB	3	40	7.5	0.065	0.079	0.086	0.009	0.002	0.009
ENDRIN ALDEHYDE	1	43	2.3	0.059	0.115	0.088	0.001	0.001	0.001
ETHYLBENZENE	10	40	25.0	1.287	7.900	3.407	50.000	0.006	3.407
FAMPHUR	2	42	4.8	0.147	0.040	0.158	0.016	0.006	0.016
FLUORANTHENE	33	41	80.5	1.599	1.734	2.055	8.400	0.051	2.055
FLUORENE	12	41	29.3	1.207	1.858	1.695	0.180	0.048	0.180
GAMMA-BHC	2	43	4.7	0.030	0.061	0.046	0.003	0.003	0.003
GAMMA-CHLORDANE (d)	7	43	16.3	0.066	0.257	0.132	1.700	0.008	0.132
HEPTACHLOR	1	43	2.3	0.030	0.061	0.0456	0.0140	0.0140	0.0140
INDENO(1,2,3-CD)PYRENE	21	41	51.2	1.325	1.761	1.789	2.300	0.045	1.789

Table 6-A-1
Analytical Summary of Detected Compounds
Production Area
Surface Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
IRGASAN DP-300	3	26	11.5	9.417	10.135	12.812	4.200	0.570	4.200
IRON	26	26	100.0	10472.308	4028.158	11821.605	21300.00	3390.000	11821.605
KEPONE	1	43	2.3	0.127	0.567	0.272	0.015	0.015	0.015
LEAD	29	31	93.5	54.308	82.536	79.464	378.000	3.600	79.464
M&P-XYLENE	27	40	67.5	10.082	63.233	27.048	400.000	0.006	27.048
MAGNESIUM	26	26	100.0	2114.923	1270.301	2540.431	5360.000	158.000	2540.431
MANGANESE	26	26	100.0	166.935	66.563	189.231	359.000	42.900	189.231
MERCURY	22	30	73.3	0.460	0.485	0.610	1.600	0.110	0.610
METHOXYCHLOR	7	43	16.3	0.354	0.783	0.555	3.600	0.120	0.555
METHYL PARATHION	2	42	4.8	0.009	0.001	0.0089	0.0065	0.0056	0.0065
METHYLENE CHLORIDE	6	40	15.0	0.269	1.033	0.546	0.010	0.005	0.010
NAPHTHALENE	14	41	34.1	0.830	1.654	1.265	0.680	0.033	0.680
NICKEL	28	31	90.3	7.774	5.722	11.62[e]	26.600	1.500	11.620
NITRATE-NITRITE AS N	22	25	88.0	1.938	2.020	2.629	7.900	0.160	2.629
NITROBENZENE	3	41	7.3	1.253	1.833	1.735	0.140	0.081	0.140
O-XYLENE	19	40	47.5	3.034	18.968	8.124	120.000	0.009	8.124
OCDD	1	5	20.0	0.001	0.000	0.0006	0.0005	0.0005	0.0005
ORTHOPHOSPHATE	20	25	80.0	5.155	8.709	8.135	43.000	0.710	8.135
PCB-1248 (d)	39	101	38.6	51.910	449.300	126.620	4500.000	0.020	126.620
PCB-1254 (d)	94	104	90.4	15.800	27.510	20.310	84.000	0.043	20.310
PCB-1260 (d)	7	83	8.4	2.940	21.930	6.960	6.100	0.130	6.1
PERCENT MOISTURE	20	20	100.0	8.300	4.777	10.147	21.000	0.000	10.147
PH	12	12	100.0	8.458	1.481	9.226	12.000	6.400	9.226
PHENANTHRENE	28	41	68.3	1.147	1.490	1.539	5.000	0.093	1.539
PHENOL	1	41	2.4	1.277	1.820	1.756	0.630	0.630	0.630
POTASSIUM	25	26	96.2	841.865	292.108	939.712	1260.000	389.000	939.712
PYRENE	32	41	78.0	1.819	1.838	2.303	6.700	0.061	2.303
SODIUM	13	26	50.0	150.623	78.024	176.759	329.000	90.200	176.759
STYRENE	2	40	5.0	0.196	1.023	0.470	0.049	0.039	0.049
SULFATE	14	25	56.0	111.900	119.742	152.876	400.000	29.000	152.876

Table 6-A-1
Analytical Summary of Detected Compounds
Production Area
Surface Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
SULFIDE	5	25	20.0	22.540	12.973	26.979	66.000	14.000	26.979
SULFOTEPP	1	42	2.4	0.021	0.011	0.0233	0.0094	0.0094	0.0094
TCDF	3	5	60.0	0.000	0.000	0.0002	0.0002	0.0001	0.0002
TETRACHLOROETHENE	1	40	2.5	0.197	1.022	0.471	0.069	0.069	0.069
THALLIUM	1	29	3.4	0.198	0.053	0.214	0.260	0.260	0.214
TIN	1	26	3.8	5.185	4.214	6.596	25.600	25.600	6.596
TINUVIN 327	1	24	4.2	9.269	9.570	12.617	5.200	5.200	5.200
TINUVIN 328	3	3	100.0	4.533	1.721	7.435	5.900	2.600	5.900
TOLUENE (d)	18	40	45.0	0.393	1.198	0.714	4.600	0.007	0.714
TOTAL ALKALINITY	24	25	96.0	2744.720	2633.962	3646.062	10000.00	150.000	3646.062
TOTAL ORGANIC CARBON	25	25	100.0	2137.600	2596.859	3026.245	9200.000	0.000	3026.245
TRCDF	2	5	40.0	0.226	0.331	0.541	0.730	0.400	0.541
TRICHLOROFLUOROMETHANE	3	40	7.5	0.413	2.124	0.983	0.330	0.071	0.330
VANADIUM	28	31	90.3	14.981	18.947	20.756	108.000	1.400	20.756
ZINC	31	31	100.0	183.606	184.509	367.87[e]	759.000	13.000	367.870

a. Arithmetic mean concentration, unless otherwise indicated.
b. 95th percentile upper confidence limit of the arithmetic mean concentration of an assumed normal distribution, unless otherwise indicated.
c. Lesser of the maximum concentration and the 95th percentile UCL of the mean concentration.
d. Shading indicates that the chemical was selected as a chemical of potential concern.
e. 95th Percentile UCL of the arithmetic mean concentration of an assumed lognormal distribution.

Table 6-A-2
Analytical Summary of Detected Compounds
Production Area
Combined Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG) [a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) [b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG) [c]
1,2,4-TRICHLOROBENZENE	3	84	3.6	0.862	1.454	1.127	0.640	0.190	0.640
1,2-DICHLOROBENZENE	3	84	3.6	0.857	1.456	1.122	0.760	0.120	0.760
1,4-DICHLOROBENZENE	1	84	1.2	0.860	1.454	1.125	0.240	0.240	0.240
1,4-DIOXANE	1	84	1.2	2.790	2.615	3.267	4.000	4.000	3.267
2,4,5-T	2	48	4.2	0.011	0.001	0.011	0.017	0.006	0.011
2,4,5-TP (SILVEX)	2	48	4.2	0.009	0.001	0.009	0.007	0.006	0.007
2,4-D	6	48	12.5	0.059	0.018	0.064	0.110	0.008	0.064
2,4-DICHLOROPHENOL	4	84	4.8	0.971	1.548	1.253	6.200	2.600	1.253
2,4-DIMETHYLPHENOL	7	84	8.3	0.885	1.457	1.150	1.700	0.080	1.150
2,6-DICHLOROPHENOL	1	84	1.2	0.891	1.468	1.159	2.800	2.800	1.159
2-BUTANONE	4	80	5.0	1.414	6.601	2.647	0.300	0.130	0.300
2-METHYLNAPHTHALENE	5	84	6.0	0.857	1.456	1.122	0.380	0.038	0.380
2-METHYLPHENOL	1	84	1.2	0.863	1.454	1.128	0.084	0.084	0.084
2-NITROANILINE (d)	9	84	10.7	4.301	7.173	5.609	4.200	0.044	4.200
2-NITROPHENOL	1	84	1.2	0.863	1.454	1.128	0.075	0.075	0.075
3&4-METHYLPHENOL	17	42	40.5	0.282	0.343	0.372	1.200	0.023	0.372
3,3'-DICHLOROBENZIDINE	1	84	1.2	1.725	2.861	2.246	0.730	0.730	0.730
4,4'-DDD	2	86	2.3	0.026	0.080	0.041	0.230	0.003	0.041
4,4'-DDE	1	86	1.2	0.031	0.107	0.051	0.710	0.710	0.051
4,4'-DDT	2	86	2.3	0.039	0.092	0.055	0.350	0.084	0.055
4-CHLOROANILINE	8	84	9.5	0.917	1.422	1.177	0.640	0.042	0.640
4-METHYLPHENOL	4	42	9.5	1.559	1.863	2.043	3.400	0.100	2.043
ACENAPHTHENE	12	84	14.3	0.840	1.462	1.107	0.280	0.049	0.280
ACENAPHTHYLENE	5	84	6.0	0.855	1.457	1.120	0.180	0.043	0.180
ACETONE	1	80	1.3	1.409	6.602	2.642	0.053	0.053	0.053
ACETOPHENONE	4	84	4.8	0.870	1.451	1.135	0.660	0.048	0.660
ALDRIN	2	86	2.3	0.023	0.064	0.034	0.440	0.004	0.034
ALPHA-BHC	5	86	5.8	0.018	0.045	0.027	0.018	0.001	0.018
ALPHA-CHLORDANE	2	86	2.3	0.018	0.045	0.026	0.010	0.003	0.010

Table 6-A-2
Analytical Summary of Detected Compounds
Production Area
Combined Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG) [a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) [b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
AMMONIA AS N	20	42	47.6	1.548	2.230	2.127	8.500	0.170	2.127
ANILINE	5	84	6.0	0.867	1.452	1.131	0.280	0.210	0.280
ANTHRACENE	32	84	38.1	0.659	1.258	0.889	1.600	0.034	0.889
ARSENIC	52	55	94.5	7.770	16.440	11.503	125.000	0.520	11.503
BARIUM	52	52	100.0	36.973	27.889	43.486	106.000	4.600	43.486
BENZO(A)ANTHRACENE	41	84	48.8	0.797	1.192	1.015	3.100	0.140	1.015
BENZO(A)PYRENE	44	84	52.4	0.872	1.277	1.104	3.100	0.024	1.104
BENZO(B)FLUORANTHENE	52	84	61.9	1.034	1.351	1.280	4.300	0.027	1.280
BENZO(G,H,I)PERYLENE	30	84	35.7	0.910	1.415	1.168	2.900	0.086	1.168
BENZO(K)FLUORANTHENE	42	84	50.0	0.970	1.418	1.228	5.500	0.036	1.228
BERYLLIUM	49	52	94.2	0.385	0.174	0.425	0.790	0.090	0.425
BETA-BHC	1	86	1.2	0.019	0.046	0.028	0.100	0.100	0.028
BICARBONATE ALKALINITY	33	42	78.6	925.988	1408.408	1291.958	7200.000	80.000	1291.958
BIS(2-CHLOROETHYL)ETHER	2	84	2.4	0.864	1.454	1.129	0.680	0.050	0.680
BIS(2-ETHYLHEXYL)PHTHALATE	36	84	42.9	0.850	1.228	1.074	4.100	0.054	1.074
BUTAZOLIDIN	1	40	2.5	7.858	9.108	10.301	5.200	5.200	5.200
BUTYLBENZYLPHTHALATE	18	84	21.4	1.130	3.781	1.819	33.000	0.042	1.819
CADMIUM	21	54	38.9	0.473	0.598	0.610	3.900	0.240	0.610
CALCIUM	43	43	100.0	13841.372	17808.579	18414.750	58500.000	104.000	18414.750
CARBONATE ALKALINITY	20	42	47.6	904.179	1495.679	1292.826	7300.000	60.000	1292.826
CATION EXCHANGE CAPACITY	42	42	100.0	5.345	2.956	6.113	13.000	1.200	6.113
CHLORIDE	21	42	50.0	52.393	65.242	69.346	260.000	10.000	69.346
CHLOROBENZENE (d)	9	80	11.3	0.666	3.240	1.271	0.280	0.018	0.280
CHLOROBENZILATE	1	85	1.2	0.813	1.511	1.087	0.098	0.098	0.098
CHLOROFORM	1	80	1.3	0.664	3.240	1.269	0.034	0.034	0.034
CHROMIUM	52	54	96.3	10.230	6.956	11.825	30.700	0.600	11.825
CHRYSENE	43	84	51.2	0.859	1.200	1.078	3.300	0.052	1.078
COBALT	49	52	94.2	3.486	1.825	3.913	8.600	0.350	3.913
COPPER	52	54	96.3	15.518	14.381	22.960	76.100	0.810	22.960
CYANIDE	14	59	23.7	1.441	2.721	2.037	13.600	0.560	2.037

Table 6-A-2
Analytical Summary of Detected Compounds
Production Area
Combined Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG) [a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) [b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
DELTA-BHC	2	86	2.3	0.019	0.046	0.028	0.086	0.002	0.028
DI-N-BUTYLPHTHALATE	14	84	16.7	0.715	1.316	0.955	1.300	0.042	0.955
DIBENZ(A,H)ANTHRACENE	12	84	14.3	0.809	1.470	1.077	0.680	0.046	0.680
DIBENZOFURAN	14	84	16.7	0.821	1.470	1.089	0.400	0.035	0.400
DIETHYLPHTHALATE	1	84	1.2	0.806	1.360	1.054	0.670	0.670	0.670
DIMETHYLPHTHALATE	1	84	1.2	0.833	1.439	1.096	0.250	0.250	0.250
DINOSEB	5	78	6.4	0.102	0.201	0.140	0.009	0.002	0.009
DISULFOTON	1	84	1.2	0.062	0.011	0.064	0.010	0.010	0.010
ENDOSULFAN SULFATE	1	86	1.2	0.072	0.134	0.096	0.096	0.096	0.096
ENDRIN ALDEHYDE	2	86	2.3	0.035	0.086	0.051	0.002	0.001	0.002
ETHYL PARATHION	1	84	1.2	0.029	0.013	0.032	0.013	0.013	0.013
ETHYLBENZENE	24	80	30.0	1.693	6.877	2.977	50.000	0.006	2.977
FAMPHUR	2	84	2.4	0.154	0.032	0.160	0.016	0.006	0.016
FLUORANTHENE	60	84	71.4	0.989	1.363	1.237	8.400	0.043	1.237
FLUORENE	17	84	20.2	0.820	1.471	1.088	0.180	0.047	0.180
GAMMA-BHC	9	86	10.5	0.019	0.045	0.027	0.036	0.002	0.027
GAMMA-CHLORDANE (d)	13	86	15.1	0.037	0.183	0.070	1.700	0.002	0.070
HEPTACHLOR	4	86	4.7	0.018	0.045	0.026	0.042	0.004	0.026
INDENO(1,2,3-CD)PYRENE	31	84	36.9	0.874	1.426	1.134	2.300	0.045	1.134
IRGASAN DP-300	7	44	15.9	24.485	71.230	42.568	390.000	0.570	42.568
IRON	43	43	100.0	10929.884	5445.449	12328.316	29900.000	925.000	12328.316
ISODRIN	2	86	2.3	0.026	0.080	0.040	0.220	0.010	0.040
KEPONE	1	86	1.2	0.073	0.403	0.146	0.015	0.015	0.015
LEAD	50	52	96.2	37.207	66.765	52.798	378.000	3.600	52.798
M&P-XYLENE	48	80	60.0	9.443	47.247	18.270	400.000	0.006	18.270
MAGNESIUM	43	43	100.0	1973.558	1271.141	2299.997	5360.000	108.000	2299.997
MANGANESE	43	43	100.0	160.349	72.774	179.038	359.000	15.200	179.038
MERCURY	32	52	61.5	0.341	0.448	0.446	1.600	0.060	0.446
METHOXYCHLOR	12	86	14.0	0.208	0.574	0.311	3.600	0.032	0.311
METHYL PARATHION	6	84	7.1	0.0085	0.0012	0.0088	0.0070	0.0056	0.0070

Table 6-A-2
Analytical Summary of Detected Compounds
Production Area
Combined Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG) [a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) [b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
METHYLENE CHLORIDE	20	80	25.0	0.791	3.271	1.402	1.500	0.005	1.402
N-OCTANE	2	5	40.0	3.263	5.226	8.246	12.000	4.300	8.246
NAPHTHALENE	29	84	34.5	0.549	1.199	0.767	0.680	0.029	0.680
NICKEL	49	54	90.7	8.156	5.607	11.500[e]	26.600	0.630	26.600
NITRATE-NITRITE AS N	35	42	83.3	2.392	2.483	3.037	9.600	0.160	3.037
NITROBENZENE	5	84	6.0	0.873	1.462	1.140	2.100	0.081	1.140
O-XYLENE	37	80	46.3	2.609	13.854	5.198	120.000	0.009	5.198
OCDD	1	9	11.1	0.000	0.000	5.14E-04	4.60E-04	4.60E-04	4.60E-04
ORTHOPHOSPHATE	35	42	83.3	10.285	16.475	14.566	77.000	0.710	14.566
P-PHENYLENEDIAMINE	1	84	1.2	4.734	7.578	6.116	15.000	15.000	6.116
PCB-1248 (d)	39	144	27.1	36.430	376.480	88.450	4500.000	0.020	88.450
PCB-1254 (d)	127	147	86.4	11.490	24.090	14.780	84.000	0.043	14.780
PCB-1260 (d)	15	126	11.9	2.170	17.830	4.810	13.000	0.070	4.810
PERCENT MOISTURE	39	39	100.0	8.374	4.620	9.630	21.000	0.000	9.630
PH	22	22	100.0	8.114	1.746	8.754	12.000	4.700	8.754
PHENANTHRENE	49	84	58.3	0.724	1.126	0.929	5.000	0.030	0.929
PHENOL	4	84	4.8	0.857	1.456	1.123	0.630	0.150	0.630
POTASSIUM	42	43	97.7	721.244	317.799	802.857	1260.000	73.000	802.857
PYRENE	58	84	69.0	1.193	1.550	1.476	6.700	0.042	1.476
SODIUM	23	43	53.5	152.714	84.149	174.324	350.000	29.500	174.324
STYRENE	5	80	6.3	0.664	3.240	1.269	0.049	0.008	0.049
SULFATE	26	42	61.9	161.798	312.621	243.031	1800.000	17.000	243.031
SULFIDE	6	42	14.3	20.500	10.714	23.284	66.000	14.000	23.284
SULFOTEPP	1	84	1.2	0.018	0.010	0.020	0.009	0.009	0.009
TCDF	4	9	44.4	0.000	0.000	1.31E-04	1.90E-04	5.60E-05	1.31E-04
TETRACHLOROETHENE	1	80	1.3	0.664	3.240	1.269	0.069	0.069	0.069
THALLIUM	1	48	2.1	0.196	0.054	0.209	0.260	0.260	0.209
TIN	1	43	2.3	4.851	3.297	5.698	25.600	25.600	5.698
TINUVIN 327	2	40	5.0	7.187	8.635	9.504	5.200	0.490	5.200
TINUVIN 328	5	5	100.0	5.600	3.890	9.309	12.000	2.400	9.309

Table 6-A-2
Analytical Summary of Detected Compounds
Production Area
Combined Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG) [a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG) [b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
TOLUENE (d)	31	80	38.8	30.822	164.400	61.536	1200.000	0.006	61.536
TOTAL ALKALINITY	38	42	90.5	1970.369	2368.800	2585.894	10000.000	87.000	2585.894
TOTAL ORGANIC CARBON	42	42	100.0	3388.000	2132.766	3942.192	9200.000	540.000	3942.192
TRCDF	3	10	30.0	0.140	0.252	0.286	0.730	0.270	0.286
TRICHLOROFLUOROMETHANE	3	80	3.8	1.336	6.606	2.570	0.330	0.071	0.330
VANADIUM	45	52	86.5	12.357	15.089	15.881	108.000	0.880	15.881
ZINC	51	54	94.4	130.719	158.038	218.500	759.000	2.200	218.500

a. Arithmetic mean concentration, unless otherwise indicated.
b. 95th percentile upper confidence limit of the arithmetic mean concentration of an assumed normal distribution, unless otherwise indicated.
c. Lesser of the maximum concentration and the 95th percentile UCL of the mean concentration.
d. Shading indicates that the chemical was selected as a chemical of potential concern.
e. 95th Percentile UCL of the arithmetic mean concentration of an assumed lognormal distribution.

Table 6-A-3
Analytical Summary of Detected Compounds
Production Area
Groundwater

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (UG/L)[a]	STANDARD DEVIATION (UG/L)	95TH% UCL (UG/L)[b]	MAX. CONC. (UG/L)	MIN. CONC. (UG/L)	SELECTED CONC. (UG/L)[c]
1,1,1-TRICHLOROETHANE	2	50	4.0	155.7300	433.4640	258.9610	89.0000	5.0000	89.0000
1,1-BIPHENYL	5	14	35.7	3.5254	2.1257	4.5315	2.1000	0.0760	2.1000
1,1-DICHLOROETHANE	5	50	10.0	153.9880	433.9124	257.3258	4.0000	1.0000	4.0000
1,2,3,4,6,7,8-HPCDD	1	26	3.8	0.0005	0.0004	0.0007	0.0007	0.0007	0.0007
1,2,3,4,6,7,8-HPCDF	1	26	3.8	0.0003	0.0003	0.0004	0.0004	0.0004	0.0004
1,2,3,4,7,8-HXCDD	1	26	3.8	0.0005	0.0003	0.0006	0.0004	0.0004	0.0004
1,2,3,4,7,8-HXCDF	1	26	3.8	0.0002	0.0001	0.0003	0.0003	0.0003	0.0003
1,2,4-TRICHLOROBENZENE	2	50	4.0	5.6370	4.0420	6.5996	3.0000	1.0000	3.0000
1,2-DICHLOROBENZENE	14	50	28.0	17.2950	37.9601	26.3353	230.0000	3.0000	26.3353
1,3-DICHLOROBENZENE	3	50	6.0	5.5036	4.1328	6.4878	1.0000	0.8800	1.0000
1,4-DICHLOROBENZENE	7	50	14.0	4.5240	1.0769	4.7805	3.0000	1.0000	3.0000
1,4-DIOXANE	3	50	6.0	2403.8480	2519.6333	3003.9076	45.0000	2.4000	45.0000
2,3,7,8-TCDF	1	26	3.8	0.0002	0.0002	0.0003	0.0012	0.0012	0.0003
2,4,5-TP (SILVEX)	2	50	4.0	0.1274	0.0917	0.1493	0.4100	0.0670	0.1493
2,4-D	2	50	4.0	0.4310	0.2192	0.4832	1.4000	0.8000	0.4832
2,4-DICHLOROPHENOL	9	49	18.4	63.4990	204.0809	112.5950	1200.0000	1.4000	112.5950
2,4-DIMETHYLPHENOL	19	49	38.8	9.1678	9.3856	11.4257	42.0000	0.4700	11.4257
2,6-DICHLOROPHENOL	1	49	2.0	5.7724	4.0094	6.7370	5.0000	5.0000	5.0000
2-BUTANONE	1	43	2.3	1029.6744	2863.7943	1765.1185	21.0000	21.0000	21.0000
2-CHLOROPHENOL	12	49	24.5	14.3231	36.3519	23.0683	240.0000	0.3800	23.0683
2-CYCLOHEXENE-1-OL	3	14	21.4	6.9307	7.8637	10.6528	0.7400	0.5900	0.7400
2-CYCLOHEXENE-1-ONE	5	14	35.7	6.3229	8.1708	10.1903	0.8700	0.4100	0.8700
2-METHYLNAPHTHALENE	3	50	6.0	5.5020	4.1419	6.4884	1.2000	0.6000	1.2000
2-METHYLPHENOL	15	49	30.6	44.7322	116.5456	72.7698	450.0000	0.7300	72.7698
2-NITROANILINE (d)	1	50	2.0	28.3700	20.0127	33.1361	12.0000	12.0000	12.0000
3&4-METHYLPHENOL	10	26	38.5	37.3115	77.0120	63.1080	340.0000	2.2000	63.1080
4,4'-DDD	2	48	4.2	0.1504	0.2215	0.2042	0.4100	0.0250	0.2042
4,4'-DDE	2	48	4.2	0.1664	0.2296	0.2222	0.3000	0.0084	0.2222
4,4'-DDT	3	48	6.3	0.2059	0.3348	0.2872	0.0230	0.0075	0.0230

Table 6-A-3
Analytical Summary of Detected Compounds
Production Area
Groundwater

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (UG/L)[a]	STANDARD DEVIATION (UG/L)	95TH% UCL (UG/L)[b]	MAX. CONC. (UG/L)	MIN. CONC. (UG/L)	SELECTED CONC. (UG/L)[c]
4-CHLORO-3-METHYLPHENOL	8	49	16.3	7.3724	7.3782	9.1474	43.0000	2.0000	9.1474
4-CHLOROANILINE	2	50	4.0	8.7060	9.2006	10.8972	10.0000	8.0000	10.0000
4-METHYLPHENOL	13	23	56.5	68.4109	227.4394	149.8385	1100.0000	1.0000	149.8385
4-NITROPHENOL	1	50	2.0	28.2900	20.0873	33.0739	8.0000	8.0000	8.0000
ACETOPHENONE	14	50	28.0	7.4450	8.1328	9.3819	43.0000	1.0000	9.3819
ALDRIN	3	48	6.3	0.1188	0.2289	0.1744	0.0130	0.0047	0.0130
ALPHA-BHC	3	49	6.1	0.1040	0.1656	0.1439	0.1400	0.0150	0.1400
ALPHA-CHLORDANE	1	48	2.1	0.1446	0.2221	0.1986	0.0130	0.0130	0.0130
AMMONIA AS N	19	24	79.2	2.3863	4.9850	4.1303	18.0000	0.0240	4.1303
ANILINE	13	50	26.0	17.2872	24.4920	23.1201	19.0000	0.2300	19.0000
ANTIMONY	1	50	2.0	11.8780	9.0711	14.0383	13.4000	13.4000	13.4000
ARSENIC	24	50	48.0	9.7540	12.0741	12.6295	61.7000	4.1000	12.6295
BARIUM	50	50	100.0	49.4900	53.9430	62.3367	268.0000	11.2000	62.3367
BENZENE	4	50	8.0	150.3700	433.7744	253.6749	100.0000	5.0000	100.0000
BENZO(A)ANTHRACENE	2	50	4.0	5.5970	4.0784	6.5683	1.0000	1.0000	1.0000
BENZO(A)PYRENE	1	50	2.0	5.6920	4.0050	6.6458	2.0000	2.0000	2.0000
BENZO(B)FLUORANTHENE	3	50	6.0	5.5170	4.1292	6.5004	1.0000	1.0000	1.0000
BENZO(G,H,I)PERYLENE	3	50	6.0	5.6170	4.0376	6.5786	3.0000	2.0000	3.0000
BENZO(K)FLUORANTHENE	3	50	6.0	5.5370	4.1093	6.5156	2.0000	1.0000	2.0000
BENZYL ALCOHOL	8	49	16.3	12.1376	27.6945	18.8001	140.0000	0.2900	18.8001
BICARBONATE ALKALINITY	15	24	62.5	95.7708	112.4121	135.1003	351.0000	27.0000	135.1003
BIOLOGICAL OXYGEN DEMAND - 5	12	12	100.0	17166.6667	23295.8574	29244.6495	76000.0000	2000.0000	29244.6495
BIS(2-CHLOROETHOXY)METHANE	1	50	2.0	5.6620	4.0273	6.6211	1.0000	1.0000	1.0000
BIS(2-CHLOROETHYL)ETHER	1	50	2.0	5.6880	4.0089	6.6427	1.8000	1.8000	1.8000
BIS(2-ETHYLHEXYL)PHTHALATE	4	50	8.0	5.5414	7.0498	7.2203	2.0000	0.6200	2.0000
BUTAZOLIDIN	7	50	14.0	66.8320	89.2374	88.0842	19.0000	3.1000	19.0000
BUTYLBENZYLPHTHALATE	1	50	2.0	5.6640	4.0361	6.6252	0.6000	0.6000	0.6000
CADMIUM	1	50	2.0	2.6120	0.7920	2.8006	8.1000	8.1000	2.8006
CALCIUM	50	50	100.0	41338.0000	24316.9034	47129.1572	96800.0000	11700.0000	47129.1572

Table 6-A-3
Analytical Summary of Detected Compounds
Production Area
Groundwater

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (UG/L)[a]	STANDARD DEVIATION (UG/L)	95TH% UCL (UG/L)[b]	MAX. CONC. (UG/L)	MIN. CONC. (UG/L)	SELECTED CONC. (UG/L)[c]
CARBONATE ALKALINITY	1	24	4.2	2.2292	8.4712	5.1930	42.0000	42.0000	5.1930
CHEMICAL OXYGEN DEMAND	16	24	66.7	60.4042	82.9488	89.4254	350.0000	7.0000	89.4254
CHLORIDE	23	24	95.8	29.5250	18.4411	35.9770	72.0000	3.9000	35.9770
CHLOROBENZENE (d)	25	50	50.0	1528.1800	4316.5653	2556.1854	21000.0000	1.0000	2556.1854
CHLOROFORM	1	50	2.0	154.0680	433.8846	257.3992	8.4000	8.4000	8.4000
CHRYSENE	1	50	2.0	5.6710	4.0326	6.6314	0.7000	0.7000	0.7000
COBALT	2	50	4.0	5.4240	2.1536	5.9369	18.0000	13.2000	5.9369
DELTA-BHC	3	49	6.1	0.1280	0.2378	0.1852	1.3000	0.0140	0.1852
DI-N-BUTYLPHTHALATE	6	50	12.0	5.2892	4.2741	6.3071	2.4000	0.3800	2.4000
DIBENZ(A,H)ANTHRACENE	1	50	2.0	5.7120	3.9887	6.6619	3.0000	3.0000	3.0000
DIELDRIN (d)	1	47	2.1	0.1541	0.2262	0.2096	0.0120	0.0120	0.0120
DIETHYLPHTHALATE	6	50	12.0	5.3004	4.2654	6.3162	1.5000	0.7200	1.5000
DIMETHOATE	3	50	6.0	2.0994	1.9336	2.5599	0.1900	0.1200	0.1900
DINOSEB	3	49	6.1	7.6052	10.2232	10.0646	0.6500	0.0660	0.6500
ENDOSULFAN II	3	48	6.3	0.2715	0.4837	0.3890	0.0069	0.0016	0.0069
ENDOSULFAN SULFATE	1	48	2.1	0.3982	0.7850	0.5890	0.0084	0.0084	0.0084
ENDRIN	1	49	2.0	0.1412	0.2167	0.1934	0.0860	0.0860	0.0860
ENDRIN ALDEHYDE	3	48	6.3	0.2485	0.5144	0.3736	3.0000	0.0110	0.3736
ETHYL PARATHION	1	50	2.0	0.3917	0.0887	0.4128	0.3700	0.3700	0.3700
ETHYLBENZENE	15	50	30.0	244.6020	460.3855	354.2445	1100.0000	8.6000	354.2445
FAMPHUR	2	50	4.0	1.0944	0.3333	1.1738	0.2700	0.1000	0.2700
FLUORANTHENE	9	50	18.0	5.3944	4.4162	6.4461	9.2000	0.1500	6.4461
GAMMA-BHC	2	48	4.2	0.1313	0.2514	0.1924	1.4000	0.4900	0.1924
GAMMA-CHLORDANE (d)	4	48	8.3	0.1962	0.3926	0.2916	2.4000	0.0160	0.2916
HEPTACHLOR	1	48	2.1	0.1062	0.1675	0.1470	0.1900	0.1900	0.1470
HPCDD	1	26	3.8	0.0005	0.0004	0.0007	0.0014	0.0014	0.0007
HPCDF	1	26	3.8	0.0005	0.0005	0.0007	0.0004	0.0004	0.0004
HXCDD	1	26	3.8	0.0005	0.0003	0.0006	0.0004	0.0004	0.0004
HXCDF	1	26	3.8	0.0003	0.0002	0.0003	0.0006	0.0006	0.0003

Table 6-A-3
Analytical Summary of Detected Compounds
Production Area
Groundwater

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (UG/L)[a]	STANDARD DEVIATION (UG/L)	95TH% UCL (UG/L)[b]	MAX. CONC. (UG/L)	MIN. CONC. (UG/L)	SELECTED CONC. (UG/L)[c]
HYDROCARBONS	10	24	41.7	2.1000	4.9112	3.8183	24.0000	0.6400	3.8183
INDENO(1,2,3-CD)PYRENE	2	50	4.0	5.6520	4.0220	6.6099	3.0000	2.0000	3.0000
IODOMETHANE	1	49	2.0	157.6020	437.8243	262.9301	630.0000	630.0000	262.9301
IRGASAN DP-300	12	50	24.0	147.6680	425.0012	248.8835	2300.0000	2.6000	248.8835
IRON	45	50	90.0	6955.8400	7692.7141	8787.8873	27000.0000	21.0000	8787.8873
ISODRIN	2	48	4.2	0.0850	0.1532	0.1222	0.0950	0.0100	0.0950
LANGLIER INDEX	26	26	100.0	-1.2800	1.2619	-0.8573	3.2000	-3.3000	-0.8573
LEAD	4	50	8.0	3.7260	7.1091	5.4191	51.9000	4.4000	5.4191
M&P-XYLENE	23	50	46.0	522.5260	1217.3515	812.4426	7800.0000	4.6000	812.4426
MAGNESIUM	50	50	100.0	4977.2000	2588.2466	5593.6002	19000.0000	1900.0000	5593.6002
MANGANESE	49	50	98.0	31916.2410	211862.0047	82371.9325	1500000.000	10.0000	82371.9325
METHYL PARATHION	1	50	2.0	0.1774	0.1217	0.2064	0.2200	0.2200	0.2064
NAPHTHALENE	21	50	42.0	14.4440	43.6811	24.8468	240.0000	0.6500	24.8468
NICKEL	3	50	6.0	16.4860	7.7241	18.3255	45.2000	25.0000	18.3255
NITRATE-NITRITE AS N	7	24	29.2	261.3333	800.8462	541.5244	3600.0000	24.0000	541.5244
O-TOLUIDINE	1	50	2.0	5.6620	4.0273	6.6211	1.0000	1.0000	1.0000
O-XYLENE	18	50	36.0	259.8680	618.4357	407.1507	3300.0000	2.1000	407.1507
OCDD	11	26	42.3	0.0015	0.0017	0.0021	0.0077	0.0003	0.0021
OCDF	1	26	3.8	0.0007	0.0005	0.0008	0.0003	0.0003	0.0003
OIL AND GREASE	11	24	45.8	2.7056	4.5975	4.3142	17.0000	0.5200	4.3142
ORTHOPHOSPHATE	9	24	37.5	0.1806	0.4129	0.3251	1.6000	0.0100	0.3251
PCB-1260 (d)	2	48	4.2	2.8892	5.8657	4.3149	30.0000	22.0000	4.3149
PECDF	1	26	3.8	0.0003	0.0002	0.0004	0.0001	0.0001	0.0001
PENTACHLOROPHENOL	1	48	2.1	28.3333	20.6146	33.3440	3.0000	3.0000	3.0000
PHENANTHRENE	4	50	8.0	5.4296	4.1852	6.4263	1.0000	0.7800	1.0000
PHENOL	15	50	30.0	32.1430	84.6859	52.3113	410.0000	4.0000	52.3113
PHORATE	1	50	2.0	0.3946	0.1117	0.4212	0.0640	0.0640	0.0640
POTASSIUM	46	50	92.0	7393.0000	7306.6627	9133.1078	37700.0000	1700.0000	9133.1078
PYRENE	10	50	20.0	5.1883	4.4139	6.2395	8.1000	0.0950	6.2395

Table 6-A-3
Analytical Summary of Detected Compounds
Production Area
Groundwater

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (UG/L)[a]	STANDARD DEVIATION (UG/L)	95TH% UCL (UG/L)[b]	MAX. CONC. (UG/L)	MIN. CONC. (UG/L)	SELECTED CONC. (UG/L)[c]
SILICA,DISSOLVED	36	36	100.0	21.0306	11.3609	24.2438	70.0000	3.4000	24.2438
SILICA,TOTAL	14	14	100.0	2839.6429	10407.7537	7765.8373	39000.0000	12.0000	7765.8373
SILVER	1	50	2.0	5.4200	2.9698	6.1273	26.0000	26.0000	6.1273
SODIUM	50	50	100.0	47540.0000	42310.9384	57616.5008	170000.0000	15000.0000	57616.5008
SULFATE	24	24	100.0	50.2583	51.5252	68.2854	220.0000	4.8000	68.2854
SULFIDE	3	24	12.5	1.1458	1.8641	1.7980	7.7000	3.3000	1.7980
SULFOTEPP	3	50	6.0	0.2665	0.0961	0.2894	0.8200	0.0570	0.2894
TCDF	3	26	11.5	0.0023	0.0074	0.0048	0.0157	0.0001	0.0048
TETRACHLOROETHENE	2	50	4.0	154.5100	433.7451	257.8079	31.0000	2.0000	31.0000
THIONAZIN	1	50	2.0	1.3592	1.1334	1.6291	0.0580	0.0580	0.0580
TIN	1	50	2.0	37.6420	12.8090	40.6925	57.1000	57.1000	40.6925
TINUVIN 327	3	50	6.0	24.3224	16.2448	28.1912	3.0000	0.2200	3.0000
TKN	18	24	75.0	2.8546	4.9920	4.6011	18.0000	0.1000	4.6011
TOLUENE (d)	21	50	42.0	8225.6080	24508.0379	14062.2846	150000.0000	1.6000	14062.2846
TOTAL ALKALINITY	50	50	100.0	148.9600	103.3462	173.5723	420.0000	6.0000	173.5723
TOTAL DISSOLVED SOLIDS	24	24	100.0	345.4167	174.5209	406.4761	840.0000	140.0000	406.4761
TOTAL HARDNESS, AS CACO3	50	50	100.0	35517.2280	70640.7368	52340.5712	270000.0000	5.4000	52340.5712
TOTAL ORGANIC CARBON	21	24	87.5	22.0583	32.1251	33.2979	140.0000	1.2000	33.2979
TOTAL ORGANIC HALIDES	23	24	95.8	0.2387	0.1584	0.2941	0.6000	0.0330	0.2941
TOTAL SUSPENDED SOLIDS	24	24	100.0	650.6667	1384.5511	1135.0779	6800.0000	40.0000	1135.0779
TRANS-1,2-DICHLOROETHENE	2	49	4.1	154.8980	437.6550	260.1853	140.0000	130.0000	140.0000
TRICHLOROETHENE	2	50	4.0	153.9820	433.9145	257.3203	4.0000	2.6000	4.0000
VINYL CHLORIDE (d)	4	49	8.2	322.1327	877.7398	533.2918	600.0000	2.2000	533.2918
ZINC	3	50	6.0	12.5570	5.7934	13.9367	38.6000	23.0000	13.9367

a. Arithmetic mean concentration, unless otherwise indicated.
b. 95th percentile upper confidence limit of the arithmetic mean concentration of an assumed normal distribution, unless otherwise indicated.
c. Lesser of the maximum concentration and the 95th percentile UCL of the mean concentration.
d. Shading indicates that the chemical was selected as a chemical of potential concern.

Table 6-A-4
Analytical Summary of Detected Compounds
Waste Water Treatment Area
Surface Soil

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX CONC. (MG/KG)	MIN CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
1,2,4-TRICHLOROBENZENE	1	18	5.6	0.791	1.2729	1.3134	0.0340	0.0340	0.034
2,3,7,8-TCDF (d)	2	6	33.3	0.000	0.0005	0.0006	0.0012	0.0001	6.2E-04
2,4,5-T	4	15	26.7	0.017	0.0121	0.0227	0.0460	0.0310	0.023
2,4-D	1	15	6.7	0.050	0.0324	0.0648	0.1200	0.1200	0.065
2,4-DICHLOROPHENOL	9	18	50.0	0.644	0.6482	0.9094	2.1000	0.1300	0.909
2-METHYLNAPHTHALENE	3	18	16.7	0.444	0.4561	0.6315	0.5000	0.2600	0.500
2-METHYLPHENOL	1	18	5.6	0.594	0.5682	0.8269	1.8000	1.8000	0.827
2-NITROANILINE	2	18	11.1	3.885	6.2719	6.4570	0.0730	0.0530	0.073
3&4-METHYLPHENOL	2	9	22.2	0.203	0.0786	0.2514	0.3800	0.0840	0.251
4,4'-DDD	4	18	22.2	0.127	0.2205	0.2173	0.3200	0.0960	0.217
4,4'-DDE	3	18	16.7	0.107	0.2146	0.1954	0.2600	0.0120	0.195
4,4'-DDT	1	18	5.6	0.095	0.2093	0.1806	0.0075	0.0075	0.008
4-CHLOROANILINE	4	18	22.2	1.337	1.6226	2.0024	5.6000	1.2000	2.002
4-METHYLPHENOL	2	9	22.2	1.270	1.6876	2.3163	0.2900	0.2900	0.290
ACENAPHTHENE	1	18	5.6	0.774	1.2674	1.2943	0.6000	0.6000	0.600
ACENAPHTHYLENE	1	18	5.6	0.792	1.2725	1.3138	0.0450	0.0450	0.045
ACETONE	4	18	22.2	0.094	0.0841	0.1290	0.2000	0.0320	0.129
ACETOPHENONE	2	18	11.1	0.782	1.2781	1.3064	0.0260	0.0230	0.026
ALPHA-BHC	2	18	11.1	0.048	0.1082	0.0926	0.0021	0.0012	0.002
AMMONIA AS N	7	10	70.0	14.185	19.8150	25.6707	51.0000	1.3000	25.671
ANILINE	4	18	22.2	0.893	1.2588	1.4088	1.6000	0.4200	1.409
ANTHRACENE	2	18	11.1	0.726	1.2373	1.2336	1.5000	0.3800	1.234
ARSENIC	15	15	100.0	5.918	3.9460	7.7122	11.7000	0.8500	7.712
BARIUM	15	15	100.0	28.807	16.4521	36.2873	75.2000	13.2000	36.287
BENZO(A)ANTHRACENE	6	18	33.3	0.861	1.4404	1.4516	3.9000	0.1600	1.452
BENZO(A)PYRENE	5	18	27.8	0.865	1.4618	1.4642	3.6000	0.0460	1.464
BENZO(B)FLUORANTHENE	11	18	61.1	1.039	1.4467	1.6323	3.2000	0.0810	1.632
BENZO(G,H,I)PERYLENE	3	18	16.7	0.898	1.3272	1.4421	2.5000	0.3000	1.442
BENZO(K)FLUORANTHENE	8	18	44.4	0.948	1.4622	1.5477	3.8000	0.1600	1.548

Table 6-A-4
Analytical Summary of Detected Compounds
Waste Water Treatment Area
Surface Soil

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX CONC. (MG/KG)	MIN CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
BERYLLIUM	14	15	93.3	0.434	0.1905	0.5209	0.7700	0.1500	0.521
BETA-BHC	1	18	5.6	0.049	0.1079	0.0932	0.0160	0.0160	0.016
BICARBONATE ALKALINITY	6	10	60.0	400.000	450.9908	661.4148	1128.0000	180.0000	661.415
BIS(2-ETHYLHEXYL)PHTHALATE (d)	5	18	27.8	1.281	1.9259	2.0712	7.2000	0.1200	2.071
BUTYLBENZYLPHTHALATE	1	18	5.6	0.777	1.2763	1.3007	0.1500	0.1500	0.150
CADMIUM	1	9	11.1	0.271	0.1767	0.3806	0.7400	0.7400	0.381
CALCIUM	9	9	100.0	8202.000	8052.5757	13194.5970	19600.0000	738.0000	13194.597
CARBONATE ALKALINITY	6	10	60.0	372.600	404.2076	606.8971	1000.0000	265.0000	606.897
CATION EXCHANGE CAPACITY	10	10	100.0	4.480	1.5978	5.4061	6.8000	2.0000	5.406
CHLORIDE	7	10	70.0	176.050	230.4204	309.6122	600.0000	11.0000	309.612
CHLOROBENZENE	10	18	55.6	1.621	4.1835	3.3366	13.0000	0.0150	3.337
CHROMIUM	15	15	100.0	10.020	4.8654	12.2322	20.5000	4.1000	12.232
CHRYSENE	9	18	50.0	0.860	1.4735	1.4640	4.1000	0.0990	1.464
COBALT	15	15	100.0	3.433	0.9759	3.8771	5.0000	1.9000	3.877
COPPER	18	18	100.0	37.228	37.4873	75.13[e]	128.0000	3.7000	75.130
CYANIDE	4	14	28.6	0.958	0.7186	1.2976	1.7000	0.3700	1.298
DELTA-BHC	1	18	5.6	0.049	0.1079	0.0935	0.0260	0.0260	0.026
DI-N-BUTYLPHTHALATE	1	18	5.6	0.795	1.2705	1.3164	0.1000	0.1000	0.100
DI-N-OCTYLPHTHALATE	1	18	5.6	0.778	1.2761	1.3011	0.1600	0.1600	0.160
DIBENZ(A,H)ANTHRACENE	1	18	5.6	1.386	3.9283	2.9969	17.0000	17.0000	2.997
DIBENZOFURAN	2	18	11.1	0.519	0.4862	0.7183	1.1000	0.4000	0.718
DIELDRIN (d)	4	18	22.2	0.104	0.2109	0.1905	0.1700	0.0077	0.170
DIETHYLPHTHALATE	4	18	22.2	0.539	0.5782	0.7762	1.7000	0.0750	0.776
DIMETHOATE	1	18	5.6	0.140	0.0863	0.1754	0.0260	0.0260	0.026
ENDRIN ALDEHYDE	3	18	16.7	0.153	0.2714	0.2640	0.8200	0.2300	0.264
ETHYL PARATHION	2	17	11.8	0.036	0.0245	0.0461	0.0540	0.0025	0.046
ETHYLBENZENE	4	18	22.2	0.276	0.6713	0.5511	2.1000	0.0240	0.551
FAMPHUR	3	17	17.6	0.169	0.0791	0.2028	0.0770	0.0140	0.077
FLUORANTHENE	12	18	66.7	0.812	1.5994	1.4679	6.9000	0.0460	1.468

Table 6-A-4
Analytical Summary of Detected Compounds
Waste Water Treatment Area
Surface Soil

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX CONC. (MG/KG)	MIN CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
FLUORENE	2	18	11.1	0.766	1.2704	1.2866	0.5100	0.1100	0.510
GAMMA-CHLORDANE (d)	7	21	33.3	1.611	4.1990	3.1917	19.0000	0.0790	3.192
HEPTACHLOR	2	18	11.1	0.053	0.1093	0.0983	0.1200	0.0045	0.098
INDENO(1,2,3-CD)PYRENE	4	18	22.2	0.852	1.3197	1.3932	2.1000	0.1200	1.393
IRGASAN DP-300	6	10	60.0	51.820	103.0482	111.5514	340.0000	5.6000	111.551
IRON	9	9	100.0	12126.667	2992.5616	13982.0549	15800.0000	8370.0000	13982.055
LEAD	15	15	100.0	36.580	27.1986	48.9469	96.7000	2.4000	48.947
M&P-XYLENE	10	18	55.6	1.018	2.6279	2.0956	8.2000	0.0150	2.096
MAGNESIUM	9	9	100.0	1403.000	344.3007	1616.4665	1960.0000	879.0000	1616.466
MANGANESE	9	9	100.0	199.511	119.2701	273.4586	488.0000	73.6000	273.459
MERCURY	6	10	60.0	0.148	0.1554	0.2376	0.4700	0.0600	0.238
METHYL PARATHION	2	17	11.8	0.012	0.0064	0.0143	0.0320	0.0038	0.014
METHYLENE CHLORIDE	7	18	38.9	0.139	0.1708	0.2087	0.0110	0.0072	0.011
NAPHTHALENE	9	18	50.0	0.568	0.7257	0.8659	2.5000	0.0660	0.866
NICKEL	14	15	93.3	7.633	3.4739	10.15[e]	13.2000	3.3000	10.150
NITRATE-NITRITE AS N	5	10	50.0	1.625	2.0757	2.8282	6.8000	1.1000	2.828
O-XYLENE	8	18	44.4	0.393	0.9892	0.7992	3.1000	0.0074	0.799
ORTHOPHOSPHATE	10	10	100.0	31.041	52.5518	61.5024	140.0000	0.9100	61.502
PCB-1254 (d)	1	18	5.6	0.955	2.0897	1.8122	0.2100	0.2100	0.210
PERCENT MOISTURE	12	12	100.0	7.942	1.4488	8.6928	10.0000	5.0000	8.693
PH	5	5	100.0	8.720	1.8404	10.4747	10.3000	6.2000	10.300
PHENANTHRENE	14	18	77.8	0.815	1.3287	1.3601	5.1000	0.0490	1.360
PHENOL	3	18	16.7	0.563	0.5779	0.7998	1.8000	0.2700	0.800
PHORATE	2	17	11.8	0.030	0.0165	0.0367	0.0170	0.0170	0.017
POTASSIUM	9	9	100.0	762.556	239.0513	910.7674	1100.0000	384.0000	910.767
PROPAZINE	1	10	10.0	6.041	8.1834	10.7840	28.0000	28.0000	10.784
PYRENE	16	18	88.9	1.499	2.2050	2.4034	6.8000	0.0390	2.403
SELENIUM	1	15	6.7	0.169	0.0604	0.1961	0.1300	0.1300	0.130
SILVER	8	18	44.4	0.964	1.0643	1.4009	4.0000	0.6600	1.401

Table 6-A-4
Analytical Summary of Detected Compounds
Waste Water Treatment Area
Surface Soil

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX CONC. (MG/KG)	MIN CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
SODIUM	5	9	55.6	167.444	99.0257	228.8404	303.0000	148.0000	228.840
STYRENE	1	18	5.6	0.039	0.0428	0.0562	0.0190	0.0190	0.019
SULFATE	8	10	80.0	164.050	101.7656	223.0380	260.0000	59.0000	223.038
SULFIDE	2	10	20.0	30.650	33.1403	49.8596	93.0000	93.0000	49.860
SULFOTEPP	5	17	29.4	0.027	0.0327	0.0411	0.1400	0.0040	0.041
TCDF	2	6	33.3	0.002	0.0036	0.0045	0.0089	0.0001	0.004
TETRACHLOROETHENE	1	18	5.6	0.038	0.0432	0.0557	0.0064	0.0064	0.006
TINUVIN 327 (d)	2	9	22.2	5.978	8.0944	10.9963	26.0000	0.5000	10.996
TOLUENE	11	18	61.1	1.159	2.7698	2.2950	8.7000	0.0140	2.295
TOTAL ALKALINITY	9	10	90.0	972.500	639.5473	1343.2107	1800.0000	180.0000	1343.211
TOTAL ORGANIC CARBON	10	10	100.0	5600.000	3534.1194	7648.5364	9100.0000	1700.0000	7648.536
VANADIUM	15	15	100.0	10.080	3.1684	11.5206	15.0000	4.7000	11.521
ZINC	15	15	100.0	286.793	305.9296	1553.55[e]	840.0000	15.2000	840.000

a. Arithmetic mean concentration, unless otherwise indicated.
b. 95th percentile upper confidence limit of the arithmetic mean concentration of an assumed normal distribution, unless otherwise indicated.
c. Lesser of the maximum concentration and the 95th percentile UCL of the mean concentration.
d. Shading indicates that the chemical was selected as a chemical of potential concern.
e. 95th Percentile UCL of the arithmetic mean concentration of an assumed lognormal distribution.

Table 6-A-5
Analytical Summary of Detected Compounds
Waste Water Treatment Area
Combined Soil

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH UCL CONC. (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
1,2,4-TRICHLOROBENZENE	1	31	3.2	0.654	1.0349	0.9697	0.034	0.034	0.034
2,3,7,8-TCDF (d)	3	7	42.9	2.12E-04	4.37E-04	5.33E-04	0.001	0.000	5.33E-04
2,4,5-T	4	23	17.4	0.015	0.0102	0.0185	0.046	0.031	0.018
2,4-D	3	23	13.0	0.057	0.0291	0.0672	0.120	0.055	0.067
2,4-DICHLOROPHENOL	15	31	48.4	0.563	0.6154	0.7501	2.100	0.049	0.750
2-METHYLNAPHTHALENE	3	31	9.7	0.453	0.4939	0.6034	0.500	0.260	0.500
2-METHYLPHENOL	1	31	3.2	0.540	0.5596	0.7102	1.800	1.800	0.710
2-NITROANILINE	2	31	6.5	3.254	5.1124	4.8123	0.073	0.053	0.073
3&4-METHYLPHENOL	3	16	18.8	0.188	0.0615	0.2150	0.380	0.084	0.215
4,4'-DDD	4	31	12.9	0.092	0.1765	0.1453	0.320	0.096	0.145
4,4'-DDE	3	31	9.7	0.080	0.1702	0.1321	0.260	0.012	0.132
4,4'-DDT	2	31	6.5	0.074	0.1649	0.1239	0.024	0.008	0.024
4-CHLOROANILINE	11	31	35.5	1.019	1.3294	1.4246	5.600	0.053	1.425
4-METHYLPHENOL	2	15	13.3	1.082	1.3655	1.7029	0.290	0.290	0.290
ACENAPHTHENE	2	31	6.5	0.641	1.0318	0.9551	0.600	0.054	0.600
ACENAPHTHYLENE	3	31	9.7	0.648	1.0382	0.9643	0.086	0.045	0.086
ACETONE	9	31	29.0	0.103	0.0735	0.1255	0.210	0.032	0.126
ACETOPHENONE	3	31	9.7	0.644	1.0404	0.9613	0.026	0.023	0.026
ALDRIN	1	31	3.2	0.037	0.0853	0.0632	0.001	0.001	0.001
ALPHA-BHC	2	31	6.5	0.037	0.0853	0.0632	0.002	0.001	0.002
ALPHA-CHLORDANE	1	31	3.2	0.037	0.0853	0.0634	0.013	0.013	0.013
AMMONIA AS N	10	16	62.5	10.350	16.3342	17.5085	51.000	1.300	17.508
ANILINE	9	31	29.0	0.715	1.0343	1.0306	1.600	0.066	1.031
ANTHRACENE	6	31	19.4	0.615	1.0073	0.9217	1.500	0.051	0.922
ARSENIC	23	23	100.0	5.220	3.3385	6.4157	11.700	0.850	6.416
BARIUM	23	23	100.0	25.622	14.4028	30.7782	75.200	12.400	30.778
BENZO(A)ANTHRACENE	8	31	25.8	0.709	1.1561	1.0617	3.900	0.160	1.062
BENZO(A)PYRENE	8	31	25.8	0.711	1.1708	1.0676	3.600	0.046	1.068
BENZO(B)FLUORANTHENE	18	31	58.1	0.832	1.1686	1.1885	3.200	0.081	1.188

Table 6-A-5
Analytical Summary of Detected Compounds
Waste Water Treatment Area
Combined Soil

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH UCL CONC. (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
BENZO(G,H,I)PERYLENE	5	31	16.1	0.713	1.0837	1.0437	2.500	0.130	1.044
BENZO(K)FLUORANTHENE	14	31	45.2	0.739	1.1855	1.1008	3.800	0.055	1.101
BERYLLIUM	20	23	87.0	0.473	0.2375	0.5581	0.810	0.150	0.558
BETA-BHC	1	31	3.2	0.038	0.0852	0.0636	0.016	0.016	0.016
BICARBONATE ALKALINITY	9	16	56.3	274.750	388.3079	444.9259	1128.000	55.000	444.926
BIS(2-ETHYLHEXYL)PHTHALATE (d)	11	31	35.5	0.940	1.5475	1.4116	7.200	0.077	1.412
BUTYLBENZYLPHTHALATE	2	31	6.5	0.642	1.0384	0.9584	0.150	0.050	0.150
CADMIUM	1	15	6.7	0.243	0.1390	0.3062	0.740	0.740	0.306
CALCIUM	15	15	100.0	5397.867	7059.7264	8607.8413	19600.000	733.000	8607.841
CARBONATE ALKALINITY	7	16	43.8	296.688	393.0669	468.9491	1000.000	265.000	468.949
CATION EXCHANGE CAPACITY	16	16	100.0	3.481	1.8371	4.2864	6.800	1.000	4.286
CHLORIDE	9	16	56.3	121.563	194.4598	206.7845	600.000	6.000	206.784
CHLOROBENZENE	11	31	35.5	0.956	3.2481	1.9462	13.000	0.015	1.946
CHROMIUM	23	23	100.0	8.922	4.2432	10.4409	20.500	4.100	10.441
CHRYSENE	14	31	45.2	0.709	1.1777	1.0679	4.100	0.078	1.068
COBALT	23	23	100.0	3.557	0.8826	3.8725	5.000	1.900	3.872
COPPER	31	31	100.0	28.258	31.4756	37.51[e]	128.000	3.700	37.852
CYANIDE	6	18	33.3	0.918	0.6765	1.1958	1.700	0.370	1.196
DELTA-BHC	3	31	9.7	0.039	0.0848	0.0648	0.026	0.019	0.026
DI-N-BUTYLPHTHALATE	4	31	12.9	0.649	1.0367	0.9647	0.410	0.076	0.410
DI-N-OCTYLPHTHALATE	1	31	3.2	0.646	1.0360	0.9622	0.160	0.160	0.160
DIBENZ(A,H)ANTHRACENE	3	31	9.7	0.982	3.0192	1.9019	17.000	0.064	1.902
DIBENZOFURAN	3	31	9.7	0.494	0.5124	0.6497	1.100	0.094	0.650
DIELDRIN (d)	6	31	19.4	0.079	0.1670	0.1296	0.170	0.001	0.130
DIETHYLPHTHALATE	4	31	12.9	0.508	0.5628	0.6794	1.700	0.075	0.679
DIMETHOATE	1	31	3.2	0.134	0.0763	0.1568	0.026	0.026	0.026
ENDOSULFAN I	1	31	3.2	0.037	0.0853	0.0634	0.013	0.013	0.013
ENDRIN	1	31	3.2	0.068	0.1658	0.1181	0.019	0.019	0.019
ENDRIN ALDEHYDE	5	31	16.1	0.138	0.2680	0.2200	1.000	0.079	0.220

Table 6-A-5
Analytical Summary of Detected Compounds
Waste Water Treatment Area
Combined Soil

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH UCL CONC. (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
ETHYL PARATHION	3	30	10.0	0.032	0.0229	0.0396	0.054	0.002	0.040
ETHYLBENZENE	4	31	12.9	0.171	0.5208	0.3301	2.100	0.024	0.330
FAMPHUR	4	30	13.3	0.164	0.0710	0.1865	0.077	0.007	0.077
FLUORANTHENE	15	31	48.4	0.728	1.2766	1.1174	6.900	0.046	1.117
FLUORENE	4	31	12.9	0.632	1.0348	0.9477	0.510	0.045	0.510
GAMMA-BHC	3	31	9.7	0.037	0.0853	0.0633	0.004	0.001	0.004
GAMMA-CHLORDANE (d)	14	34	41.2	1.228	3.3420	2.2004	19.000	0.079	2.200
HEPTACHLOR	3	31	9.7	0.040	0.0865	0.0667	0.120	0.005	0.067
INDENO(1,2,3-CD)PYRENE	10	31	32.3	0.686	1.0752	1.0140	2.100	0.049	1.014
IRGASAN DP-300	8	18	44.4	34.983	78.7586	67.2840	340.000	5.600	67.284
IRON	15	15	100.0	12089.333	2393.2658	13177.5231	15800.000	8370.000	13177.523
KEPONE	1	31	3.2	0.323	0.8634	0.5860	0.005	0.005	0.005
LEAD	23	23	100.0	28.557	25.1356	37.5555	96.700	2.400	37.556
M&P-XYLENE	11	31	35.5	0.610	2.0379	1.2311	8.200	0.015	1.231
MAGNESIUM	15	15	100.0	1429.800	309.0890	1570.3391	1990.000	879.000	1570.339
MANGANESE	15	15	100.0	186.640	91.9548	228.4508	488.000	73.600	228.451
MERCURY	7	17	41.2	0.107	0.1329	0.1636	0.470	0.060	0.164
METHYL PARATHION	3	30	10.0	0.010	0.0050	0.0118	0.032	0.004	0.012
METHYLENE CHLORIDE	12	31	38.7	0.101	0.1402	0.1437	0.011	0.006	0.011
NAPHTHALENE	11	31	35.5	0.520	0.6565	0.7203	2.500	0.041	0.720
NICKEL	19	23	82.6	6.402	3.4368	8.45[e]	13.200	3.300	7.633
NITRATE-NITRITE AS N	9	16	56.3	1.728	1.7757	2.5063	6.800	1.100	2.506
O-XYLENE	9	31	29.0	0.244	0.7663	0.4771	3.100	0.007	0.477
ORTHOPHOSPHATE	13	16	81.3	19.828	43.3738	38.8367	140.000	0.420	38.837
PCB-1254 (d)	2	31	6.5	0.736	1.6496	1.2387	0.210	0.057	0.210
PERCENT MOISTURE	19	19	100.0	7.784	1.9196	8.5478	13.000	4.900	8.548
PH	8	8	100.0	7.663	2.0184	9.0148	10.300	5.800	9.015
PHENANTHRENE	21	31	67.7	0.739	1.0839	1.0698	5.100	0.049	1.070
PHENOL	4	31	12.9	0.509	0.5682	0.6825	1.800	0.120	0.683

Table 6-A-5
Analytical Summary of Detected Compounds
Waste Water Treatment Area
Combined Soil

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH UCL CONC. (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
PHORATE	2	30	6.7	0.030	0.0175	0.0358	0.017	0.017	0.017
POTASSIUM	15	15	100.0	845.467	222.2945	946.5414	1170.000	384.000	946.541
PROPAZINE	2	18	11.1	4.772	6.5270	7.4491	28.000	0.640	7.449
PYRENE	23	31	74.2	1.191	1.7480	1.7235	6.800	0.039	1.724
SELENIUM	2	23	8.7	0.186	0.0756	0.2129	0.430	0.130	0.213
SILVER	13	31	41.9	0.725	0.8557	0.9856	4.000	0.310	0.986
SODIUM	8	15	53.3	147.333	89.0631	187.8293	303.000	139.000	187.829
STYRENE	2	31	6.5	0.034	0.0368	0.0452	0.019	0.009	0.019
SULFATE	11	16	68.8	110.031	106.9541	156.9039	260.000	6.200	156.904
SULFIDE	2	16	12.5	24.219	27.4888	36.2657	93.000	93.000	36.266
SULFOTEPP	5	30	16.7	0.024	0.0262	0.0319	0.140	0.004	0.032
TCDF	3	7	42.9	1.63E-03	0.0033	0.0041	0.009	0.000	0.004
TETRACHLOROETHENE	1	31	3.2	0.033	0.0372	0.0447	0.006	0.006	0.006
THIONAZIN	1	30	3.3	0.164	0.1900	0.2225	0.006	0.006	0.006
TINUVIN 327 (d)	2	15	13.3	5.220	6.5633	8.2043	26.000	0.500	8.204
TINUVIN 328	3	3	100.0	173.333	35.1188	232.5389	210.000	140.000	210.000
TOLUENE	15	31	48.4	0.687	2.1603	1.3452	8.700	0.006	1.345
TOTAL ALKALINITY	12	16	75.0	691.000	659.0468	979.8273	1800.000	130.000	979.827
TOTAL ORGANIC CARBON	16	16	100.0	4150.000	3353.8890	5619.8419	9100.000	1200.000	5619.842
TRCDF	2	3	66.7	0.767	0.6658	1.8892	1.200	1.100	1.200
VANADIUM	23	23	100.0	9.252	2.8515	10.2730	15.000	4.700	10.273
ZINC	23	23	100.0	215.935	266.0507	505.73[e]	840.000	15.200	505.730

a. Arithmetic mean concentration, unless otherwise indicated.
b. 95th percentile upper confidence limit of the arithmetic mean concentration of an assumed normal distribution, unless otherwise indicated.
c. Lesser of the maximum concentration and the 95th percentile UCL of the mean concentration.
d. Shading indicates that the chemical was selected as a chemical of potential concern.
e. 95th Percentile UCL of the arithmetic mean concentration of an assumed lognormal distribution.

Table 6-A-6
Analytical Summary of Detected Compounds
Waste Water Treatment Area
Groundwater

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (UG/L)[a]	STANDARD DEVIATION (UG/L)	95TH% UCL (UG/L)[b]	MAX. CONC. (UG/L)	MIN. CONC. (UG/L)	SELECTED CONC. (UG/L)[c]
1,1,1-TRICHLOROETHANE	2	24	8.3	2.5375	0.3899	2.6739	4.2000	1.7000	2.6739
1,1-DICHLOROETHANE	4	24	16.7	2.5833	1.0507	2.9510	7.2000	1.0000	2.9510
1,2,3,4,6,7,8-HPCDF	2	12	16.7	1.45E-04	7.73E-05	1.85E-04	7.85E-05	6.20E-05	7.85E-05
1,2-DICHLOROBENZENE	3	24	12.5	4.4708	1.2992	4.9254	2.0000	0.5000	2.0000
2,4-DICHLOROPHENOL	4	24	16.7	6.1125	4.9878	7.8576	29.0000	4.4000	7.8576
2-METHYLNAPHTHALENE	4	24	16.7	4.8583	0.4093	5.0015	6.0000	4.0000	5.0015
3&4-METHYLPHENOL	1	12	8.3	4.6250	1.2990	5.2985	0.5000	0.5000	0.5000
4,4'-DDE	1	24	4.2	0.0970	0.2177	0.1732	0.0120	0.0120	0.0120
4,4'-DDT	1	24	4.2	0.1203	0.2680	0.2141	0.0070	0.0070	0.0070
4-CHLORO-3-METHYLPHENOL	1	24	4.2	4.7583	0.8918	5.0704	0.6000	0.6000	0.6000
4-CHLOROANILINE	2	24	8.3	6.4083	2.3746	7.2391	6.0000	4.0000	6.0000
4-METHYLPHENOL	2	12	16.7	13.0667	19.9076	23.3880	67.0000	41.0000	23.3880
ACENAPHTHENE	2	24	8.3	4.5971	1.1562	5.0016	1.1000	0.6300	1.1000
ACETOPHENONE	1	24	4.2	4.8667	0.4101	5.0102	3.0000	3.0000	3.0000
ALDRIN	2	24	8.3	0.0605	0.1339	0.1074	0.0110	0.0054	0.0110
ALPHA-BHC	2	24	8.3	0.0610	0.1337	0.1077	0.0150	0.0120	0.0150
ALPHA-CHLORDANE	1	24	4.2	0.0706	0.1378	0.1188	0.0031	0.0031	0.0031
AMMONIA AS N	9	12	75.0	2.7724	4.8253	5.2742	14.0000	0.0470	5.2742
ANILINE	2	24	8.3	21.2833	20.8918	28.5927	86.0000	76.0000	28.5927
ANTHRACENE	3	24	12.5	4.4383	1.3809	4.9215	1.0000	0.6200	1.0000
ARSENIC	8	24	33.3	6.6833	7.1635	9.1896	32.0000	5.2000	9.1896
BARIUM	24	24	100.0	56.8958	45.6004	72.8500	215.0000	14.0000	72.8500
BENZO(A)ANTHRACENE	2	24	8.3	4.6542	1.0133	5.0087	1.8000	1.0000	1.8000
BENZO(A)PYRENE	2	24	8.3	4.6500	1.0205	5.0071	1.4000	1.3000	1.4000
BENZO(B)FLUORANTHENE	3	24	12.5	4.5417	1.1177	4.9327	2.3000	1.0000	2.3000
BENZO(K)FLUORANTHENE	2	24	8.3	4.7417	0.7239	4.9949	2.9000	2.0000	2.9000
BICARBONATE ALKALINITY	8	13	61.5	38.5000	40.7226	58.6267	123.0000	11.0000	58.6267
BIOLOGICAL OXYGEN DEMAND - 5	6	6	100.0	3000.0000	1673.3201	4376.5071	6000.0000	2000.0000	4376.5071
BIS(2-ETHYLHEXYL)PHTHALATE (d)	2	24	8.3	10.2500	26.4176	19.4927	130.0000	36.0000	19.4927

Table 6-A-6
Analytical Summary of Detected Compounds
Waste Water Treatment Area
Groundwater

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (UG/L)[a]	STANDARD DEVIATION (UG/L)	95TH% UCL (UG/L)[b]	MAX. CONC. (UG/L)	MIN. CONC. (UG/L)	SELECTED CONC. (UG/L)[c]
BUTAZOLIDIN	3	24	12.5	69.9833	94.4595	103.0318	20.0000	7.0000	20.0000
CALCIUM	24	24	100.0	25079.1667	11574.2961	29128.6516	53400.0000	8600.0000	29128.6516
CARBONATE ALKALINITY	1	13	7.7	1.7115	4.2937	3.8336	16.0000	16.0000	3.8336
CHEMICAL OXYGEN DEMAND	6	12	50.0	12.3417	12.9703	19.0662	36.0000	7.0000	19.0662
CHLORIDE	12	12	100.0	18.6667	5.1581	21.3409	28.0000	12.0000	21.3409
CHLOROBENZENE	4	24	16.7	3.4000	2.8587	4.4002	14.0000	2.4000	4.4002
CHLOROBENZILATE	1	24	4.2	2.8167	2.3154	3.6267	0.5000	0.5000	0.5000
CHLOROETHANE	1	24	4.2	5.5000	2.4495	6.3570	17.0000	17.0000	6.3570
CHLOROFORM	3	24	12.5	2.8333	1.0341	3.1951	7.1000	3.8000	3.1951
CHRYSENE	3	24	12.5	4.5292	1.1448	4.9297	1.9000	1.0000	1.9000
COBALT	1	24	4.2	5.2417	1.1839	5.6559	10.8000	10.8000	5.6559
DELTA-BHC	3	24	12.5	0.0755	0.1505	0.1282	0.4000	0.0120	0.1282
DI-N-BUTYLPHTHALATE	2	24	8.3	4.6067	1.1527	5.0100	1.0000	0.7600	1.0000
DI-N-OCTYLPHTHALATE	1	24	4.2	4.7896	0.7918	5.0666	1.1000	1.1000	1.1000
DIBENZOFURAN	1	24	4.2	4.7500	0.9324	5.0762	0.4000	0.4000	0.4000
DIELDRIN (d)	3	24	12.5	0.1155	0.2298	0.1959	0.4700	0.0160	0.1959
DIETHYLPHTHALATE	1	24	4.2	4.7833	0.8121	5.0675	1.0000	1.0000	1.0000
DINOSEB	1	24	4.2	7.5337	10.5045	11.2089	0.0380	0.0380	0.0380
ENDRIN ALDEHYDE	1	24	4.2	0.1475	0.3692	0.2767	1.6000	1.6000	0.2767
ETHYLBENZENE	3	24	12.5	2.6208	0.4961	2.7944	4.9000	2.5000	2.7944
FLUORANTHENE	4	24	16.7	4.6417	0.7794	4.9144	4.2000	1.9000	4.2000
FLUORENE	1	24	4.2	4.7542	0.9121	5.0733	0.5000	0.5000	0.5000
GAMMA-CHLORDANE (d)	3	24	12.5	0.1028	0.2552	0.1921	1.2000	0.0062	0.1921
HEPTACHLOR	1	24	4.2	0.0619	0.1335	0.1086	0.0450	0.0450	0.0450
HPCDF	2	12	16.7	4.46E-04	8.44E-04	8.83E-04	6.20E-04	7.85E-05	6.20E-04
HYDROCARBONS	5	13	38.5	1.1319	1.4018	1.8248	5.0000	0.7400	1.8248
IRGASAN DP-300	5	24	20.8	79.6250	146.6169	130.9217	670.0000	26.0000	130.9217
IRON	20	24	83.3	6172.6354	4295.8398	7675.6157	13000.0000	29.0000	7675.6157
ISODRIN	3	24	12.5	0.0584	0.1351	0.1056	0.1400	0.0086	0.1056

Table 6-A-6
Analytical Summary of Detected Compounds
Waste Water Treatment Area
Groundwater

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (UG/L)[a]	STANDARD DEVIATION (UG/L)	95TH% UCL (UG/L)[b]	MAX. CONC. (UG/L)	MIN. CONC. (UG/L)	SELECTED CONC. (UG/L)[c]
LANGLIER INDEX	12	12	100.0	-2.0725	0.6808	-1.7195	-1.2000	-3.3000	-1.7195
M&P-XYLENE	2	24	8.3	3.7917	4.6178	5.4073	23.0000	13.0000	5.4073
MAGNESIUM	24	24	100.0	4115.0000	1326.9022	4579.2417	6290.0000	1200.0000	4579.2417
MANGANESE	24	24	100.0	992.0000	592.9304	1199.4478	2400.0000	24.0000	1199.4478
N-NITROSODIMETHYLAMINE	1	24	4.2	4.9833	0.2403	5.0674	6.0000	6.0000	5.0674
NAPHTHALENE	6	24	25.0	6.0333	3.4557	7.2424	14.0000	1.0000	7.2424
NICKEL	1	24	4.2	16.4500	7.8369	19.1919	44.8000	44.8000	19.1919
NITRATE-NITRITE AS N	5	12	41.7	283.7500	592.6409	591.0109	1600.0000	60.0000	591.0109
O-XYLENE	2	24	8.3	3.0000	1.8474	3.6464	11.0000	6.0000	3.6464
OCDD	4	12	33.3	0.0009	0.0006	0.0012	0.0017	0.0003	0.0012
OCDF	2	12	16.7	2.88E-04	1.92E-04	3.88E-04	2.01E-04	1.38E-04	2.01E-04
OIL AND GREASE	6	12	50.0	1.0279	0.9466	1.5187	3.3000	0.6600	1.5187
ORTHOPHOSPHATE	2	12	16.7	0.0125	0.0217	0.0237	0.0800	0.0200	0.0237
PHENANTHRENE	2	24	8.3	4.6250	1.1049	5.0116	1.1000	1.0000	1.1000
PHENOL	2	24	8.3	9.1167	14.2426	14.0997	61.0000	49.0000	14.0997
POTASSIUM	19	24	79.2	4422.9167	3561.7454	5669.0602	14200.0000	1500.0000	5669.0602
PROPAZINE	3	24	12.5	45.6667	43.4338	60.8628	170.0000	43.0000	60.8628
PYRENE	4	24	16.7	4.5333	0.9857	4.8782	3.2000	1.5000	3.2000
SILICA,DISSOLVED	24	24	100.0	20.6125	10.5700	24.3106	56.0000	3.7000	24.3106
SILVER	1	24	4.2	5.2250	1.1023	5.6107	10.4000	10.4000	5.6107
SODIUM	24	24	100.0	17258.3333	5665.2961	19240.4436	30600.0000	11000.0000	19240.4436
SULFATE	12	12	100.0	45.3333	22.9875	57.2514	101.0000	20.0000	57.2514
TCDD	1	12	8.3	0.0040	0.0048	0.0065	0.0001	0.0001	0.0001
TCDF	3	12	25.0	0.0005	0.0009	0.0010	0.0034	0.0000	0.0010
TINUVIN 328	1	6	16.7	20.8333	38.7836	52.7375	100.0000	100.0000	52.7375
TKN	7	12	58.3	2.6942	4.8080	5.1869	14.0000	0.1500	5.1869
TOFRANIL	3	24	12.5	30.0000	15.0398	35.2620	67.0000	2.5000	35.2620
TOLUENE	3	24	12.5	3.6500	5.6189	5.6159	30.0000	1.7000	5.6159
TOTAL ALKALINITY	24	24	100.0	60.5000	37.2740	73.5410	170.0000	11.0000	73.5410

Table 6-A-6
Analytical Summary of Detected Compounds
Waste Water Treatment Area
Groundwater

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (UG/L)[a]	STANDARD DEVIATION (UG/L)	95TH% UCL (UG/L)[b]	MAX. CONC. (UG/L)	MIN. CONC. (UG/L)	SELECTED CONC. (UG/L)[c]
TOTAL DISSOLVED SOLIDS	12	12	100.0	210.8333	79.2531	251.9230	350.0000	110.0000	251.9230
TOTAL HARDNESS, AS CACO3	20	20	100.0	24973.7500	49261.0670	44018.8742	160000.0000	26.0000	44018.8742
TOTAL ORGANIC CARBON	11	12	91.7	13.9083	14.0020	21.1678	40.0000	1.2000	21.1678
TOTAL ORGANIC HALIDES	12	12	100.0	0.1516	0.0408	0.1727	0.2300	0.0990	0.1727
TOTAL SUSPENDED SOLIDS	12	12	100.0	452.2500	347.7471	632.5431	1200.0000	87.0000	632.5431
TRCDF	1	6	16.7	1.5167	3.7151	4.5728	9.1000	9.1000	4.5728
TRICHLOROETHENE	1	24	4.2	2.5167	0.0816	2.5452	2.9000	2.9000	2.5452
ZINC	3	24	12.5	16.0333	17.1445	22.0317	90.8000	24.0000	22.0317

a. Arithmetic mean concentration, unless otherwise indicated.
b. 95th percentile upper confidence limit of the arithmetic mean concentration of an assumed normal distribution, unless otherwise indicated.
c. Lesser of the maximum concentration and the 95th percentile UCL of the mean concentration.
d. Shading indicates that the chemical was selected as a chemical of potential concern.
e. 95th Percentile UCL of the arithmetic mean concentration of an assumed lognormal distribution.

Table 6-A-7
Analytical Summary of Detected Compounds
Warwick Area
Surface Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
1,1-BIPHENYL	1	2	50.0	1.0375	1.2198	6.4833	1.9000	1.9000	1.9000
1,1-DICHLOROETHANE	1	32	3.1	0.0478	0.0303	0.0568	0.0440	0.0440	0.0440
1,2-DICHLOROBENZENE	1	31	3.2	1.3803	2.1297	2.0294	0.1800	0.1800	0.1800
1,4-DICHLOROBENZENE	2	31	6.5	1.3865	2.1281	2.0351	0.0390	0.0320	0.0390
2,4,5-T	1	31	3.2	0.0121	0.0070	0.0143	0.0095	0.0095	0.0095
2,4,5-TP (SILVEX)	4	31	12.9	0.0314	0.0698	0.0527	0.3400	0.0200	0.0527
2-BUTANONE	2	32	6.3	0.1031	0.0637	0.1222	0.2300	0.1800	0.1222
2-METHYLNAPHTHALENE	6	31	19.4	1.3441	2.1482	1.9989	0.3600	0.0140	0.3600
2-NITROANILINE (d)	2	31	6.5	7.1542	10.6697	10.4062	7.0000	0.9800	7.0000
3&4-METHYLPHENOL	1	6	16.7	0.1520	0.0640	0.2046	0.0220	0.0220	0.0220
3,3'-DIMETHYLBENZIDINE	1	31	3.2	2.9161	4.3040	4.2279	6.6000	6.6000	4.2279
4,4'-DDD	2	32	6.3	0.0654	0.2290	0.1341	0.4100	0.0180	0.1341
4,4'-DDE	6	33	18.2	0.0774	0.2414	0.1487	0.6500	0.0040	0.1487
4,4'-DDT	9	32	28.1	0.1185	0.4461	0.2523	0.5100	0.0010	0.2523
4-CHLOROANILINE	10	31	32.3	1.4424	1.8587	2.0089	7.4000	0.3100	2.0089
ACENAPHTHENE	3	31	9.7	1.3716	2.1350	2.0223	0.1600	0.0160	0.1600
ACENAPHTHYLENE	3	31	9.7	1.3864	2.1281	2.0350	0.1100	0.0610	0.1100
ACETONE	1	32	3.1	0.1047	0.0700	0.1257	0.3200	0.3200	0.1257
ALDRIN (d)	3	33	9.1	0.1120	0.3252	0.2080	1.1000	0.1300	0.2080
ALPHA-BHC	6	33	18.2	0.0949	0.2965	0.1825	1.2000	0.0010	0.1825
ALPHA-CHLORDANE	3	32	9.4	0.0550	0.2201	0.1210	0.0770	0.0040	0.0770
AMMONIA AS N	5	25	20.0	0.9690	1.3063	1.4160	5.2000	1.1000	1.4160
ANTHRACENE	10	31	32.3	1.3073	2.1638	1.9668	0.3200	0.0310	0.3200
ANTIMONY	6	23	26.1	2.2654	8.6269	5.3540	41.8000	0.8600	5.3540
ARSENIC	27	27	100.0	9.0037	3.7988	10.2509	16.2000	2.4000	10.2509
BARIUM	31	31	100.0	113.6484	227.7039	183.0503	1270.0000	7.3000	183.0503
BENZENE	1	32	3.1	0.0474	0.0304	0.0565	0.0340	0.0340	0.0340
BENZO(A)ANTHRACENE	15	31	48.4	0.9723	1.2572	1.3554	1.6000	0.1400	1.3554
BENZO(A)PYRENE	13	31	41.9	1.1502	1.5519	1.6232	1.7000	0.0250	1.6232

Table 6-A-7
Analytical Summary of Detected Compounds
Warwick Area
Surface Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
BENZO(B)FLUORANTHENE	14	31	45.2	1.2336	1.3580	1.6475	2.8000	0.0420	1.6475
BENZO(G,H,I)PERYLENE	9	31	29.0	1.4471	2.1041	2.0884	1.2000	0.0640	1.2000
BENZO(K)FLUORANTHENE	13	31	41.9	1.3034	1.5083	1.7631	3.6000	0.0620	1.7631
BERYLLIUM (d)	31	31	100.0	NA	NA	0.818[e]	2.0000	0.3200	2.0000
BETA-BHC	2	32	6.3	0.0523	0.2202	0.1183	0.0096	0.0091	0.0096
BICARBONATE ALKALINITY	17	25	68.0	230.9600	222.9096	307.2397	940.0000	97.0000	307.2397
BIS(2-ETHYLHEXLYL)PHTHALATE	2	31	6.5	1.3797	2.1293	2.0287	0.4300	0.3300	0.4300
BIS(2-CHLOROETHYL)ETHER (d)	14	31	45.2	10.4613	31.4433	20.0449	140.0000	0.1000	20.0449
BUTYLBENZYLPHTHALATE	6	31	19.4	1.3891	2.1279	2.0377	0.7800	0.0460	0.7800
CADMIUM	15	31	48.4	1.5608	2.1672	2.2214	6.9000	0.2800	2.2214
CALCIUM	25	25	100.0	1465.8800	792.1142	1736.9415	3730.0000	363.0000	1736.9415
CATION EXCHANGE CAPACITY	25	25	100.0	7.1040	4.9712	8.8051	18.0000	1.4000	8.8051
CHLORIDE	13	25	52.0	71.7000	86.9734	101.4623	300.0000	19.0000	101.4623
CHLOROBENZENE	9	32	28.1	0.4073	0.8703	0.6684	3.6000	0.0057	0.6684
CHLOROFORM	1	32	3.1	0.0479	0.0303	0.0570	0.0480	0.0480	0.0480
CHROMIUM	31	31	100.0	68.1548	107.8930	101.0396	357.0000	1.3000	101.0396
CHRYSENE	14	31	45.2	1.0750	1.2914	1.4686	2.3000	0.1200	1.4686
COBALT	27	27	100.0	4.4259	1.7819	5.0110	7.8000	1.0000	5.0110
COPPER	30	31	96.8	118.0161	351.7469	225.2251	1960.0000	2.9000	225.2251
CYANIDE	9	30	30.0	1.8620	2.7130	2.7035	9.4000	0.8000	2.7035
DELTA-BHC	3	33	9.1	0.0582	0.2199	0.1231	0.2600	0.0020	0.1231
DI-N-BUTYLPHTHALATE	1	31	3.2	1.3780	2.1315	2.0276	0.0570	0.0570	0.0570
DI-N-OCTYLPHTHALATE	8	31	25.8	2.5247	5.2476	4.1241	23.0000	0.0400	4.1241
DIBENZ(A,H)ANTHRACENE	3	31	9.7	1.3817	2.1308	2.0312	0.1300	0.0830	0.1300
DIBENZOFURAN	3	31	9.7	1.3598	2.1403	2.0122	0.2000	0.0520	0.2000
DIELDRIN (d)	5	32	15.6	0.0806	0.2667	0.1606	0.9100	0.0019	0.1606
DINOSEB	1	31	3.2	0.0431	0.0675	0.0637	0.0720	0.0720	0.0637
DISULFOTON	3	30	10.0	0.0569	0.0246	0.0645	0.0077	0.0059	0.0077
ENDOSULFAN I	3	32	9.4	0.0524	0.2202	0.1185	0.0180	0.0100	0.0180

Table 6-A-7
Analytical Summary of Detected Compounds
Warwick Area
Surface Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
ENDOSULFAN II	1	32	3.1	0.1524	0.6613	0.3508	0.0180	0.0180	0.0180
ENDOSULFAN SULFATE	3	32	9.4	0.2464	1.0589	0.5640	0.2900	0.0075	0.2900
ENDRIN	6	32	18.8	0.0702	0.2362	0.1411	0.5400	0.0031	0.1411
ENDRIN ALDEHYDE	5	32	15.6	0.2206	0.7449	0.4441	3.5000	0.0021	0.4441
ETHYL PARATHION	2	30	6.7	0.0359	0.0129	0.0399	0.0064	0.0056	0.0064
ETHYLBENZENE	1	32	3.1	0.0472	0.0305	0.0564	0.0270	0.0270	0.0270
FLUORANTHENE	17	31	54.8	1.2277	1.1463	1.5771	3.7000	0.0380	1.5771
FLUORENE	7	31	22.6	1.3418	2.1494	1.9969	0.2300	0.0350	0.2300
GAMMA-BHC	2	32	6.3	0.0518	0.2204	0.1179	0.0017	0.0014	0.0017
GAMMA-CHLORDANE	9	32	28.1	0.0783	0.2345	0.1487	0.5000	0.0040	0.1487
HEPTACHLOR	1	32	3.1	0.0518	0.2203	0.1179	0.0032	0.0032	0.0032
HEPTACHLOR EPOXIDE	7	32	21.9	0.1016	0.3000	0.1916	1.2000	0.0022	0.1916
INDENO(1,2,3-CD)PYRENE	8	31	25.8	1.4132	2.1159	2.0581	0.8600	0.0700	0.8600
IRON	25	25	100.0	12327.6000	3723.4228	13601.7553	20000.0000	4610.0000	13601.7553
ISODRIN	2	32	6.3	0.0769	0.2612	0.1553	0.8500	0.0072	0.1553
KEPONE	1	32	3.1	0.0808	0.2343	0.1511	0.2600	0.2600	0.1511
LEAD	26	27	96.3	84.0056	102.6756	117.7160	428.0000	2.8000	117.7160
M&P-XYLENE	8	32	25.0	0.0456	0.0300	0.0546	0.0700	0.0065	0.0546
MAGNESIUM	25	25	100.0	1713.5600	1030.6747	2066.2569	5360.0000	290.0000	2066.2569
MANGANESE	25	25	100.0	221.1360	68.3698	244.5321	416.0000	83.4000	244.5321
MERCURY	15	31	48.4	0.2261	0.2792	0.3112	0.9400	0.1300	0.3112
METHOXYCHLOR	13	34	38.2	110.3893	417.4362	231.8771	2200.0000	0.0460	231.8771
METHYLENE CHLORIDE	3	32	9.4	0.2386	0.3910	0.3559	0.0250	0.0110	0.0250
N-NITROSO-DI-N-PROPYLAMINE	1	31	3.2	1.3684	2.1372	2.0198	0.1700	0.1700	0.1700
NAPHTHALENE	16	31	51.6	1.1290	1.4793	1.5799	3.5000	0.0360	1.5799
NICKEL	28	31	90.3	17.0097	34.6186	26.26[e]	199.0000	2.7000	199.0000
NITRATE-NITRITE AS N	22	25	88.0	1.5752	1.5060	2.0906	6.4000	0.3400	2.0906
NITROBENZENE	2	31	6.5	1.4774	2.1294	2.1264	2.9000	0.4800	2.1264
O-XYLENE	4	32	12.5	0.0445	0.0306	0.0537	0.0460	0.0170	0.0460

Table 6-A-7
Analytical Summary of Detected Compounds
Warwick Area
Surface Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
ORTHOPHOSPHATE	20	25	80.0	4.5704	6.7629	6.8847	29.0000	0.3600	6.8847
PCB-1248 (d)	3	34	8.8	6.8904	28.3977	15.1550	160.0000	8.1000	15.1550
PCB-1254 (d)	15	32	46.9	2.8607	7.7752	5.1932	36.0000	0.0320	5.1932
PERCENT MOISTURE	24	24	100.0	12.3333	8.2031	15.2033	28.0000	0.0000	15.2033
PH	15	15	100.0	6.1800	0.4931	6.4042	7.1000	5.6000	6.4042
PHENACETIN	1	31	3.2	1.4084	2.1187	2.0541	1.0000	1.0000	1.0000
PHENANTHRENE	17	31	54.8	0.8669	0.8585	1.1286	1.7000	0.1800	1.1286
PHENOL	3	31	9.7	1.1845	1.8565	1.7503	0.8900	0.3500	0.8900
PHORATE	1	30	3.3	0.0369	0.0112	0.0404	0.0096	0.0096	0.0096
POTASSIUM	25	25	100.0	767.4400	853.6258	1059.5508	4630.0000	331.0000	1059.5508
PROPAZINE	1	25	4.0	8.2420	11.0038	12.0075	24.0000	24.0000	12.0075
PYRENE	18	31	58.1	1.2765	1.0767	1.6047	3.0000	0.0530	1.6047
SAFROLE	4	31	12.9	2.4648	5.2075	4.0520	28.0000	0.7000	4.0520
SODIUM	15	25	60.0	117.2300	60.5521	137.9509	217.0000	118.0000	137.9509
SULFATE	11	25	44.0	128.8200	199.8416	197.2058	890.0000	13.0000	197.2058
SULFIDE	5	25	20.0	92.1200	335.4909	206.9250	1700.0000	35.0000	206.9250
SULFOTEPP	1	31	3.2	0.0241	0.0085	0.0267	0.0041	0.0041	0.0041
TETRACHLOROETHENE	9	32	28.1	0.1261	0.4190	0.2518	2.4000	0.0090	0.2518
THIONAZIN	1	30	3.3	0.2207	0.1157	0.2566	0.0058	0.0058	0.0058
TIN	6	31	19.4	7.4226	7.6452	9.7528	37.8000	14.7000	9.7528
TINUVIN 327	8	25	32.0	6.3408	8.5561	9.2687	18.0000	0.5700	9.2687
TOLUENE	18	32	56.3	0.2324	0.4102	0.3555	1.8000	0.0069	0.3555
TOTAL ALKALINITY	17	25	68.0	230.9600	222.9096	307.2397	940.0000	97.0000	307.2397
TOTAL ORGANIC CARBON	25	25	100.0	11732.0000	11172.9005	15555.3666	28000.0000	240.0000	15555.3666
TRICHLOROETHENE	2	32	6.3	0.0497	0.0335	0.0597	0.1300	0.0410	0.0597
VANADIUM	19	27	70.4	10.5074	5.2704	12.2378	22.0000	1.7000	12.2378
ZINC	28	31	90.3	2538.6726	4505.7211	3911.9722	16100.0000	24.8000	3911.9722

a. Arithmetic mean concentration, unless otherwise indicated.

Table 6-A-7
Analytical Summary of Detected Compounds
Warwick Area
Surface Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]

b. 95th percentile upper confidence limit of the arithmetic mean concentration of an assumed normal distribution, unless otherwise indicated.

c. Lesser of the maximum concentration and the 95th percentile UCL of the mean concentration.

d. Shading indicates that the chemical was selected as a chemical of potential concern.

e. 95th Percentile UCL of the arithmetic mean concentration of an assumed lognormal distribution.

Table 6-A-8
Analytical Summary of Detected Compounds
Warwick Area
Combined Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
1,1-BIPHENYL	1	2	50.0	1.0375	1.2198	6.483	1.900	1.900	1.900
1,1-DICHLOROETHANE	1	52	1.9	0.0416	0.0617	0.056	0.044	0.044	0.044
1,2-DICHLOROBENZENE	1	41	2.4	1.0983	1.9124	1.601	0.180	0.180	0.180
1,4-DICHLOROBENZENE	2	41	4.9	1.1030	1.9118	1.606	0.039	0.032	0.039
2,4,5-T	1	38	2.6	0.0129	0.0095	0.015	0.010	0.010	0.010
2,4,5-TP (SILVEX)	4	38	10.5	0.0285	0.0636	0.046	0.340	0.020	0.046
2-BUTANONE	2	52	3.8	0.1147	0.2882	0.182	0.230	0.180	0.182
2-CHLOROPHENOL	1	41	2.4	1.1102	1.9079	1.612	0.240	0.240	0.240
2-METHYLNAPHTHALENE	11	41	26.8	1.0517	1.9336	1.560	0.360	0.010	0.360
2-NITROANILINE (d)	3	41	7.3	5.6817	9.6092	8.209	7.000	0.920	7.000
3&4-METHYLPHENOL	2	15	13.3	0.1637	0.0576	0.190	0.028	0.022	0.028
3,3'-DIMETHYLBENZIDINE	1	41	2.4	2.2555	3.9115	3.284	6.600	6.600	3.284
4,4'-DDD	4	52	7.7	0.0505	0.1844	0.094	0.410	0.017	0.094
4,4'-DDE	10	53	18.9	0.0583	0.1954	0.103	0.650	0.004	0.103
4,4'-DDT	15	52	28.8	0.0880	0.3551	0.171	0.510	0.001	0.171
4-CHLOROANILINE	13	41	31.7	1.1842	1.6765	1.625	7.400	0.098	1.625
ACENAPHTHENE	5	41	12.2	1.0934	1.9155	1.597	0.410	0.016	0.410
ACENAPHTHYLENE	6	41	14.6	1.0959	1.9154	1.600	0.160	0.042	0.160
ACETONE	1	52	1.9	0.1157	0.2890	0.183	0.320	0.320	0.183
ALDRIN (d)	3	53	5.7	0.0765	0.2611	0.137	1.100	0.130	0.137
ALPHA-BHC	7	53	13.2	0.0659	0.2377	0.121	1.200	0.001	0.121
ALPHA-CHLORDANE	6	52	11.5	0.0416	0.1753	0.083	0.077	0.002	0.077
AMMONIA AS N	5	26	19.2	0.9529	1.2825	1.382	5.200	1.100	1.382
ANTHRACENE	14	41	34.1	1.0405	1.9344	1.549	0.320	0.031	0.320
ANTIMONY	7	30	23.3	1.8440	7.5631	4.190	41.800	0.860	4.190
ARSENIC	34	34	100.0	7.6044	4.4865	8.910	16.200	0.550	8.910
BARIUM	44	44	100.0	90.2682	195.0021	139.774	1270.000	7.300	139.774
BENZENE	1	52	1.9	0.0414	0.0617	0.056	0.034	0.034	0.034
BENZO(A)ANTHRACENE	22	41	53.7	0.8341	1.1339	1.132	1.600	0.066	1.132

Table 6-A-8
Analytical Summary of Detected Compounds
Warwick Area
Combined Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
BENZO(A)PYRENE	20	41	48.8	0.9667	1.3988	1.335	1.700	0.025	1.335
BENZO(B)FLUORANTHENE	21	41	51.2	1.0710	1.2517	1.400	2.800	0.042	1.400
BENZO(G,H,I)PERYLENE	15	41	36.6	1.1703	1.8912	1.668	1.200	0.064	1.200
BENZO(K)FLUORANTHENE	19	41	46.3	1.0503	1.3890	1.416	3.600	0.062	1.416
BERYLLIUM (d)	43	44	97.7	0.6759	0.3732	0.829[e]	2.000	0.230	2.000
BETA-BHC	2	52	3.8	0.0391	0.1754	0.080	0.010	0.009	0.010
BICARBONATE ALKALINITY	17	26	65.4	223.1346	222.0209	297.504	940.000	97.000	297.504
BIS(2-CHLOROETHYL)ETHER (d)	2	41	4.9	1.0978	1.9121	1.601	0.430	0.330	0.430
BIS(2-ETHYLHEXYL)PHTHALATE	18	41	43.9	11.8848	36.3537	21.446	160.000	0.100	21.446
BUTYLBENZYLPHTHALATE	7	41	17.1	1.1025	1.9132	1.606	0.780	0.046	0.780
CADMIUM	17	38	44.7	1.5504	2.2483	2.169	7.600	0.280	2.169
CALCIUM	26	26	100.0	1431.7692	795.3611	1698.188	3730.000	363.000	1698.188
CATION EXCHANGE CAPACITY	26	26	100.0	6.9731	4.9163	8.620	18.000	1.400	8.620
CHLORIDE	13	26	50.0	70.0000	85.6559	98.692	300.000	19.000	98.692
CHLOROBENZENE	12	52	23.1	10.0627	70.6917	26.571	510.000	0.006	26.571
CHLOROBENZILATE	1	55	1.8	0.8584	1.7274	1.251	0.046	0.046	0.046
CHLOROFORM	1	52	1.9	0.0416	0.0617	0.056	0.048	0.048	0.048
CHROMIUM	44	44	100.0	61.3545	114.0027	90.297	478.000	1.300	90.297
CHRYSENE	21	41	51.2	0.9178	1.1717	1.226	2.300	0.061	1.226
COBALT	34	34	100.0	4.2118	1.8383	4.747	7.800	1.000	4.747
COPPER	43	44	97.7	94.1318	300.5219	170.426	1960.000	1.800	170.426
CYANIDE	10	40	25.0	1.6928	2.3652	2.327	9.400	0.800	2.327
DELTA-BHC	4	53	7.5	0.0430	0.1764	0.084	0.260	0.002	0.084
DI-N-BUTYLPHTHALATE	1	41	2.4	1.0965	1.9137	1.600	0.057	0.057	0.057
DI-N-OCTYLPHTHALATE	10	41	24.4	4.1278	14.3585	7.904	89.000	0.040	7.904
DIBENZ(A,H)ANTHRACENE	6	41	14.6	1.0787	1.9240	1.585	0.130	0.045	0.130
DIBENZOFURAN	6	41	14.6	1.0720	1.9246	1.578	0.200	0.034	0.200
DIELDRIN (d)	6	52	11.5	0.0629	0.2179	0.114	0.910	0.002	0.114
DINOSEB	1	41	2.4	0.0740	0.0850	0.096	0.072	0.072	0.072

Table 6-A-8
Analytical Summary of Detected Compounds
Warwick Area
Combined Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
DIPHENYLAMINE	1	41	2.4	1.1078	1.9091	1.610	0.140	0.140	0.140
DISULFOTON	3	40	7.5	0.0695	0.0633	0.087	0.008	0.006	0.008
ENDOSULFAN I	4	52	7.7	0.0392	0.1754	0.080	0.018	0.002	0.018
ENDOSULFAN II	1	52	1.9	0.1071	0.5224	0.229	0.018	0.018	0.018
ENDOSULFAN SULFATE	4	52	7.7	0.1650	0.8343	0.360	0.290	0.007	0.290
ENDRIN	6	52	11.5	0.0565	0.1948	0.102	0.540	0.003	0.102
ENDRIN ALDEHYDE	6	52	11.5	0.1491	0.5911	0.287	3.500	0.002	0.287
ETHYL PARATHION	2	40	5.0	0.0344	0.0180	0.039	0.006	0.006	0.006
ETHYLBENZENE	1	52	1.9	0.0412	0.0617	0.056	0.027	0.027	0.027
FLUORANTHENE	24	41	58.5	1.0593	1.0647	1.339	3.700	0.038	1.339
FLUORENE	10	41	24.4	1.0714	1.9245	1.578	0.540	0.035	0.540
GAMMA-BHC	2	52	3.8	0.0388	0.1755	0.080	0.002	0.001	0.002
GAMMA-CHLORDANE	13	52	25.0	0.0594	0.1906	0.104	0.500	0.002	0.104
HEPTACHLOR	1	52	1.9	0.0388	0.1755	0.080	0.003	0.003	0.003
HEPTACHLOR EPOXIDE	8	52	15.4	0.0695	0.2396	0.125	1.200	0.002	0.125
INDENO(1,2,3-CD)PYRENE	13	41	31.7	1.1297	1.9040	1.630	0.860	0.070	0.860
IRON	26	26	100.0	12215.0000	3693.0976	13452.063	20000.000	4610.000	13452.063
ISODRIN	2	52	3.8	0.0606	0.2136	0.110	0.850	0.007	0.110
KEPONE	1	52	1.9	0.1186	0.3691	0.205	0.260	0.260	0.205
LEAD	33	34	97.1	69.0103	96.0971	96.978	428.000	2.800	96.978
M&P-XYLENE	12	52	23.1	0.2441	1.5211	0.599	11.000	0.006	0.599
MAGNESIUM	26	26	100.0	1691.5000	1016.0961	2031.858	5360.000	290.000	2031.858
MANGANESE	26	26	100.0	228.1308	75.8917	253.552	416.000	83.400	253.552
MERCURY	20	38	52.6	0.2373	0.3122	0.323	1.300	0.090	0.323
METHOXYCHLOR	22	54	40.7	105.8591	408.0589	199.371	2200.000	0.019	199.371
METHYLENE CHLORIDE	10	52	19.2	0.1796	0.3603	0.264	1.300	0.005	0.264
N-NITROSO-DI-N-PROPYLAMINE	1	41	2.4	1.0730	1.9254	1.579	0.170	0.170	0.170
NAPHTHALENE	21	41	51.2	1.6720	5.0374	2.997	32.000	0.036	2.997
NICKEL	41	44	93.2	13.7636	29.4651	17.57[e]	199.000	2.200	17.570

Table 6-A-8
Analytical Summary of Detected Compounds
Warwick Area
Combined Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
NITRATE-NITRITE AS N	22	26	84.6	1.5358	1.4892	2.035	6.400	0.340	2.035
NITROBENZENE	2	41	4.9	1.1717	1.9237	1.678	2.900	0.480	1.678
O-XYLENE	5	52	9.6	0.1605	0.9252	0.377	6.700	0.017	0.377
OCDD	2	8	25.0	0.0007	0.0013	0.002	0.004	0.000	0.002
ORTHOPHOSPHATE	21	26	80.8	4.4146	6.6737	6.650	29.000	0.360	6.650
PCB-1248 (d)	3	54	5.6	4.4661	22.6419	9.655	160.000	8.100	9.655
PCB-1254 (d)	17	52	32.7	1.8958	6.2158	3.347	36.000	0.029	3.347
PERCENT MOISTURE	31	31	100.0	12.8000	8.6040	15.422	29.000	0.000	15.422
PH	16	16	100.0	6.1938	0.4795	6.404	7.100	5.600	6.404
PHENACETIN	1	41	2.4	1.1195	1.9065	1.621	1.000	1.000	1.000
PHENANTHRENE	25	41	61.0	0.7915	0.8232	1.008	2.300	0.033	1.008
PHENOL	4	41	9.8	1.0668	1.7734	1.533	5.000	0.350	1.533
PHORATE	1	40	2.5	0.0352	0.0173	0.040	0.010	0.010	0.010
POTASSIUM	26	26	100.0	755.5769	838.5637	1036.468	4630.000	331.000	1036.468
PROPAZINE	1	26	3.8	8.0269	10.8371	11.657	24.000	24.000	11.657
PYRENE	25	41	61.0	1.2800	1.3335	1.631	6.400	0.053	1.631
SAFROLE	4	41	9.8	1.9183	4.6142	3.132	28.000	0.700	3.132
SODIUM	16	26	61.5	119.6827	60.6326	139.993	217.000	118.000	139.993
SULFATE	11	26	42.3	124.9231	196.8096	190.848	890.000	13.000	190.848
SULFIDE	5	26	19.2	89.4423	328.9961	199.645	1700.000	35.000	199.645
SULFOTEPP	1	41	2.4	0.0221	0.0108	0.025	0.004	0.004	0.004
TETRACHLOROETHENE	14	52	26.9	0.1227	0.4336	0.224	2.400	0.007	0.224
THIONAZIN	1	40	2.5	0.1788	0.1301	0.214	0.006	0.006	0.006
TIN	6	38	15.8	6.7145	7.1763	8.690	37.800	14.700	8.690
TINUVIN 327	8	26	30.8	6.1988	8.4144	9.017	18.000	0.570	9.017
TINUVIN 328	1	5	20.0	0.2600	0.1623	0.415	0.550	0.550	0.415
TOLUENE	23	52	44.2	2.0701	13.8508	5.305	100.000	0.007	5.305
TOTAL ALKALINITY	17	26	65.4	223.1346	222.0209	297.504	940.000	97.000	297.504
TOTAL ORGANIC CARBON	26	26	100.0	11732.0000	11172.9005	15474.546	28000.000	240.000	15474.546

Table 6-A-8
Analytical Summary of Detected Compounds
Warwick Area
Combined Soil

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)	SELECTED CONC. (MG/KG)[c]
TRICHLOROETHENE	2	52	3.8	0.0428	0.0628	0.057	0.130	0.041	0.057
VANADIUM	26	34	76.5	9.5941	5.0737	11.071	22.000	1.700	11.071
ZINC	41	44	93.2	2000.6148	4000.9642	3016.350	16100.000	18.300	3016.350

a. Arithmetic mean concentration, unless otherwise indicated.
b. 95th percentile upper confidence limit of the arithmetic mean concentration of an assumed normal distribution, unless otherwise indicated.
c. Lesser of the maximum concentration and the 95th percentile UCL of the mean concentration.
d. Shading indicates that the chemical was selected as a chemical of potential concern.
e. 95th Percentile UCL of the arithmetic mean concentration of an assumed lognormal distribution.

Table 6-A-9
Analytical Summary of Detected Compounds
Warwick Area
Groundwater

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (UG/L)[a]	STANDARD DEVIATION (UG/L)	95TH% UCL (UG/L)[b]	MAX. CONC. (UG/L)	MIN. CONC. (UG/L)	SELECTED CONC. (UG/L)[c]
1,1,1-TRICHLOROETHANE	5	27	18.5	5.6037	7.4246	8.0413	34.0000	2.9000	8.0413
1,1-DICHLOROETHANE	2	27	7.4	3.2852	2.6752	4.1635	4.1000	2.1000	4.1000
2,2'-OXYBIS(1-CHLOROPROPANE)	3	27	11.1	5.9667	4.4434	7.4255	28.0000	7.0000	7.4255
2,4,5-T	1	27	3.7	0.1750	0.0710	0.1983	0.1100	0.1100	0.1100
2-CHLOROPHENOL	5	27	18.5	5.0222	1.3914	5.4791	11.0000	1.7000	5.4791
4,4'-DDT	1	27	3.7	0.0391	0.0214	0.0461	0.0230	0.0230	0.0230
4-CHLOROANILINE	4	27	14.8	7.3222	3.9841	8.6303	21.0000	0.9000	8.6303
ACETOPHENONE	1	27	3.7	4.8222	0.6905	5.0489	1.4000	1.4000	1.4000
ALDRIN (d)	3	27	11.1	0.0238	0.0164	0.0292	0.0890	0.0250	0.0292
ALPHA-BHC	2	27	7.4	0.0207	0.0109	0.0243	0.0370	0.0150	0.0243
AMMONIA AS N	8	9	88.9	0.7773	0.9893	1.3907	2.7000	0.0510	1.3907
ANILINE	2	27	7.4	17.5185	9.9653	20.7903	7.0000	2.0000	7.0000
ARSENIC	4	27	14.8	5.2000	4.8127	6.7801	20.0000	10.3000	6.7801
BARIUM	24	27	88.9	52.8222	74.8553	77.3987	305.0000	13.0000	77.3987
BENZENE	2	27	7.4	4.8333	6.8486	7.0819	35.0000	13.0000	7.0819
BERYLLIUM (d)	1	27	3.7	2.0370	0.7712	2.2902	3.5000	3.5000	2.2902
BICARBONATE ALKALINITY	9	13	69.2	32.6923	34.0316	49.5120	92.0000	17.0000	49.5120
BIOLOGICAL OXYGEN DEMAND - 5	4	4	100.0	23250.0000	39852.8544	70136.8831	83000.0000	2000.0000	70136.8831
BIS(2-CHLOROETHYL)ETHER (d)	4	27	14.8	4.4333	1.2948	4.8584	2.0000	0.7000	2.0000
CADMIUM	2	27	7.4	3.3741	3.2370	4.4368	17.0000	11.6000	4.4368
CALCIUM	25	25	100.0	61104.0000	103107.0862	96387.2449	430000.0000	13000.0000	96387.2449
CARBON DISULFIDE	1	27	3.7	36.8519	176.5094	94.8034	920.0000	920.0000	94.8034
CARBONATE ALKALINITY	3	13	23.1	5.0769	9.8178	9.9293	32.0000	8.0000	9.9293
CHEMICAL OXYGEN DEMAND	5	9	55.6	22.7778	29.8968	41.3138	95.0000	16.0000	41.3138
CHLORIDE	9	9	100.0	20.3333	7.2801	24.8470	32.0000	11.0000	24.8470
CHLOROBENZENE (d)	7	27	25.9	221.5889	692.8727	449.0727	3500.0000	1.1000	449.0727
CHROMIUM	1	27	3.7	5.2704	1.4049	5.7316	12.3000	12.3000	5.7316
DELTA-BHC	1	27	3.7	0.0198	0.0106	0.0232	0.0170	0.0170	0.0170
DI-N-BUTYLPHTHALATE	1	27	3.7	4.7963	0.8241	5.0669	0.7000	0.7000	0.7000

Table 6-A-9
Analytical Summary of Detected Compounds
Warwick Area
Groundwater

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (UG/L)[a]	STANDARD DEVIATION (UG/L)	95TH% UCL (UG/L)[b]	MAX. CONC. (UG/L)	MIN. CONC. (UG/L)	SELECTED CONC. (UG/L)[c]
DI-N-OCTYLPHTHALATE	2	27	7.4	4.6593	1.0589	5.0069	1.0000	1.0000	1.0000
DIETHYLPHTHALATE	2	27	7.4	4.6963	0.9523	5.0090	2.3000	0.7000	2.3000
DIMETHOATE	1	27	3.7	2.7694	2.1049	3.4605	0.4400	0.4400	0.4400
DINOSEB	1	26	3.8	7.1390	9.1650	10.2090	0.0400	0.0400	0.0400
ETHYLBENZENE	1	27	3.7	3.2481	2.6669	4.1238	2.7000	2.7000	2.7000
FLUORANTHENE	2	27	7.4	4.6685	1.0609	5.0168	1.0000	1.0000	1.0000
GAMMA-BHC	2	27	7.4	0.0248	0.0252	0.0331	0.1400	0.0180	0.0331
GAMMA-CHLORDANE	2	27	7.4	0.0253	0.0170	0.0309	0.0520	0.0160	0.0309
HYDROCARBONS	6	14	42.9	0.4754	0.4920	0.7082	2.0000	0.0500	0.7082
IRON	14	24	58.3	1151.7813	2005.5095	1853.4464	5700.0000	8.4000	1853.4464
LANGLIER INDEX	15	15	100.0	-1.3427	2.4636	-0.2225	6.2000	-3.1100	-0.2225
LEAD	1	27	3.7	2.2444	0.4828	2.4030	3.4000	3.4000	2.4030
M&P-XYLENE	1	27	3.7	465.7148	2405.0769	1255.3493	1.8000	1.8000	1.8000
MAGNESIUM	22	25	88.0	2935.2800	1769.6272	3540.8464	6600.0000	960.0000	3540.8464
MANGANESE	21	24	87.5	295.2854	222.8864	373.2664	750.0000	3.1000	373.2664
NAPHTHALENE	2	27	7.4	4.7778	0.7824	5.0347	4.0000	1.0000	4.0000
NITRATE-NITRITE AS N	6	9	66.7	1243.8889	1806.6401	2364.0058	4100.0000	50.0000	2364.0058
O-TOLUIDINE	1	27	3.7	4.9926	0.2226	5.0657	6.0000	6.0000	5.0657
O-XYLENE	1	27	3.7	3.1778	2.7067	4.0665	0.8000	0.8000	0.8000
OCDD	2	16	12.5	0.0008	0.0006	0.0010	0.0012	0.0003	0.0010
OIL AND GREASE	4	9	44.4	0.5850	0.2225	0.7230	1.0000	0.6400	0.7230
ORTHOPHOSPHATE	3	9	33.3	0.0500	0.0853	0.1029	0.2600	0.0700	0.1029
PECDD	1	16	6.3	0.0003	0.0002	0.0004	0.0004	0.0004	0.0004
PHENOL	2	27	7.4	4.7593	0.7579	5.0081	3.0000	1.5000	3.0000
POTASSIUM	23	24	95.8	5491.6667	4166.6478	6949.4467	17100.0000	2200.0000	6949.4467
PROPAZINE	6	27	22.2	29.0185	30.9896	39.1930	35.0000	7.0000	35.0000
PYRENE	3	27	11.1	4.5404	1.2305	4.9444	2.0000	0.6000	2.0000
SILICA,DISSOLVED	25	25	100.0	13.5964	8.6285	16.5491	43.0000	0.3100	16.5491
SILVER	1	27	3.7	5.3370	1.7513	5.9120	14.1000	14.1000	5.9120

Table 6-A-9
Analytical Summary of Detected Compounds
Warwick Area
Groundwater

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (UG/L)[a]	STANDARD DEVIATION (UG/L)	95TH% UCL (UG/L)[b]	MAX. CONC. (UG/L)	MIN. CONC. (UG/L)	SELECTED CONC. (UG/L)[c]
SODIUM	24	24	100.0	17695.8333	11362.6956	21671.2858	61300.0000	8200.0000	21671.2858
SULFATE	9	9	100.0	47.2222	25.1236	62.7988	94.0000	16.0000	62.7988
TETRACHLOROETHENE	2	27	7.4	3.5148	2.8369	4.4462	8.4000	4.0000	4.4462
TINUVIN 327	4	15	26.7	25.2200	18.1091	33.4540	4.0000	0.8000	4.0000
TKN	5	10	50.0	1.1500	1.3622	1.9396	3.2000	0.5500	1.9396
TOLUENE	2	27	7.4	5.1111	7.1647	7.4634	31.0000	2.0000	7.4634
TOTAL ALKALINITY	24	24	100.0	142.7500	257.4794	232.8340	940.0000	17.0000	232.8340
TOTAL DISSOLVED SOLIDS	9	9	100.0	337.7778	256.8452	497.0218	850.0000	180.0000	497.0218
TOTAL HARDNESS, AS CACO3	23	23	100.0	3530.0000	16234.8975	9342.4058	78000.0000	46.0000	9342.4058
TOTAL ORGANIC CARBON	9	9	100.0	13.5444	16.3176	23.6613	48.0000	1.7000	23.6613
TOTAL ORGANIC HALIDES	10	10	100.0	0.1528	0.0625	0.1890	0.2700	0.0590	0.1890
TOTAL SUSPENDED SOLIDS	9	9	100.0	487.8889	435.8527	758.1176	1300.0000	50.0000	758.1176
TRICHLOROETHENE	3	27	11.1	3.1630	2.7032	4.0505	2.1000	1.3000	2.1000
ZINC	12	27	44.4	221.6963	438.3930	365.6294	1530.0000	31.0000	365.6294

a. Arithmetic mean concentration, unless otherwise indicated.
b. 95th percentile upper confidence limit of the arithmetic mean concentration of an assumed normal distribution, unless otherwise indicated.
c. Lesser of the maximum concentration and the 95th percentile UCL of the mean concentration.
d. Shading indicates that the chemical was selected as a chemical of potential concern.
e. 95th Percentile UCL of the arithmetic mean concentration of an assumed lognormal distribution.

Table 6-A-10
Analytical Summary of Detected Compounds
Background
Surface Soils

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)
1,2,3,4,6,7,8-HPCDF	1	4	25.0	4.85E-05	4.23E-05	9.83E-05	1.10E-04	1.10E-04
2-BUTANONE	1	12	8.3	0.087	0.057	0.117	0.170	0.170
2-METHYLNAPHTHALENE	2	12	16.7	0.664	1.220	1.297	4.500	0.570
4,4'-DDD	4	12	33.3	0.093	0.317	0.257	1.100	0.001
4,4'-DDE	6	12	50.0	0.105	0.247	0.233	0.810	0.001
4,4'-DDT	6	12	50.0	0.783	2.682	2.173	9.300	0.004
ACENAPHTHENE	5	12	41.7	0.690	1.501	1.469	5.400	0.031
ACENAPHTHYLENE	4	12	33.3	0.296	0.200	0.400	0.610	0.044
ALPHA-CHLORDANE	1	12	8.3	0.001	0.001	0.002	0.003	0.003
ANTHRACENE	7	12	58.3	2.166	5.735	5.139	20.000	0.041
ARSENIC	11	12	91.7	10.847	10.128	16.098	36.900	2.700
BARIUM	12	12	100.0	44.033	73.765	82.278	275.000	8.700
BENZENE	1	12	8.3	0.039	0.028	0.054	0.043	0.043
BENZO(A)ANTHRACENE	7	12	58.3	3.198	8.016	7.354	28.000	0.280
BENZO(A)PYRENE	8	12	66.7	2.566	6.266	5.814	22.000	0.130
BENZO(B)FLUORANTHENE	9	12	75.0	4.160	10.273	9.486	36.000	0.026
BENZO(G,H,I)PERYLENE	8	12	66.7	1.570	3.429	3.348	12.000	0.080
BENZO(K)FLUORANTHENE	8	12	66.7	4.700	12.292	11.073	43.000	0.079
BERYLLIUM	12	12	100.0	0.487	0.238	0.610	0.980	0.160
BETA-BHC	1	12	8.3	0.023	0.075	0.061	0.260	0.260
BIS(2-ETHYLHEXYL)PHTHALATE	2	11	18.2	0.367	0.321	0.542	0.120	0.087
BUTYLBENZYLPHTHALATE	1	11	9.1	0.303	0.191	0.407	0.050	0.050
CADMIUM	2	8	25.0	0.333	0.208	0.473	0.780	0.520
CALCIUM	8	8	100.0	926.375	322.369	1142.357	1440.000	560.000
CHLOROFORM	1	12	8.3	0.039	0.028	0.053	0.032	0.032
CHROMIUM	12	12	100.0	10.292	4.546	12.649	20.000	4.800
CHRYSENE	9	12	75.0	3.445	8.534	7.869	30.000	0.140
COBALT	12	12	100.0	3.467	1.690	4.343	7.000	1.800
COPPER	12	12	100.0	10.425	8.809	14.992	32.400	3.700

Table 6-A-10
Analytical Summary of Detected Compounds
Background
Surface Soils

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)
CYANIDE	1	8	12.5	0.964	0.909	1.573	3.000	3.000
DIBENZ(A,H)ANTHRACENE	3	12	25.0	0.612	1.010	1.135	3.700	0.120
DIBENZOFURAN	4	12	33.3	1.116	2.732	2.532	9.700	0.043
DIELDRIN	2	12	16.7	0.002	0.002	0.003	0.004	0.001
DINOSEB	1	12	8.3	0.063	0.085	0.107	0.003	0.003
DISULFOTON	1	12	8.3	0.044	0.026	0.057	0.002	0.002
ENDOSULFAN SULFATE	1	12	8.3	0.003	0.002	0.004	0.006	0.006
ENDRIN	1	12	8.3	0.001	0.002	0.003	0.000	0.000
ENDRIN ALDEHYDE	1	12	8.3	0.002	0.002	0.003	0.001	0.001
ETHYL PARATHION	2	12	16.7	0.018	0.012	0.024	0.004	0.003
FLUORANTHENE	12	12	100.0	6.592	16.337	15.062	57.000	0.043
FLUORENE	5	12	41.7	1.105	2.651	2.479	9.400	0.053
GAMMA-CHLORDANE	1	12	8.3	0.001	0.002	0.002	0.005	0.005
HPCDF	1	4	25.0	0.000	0.000	0.000	0.000	0.000
INDENO(1,2,3-CD)PYRENE	7	12	58.3	1.830	4.029	3.919	14.000	0.230
IRON	8	8	100.0	12772.500	6867.927	17373.899	28300.000	7240.000
ISODRIN	1	12	8.3	0.002	0.002	0.003	0.003	0.003
KEPONE	2	12	16.7	0.022	0.052	0.049	0.180	0.055
LEAD	12	12	100.0	98.025	138.477	169.820	471.000	11.800
MAGNESIUM	8	8	100.0	1392.375	653.059	1829.914	2450.000	683.000
MANGANESE	8	8	100.0	202.525	145.874	300.258	476.000	53.200
MERCURY	4	12	33.3	0.133	0.226	0.250	0.810	0.060
METHYL PARATHION	4	12	33.3	0.005	0.003	0.007	0.005	0.003
METHYLENE CHLORIDE	5	12	41.7	0.295	0.665	0.639	0.120	0.010
NAPHTHALENE	4	12	33.3	0.861	2.042	1.920	7.300	0.023
NICKEL	8	12	66.7	5.608	3.277	7.307	13.300	3.200
OCDD	3	4	75.0	0.000	0.000	0.001	0.001	0.000
OCDF	1	4	25.0	0.000	0.000	0.000	0.000	0.000
P-PHENYLENEDIAMINE	1	11	9.1	2.041	1.545	2.885	5.900	5.900

Table 6-A-10
Analytical Summary of Detected Compounds
Background
Surface Soils

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)
PERCENT MOISTURE	12	12	100.0	12.317	6.833	15.859	22.000	2.000
PHENANTHRENE	11	12	91.7	7.210	19.742	17.446	69.000	0.052
POTASSIUM	8	8	100.0	504.250	144.534	601.086	786.000	349.000
PYRENE	12	12	100.0	6.284	16.002	14.580	56.000	0.038
SAFROLE	1	11	9.1	0.301	0.193	0.406	0.042	0.042
SELENIUM	3	12	25.0	0.350	0.284	0.497	1.100	0.490
SILVER	1	12	8.3	0.365	0.136	0.436	0.310	0.310
SODIUM	4	8	50.0	124.000	87.648	182.723	230.000	182.000
TETRACHLOROETHENE	1	12	8.3	0.042	0.028	0.056	0.011	0.011
THALLIUM	2	12	16.7	0.181	0.080	0.222	0.170	0.100
TIN	2	8	25.0	28.331	44.008	57.816	102.000	97.200
TOLUENE	4	12	33.3	0.140	0.337	0.315	1.200	0.027
VANADIUM	12	12	100.0	16.158	5.593	19.058	27.300	8.500
ZINC	12	12	100.0	46.442	56.900	75.942	219.000	13.300

a. Arithmetic mean concentration, unless otherwise indicated.

b. 95th percentile upper confidence limit of the arithmetic mean concentration of an assumed normal distribution, unless otherwise indicated.

Table 6-A-11
Analytical Summary of Detected Compounds
Background
Combined Soils

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)
1,2,3,4,6,7,8-HPCDF	1	9	11.1	3.81E-05	2.97E-05	5.65E-05	1.10E-04	1.10E-04
2-BUTANONE	1	17	5.9	0.066	0.058	0.091	0.170	0.170
2-METHYLNAPHTHALENE	2	17	11.8	0.525	1.036	0.963	4.500	0.570
4,4'-DDD	4	17	23.5	0.066	0.266	0.179	1.100	0.001
4,4'-DDE	6	17	35.3	0.075	0.210	0.164	0.810	0.001
4,4'-DDT	7	17	41.2	0.553	2.254	1.508	9.300	0.004
7,12-DIMETHYLBENZ(A)ANTHRACENE	1	16	6.3	0.929	1.116	1.418	0.160	0.160
ACENAPHTHENE	5	17	29.4	0.543	1.267	1.080	5.400	0.031
ACENAPHTHYLENE	4	17	23.5	0.265	0.173	0.338	0.610	0.044
ALPHA-CHLORDANE	1	17	5.9	0.001	0.001	0.002	0.003	0.003
ANTHRACENE	8	17	47.1	1.575	4.848	3.628	20.000	0.036
ARSENIC	14	17	82.4	8.036	9.562	12.085	36.900	0.740
BARIUM	17	17	100.0	35.124	62.876	61.750	275.000	6.400
BENZENE	1	17	5.9	0.029	0.029	0.041	0.043	0.043
BENZO(A)ANTHRACENE	9	17	52.9	2.293	6.802	5.173	28.000	0.088
BENZO(A)PYRENE	10	17	58.8	1.847	5.321	4.100	22.000	0.086
BENZO(B)FLUORANTHENE	11	17	64.7	2.976	8.726	6.671	36.000	0.026
BENZO(G,H,I)PERYLENE	9	17	52.9	1.156	2.919	2.393	12.000	0.045
BENZO(K)FLUORANTHENE	10	17	58.8	3.348	10.418	7.760	43.000	0.051
BERYLLIUM	16	17	94.1	0.490	0.262	0.601	0.980	0.160
BETA-BHC	1	17	5.9	0.016	0.063	0.043	0.260	0.260
BIS(2-ETHYLHEXYL)PHTHALATE	2	16	12.5	0.312	0.276	0.432	0.120	0.087
BUTYLBENZYLPHTHALATE	1	16	6.3	0.268	0.165	0.340	0.050	0.050
CADMIUM	2	9	22.2	0.313	0.204	0.440	0.780	0.520
CALCIUM	8	8	100.0	926.375	322.369	1142.357	1440.000	560.000
CHLOROFORM	2	17	11.8	0.028	0.028	0.040	0.032	0.007
CHROMIUM	17	17	100.0	8.788	4.591	10.733	20.000	3.200
CHRYSENE	11	17	64.7	2.482	7.241	5.548	30.000	0.088
COBALT	17	17	100.0	3.182	1.559	3.842	7.000	1.700

Table 6-A-11
Analytical Summary of Detected Compounds
Background
Combined Soils

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)
COPPER	17	17	100.0	9.082	7.632	12.314	32.400	3.700
CYANIDE	1	13	7.7	0.978	0.700	1.324	3.000	3.000
DIBENZ(A,H)ANTHRACENE	3	17	17.6	0.466	0.869	0.834	3.700	0.120
DIBENZOFURAN	4	17	23.5	0.844	2.306	1.820	9.700	0.043
DIELDRIN	2	17	11.8	0.002	0.002	0.003	0.004	0.001
DINOSEB	1	16	6.3	0.096	0.093	0.137	0.003	0.003
DISULFOTON	1	16	6.3	0.052	0.026	0.064	0.002	0.002
ENDOSULFAN SULFATE	1	17	5.9	0.003	0.002	0.004	0.006	0.006
ENDRIN	1	17	5.9	0.002	0.002	0.002	0.000	0.000
ENDRIN ALDEHYDE	1	17	5.9	0.002	0.002	0.003	0.001	0.001
ETHYL PARATHION	2	16	12.5	0.019	0.010	0.023	0.004	0.003
FLUORANTHENE	16	17	94.1	4.697	13.880	10.575	57.000	0.043
FLUORENE	5	17	29.4	0.836	2.239	1.784	9.400	0.053
GAMMA-CHLORDANE	1	17	5.9	0.001	0.001	0.002	0.005	0.005
HPCDD	1	9	11.1	0.000	0.000	0.000	0.000	0.000
HPCDF	1	9	11.1	0.000	0.000	0.000	0.000	0.000
INDENO(1,2,3-CD)PYRENE	9	17	52.9	1.320	3.438	2.776	14.000	0.050
IRON	8	8	100.0	12772.500	6867.927	17373.899	28300.000	7240.000
ISODRIN	1	17	5.9	0.002	0.002	0.003	0.003	0.003
KEPONE	2	17	11.8	0.019	0.044	0.037	0.180	0.055
LEAD	17	17	100.0	72.235	122.088	123.936	471.000	2.900
MAGNESIUM	8	8	100.0	1392.375	653.059	1829.914	2450.000	683.000
MANGANESE	8	8	100.0	202.525	145.874	300.258	476.000	53.200
MERCURY	4	17	23.5	0.107	0.192	0.188	0.810	0.060
METHOXYCHLOR	2	17	11.8	0.058	0.199	0.142	0.830	0.034
METHYL PARATHION	4	16	25.0	0.007	0.003	0.008	0.005	0.003
METHYLENE CHLORIDE	7	17	41.2	0.210	0.567	0.450	0.120	0.010
NAPHTHALENE	4	17	23.5	0.664	1.722	1.393	7.300	0.023
NICKEL	13	17	76.5	5.124	2.925	6.362	13.300	2.600

Table 6-A-11
Analytical Summary of Detected Compounds
Background
Combined Soils

COMPOUND	NUMBER OF DETECTIONS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC. (MG/KG)[a]	STANDARD DEVIATION (MG/KG)	95TH% UCL (MG/KG)[b]	MAX. CONC. (MG/KG)	MIN. CONC. (MG/KG)
OCDD	4	9	44.4	0.000	0.000	0.001	0.001	0.000
OCDF	1	9	11.1	0.000	0.000	0.000	0.000	0.000
P-PHENYLENEDIAMINE	1	16	6.3	1.706	1.362	2.303	5.900	5.900
PERCENT MOISTURE	15	15	100.0	12.253	6.480	15.200	22.000	2.000
PHENANTHRENE	13	17	76.5	5.141	16.700	12.213	69.000	0.052
POTASSIUM	8	8	100.0	504.250	144.534	601.086	786.000	349.000
PYRENE	16	17	94.1	4.482	13.576	10.231	56.000	0.037
SAFROLE	1	16	6.3	0.266	0.166	0.339	0.042	0.042
SELENIUM	3	17	17.6	0.349	0.290	0.472	1.100	0.490
SILVER	2	17	11.8	0.313	0.148	0.375	0.350	0.310
SODIUM	4	8	50.0	124.000	87.648	182.723	230.000	182.000
TETRACHLOROETHENE	1	17	5.9	0.031	0.029	0.043	0.011	0.011
THALLIUM	3	17	17.6	0.170	0.099	0.212	0.210	0.100
TIN	2	9	22.2	30.961	41.915	56.948	102.000	97.200
TOLUENE	4	17	23.5	0.100	0.287	0.221	1.200	0.027
VANADIUM	17	17	100.0	13.412	6.559	16.189	27.300	4.300
ZINC	17	17	100.0	40.241	48.538	60.795	219.000	12.700

a. Arithmetic mean concentration, unless otherwise indicated.

b. 95th percentile upper confidence limit of the arithmetic mean concentration of an assumed normal distribution, unless otherwise indicated.

Table 6-A-12
Analytical Summary of Detected Compounds
Background
Groundwater

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC.[a] (UG/L)	STANDARD DEVIATION (UG/L)	95TH% UCL[b] (UG/L)	MAX. CONC. (UG/L)	MIN. CONC. (UG/L)
2,4-DIMETHYLPHENOL	1	14	7.1	5.1250	0.7493	5.4797	7.7	7.7
2-METHYLNAPHTHALENE	2	14	14.3	8.0571	12.9836	14.2025	53	0.75
3&4-METHYLPHENOL	1	8	12.5	4.9625	0.1061	5.0336	4.7	4.7
4,4'-DDE	1	13	7.7	0.0329	0.0226	0.0440	0.0081	0.0081
4,4'-DDT	1	13	7.7	0.0356	0.0193	0.0452	0.024	0.024
ACENAPHTHENE	1	14	7.1	10.5750	21.1335	20.5779	84	84
ACENAPHTHYLENE	1	14	7.1	5.1321	0.7757	5.4993	7.8	7.8
ALDRIN	2	14	14.3	0.0196	0.0113	0.0249	0.042	0.013
ALPHA-BHC	2	13	15.4	0.0185	0.0090	0.0230	0.016	0.01
AMMONIA AS N	1	6	16.7	0.0108	0.0097	0.0188	0.03	0.03
ANTHRACENE	1	14	7.1	4.6679	0.9756	5.1296	1.3	1.3
ARSENIC	1	14	7.1	3.6143	2.0111	4.5662	8.6	8.6
BARIUM	13	14	92.9	24.8643	14.3922	31.6764	54	10.8
BENZO(A)PYRENE	1	14	7.1	4.6893	0.8960	5.1134	1.6	1.6
BENZO(B)FLUORANTHENE	1	14	7.1	4.7536	0.6579	5.0650	2.5	2.5
BENZO(G,H,I)PERYLENE	1	14	7.1	4.6607	1.0021	5.1351	1.2	1.2
BENZO(K)FLUORANTHENE	1	14	7.1	4.6364	1.0925	5.1535	0.86	0.86
BICARBONATE ALKALINITY	3	6	50.0	23.2500	25.9668	44.6108	57	34
BIOLOGICAL OXYGEN DEMAND - 5	3	3	100.0	2000.0000	0.0000	2000.0000	2000	2000
BIS(2-ETHYLHEXYL)PHTHALATE	1	14	7.1	4.8250	1.7933	5.6738	5.3	5.3
CALCIUM	9	9	100.0	24733.3333	9782.1266	30798.2518	40000	12700
CHEMICAL OXYGEN DEMAND	2	6	33.3	8.1667	8.7845	15.3929	20	19
CHLORIDE	6	6	100.0	26.5000	11.7260	36.1461	40	14
CHLOROBENZENE	1	14	7.1	2.4714	0.1069	2.5220	2.1	2.1
CHROMIUM	2	14	14.3	6.2929	3.6333	8.0126	18	10.1
CHRYSENE	1	14	7.1	4.7179	0.7900	5.0918	2	2
DIBENZOFURAN	2	14	14.3	7.6750	11.3578	13.0509	47	1.4
DIETHYLPHTHALATE	1	14	7.1	4.6464	1.0553	5.1459	1	1
ENDOSULFAN II	1	14	7.1	0.0342	0.0192	0.0432	0.0041	0.0041

Table 6-A-12
Analytical Summary of Detected Compounds
Background
Groundwater

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC.[a] (UG/L)	STANDARD DEVIATION (UG/L)	95TH% UCL[b] (UG/L)	MAX. CONC. (UG/L)	MIN. CONC. (UG/L)
ETHYLBENZENE	1	14	7.1	3.0357	2.0045	3.9845	10	10
FLUORANTHENE	3	14	21.4	4.5321	1.3022	5.1485	6.2	1.2
FLUORENE	2	14	14.3	7.2679	9.7472	11.8814	41	1.7
HEPTACHLOR EPOXIDE	1	13	7.7	0.0217	0.0156	0.0294	0.063	0.063
HPCDF	1	8	12.5	0.0002	0.0001	0.0003	0.00052	0.00052
HYDROCARBONS	1	6	16.7	0.3800	0.1495	0.5030	0.39	0.39
INDENO(1,2,3-CD)PYRENE	1	14	7.1	4.6536	1.0287	5.1405	1.1	1.1
IRON	5	9	55.6	2029.5000	3217.3990	4024.2874	8360	121
LANGLIER INDEX	3	3	100.0	-1.9733	1.0854	-0.1435	-0.72	-2.6
MAGNESIUM	9	9	100.0	3447.7778	1167.8161	4171.8238	5170	2100
MANGANESE	9	9	100.0	636.1111	679.7473	1057.5544	1920	71
NAPHTHALENE	2	14	14.3	38.5750	127.0552	98.7127	480	1
NICKEL	4	14	28.6	99.0000	157.4352	173.5172	480	139
NITRATE-NITRITE AS N	3	6	50.0	593.3333	828.3638	1274.7623	1800	230
NITROBENZENE	1	14	7.1	4.6307	1.1138	5.1579	0.78	0.78
OCDD	3	8	37.5	0.0012	0.0011	0.0019	0.0037	0.00036077
OIL AND GREASE	1	6	16.7	0.4167	0.1769	0.5622	0.61	0.61
ORTHOPHOSPHATE	5	6	83.3	0.1097	0.1403	0.2251	0.38	0.01
PHENANTHRENE	3	14	21.4	6.0036	3.8742	7.8373	19	3
POTASSIUM	7	9	77.8	3431.1111	1378.9438	4286.0563	5250	2400
PYRENE	2	14	14.3	4.7750	0.8394	5.1723	5.8	2
SILICA,DISSOLVED	9	9	100.0	28.2222	21.4288	41.5081	77	12
SODIUM	9	9	100.0	16512.2222	8299.7527	21658.0689	30200	8390
SULFATE	6	6	100.0	24.3167	10.5319	32.9805	35	7.9
TCDD	1	8	12.5	0.0050	0.0074	0.0100	0.01958771	0.01958771
TKN	4	6	66.7	0.2400	0.2952	0.4829	0.83	0.15
TOLUENE	1	14	7.1	2.5714	0.2673	2.6979	3.5	3.5
TOTAL ALKALINITY	9	9	100.0	51.2222	25.5038	67.0346	98	23
TOTAL DISSOLVED SOLIDS	6	6	100.0	186.6667	33.2666	214.0324	240	140

Table 6-A-12
Analytical Summary of Detected Compounds
Background
Groundwater

COMPOUND	NUMBER OF DETECTS	NUMBER OF SAMPLES	FREQUENCY OF DETECTION (%)	MEAN CONC.[a] (UG/L)	STANDARD DEVIATION (UG/L)	95TH% UCL[b] (UG/L)	MAX. CONC. (UG/L)	MIN. CONC. (UG/L)
TOTAL HARDNESS, AS CACO3	9	9	100.0	17427.5444	34573.5885	38863.1693	86000	1.9
TOTAL ORGANIC CARBON	5	6	83.3	5.1667	6.1138	10.1960	14	1.4
TOTAL ORGANIC HALIDES	6	6	100.0	0.1097	0.0637	0.1620	0.21	0.019
TOTAL SUSPENDED SOLIDS	6	6	100.0	1783.3333	1814.9674	3276.3624	4400	150
ZINC	7	14	50.0	27.5429	27.5469	40.5814	110	20

a. Arithmetic mean concentration, unless otherwise indicated.

b. 95th percentile upper confidence limit of the arithmetic mean concentration of an assumed normal distribution, unless otherwise indicated.

Appendix 6-B

Chemicals of Potential Concern Selection Process and Results

6-B1.0 Introduction

Chemicals of potential concern (COPCs) for the Public Health Risk Assessment (PHRA) were selected separately for the Production, Waste Water Treatment, and Warwick Areas. The selection process was based on USEPA guidance (USEPA, 1989a), previous discussions with USEPA Region I and an evaluation of the analytical data. The purpose of this selection process is to limit the PHRA to those chemicals which represent the dominant human health risks. The selection process was used for both chemicals present in near-site background soils, and for COPCs. The data evaluated include Phase I (Rounds 1 and 2) and Phase II (Rounds 1 and 2) RCRA Facility Investigation (RFI) soils data, and Phase I (Rounds 1 through 3) and Phase II (Rounds "S", 1, and 2) RFI groundwater data.

6-B2.0 Methodology

Only those chemicals which were detected in at least 5 percent of the surface soil samples from a Site area were included in the selection process. Chemicals that are infrequently detected may be artifacts in the data due to sampling errors, analytical errors, or other problems.

The first step in the selection of COPCs was the evaluation of background chemical concentrations. Background levels were evaluated for inorganic chemicals and PAHs. Inorganic chemicals are naturally present in soil. PAHs are ubiquitous in surface soil due to emissions from non-site-related combustion sources such as automobiles, industrial burners, and charcoal grills.

Background chemical concentrations were evaluated separately in each of the three Site areas using a quantitative concentration/toxicity relative ranking system (USEPA, 1989). The objective of this ranking procedure was to identify the PAHs and inorganics that are most likely to contribute significantly to risks at the Site. The ranking procedure has three steps. First a ranking factor was calculated for each chemical. This ranking factor is based on the soil or groundwater concentrations detected in each of the three Site areas and toxicity of the chemical. The ranking factor for each chemical in each medium was calculated as shown below:

$$R_i = (C_i)(T_i)$$

Where:

R_i = Ranking factor for chemical *i*.
C_i = Concentration of chemical *i*.
T_i = Toxicity criterion of chemical *i* (either the CSF or 1/RfD of chemical *i* see "Toxicity Assessment" (Section 6.0 of the PHRA) for description).

The concentration used is the 95th percentile UCL of the mean of the surface soil sample concentrations. For samples in which a chemical was not detected, one-half the sample detection or quantitation limit was assumed to be present. The Integrated Risk Information System (IRIS) on-line database, Health Effect Assessment Summary Tables (USEPA, 1994), Appendix X of the 1993 Revised Cranston RFI Interim Report and Phase 2 Work Plan (Ciba, 1993), and information from Ciba were the sources for toxicity criteria.

Next, a total score was calculated by summing the chemical-specific ranking factor values:

$$TS = R_i + R_{ii} + R_{iii} + \ldots + R_n$$

Where:

TS = Total score for all chemicals.
R_i = Ranking factor for chemical *i*.

Finally the relative ranking score of each chemical was determined by dividing its ranking factor by the total score:

$$RRS_i = R/TS$$

Where:

RRS_i= Relative ranking score of chemical *i*. (R_i and TS are as described above.)

Separate RRS values were calculated for cancer and noncancer effects. The results of the relative ranking system were used to select the inorganics and PAHs to be further evaluated. Inorganics and PAHs for each of the three media associated with a Site area were ordered, beginning with

the highest cancer- and noncancer-effects RRS values. Those comprising a cumulative RRS of 0.9 were the initial candidates for further evaluation. The COPCs for the Site areas were selected from those compounds with the highest RRS values among the three media.

The chemicals comprising a cumulative RRS of 0.9 were virtually the same for the surface and combined soils in each of three Site areas; this is true for both cancer-effects and noncancer-effects RRS . The chemicals which summed to a cumulative RRS of 0.9 for groundwater differed somewhat from those of the soil media in the same Site area. Because much more of the potential risk associated with the Site involves exposure to soil than exposure to groundwater (indirectly as surface water) and soil represents the source of groundwater impact, more emphasis was placed on inorganics and PAHs with high soil RRS than those with high RRS in groundwater.

The inorganics and PAHs with the greatest contributions to the RRS in each of the Site media are shown below:

Production Area

Arsenic	Benzo(a)pyrene	Chromium
Dibenz(a,h)anthracene	Nickel	

Waste Water Treatment Area

Arsenic	Benzo(a)pyrene	Beryllium
Chromium	Dibenz(a,h)anthracene	Manganese
Vanadium		

Warwick Area

Antimony	Arsenic	Beryllium
Benzo(a)pyrene	Cadmium	Chromium
Manganese	Zinc	

t-Tests were run, comparing the concentrations of each data set to those found in the background samples. The findings of the *t*-test results are summarized below:

Production Area:

- Nickel was the only selected inorganic or PAH that tested to be significantly above background concentrations. It was found to be above background in the combined surface and subsurface soil only.

Waste Water Treatment Area:

- No selected inorganic or PAH tested as being significantly above background.

Warwick Area:

- Five of the selected inorganics and none of the PAHs were determined to be present in surface and combine soils at above-background concentrations. These are:

  -antimony,
  -beryllium,
  -cadmium,
  -chromium, and
  -zinc.

  The latter four inorganics tested as being significantly above background concentrations. Antimony could not be tested statistically because all of the background soil data for antimony were rejected during data validation. Thus, it was assumed to be present in Warwick Area soils at above-background concentrations.

Concentration/toxicity screenings for organics, as described above for inorganics and PAHs, were performed to identify the COPCs for each Site area. Organic compounds, other than PAHs, were assumed for the purpose of risk characterization to be site-related. Based on concentration/toxicity screening and *t*-test results described above for inorganics and PAHs, nickel in Production Area surface soil, combined soil, and groundwater was assumed to be site-related, as were antimony, beryllium, cadmium, chromium, and zinc in the Warwick Area. These inorganics were included in the concentration/toxicity screens performed for COPC identification. Tables 6-B-28 through 6-B-54 show the concentration/toxicity screens used for the identification of COPCs.

The basic concentration/toxicity approach used for COPC identification is identical to that described above for background inorganics and PAHs. A maximum of eight COPCs was

selected for each Site area, including a minimum of three COPCs identified based on cancer effects, and three based on noncancer effects. More emphasis was placed on chemicals with higher soil RRS values than groundwater RRS values because soil represents the source of groundwater impact and much more potential exposure is associated with soil than with groundwater (indirectly as surface water). However, for each of the Site areas, the chemicals with the highest groundwater RRS values were selected as COPCs.

To ensure that all chemicals which contribute significantly to risk at the Site were included in the PHRA, the following iterative evaluation process was performed after completion of the risk analyses during the risk characterization process.

- For noncarcinogens--if a total hazard index of 1 is exceeded under any exposure scenario in the risk calculations of the selected COPCs, noncarcinogens with the highest remaining RRS values are added sequentially to the list of COPCs until two additional compounds with estimated hazard quotients of less than 0.5 are included on the list.

- For carcinogens--if the total incremental lifetime cancer risk (ILCR) associated with the selected COPCs exceeds 1×10^{-4}, under any exposure scenario, carcinogens with the highest remaining RRS values are added sequentially to the list of COPCs until two additional compounds with estimated ILCR values of less than 1×10^{-5} are included on the list.

6-B3.0 Results

The COPCs selected for the Production, Warwick, and Waste Water Treatment Areas are as follows:

PRODUCTION AREA

Noncarcinogens	*Carcinogens*
PCB 1248	PCB 1260
PCB 1254	*gamma*-Chlordane
2-Nitroaniline	Vinyl chloride
Chlorobenzene	
Toluene	

WARWICK AREA

Noncarcinogens	*Carcinogens*
PCB 1248	Aldrin
PCB 1254	Beryllium
2-Nitroaniline	Dieldrin
Chlorobenzene	*bis*(2-Chloroethyl)ether

WASTE WATER TREATMENT AREA

Noncarcinogens	*Carcinogens*
gamma-Chlordane	2,3,7,8-TCDF
PCB 1254	Dieldrin
Tinuvin 327	*bis*(2-Ethylhexyl)phthalate

Summaries of the data, background screening, and COPC screening are provided in Tables 6-B-1 through 6-B-54. These tables provide the following information for the environmental media in each area:

- Frequency of detection;
- Mean concentration;
- Reasonable Maximum Exposure (RME) concentration;

- Cancer and/or noncancer toxicity criteria; and
- A relative ranking score based on the above information.

The results of the COPC selection process were reviewed to ensure that the COPCs included chemicals known to be previously used or produced at the Site and those identified as concerns during previous discussions with USEPA, such as polychlorinated biphenyls (PCBs).

Table 6-B-1
Ranking Process for Potential Background Chemicals
Production Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RfD[e]	RRS-C[f]	RRS-N[g]
PAHs									
ACENAPHTHENE	10	41	24%	1.24	0.21	0.00E+00	6.0E-2	0.000	0.000
ACENAPHTHYLENE	5	41	12%	1.26	0.18	0.00E+00	4.0E-2	0.000	0.000
ANTHRACENE	24	41	59%	0.88	1.29	0.00E+00	3.0E-1	0.000	0.000
BENZO(A)ANTHRACENE	28	41	68%	1.14	1.52	7.30E-01	0.0E+0	0.022	0.000
BENZO(A)PYRENE	27	41	66%	1.29	1.69	7.30E+00	0.0E+0	0.246	0.000
BENZO(B)FLUORANTHENE	30	41	73%	1.57	1.98	7.30E-01	0.0E+0	0.029	0.000
BENZO(G,H,I)PERYLENE	21	41	51%	1.35	1.81	0.00E+00	4.0E-2	0.000	0.001
BENZO(K)FLUORANTHENE	27	41	66%	1.50	1.95	7.30E-02	0.0E+0	0.003	0.000
CHRYSENE	28	41	68%	1.24	1.62	7.30E-03	0.0E+0	0.000	0.000
DIBENZ(A,H)ANTHRACENE	10	41	24%	1.23	0.68	7.30E+00	0.0E+0	0.099	0.000
FLUORANTHENE	33	41	80%	1.60	2.05	0.00E+00	4.0E-2	0.000	0.001
FLUORENE	12	41	29%	1.21	0.18	0.00E+00	4.0E-2	0.000	0.000
INDENO(1,2,3-CD)PYRENE	21	41	51%	1.33	1.79	7.30E-01	0.0E+0	0.026	0.000
2-METHYLNAPHTHALENE	4	41	10%	1.26	0.38	0.00E+00	0.0E+0	0.000	0.000
NAPHTHALENE	14	41	34%	0.83	0.68	0.00E+00	4.0E-2	0.000	0.000
PHENANTHRENE	28	41	68%	1.15	1.54	0.00E+00	4.0E-2	0.000	0.001
PYRENE	32	41	78%	1.82	2.30	0.00E+00	3.0E-2	0.000	0.001
INORGANICS									
ARSENIC	29	32	91%	9.01	15.43	1.75E+00	3.0E-4	0.537	0.777
BARIUM	31	31	100%	46.54	56.20	0.00E+00	7.0E-2	0.000	0.012
BERYLLIUM	30	31	97%	0.40	0.45	4.30E+00	5.0E-3	0.039	0.001
CADMIUM	18	31	58%	0.65	0.87	0.00E+00	5.0E-4	0.000	0.026
CHROMIUM	29	31	94%	11.13	13.66	0.00E+00	5.0E-3	0.000	0.041
COBALT	29	31	94%	3.04	3.44	0.00E+00	6.0E-2	0.000	0.001
COPPER	29	31	94%	17.62	30.53	0.00E+00	3.7E-2	0.000	0.012
CYANIDE	11	34	32%	1.32	1.98	0.00E+00	2.0E-2	0.000	0.001
IRON	26	26	100%	10472.31	11821.61	0.00E+00	0.0E+0	0.000	0.000
LEAD	29	31	94%	54.31	79.46	0.00E+00	0.0E+0	0.000	0.000
MAGNESIUM	26	26	100%	2114.92	2540.43	0.00E+00	0.0E+0	0.000	0.000

Table 6-B-1
Ranking Process for Potential Background Chemicals
Production Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RfD[e]	RRS-C[f]	RRS-N[g]
MANGANESE	26	26	100%	166.93	189.23	0.00E+00	1.4E-1	0.000	0.020
MERCURY	22	30	73%	0.46	0.61	0.00E+00	3.0E-4	0.000	0.031
NICKEL	28	31	90%	7.77	11.62	0.00E+00	2.0E-2	0.000	0.009
POTASSIUM	25	26	96%	841.87	939.71	0.00E+00	0.0E+0	0.000	0.000
SODIUM	13	26	50%	150.62	176.76	0.00E+00	0.0E+0	0.000	0.000
VANADIUM	28	31	90%	14.98	20.76	0.00E+00	7.0E-3	0.000	0.045
ZINC	31	31	100%	183.61	367.87	0.00E+00	3.0E-1	0.000	0.019

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Cancer slope factor. "0" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[e]Chronic reference dose; "0" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

[f]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

[g]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-2
Ranking Process for Potential Background Chemicals Summary of Carcinogens
Production Area
Surface Soil

Detected Compound[a]	Detects No. of	Samples No. of	Frequency of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RRS-C[e]	Cumulative RRS-C
ARSENIC	29	32	91%	9.01	15.43	1.75E+00	0.537	0.537
BENZO(A)PYRENE	27	41	66%	1.29	1.69	7.30E+00	0.246	0.783
DIBENZ(A,H)ANTHRACENE	10	41	24%	1.23	0.68	7.30E+00	0.099	0.881
BERYLLIUM	30	31	97%	0.40	0.45	4.30E+00	0.039	0.920
BENZO(B)FLUORANTHENE	30	41	73%	1.57	1.98	7.30E-01	0.029	0.949
INDENO(1,2,3-CD)PYRENE	21	41	51%	1.33	1.79	7.30E-01	0.026	0.975
BENZO(A)ANTHRACENE	28	41	68%	1.14	1.52	7.30E-01	0.022	0.997
BENZO(K)FLUORANTHENE	27	41	66%	1.50	1.95	7.30E-02	0.003	1.000
CHRYSENE	28	41	68%	1.24	1.62	7.30E-03	0.000	1.000

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Cancer slope factor. "0" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[e]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-3
Ranking Process for Potential Background Chemicals Summary of Noncarcinogens
Production Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc[b] (mg/kg)	RME Conc.[c] (mg/kg)	RfD[d]	RRS-N[e]	Cumulative RRS-N
ARSENIC	29	32	91%	9.01	15.43	3.0E-4	0.786	0.786
VANADIUM	28	31	90%	14.98	20.76	7.0E-3	0.045	0.831
CHROMIUM	29	31	94%	11.13	13.66	5.0E-3	0.042	0.873
MERCURY	22	30	73%	0.46	0.61	3.0E-4	0.031	0.904
CADMIUM	18	31	58%	0.65	0.87	5.0E-4	0.027	0.931
MANGANESE	26	26	100%	166.93	189.23	1.4E-1	0.021	0.951
BARIUM	31	31	100%	46.54	56.20	7.0E-2	0.012	0.964
ZINC	31	31	100%	183.61	367.87	3.0E-1	0.012	0.976
COPPER	29	31	94%	17.62	30.53	3.7E-2	0.009	0.985
NICKEL	28	31	90%	7.77	11.62	2.0E-2	0.007	0.992
CYANIDE	11	34	32%	1.32	1.98	2.0E-2	0.002	0.994
BERYLLIUM	30	31	97%	0.40	0.45	5.0E-3	0.001	0.995
PYRENE	32	41	78%	1.82	2.30	3.0E-2	0.001	0.997
COBALT	29	31	94%	3.04	3.44	6.0E-2	0.001	0.997
FLUORANTHENE	33	41	80%	1.60	2.05	4.0E-2	0.001	0.998
BENZO(G,H,I)PERYLENE	21	41	51%	1.35	1.81	4.0E-2	0.001	0.999
PHENANTHRENE	28	41	68%	1.15	1.54	4.0E-2	0.001	0.999
NAPHTHALENE	14	41	34%	0.83	0.68	4.0E-2	0.000	1.000
ACENAPHTHYLENE	5	41	12%	1.26	0.18	4.0E-2	0.000	1.000
FLUORENE	12	41	29%	1.21	0.18	4.0E-2	0.000	1.000
ANTHRACENE	24	41	59%	0.88	1.29	3.0E-1	0.000	1.000
ACENAPHTHENE	10	41	24%	1.24	0.21	6.0E-2	0.000	1.000

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

Table 6-B-3
Ranking Process for Potential Background Chemicals Summary of Noncarcinogens
Production Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc[b] (mg/kg)	RME Conc.[c] (mg/kg)	RfD[d]	RRS-N[e]	Cumulative RRS-N

[d]Chronic reference dose; "0" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitue RfDs.

[e]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-4
Ranking Process for Potential Background Chemicals
Production Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RfD[e] (mg/kg-day)	RRS-C[f]	RRS-N[g]
PAHs									
ACENAPHTHENE	12	84	14%	0.840	0.280	0.0E+0	6.0E-2	0.000	0.000
ACENAPHTHYLENE	5	84	6%	0.855	0.180	0.0E+0	4.0E-2	0.000	0.000
ANTHRACENE	32	84	38%	0.659	0.889	0.0E+0	3.0E-1	0.000	0.000
BENZO(A)ANTHRACENE	41	84	49%	0.797	1.015	7.3E-1	0.0E+0	0.020	0.000
BENZO(A)PYRENE	44	84	52%	0.872	1.104	7.3E+0	0.0E+0	0.214	0.000
BENZO(B)FLUORANTHENE	52	84	62%	1.034	1.280	7.3E-1	0.0E+0	0.025	0.000
BENZO(G,H,I)PERYLENE	30	84	36%	0.910	1.168	0.0E+0	4.0E-2	0.000	0.000
BENZO(K)FLUORANTHENE	42	84	50%	0.970	1.228	7.3E-2	0.0E+0	0.002	0.000
CHRYSENE	43	84	51%	0.859	1.078	7.3E-3	0.0E+0	0.000	0.000
DIBENZ(A,H)ANTHRACENE	12	84	14%	0.809	0.680	7.3E+0	0.0E+0	0.132	0.000
FLUORANTHENE	60	84	71%	0.989	1.237	0.0E+0	4.0E-2	0.000	0.001
FLUORENE	17	84	20%	0.820	0.180	0.0E+0	4.0E-2	0.000	0.000
INDENO(1,2,3-CD)PYRENE	31	84	37%	0.874	1.134	7.3E-1	0.0E+0	0.022	0.000
2-METHYLNAPHTHALENE	5	84	6%	0.857	0.380	0.0E+0	0.0E+0	0.000	0.000
NAPHTHALENE	29	84	35%	0.549	0.680	0.0E+0	4.0E-2	0.000	0.000
PHENANTHRENE	49	84	58%	0.724	0.929	0.0E+0	4.0E-2	0.000	0.000
PYRENE	58	84	69%	1.193	1.476	0.0E+0	3.0E-2	0.000	0.001
INORGANICS									
ARSENIC	52	55	95%	7.77	11.50	1.8E+0	3.0E-4	0.536	0.775
BARIUM	52	52	100%	36.97	43.49	0.0E+0	7.0E-2	0.000	0.013
BERYLLIUM	49	52	94%	0.38	0.43	4.3E+0	5.0E-3	0.049	0.002
CADMIUM	21	54	39%	0.47	0.61	0.0E+0	5.0E-4	0.000	0.025
CHROMIUM	52	54	96%	10.23	11.82	0.0E+0	5.0E-3	0.000	0.048
COBALT	49	52	94%	3.49	3.91	0.0E+0	6.0E-2	0.000	0.001
COPPER	52	54	96%	15.52	22.96	0.0E+0	3.7E-2	0.000	0.010
CYANIDE	14	59	24%	1.44	2.04	0.0E+0	2.0E-2	0.000	0.002
LEAD	50	52	96%	37.21	52.80	0.0E+0	0.000	0.000	0.000
MAGNESIUM	43	43	100%	1973.56	2300.00	0.0E+0	0.000	0.000	0.000
MANGANESE	43	43	100%	160.35	179.04	0.0E+0	1.4E-1	0.000	0.026

Table 6-B-4
Ranking Process for Potential Background Chemicals
Production Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RfD[e] (mg/kg-day)	RRS-C[f]	RRS-N[g]
MERCURY	32	52	62%	0.34	0.45	0.0E+0	3.0E-4	0.000	0.030
NICKEL	49	54	91%	8.16	11.50	0.0E+0	2.0E-2	0.000	0.010
THALLIUM	1	48	2%	0.20	0.21	0.0E+0	0.0E+0	NA	NA
TIN	1	43	2%	4.85	5.70	0.0E+0	6.0E-1	NA	NA
VANADIUM	45	52	87%	12.36	15.88	0.0E+0	7.0E-3	0.000	0.046
ZINC	51	54	94%	130.72	218.50	0.0E+0	3.0E-1	0.000	0.011

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Cancer slope factor. "0" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[e]Chronic reference dose; "0" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

[f]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

[g]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-5
Ranking Process for Potential Background Chemicals Summary of Carcinogens
Production Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RRS-C[e]	Cumulative RRS-C
ARSENIC	52	55	95%	7.77	11.50	1.8E+0	0.536	0.536
BENZO(A)PYRENE	44	84	52%	0.872	1.104	7.3E+0	0.214	0.750
DIBENZ(A,H)ANTHRACENE	12	84	14%	0.809	0.680	7.3E+0	0.132	0.882
BERYLLIUM	49	52	94%	0.38	0.43	4.3E+0	0.049	0.931
BENZO(B)FLUORANTHENE	52	84	62%	1.034	1.280	7.3E-1	0.025	0.956
INDENO(1,2,3-CD)PYRENE	31	84	37%	0.874	1.134	7.3E-1	0.022	0.978
BENZO(A)ANTHRACENE	41	84	49%	0.797	1.015	7.3E-1	0.020	0.997
BENZO(K)FLUORANTHENE	42	84	50%	0.970	1.228	7.3E-2	0.002	1.000
CHRYSENE	43	84	51%	0.859	1.078	7.3E-3	0.000	1.000

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Cancer slope factor. "0" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[e]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-6
Ranking Process for Potential Background Chemicals Summary of Noncarcinogens
Production Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	RfD[d] (mg/kg-day)	RRS-N[e]	Cumulative RRS-N
ARSENIC	52	55	95%	7.77	11.50	3.0E-4	0.775	0.775
CHROMIUM	52	54	96%	10.23	11.82	5.0E-3	0.048	0.823
VANADIUM	45	52	87%	12.36	15.88	7.0E-3	0.046	0.869
MERCURY	32	52	62%	0.34	0.45	3.0E-4	0.030	0.899
MANGANESE	43	43	100%	160.35	179.04	1.4E-1	0.026	0.924
CADMIUM	21	54	39%	0.47	0.61	5.0E-4	0.025	0.949
BARIUM	52	52	100%	36.97	43.49	7.0E-2	0.013	0.962
ZINC	51	54	94%	130.72	218.50	3.0E-1	0.011	0.973
COPPER	52	54	96%	15.52	22.96	3.7E-2	0.010	0.983
NICKEL	49	54	91%	8.16	11.50	2.0E-2	0.010	0.993
CYANIDE	14	59	24%	1.44	2.04	2.0E-2	0.002	0.995
BERYLLIUM	49	52	94%	0.38	0.43	5.0E-3	0.002	0.996
COBALT	49	52	94%	3.49	3.91	6.0E-2	0.001	0.998
PYRENE	58	84	69%	1.193	1.476	3.0E-2	0.001	0.999
FLUORANTHENE	60	84	71%	0.989	1.237	4.0E-2	0.001	0.999
NAPHTHALENE	29	84	35%	0.549	0.680	4.0E-2	0.000	1.000
ACENAPHTHENE	12	84	14%	0.840	0.280	6.0E-2	0.000	1.000
FLUORENE	17	84	20%	0.820	0.180	4.0E-2	0.000	1.000
ANTHRACENE	32	84	38%	0.659	0.889	3.0E-1	0.000	1.000

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Chronic reference dose; "0" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitue RfDs.

[e]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-7
Ranking Process for Potential Background Chemicals
Production Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] $(mg/kg\text{-}day)^{-1}$	RfD[e] (mg/kg-day)	RRS-C[f]	RRS-N[g]
PAHs									
BENZO(A)ANTHRACENE	2	50	4%	5.60	1.00	7.3E-1	0.0E+0	NA	NA
BENZO(A)PYRENE	1	50	2%	5.69	2.00	7.3E+0	0.0E+0	NA	NA
BENZO(B)FLUORANTHENE	3	50	6%	5.52	1.00	7.3E-1	0.0E+0	0.032	0.000
BENZO(G,H,I)PERYLENE	3	50	6%	5.62	3.00	0.0E+0	4.0E-2	0.000	0.002
BENZO(K)FLUORANTHENE	3	50	6%	5.54	2.00	7.3E-2	0.0E+0	0.006	0.000
CHRYSENE	1	50	2%	5.67	0.70	7.3E-3	0.0E+0	NA	NA
DIBENZ(A,H)ANTHRACENE	1	50	2%	5.71	3.00	7.3E+0	0.0E+0	NA	NA
INDENO(1,2,3-CD)PYRENE	2	50	4%	5.65	3.00	7.3E-1	0.0E+0	NA	NA
2-METHYLNAPHTHALENE	3	50	6%	5.50	1.20	0.0E+0	0.0E+0	0.000	0.000
NAPHTHALENE	21	50	42%	14.44	24.85	0.0E+0	4.0E-2	0.000	0.014
PHENANTHRENE	4	50	8%	5.43	1.00	0.0E+0	4.0E-2	0.000	0.001
PYRENE	10	50	20%	5.19	6.24	0.0E+0	3.0E-2	0.000	0.005
INORGANICS									
ANTIMONY	1	50	2%	11.88	13.40	0.0E+0	4.0E-4	NA	NA
ARSENIC	24	50	48%	9.75	12.63	1.8E+0	3.0E-4	0.962	0.938
BARIUM	50	50	100%	49.49	62.34	0.0E+0	7.0E-2	0.000	0.020
CADMIUM	1	50	2%	2.61	2.80	0.0E+0	5.0E-4	NA	NA
COBALT	2	50	4%	5.42	5.94	0.0E+0	6.0E-2	NA	NA
LEAD	4	50	8%	3.73	5.42	0.0E+0	0.0E+0	0.000	0.000
NICKEL	3	50	6%	16.49	18.33	0.0E+0	2.0E-2	0.000	0.020
SILVER	1	50	2%	5.42	6.13	0.0E+0	5.0E-3	NA	NA
TIN	1	50	2%	37.64	40.69	0.0E+0	6.0E-1	NA	NA
ZINC	3	50	6%	12.56	13.94	0.0E+0	3.0E-1	0.000	0.001

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum

Table 6-B-7
Ranking Process for Potential Background Chemicals
Production Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RfD[e] (mg/kg-day)	RRS-C[f]	RRS-N[g]

detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Cancer slope factor. "0" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[e]Chronic reference dose; "0" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

[f]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

[g]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-8
Ranking Process for Potential Background Chemicals Summary of Carcinogens
Production Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RRS-C[e]	Cumulative RRS-C
ARSENIC	24	50	48%	9.75	12.63	1.75E+00	0.962	0.962
BENZO(B)FLUORANTHENE	3	50	6%	5.52	1.00	7.30E-01	0.032	0.994
BENZO(K)FLUORANTHENE	3	50	6%	5.54	2.00	7.30E-02	0.006	1.000

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Cancer slope factor. "0" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[e]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-9
Ranking Process for Potential Background Chemicals Summary of Noncarcinogens
Production Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	RfD[d] (mg/kg-day)	RRS-N[e]	Cumulative RRS-N
ARSENIC	24	50	48%	9.75	12.63	3.0E-4	0.940	0.940
NICKEL	3	50	6%	16.49	18.33	2.0E-2	0.020	0.961
BARIUM	50	50	100%	49.49	62.34	7.0E-2	0.020	0.980
NAPHTHALENE	21	50	42%	14.44	24.85	4.0E-2	0.014	0.994
PYRENE	10	50	20%	5.19	6.24	3.0E-2	0.005	0.999
ZINC	3	50	6%	12.56	13.94	3.0E-1	0.001	1.000

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Chronic reference dose; "0" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitue RfDs.

[e]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-10
Ranking Process for Potential Background Chemicals
Waste Water Treatment Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RfD[e] (mg/kg-day)	RRS-C[f]	RRS-N[g]
PAHs									
ACENAPHTHENE	1	18	6%	0.774	0.600	0.0E+0	6.0E-2	0.000	0.000
ACENAPHTHYLENE	1	18	6%	0.792	0.045	0.0E+0	4.0E-2	0.000	0.000
ANTHRACENE	2	18	11%	0.726	1.234	0.0E+0	3.0E-1	0.000	0.000
BENZO(A)ANTHRACENE	6	18	33%	0.861	1.452	7.3E-1	0.0E+0	0.020	0.000
BENZO(A)PYRENE	5	18	28%	0.865	1.464	7.3E+0	0.0E+0	0.207	0.000
BENZO(B)FLUORANTHENE	11	18	61%	1.039	1.632	7.3E-1	0.0E+0	0.023	0.000
BENZO(G,H,I)PERYLENE	3	18	17%	0.898	1.442	0.0E+0	4.0E-2	0.000	0.001
BENZO(K)FLUORANTHENE	8	18	44%	0.948	1.548	7.3E-2	0.0E+0	0.002	0.000
CHRYSENE	9	18	50%	0.860	1.464	7.3E-3	0.0E+0	0.000	0.000
DIBENZ(A,H)ANTHRACENE	1	18	6%	1.386	2.997	7.3E+0	0.0E+0	0.423	0.000
FLUORANTHENE	12	18	67%	0.812	1.468	0.0E+0	4.0E-2	0.000	0.001
FLUORENE	2	18	11%	0.766	0.510	0.0E+0	4.0E-2	0.000	0.000
INDENO(1,2,3-CD)PYRENE	4	18	22%	0.852	1.393	7.3E-1	0.0E+0	0.020	0.000
2-METHYLNAPHTHALENE	3	18	17%	0.444	0.500	0.0E+0	0.0E+0	0.000	0.000
NAPHTHALENE	9	18	50%	0.568	0.866	0.0E+0	4.0E-2	0.000	0.001
PHENANTHRENE	14	18	78%	0.815	1.360	0.0E+0	4.0E-2	0.000	0.001
PYRENE	16	18	89%	1.499	2.403	0.0E+0	3.0E-2	0.000	0.002
INORGANICS									
ARSENIC	15	15	100%	5.918	7.712	1.8E+0	3.0E-4	0.261	0.644
BARIUM	15	15	100%	28.807	36.287	0.0E+0	7.0E-2	0.000	0.013
BERYLLIUM	14	15	93%	0.434	0.521	4.3E+0	5.0E-3	0.043	0.003
CADMIUM	1	9	11%	0.271	0.381	0.0E+0	5.0E-4	0.000	0.019
COBALT	15	15	100%	3.433	3.877	0.0E+0	6.0E-2	0.000	0.002
CHROMIUM	15	15	100%	10.020	12.232	0.0E+0	5.0E-3	0.000	0.061
COPPER	18	18	100%	37.228	75.130	0.0E+0	3.7E-2	0.000	0.051
CYANIDE	4	14	29%	0.958	1.298	0.0E+0	2.0E-2	0.000	0.002
IRON	9	9	100%	12126.667	13982.055	0.0E+0	0.0E+0	0.000	0.000
LEAD	15	15	100%	36.580	48.947	0.0E+0	0.0E+0	0.000	0.000
MAGNESIUM	9	9	100%	1403.000	1616.466	0.0E+0	0.0E+0	0.000	0.000

Table 6-B-10
Ranking Process for Potential Background Chemicals
Waste Water Treatment Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RfD[e] (mg/kg-day)	RRS-C[f]	RRS-N[g]
MANGANESE	9	9	100%	199.511	273.459	0.0E+0	1.4E-1	0.000	0.049
MERCURY	6	10	60%	0.148	0.238	0.0E+0	3.0E-4	0.000	0.020
NICKEL	14	15	93%	7.633	10.150	0.0E+0	2.0E-2	0.000	0.013
SELENIUM	1	15	7%	0.169	0.130	0.0E+0	5.0E-3	0.000	0.001
SILVER	8	18	44%	0.964	1.401	0.0E+0	5.0E-3	0.000	0.007
VANADIUM	15	15	100%	10.080	11.521	0.0E+0	7.0E-3	0.000	0.041
ZINC	15	15	100%	286.793	840.000	0.0E+0	3.0E-1	0.000	0.070

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Cancer slope factor. "0" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[e]Chronic reference dose; "0" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

[f]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

[g]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-11
Ranking Process for Potential Background Chemicals Summary of Carcinogens
Waste Water Treatment Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RRS-C[e]	Cumulative RRS-C
DIBENZ(A,H)ANTHRACENE	1	18	6%	1.386	2.997	7.3E+0	0.423	0.423
ARSENIC	15	15	100%	5.918	7.712	1.8E+0	0.261	0.684
BENZO(A)PYRENE	5	18	28%	0.865	1.464	7.3E+0	0.207	0.891
BERYLLIUM	14	15	93%	0.434	0.521	4.3E+0	0.043	0.934
BENZO(B)FLUORANTHENE	11	18	61%	1.039	1.632	7.3E-1	0.023	0.957
BENZO(A)ANTHRACENE	6	18	33%	0.861	1.452	7.3E-1	0.020	0.978
INDENO(1,2,3-CD)PYRENE	4	18	22%	0.852	1.393	7.3E-1	0.020	0.998
BENZO(K)FLUORANTHENE	8	18	44%	0.948	1.548	7.3E-2	0.002	1.000
CHRYSENE	9	18	50%	0.860	1.464	7.3E-3	0.000	1.000

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Cancer slope factor. "0" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[e]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-12
Ranking Process for Potential Background Chemicals Summary of Noncarcinogens
Waste Water Treatment Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	RfD[d] (mg/kg-day)	RRS-N[e]	Cumulative RRS-N
ARSENIC	15	15	100%	5.918	7.712	3.0E-4	0.678	0.678
CHROMIUM	15	15	100%	10.020	12.232	5.0E-3	0.065	0.743
MANGANESE	9	9	100%	199.511	273.459	1.4E-1	0.052	0.794
VANADIUM	15	15	100%	10.080	11.521	7.0E-3	0.043	0.838
ZINC	15	15	100%	286.793	840.000	3.0E-1	0.037	0.875
COPPER	18	18	100%	37.228	75.130	3.7E-2	0.037	0.913
MERCURY	6	10	60%	0.148	0.238	3.0E-4	0.021	0.934
CADMIUM	1	9	11%	0.271	0.381	5.0E-4	0.020	0.954
BARIUM	15	15	100%	28.807	36.287	7.0E-2	0.014	0.967
NICKEL	14	15	93%	7.633	10.150	2.0E-2	0.012	0.980
SILVER	8	18	44%	0.964	1.401	5.0E-3	0.007	0.987
BERYLLIUM	14	15	93%	0.434	0.521	5.0E-3	0.003	0.990
PYRENE	16	18	89%	1.499	2.403	3.0E-2	0.002	0.992
CYANIDE	4	14	29%	0.958	1.298	2.0E-2	0.002	0.993
COBALT	15	15	100%	3.433	3.877	6.0E-2	0.002	0.995
FLUORANTHENE	12	18	67%	0.812	1.468	4.0E-2	0.001	0.996
BENZO(G,H,I)PERYLENE	3	18	17%	0.898	1.442	4.0E-2	0.001	0.997
PHENANTHRENE	14	18	78%	0.815	1.360	4.0E-2	0.001	0.998
SELENIUM	1	15	7%	0.169	0.130	5.0E-3	0.001	0.999
NAPHTHALENE	9	18	50%	0.568	0.866	4.0E-2	0.001	0.999
FLUORENE	2	18	11%	0.766	0.510	4.0E-2	0.000	1.000
ACENAPHTHENE	1	18	6%	0.774	0.600	6.0E-2	0.000	1.000
ANTHRACENE	2	18	11%	0.726	1.234	3.0E-1	0.000	1.000
ACENAPHTHYLENE	1	18	6%	0.792	0.045	4.0E-2	0.000	1.000

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation

Table 6-B-12
Ranking Process for Potential Background Chemicals Summary of Noncarcinogens
Waste Water Treatment Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	RfD[d] (mg/kg-day)	RRS-N[e]	Cumulative RRS-N

limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Chronic reference dose; "0" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitue RfDs.

[e]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-13
Ranking Process for Potential Background Chemicals
Waste Water Treatment Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RfD[e] (mg/kg-day)	RRS-C[f]	RRS-N[g]
PAHs									
ACENAPHTHENE	2	31	6%	0.641	0.600	0.0E+0	6.0E-2	0.000	0.000
ACENAPHTHYLENE	3	31	10%	0.648	0.086	0.0E+0	4.0E-2	0.000	0.000
ANTHRACENE	6	31	19%	0.615	0.922	0.0E+0	3.0E-1	0.000	0.000
BENZO(A)ANTHRACENE	8	31	26%	0.709	1.062	7.3E-1	0.0E+0	0.021	0.000
BENZO(A)PYRENE	8	31	26%	0.711	1.068	7.3E+0	0.0E+0	0.206	0.000
BENZO(B)FLUORANTHENE	18	31	58%	0.832	1.188	7.3E-1	0.0E+0	0.023	0.000
BENZO(G,H,I)PERYLENE	5	31	16%	0.713	1.044	0.0E+0	4.0E-2	0.000	0.001
BENZO(K)FLUORANTHENE	14	31	45%	0.739	1.101	7.3E-2	0.0E+0	0.002	0.000
CHRYSENE	14	31	45%	0.709	1.068	7.3E-3	0.0E+0	0.000	0.000
DIBENZ(A,H)ANTHRACENE	3	31	10%	0.982	1.902	7.3E+0	0.0E+0	0.368	0.000
FLUORANTHENE	15	31	48%	0.728	1.117	0.0E+0	4.0E-2	0.000	0.001
FLUORENE	4	31	13%	0.632	0.510	0.0E+0	4.0E-2	0.000	0.000
INDENO(1,2,3-CD)PYRENE	10	31	32%	0.686	1.014	7.3E-1	0.0E+0	0.020	0.000
2-METHYLNAPHTHALENE	3	31	10%	0.453	0.500	0.0E+0	0.0E+0	0.000	0.000
NAPHTHALENE	11	31	35%	0.520	0.720	0.0E+0	4.0E-2	0.000	0.001
PHENANTHRENE	21	31	68%	0.739	1.070	0.0E+0	4.0E-2	0.000	0.001
PYRENE	23	31	74%	1.191	1.724	0.0E+0	3.0E-2	0.000	0.002
INORGANICS									
ARSENIC	23	23	100%	5.220	6.416	1.8E+0	3.0E-4	0.297	0.669
BARIUM	23	23	100%	25.622	30.778	0.0E+0	7.0E-2	0.000	0.014
BERYLLIUM	20	23	87%	0.473	0.558	4.3E+0	5.0E-3	0.064	0.003
CADMIUM	1	15	7%	0.243	0.306	0.0E+0	5.0E-4	0.000	0.019
CHROMIUM	23	23	100%	8.922	10.441	0.0E+0	5.0E-3	0.000	0.065
COBALT	23	23	100%	3.557	3.872	0.0E+0	6.0E-2	0.000	0.002
COPPER	31	31	100%	28.258	37.510	0.0E+0	3.7E-2	0.000	0.032
CYANIDE	6	18	33%	0.918	1.196	0.0E+0	2.0E-2	0.000	0.002
IRON	15	15	100%	12089.333	13177.523	0.0E+0	0.00	0.000	0.000
LEAD	23	23	100%	28.557	37.556	0.0E+0	0.00	0.000	0.000

Table 6-B-13
Ranking Process for Potential Background Chemicals
Waste Water Treatment Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RfD[e] (mg/kg-day)	RRS-C[f]	RRS-N[g]
MAGNESIUM	15	15	100%	1429.800	1570.339	0.0E+0	0.00	0.000	0.000
MANGANESE	15	15	100%	186.640	228.451	0.0E+0	1.4E-1	0.000	0.051
MERCURY	7	17	41%	0.107	0.164	0.0E+0	3.0E-4	0.000	0.017
NICKEL	19	23	83%	6.402	8.450	0.0E+0	2.0E-2	0.000	0.013
POTASSIUM	15	15	100%	845.467	946.541	0.0E+0	0.00	0.000	0.000
SELENIUM	2	23	9%	0.186	0.213	0.0E+0	5.0E-3	0.000	0.001
SILVER	13	31	42%	0.725	0.986	0.0E+0	5.0E-3	0.000	0.006
VANADIUM	23	23	100%	9.252	10.273	0.0E+0	7.0E-3	0.000	0.046
ZINC	23	23	100%	215.935	505.730	0.0E+0	3.0E-1	0.000	0.053

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Cancer slope factor. "0" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[e]Chronic reference dose; "0" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

[f]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

[g]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-14
Ranking Process for Potential Background Chemicals Summary of Carcinogens
Waste Water Treatment Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RRS-C[e]	Cumulative RRS-C
DIBENZ(A,H)ANTHRACENE	3	31	10%	0.982	1.902	7.3E+0	0.368	0.368
ARSENIC	23	23	100%	5.220	6.416	1.8E+0	0.297	0.665
BENZO(A)PYRENE	8	31	26%	0.711	1.068	7.3E+0	0.206	0.871
BERYLLIUM	20	23	87%	0.473	0.558	4.3E+0	0.064	0.935
BENZO(B)FLUORANTHENE	18	31	58%	0.832	1.188	7.3E-1	0.023	0.958
BENZO(A)ANTHRACENE	8	31	26%	0.709	1.062	7.3E-1	0.021	0.978
INDENO(1,2,3-CD)PYRENE	10	31	32%	0.686	1.014	7.3E-1	0.020	0.998
BENZO(K)FLUORANTHENE	14	31	45%	0.739	1.101	7.3E-2	0.002	1.000
CHRYSENE	14	31	45%	0.709	1.068	7.3E-3	0.000	1.000

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Cancer slope factor. "0" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[e]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-15
Ranking Process for Potential Background Chemicals Summary of Noncarcinogens
Waste Water Treatment Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	RfD[d] (mg/kg-day)	RRS-N[e]	Cumulative RRS-N
ARSENIC	23	23	100%	5.220	6.416	3.0E-4	0.684	0.684
CHROMIUM	23	23	100%	8.922	10.441	5.0E-3	0.067	0.751
MANGANESE	15	15	100%	186.640	228.451	1.4E-1	0.052	0.803
VANADIUM	23	23	100%	9.252	10.273	7.0E-3	0.047	0.850
ZINC	23	23	100%	215.935	505.730	3.0E-1	0.033	0.883
COPPER	31	31	100%	28.258	37.510	3.7E-2	0.033	0.916
CADMIUM	1	15	7%	0.243	0.306	5.0E-4	0.020	0.935
MERCURY	7	17	41%	0.107	0.164	3.0E-4	0.017	0.953
BARIUM	23	23	100%	25.622	30.778	7.0E-2	0.014	0.967
NICKEL	19	23	83%	6.402	8.450	2.0E-2	0.012	0.979
SILVER	13	31	42%	0.725	0.986	5.0E-3	0.006	0.985
BERYLLIUM	20	23	87%	0.473	0.558	5.0E-3	0.004	0.989
COBALT	23	23	100%	3.557	3.872	6.0E-2	0.002	0.991
CYANIDE	6	18	33%	0.918	1.196	2.0E-2	0.002	0.993
PYRENE	23	31	74%	1.191	1.724	3.0E-2	0.002	0.995
SELENIUM	2	23	9%	0.186	0.213	5.0E-3	0.001	0.996
FLUORANTHENE	15	31	48%	0.728	1.117	4.0E-2	0.001	0.997
PHENANTHRENE	21	31	68%	0.739	1.070	4.0E-2	0.001	0.998
BENZO(G,H,I)PERYLENE	5	31	16%	0.713	1.044	4.0E-2	0.001	0.999
NAPHTHALENE	11	31	35%	0.520	0.720	4.0E-2	0.001	0.999
FLUORENE	4	31	13%	0.632	0.510	4.0E-2	0.000	1.000
ACENAPHTHENE	2	31	6%	0.641	0.600	6.0E-2	0.000	1.000
ANTHRACENE	6	31	19%	0.615	0.922	3.0E-1	0.000	1.000
ACENAPHTHYLENE	3	31	10%	0.648	0.086	4.0E-2	0.000	1.000

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

Table 6-B-15
Ranking Process for Potential Background Chemicals Summary of Noncarcinogens
Waste Water Treatment Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	RfD[d] (mg/kg-day)	RRS-N[e]	Cumulative RRS-N

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Chronic reference dose; "0" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitue RfDs.

[e]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-16
Ranking Process for Potential Background Chemicals
Waste Water Treatment Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RfD[e] (mg/kg-day)	RRS-C[f]	RRS-N[g]
PAHs									
ACENAPHTHENE	2	24	8%	4.597	1.100	0.0E+0	6.0E-2	0.000	0.000
ANTHRACENE	3	24	13%	4.438	1.000	0.0E+0	3.0E-1	0.000	0.000
BENZO(A)ANTHRACENE	2	24	8%	4.654	1.800	7.3E-1	0.0E+0	0.045	0.000
BENZO(A)PYRENE	2	24	8%	4.650	1.400	7.3E+0	0.0E+0	0.346	0.000
BENZO(B)FLUORANTHENE	3	24	13%	4.542	2.300	7.3E-1	0.0E+0	0.057	0.000
BENZO(K)FLUORANTHENE	2	24	8%	4.742	2.900	7.3E-2	0.0E+0	0.007	0.000
CHRYSENE	3	24	13%	4.529	1.900	7.3E-3	0.0E+0	0.000	0.000
FLUORANTHENE	4	24	17%	4.642	4.200	0.0E+0	4.0E-2	0.000	0.000
FLUORENE	1	24	4%	4.754	0.500	0.0E+0	4.0E-2	NA	NA
2-METHYLNAPHTHALENE	4	24	17%	4.858	5.002	0.0E+0	0.0E+0	0.000	0.000
NAPHTHALENE	6	24	25%	6.033	7.242	0.0E+0	4.0E-2	0.000	0.001
PHENANTHRENE	2	24	8%	4.625	1.100	0.0E+0	4.0E-2	0.000	0.000
PYRENE	4	24	17%	4.533	3.200	0.0E+0	3.0E-2	0.000	0.000
INORGANICS									
ARSENIC	8	24	33%	6.683	9.190	1.8E+0	3.0E-4	0.545	0.113
BARIUM	24	24	100%	56.896	72.850	0.0E+0	7.0E-2	0.000	0.004
COBALT	1	24	4%	5.242	5.656	0.0E+0	6.0E-2	NA	NA
IRON	20	24	83%	6172.635	7675.616	0.0E+0	0.0E+0	0.000	0.000
MAGNESIUM	24	24	100%	4115.000	4579.242	0.0E+0	0.0E+0	0.000	0.000
MANGANESE	24	24	100%	992.000	1199.448	0.0E+0	5.0E-3	0.000	0.882
NICKEL	1	24	4%	16.450	19.192	0.0E+0	2.0E-2	NA	NA
SILVER	1	24	4%	5.225	5.611	0.0E+0	5.0E-3	NA	NA
ZINC	3	24	13%	16.033	22.032	0.0E+0	3.0E-1	0.000	0.000

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation

Table 6-B-16
Ranking Process for Potential Background Chemicals
Waste Water Treatment Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RfD[e] (mg/kg-day)	RRS-C[f]	RRS-N[g]

limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Cancer slope factor. "0" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[e]Chronic reference dose; "0" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

[f]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

[g]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-17
Ranking Process for Potential Background Chemicals Summary of Carcinogens
Waste Water Treatment Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RRS-C[e]	Cumulative RRS-C
ARSENIC	8	24	33%	6.683	9.190	1.8E+0	0.545	0.545
BENZO(A)PYRENE	2	24	8%	4.650	1.400	7.3E+0	0.346	0.891
BENZO(B)FLUORANTHENE	3	24	13%	4.542	2.300	7.3E-1	0.057	0.948
BENZO(A)ANTHRACENE	2	24	8%	4.654	1.800	7.3E-1	0.045	0.992
BENZO(K)FLUORANTHENE	2	24	8%	4.742	2.900	7.3E-2	0.007	1.000
CHRYSENE	3	24	13%	4.529	1.900	7.3E-3	0.000	1.000

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Cancer slope factor. "0" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[e]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-18
Ranking Process for Potential Background Chemicals Summary of Noncarcinogens
Waste Water Treatment Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	RfD[d] (mg/kg-day)	RRS-N[e]	Cumulative RRS-N
MANGANESE	24	24	100%	992.000	1199.448	5.0E-3	0.882	0.882
ARSENIC	8	24	33%	6.683	9.190	3.0E-4	0.113	0.994
BARIUM	24	24	100%	56.896	72.850	7.0E-2	0.004	0.998
NAPHTHALENE	6	24	25%	6.033	7.242	4.0E-2	0.001	0.999
PYRENE	4	24	17%	4.533	3.200	3.0E-2	0.000	0.999
FLUORANTHENE	4	24	17%	4.642	4.200	4.0E-2	0.000	1.000
ZINC	3	24	13%	16.033	22.032	3.0E-1	0.000	1.000
PHENANTHRENE	2	24	8%	4.625	1.100	4.0E-2	0.000	1.000
ACENAPHTHENE	2	24	8%	4.597	1.100	6.0E-2	0.000	1.000
ANTHRACENE	3	24	13%	4.438	1.000	3.0E-1	0.000	1.000

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Chronic reference dose; "0" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitue RfDs.

[e]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-19
Ranking Process for Potential Background Chemicals
Warwick Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RfD[e] (mg/kg-day)	RRS-C[f]	RRS-N[g]
PAHs									
ACENAPHTHENE	3	31	10%	1.37	0.16	0.0E+0	6.0E-2	0.000	0.000
ACENAPHTHYLENE	3	31	10%	1.39	0.11	0.0E+0	4.0E-2	0.000	0.000
ANTHRACENE	10	31	32%	1.31	0.32	0.0E+0	3.0E-1	0.000	0.000
BENZO(A)ANTHRACENE	15	31	48%	0.97	1.36	7.3E-1	0.0E+0	0.027	0.000
BENZO(A)PYRENE	13	31	42%	1.15	1.62	7.3E+0	0.0E+0	0.318	0.000
BENZO(B)FLUORANTHENE	14	31	45%	1.23	1.65	7.3E-1	0.0E+0	0.032	0.000
BENZO(G,H,I)PERYLENE	9	31	29%	1.45	1.20	0.0E+0	4.0E-2	0.000	0.000
BENZO(K)FLUORANTHENE	13	31	42%	1.30	1.76	7.3E-2	0.0E+0	0.003	0.000
CHRYSENE	14	31	45%	1.08	1.47	7.3E-3	0.0E+0	0.000	0.000
DIBENZ(A,H)ANTHRACENE	3	31	10%	1.38	0.13	7.3E+0	0.0E+0	0.025	0.000
FLUORANTHENE	17	31	55%	1.23	1.58	0.0E+0	4.0E-2	0.000	0.000
FLUORENE	7	31	23%	1.34	0.23	0.0E+0	4.0E-2	0.000	0.000
INDENO(1,2,3-CD)PYRENE	8	31	26%	1.41	0.86	7.3E-1	0.0E+0	0.017	0.000
2-METHYLNAPHTHALENE	6	31	19%	1.34	0.36	0.0E+0	0.0E+0	0.000	0.000
NAPHTHALENE	16	31	52%	1.13	1.58	0.0E+0	4.0E-2	0.000	0.000
PHENANTHRENE	17	31	55%	0.87	1.13	0.0E+0	4.0E-2	0.000	0.000
PYRENE	18	31	58%	1.28	1.60	0.0E+0	3.0E-2	0.000	0.001
INORGANICS									
ANTIMONY	6	23	26%	2.27	5.35	0.0E+0	4.0E-4	0.000	0.133
ARSENIC	27	27	100%	9.00	10.25	1.8E+0	3.0E-4	0.482	0.341
BARIUM	31	31	100%	113.65	183.05	0.0E+0	7.0E-2	0.000	0.026
BERYLLIUM	31	31	100%	0.70	0.82	4.3E+0	5.0E-3	0.095	0.002
CADMIUM	15	31	48%	1.56	2.22	0.0E+0	5.0E-4	0.000	0.044
CHROMIUM	31	31	100%	68.15	101.04	0.0E+0	5.0E-3	0.000	0.201
COBALT	27	27	100%	4.43	5.01	0.0E+0	6.0E-2	0.000	0.001
COPPER	30	31	97%	118.02	225.23	0.0E+0	3.7E-2	0.000	0.061
CYANIDE	9	30	30%	1.86	2.70	0.0E+0	4.0E-2	0.000	0.001
IRON	25	25	100%	12327.60	13601.76	0.0E+0	0.0E+0	0.000	0.000
LEAD	26	27	96%	84.01	117.72	0.0E+0	0.0E+0	0.000	0.000
MAGNESIUM	25	25	100%	1713.56	2066.26	0.0E+0	0.0E+0	0.000	0.000
MANGANESE	25	25	100%	221.14	244.53	0.0E+0	1.4E-1	0.000	0.017
MERCURY	15	31	48%	0.23	0.31	0.0E+0	3.0E-4	0.000	0.010
NICKEL	28	31	90%	17.01	26.26	0.0E+0	2.0E-2	0.000	0.013

Table 6-B-19
Ranking Process for Potential Background Chemicals
Warwick Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RfD[e] (mg/kg-day)	RRS-C[f]	RRS-N[g]
POTASSIUM	25	25	100%	767.44	1059.55	0.0E+0	0.0E+0	0.000	0.000
SODIUM	15	25	60%	117.23	137.95	0.0E+0	0.0E+0	0.000	0.000
TIN	6	31	19%	7.42	9.75	0.0E+0	6.0E-1	0.000	0.000
VANADIUM	19	27	70%	10.51	12.24	0.0E+0	7.0E-3	0.000	0.017
ZINC	28	31	90%	2538.67	3911.97	0.0E+0	3.0E-1	0.000	0.130

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Cancer slope factor. "0" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[e]Chronic reference dose; "0" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

[f]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

[g]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-20
Ranking Process for Potential Background Chemicals Summary of Carcinogens
Warwick Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RRS-C[e]	Cumulative RRS-C
ARSENIC	27	27	100%	9.00	10.25	1.8E+0	0.482	0.482
BENZO(A)PYRENE	13	31	42%	1.15	1.62	7.3E+0	0.318	0.801
BERYLLIUM	31	31	100%	0.70	0.82	4.3E+0	0.094	0.895
BENZO(B)FLUORANTHENE	14	31	45%	1.23	1.65	7.3E-1	0.032	0.927
BENZO(A)ANTHRACENE	15	31	48%	0.97	1.36	7.3E-1	0.027	0.954
DIBENZ(A,H)ANTHRACENE	3	31	10%	1.38	0.13	7.3E+0	0.026	0.979
INDENO(1,2,3-CD)PYRENE	8	31	26%	1.41	0.86	7.3E-1	0.017	0.996
BENZO(K)FLUORANTHENE	13	31	42%	1.30	1.76	7.3E-2	0.003	1.000
CHRYSENE	14	31	45%	1.08	1.47	7.3E-3	0.000	1.000

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Cancer slope factor. "0" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[e]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-21
Ranking Process for Potential Background Chemicals Summary of Noncarcinogens
Warwick Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	RfD[d] (mg/kg-day)	RRS-N[e]	Cumulative RRS-N
ARSENIC	27	27	100%	9.00	10.25	3.0E-4	0.341	0.341
CHROMIUM	31	31	100%	68.15	101.04	5.0E-3	0.201	0.542
ANTIMONY	6	23	26%	2.27	5.35	4.0E-4	0.133	0.676
ZINC	28	31	90%	2538.67	3911.97	3.0E-1	0.130	0.806
COPPER	30	31	97%	118.02	225.23	3.7E-2	0.061	0.866
CADMIUM	15	31	48%	1.56	2.22	5.0E-4	0.044	0.910
BARIUM	31	31	100%	113.65	183.05	7.0E-2	0.026	0.936
VANADIUM	19	27	70%	10.51	12.24	7.0E-3	0.017	0.954
MANGANESE	25	25	100%	221.14	244.53	1.4E-1	0.017	0.971
NICKEL	28	31	90%	17.01	26.26	2.0E-2	0.013	0.984
MERCURY	15	31	48%	0.23	0.31	3.0E-4	0.010	0.995
BERYLLIUM	31	31	100%	0.70	0.82	5.0E-3	0.002	0.996
COBALT	27	27	100%	4.43	5.01	6.0E-2	0.001	0.997
CYANIDE	9	30	30%	1.86	2.70	4.0E-2	0.001	0.998
PYRENE	18	31	58%	1.28	1.60	3.0E-2	0.001	0.998
NAPHTHALENE	16	31	52%	1.13	1.58	4.0E-2	0.000	0.999
FLUORANTHENE	17	31	55%	1.23	1.58	4.0E-2	0.000	0.999
TIN	6	31	19%	7.42	9.75	6.0E-1	0.000	0.999
FLUORENE	7	31	23%	1.34	0.23	4.0E-2	0.000	0.999
ACENAPHTHENE	3	31	10%	1.37	0.16	6.0E-2	0.000	0.999
ANTHRACENE	10	31	32%	1.31	0.32	3.0E-1	0.000	0.999
ACENAPHTHYLENE	3	31	10%	1.39	0.11	4.0E-2	0.000	0.999
BENZO(G,H,I)PERYLENE	9	31	29%	1.45	1.20	4.0E-2	0.000	1.000
PHENANTHRENE	17	31	55%	0.87	1.13	4.0E-2	0.000	1.000

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Chronic reference dose; "0" means no value available. Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitue RfDs.

[e]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-22
Ranking Process for Potential Background Chemicals
Warwick Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RfD[e] (mg/kg-day)	RRS-C[f]	RRS-N[g]
PAHs									
ACENAPHTHENE	5	41	12%	1.093	0.410	0.0E+0	6.0E-2	0.000	0.000
ACENAPHTHYLENE	6	41	15%	1.096	0.160	0.0E+0	4.0E-2	0.000	0.000
ANTHRACENE	14	41	34%	1.040	0.320	0.0E+0	3.0E-1	0.000	0.000
BENZO(A)ANTHRACENE	22	41	54%	0.834	1.132	7.3E-1	0.0E+0	0.025	0.000
BENZO(A)PYRENE	20	41	49%	0.967	1.335	7.3E+0	0.0E+0	0.300	0.000
BENZO(B)FLUORANTHENE	21	41	51%	1.071	1.400	7.3E-1	0.0E+0	0.032	0.000
BENZO(G,H,I)PERYLENE	15	41	37%	1.170	1.200	0.0E+0	4.0E-2	0.000	0.000
BENZO(K)FLUORANTHENE	19	41	46%	1.050	1.416	7.3E-2	0.0E+0	0.003	0.000
CHRYSENE	21	41	51%	0.918	1.226	7.3E-3	0.0E+0	0.000	0.000
DIBENZ(A,H)ANTHRACENE	6	41	15%	1.079	0.130	7.3E+0	0.0E+0	0.029	0.000
FLUORANTHENE	24	41	59%	1.059	1.339	0.0E+0	4.0E-2	0.000	0.000
FLUORENE	10	41	24%	1.071	0.540	0.0E+0	4.0E-2	0.000	0.000
INDENO(1,2,3-CD)PYRENE	13	41	32%	1.130	0.860	7.3E-1	0.0E+0	0.019	0.000
2-METHYLNAPHTHALENE	11	41	27%	1.052	0.360	0.0E+0	0.0E+0	0.000	0.000
NAPHTHALENE	21	41	51%	1.672	2.997	0.0E+0	4.0E-2	0.000	0.001
PHENANTHRENE	25	41	61%	0.791	1.008	0.0E+0	4.0E-2	0.000	0.000
PYRENE	25	41	61%	1.280	1.631	0.0E+0	3.0E-2	0.000	0.001
INORGANICS									
ANTIMONY	7	30	23%	1.84	4.19	0.0E+0	4.0E-4	0.000	0.123
ARSENIC	34	34	100%	7.60	8.91	1.8E+0	3.0E-4	0.481	0.349
BARIUM	44	44	100%	90.27	139.77	0.0E+0	7.0E-2	0.000	0.023
BERYLLIUM	43	44	98%	0.68	0.83	4.3E+0	5.0E-3	0.110	0.002
CADMIUM	17	38	45%	1.55	2.17	0.0E+0	5.0E-4	0.000	0.051
CHROMIUM	44	44	100%	61.35	90.30	0.0E+0	5.0E-3	0.000	0.212
COBALT	34	34	100%	4.21	4.75	0.0E+0	6.0E-2	0.000	0.001
COPPER	43	44	98%	94.13	170.43	0.0E+0	3.7E-2	0.000	0.054
CYANIDE	10	40	25%	1.69	2.33	0.0E+0	2.0E-2	0.000	0.001
LEAD	33	34	97%	69.01	96.98	0.0E+0	0.0E+0	0.000	0.000
MAGNESIUM	26	26	100%	1691.50	2031.86	0.0E+0	0.0E+0	0.000	0.000

Table 6-B-22
Ranking Process for Potential Background Chemicals
Warwick Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RfD[e] (mg/kg-day)	RRS-C[f]	RRS-N[g]
MANGANESE	26	26	100%	228.13	253.55	0.0E+0	1.4E-1	0.000	0.021
MERCURY	20	38	53%	0.24	0.32	0.0E+0	3.0E-4	0.000	0.013
NICKEL	41	44	93%	13.76	17.57	0.0E+0	2.0E-2	0.000	0.010
TIN	6	38	16%	6.71	8.69	0.0E+0	6.0E-1	0.000	0.000
VANADIUM	26	34	76%	9.59	11.07	0.0E+0	7.0E-3	0.000	0.019
ZINC	41	44	93%	2000.61	3016.35	0.0E+0	3.0E-1	0.000	0.118

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Cancer slope factor. "0" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[e]Chronic reference dose; "0" means no value available. Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

[f]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

[g]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

were eliminated as COPCs and were not carried through the ranking process.

Table 6-B-23
Ranking Process for Potential Background Chemicals Summary of Carcinogens
Warwick Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RRS-C[e]	Cumulative RRS-C
ARSENIC	34	34	100%	7.60	8.91	1.8E+0	0.484	0.484
BENZO(A)PYRENE	20	41	49%	0.967	1.335	7.3E+0	0.303	0.787
BERYLLIUM	43	44	98%	0.68	0.83	4.3E+0	0.103	0.890
BENZO(B)FLUORANTHENE	21	41	51%	1.071	1.400	7.3E-1	0.032	0.922
DIBENZ(A,H)ANTHRACENE	6	41	15%	1.079	0.130	7.3E+0	0.029	0.951
BENZO(A)ANTHRACENE	22	41	54%	0.834	1.132	7.3E-1	0.026	0.977
INDENO(1,2,3-CD)PYRENE	13	41	32%	1.130	0.860	7.3E-1	0.020	0.997
BENZO(K)FLUORANTHENE	19	41	46%	1.050	1.416	7.3E-2	0.003	1.000
CHRYSENE	21	41	51%	0.918	1.226	7.3E-3	0.000	1.000

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Cancer slope factor. "0" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[e]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-24
Ranking Process for Potential Background Chemicals Summary of Noncarcinogens
Warwick Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	RfD[d] (mg/kg-day)	RRS-N[e]	Cumulative RRS-N
ARSENIC	34	34	100%	7.60	8.91	3.0E-4	0.348	0.348
CHROMIUM	44	44	100%	61.35	90.30	5.0E-3	0.212	0.560
ANTIMONY	7	30	23%	1.84	4.19	4.0E-4	0.123	0.683
ZINC	41	44	93%	2000.61	3016.35	3.0E-1	0.118	0.801
COPPER	43	44	98%	94.13	170.43	3.7E-2	0.054	0.854
CADMIUM	17	38	45%	1.55	2.17	5.0E-4	0.051	0.905
BARIUM	44	44	100%	90.27	139.77	7.0E-2	0.023	0.929
MANGANESE	26	26	100%	228.13	253.55	1.4E-1	0.021	0.950
VANADIUM	26	34	76%	9.59	11.07	7.0E-3	0.019	0.968
MERCURY	20	38	53%	0.24	0.32	3.0E-4	0.013	0.981
NICKEL	41	44	93%	13.76	17.57	2.0E-2	0.012	0.994
BERYLLIUM	43	44	98%	0.68	0.83	5.0E-3	0.002	0.995
CYANIDE	10	40	25%	1.69	2.33	2.0E-2	0.001	0.997
COBALT	34	34	100%	4.21	4.75	6.0E-2	0.001	0.998
NAPHTHALENE	21	41	51%	1.672	2.997	4.0E-2	0.001	0.999
PYRENE	25	41	61%	1.280	1.631	3.0E-2	0.001	0.999
FLUORANTHENE	24	41	59%	1.059	1.339	4.0E-2	0.000	1.000
TIN	6	38	16%	6.71	8.69	6.0E-1	0.000	1.000
FLUORENE	10	41	24%	1.071	0.540	4.0E-2	0.000	1.000
ACENAPHTHENE	5	41	12%	1.093	0.410	6.0E-2	0.000	1.000
ANTHRACENE	14	41	34%	1.040	0.320	3.0E-1	0.000	1.000
ACENAPHTHYLENE	6	41	15%	1.096	0.160	4.0E-2	0.000	1.000
BENZO(G,H,I)PERYLENE	15	41	37%	1.170	1.200	4.0E-2	0.000	1.000
PHENANTHRENE	25	41	61%	0.791	1.008	4.0E-2	0.000	1.000

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation

Table 6-B-24
Ranking Process for Potential Background Chemicals Summary of Noncarcinogens
Warwick Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	RfD[d] (mg/kg-day)	RRS-N[e]	Cumulative RRS-N

limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected). If the 95% UCL is greater than the maximum detected concentration, the RME concentration is the maximum detected concentration.

[d]Chronic reference dose; NA - not applicable - no RfD is reported because the sample concentration did not exceed background (for inorganics and PAHs only). Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

[e]Relative ranking score--noncarcinogenic.

Table 6-B-25
Ranking Process for Potential Background Chemicals
Warwick Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] $(mg/kg\text{-}day)^{-1}$	RfD[e] (mg/kg-day)	RRS-C[f]	RRS-N[g]
PAHs									
FLUORANTHENE	2	27	7%	4.669	1.000	0.0E+0	4.0E-2	0.000	0.000
NAPHTHALENE	2	27	7%	4.778	4.000	0.0E+0	4.0E-2	0.000	0.001
PYRENE	3	27	11%	4.540	2.000	0.0E+0	3.0E-2	0.000	0.001
INORGANICS									
ARSENIC	4	27	15%	5.20	6.78	1.8E+0	3.0E-4	1.000	0.208
BARIUM	24	27	89%	52.82	77.40	0.0E+0	7.0E-2	0.000	0.010
BERYLLIUM	1	27	4%	2.04	2.29	4.3E+0	5.0E-3	NA	NA
CADMIUM	2	27	7%	3.37	4.44	0.0E+0	5.0E-4	0.000	0.082
CHROMIUM	1	27	4%	5.27	5.73	0.0E+0	5.0E-3	NA	NA
LEAD	1	27	4%	2.24	2.40	0.0E+0	0.0E+0	NA	NA
MAGNESIUM	22	25	88%	2935.28	3540.85	0.0E+0	0.0E+0	0.000	0.000
MANGANESE	21	24	88%	295.29	373.27	0.0E+0	5.0E-3	0.000	0.687
SILVER	1	27	4%	5.34	5.91	0.0E+0	5.0E-3	NA	NA
ZINC	12	27	44%	221.70	365.63	0.0E+0	3.0E-1	0.000	0.011

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Cancer slope factor. "0" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[e]Chronic reference dose; "0" means no value available. Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

[f]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

[g]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-26
Ranking Process for Potential Background Chemicals Summary of Carcinogens
Warwick Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	CSF[d] (mg/kg-day)$^{-1}$	RRS-C[e]	Cumulative RRS-C
ARSENIC	4	27	15%	5.20	6.78	1.8E+0	1.000	1.000

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Cancer slope factor. "0" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[e]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-27
Ranking Process for Potential Background Chemicals Summary of Noncarcinogens
Warwick Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	RfD[d] (mg/kg-day)	RRS-N[e]	Cumulative RRS-N
MANGANESE	21	24	88%	295.29	373.27	5.0E-3	0.687	0.687
ARSENIC	4	27	15%	5.20	6.78	3.0E-4	0.208	0.895
CADMIUM	2	27	7%	3.37	4.44	5.0E-4	0.082	0.977
ZINC	12	27	44%	221.70	365.63	3.0E-1	0.011	0.988
BARIUM	24	27	89%	52.82	77.40	7.0E-2	0.010	0.998
NAPHTHALENE	2	27	7%	4.778	4.000	4.0E-2	0.001	0.999
PYRENE	3	27	11%	4.540	2.000	3.0E-2	0.001	1.000
FLUORANTHENE	2	27	7%	4.669	1.000	4.0E-2	0.000	1.000

[a]Includes polycyclic aromatic hydrocarbons (PAHs) and inorganics.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]Chronic reference dose; "0" means no value available. Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitue RfDs.

[e]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-28
Ranking Process for Chemicals of Potential Concern Selection
Production Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]
BIS(2-ETHYLHEXYL)PHTHALATE	16	41	39%	1.09	1.49	--	1.4E-2	2.0E-2	0.000	0.000
BUTYLBENZYLPHTHALATE	13	41	32%	1.83	3.22	--	--	2.0E-1	--	0.000
GAMMA-CHLORDANE	7	43	16%	0.07	0.13	--	1.3E+0	6.0E-5	0.004	0.000
4-CHLOROANILINE	5	41	12%	1.32	0.64	--	--	4.0E-3	--	0.000
CHLOROBENZENE	7	40	18%	0.20	0.28	--	--	2.0E-2	--	0.000
DI-N-BUTYLPHTHALATE	8	41	20%	1.00	1.30	--	--	1.0E-1	--	0.000
DIBENZOFURAN	12	41	29%	1.20	0.13	--	--	1.0E-2	--	0.000
DINOSEB	3	40	8%	0.07	0.01	--	--	1.0E-3	--	0.000
ETHYLBENZENE	10	40	25%	1.29	3.41	--	--	1.0E-1	--	0.000
IRGASAN DP-300	3	26	12%	9.42	4.20	--	--	3.0E-1	--	0.000
METHOXYCHLOR	7	43	16%	0.35	0.55	--	--	5.0E-3	--	0.000
METHYLENE CHLORIDE	6	40	15%	0.27	0.01	--	7.5E-3	6.0E-2	0.000	0.000
3&4-METHYLPHENOL	5	16	31%	0.18	0.25	--	--	5.0E-2	--	0.000
NICKEL	28	31	90%	7.77	11.62	--	--	2.0E-2	0.000	0.000
2-NITROANILINE	8	41	20%	6.25	0.89	--	--	6.0E-5	--	0.003
NITROBENZENE	3	41	7%	1.25	0.14	--	--	5.0E-4	--	0.000
OCDD	1	5	20%	0.00	0.00	--	1.6E+2	--	0.002	--
ORTHOPHOSPHATE	20	25	80%	5.15	8.13	--	--	--	--	--
PCB-1248	39	101	39%	51.91	126.62	--	--	3.0E-5	--	0.803
PCB-1254	94	104	90%	15.80	20.31	--	--	2.0E-5	--	0.193
PCB-1260	7	83	8%	2.94	6.10	--	7.7E+0	--	0.994	--
TCDF	3	5	60%	0.00	0.00	--	1.6E+2	--	0.001	--
TOLUENE	18	40	45%	0.39	0.71	--	--	2.0E-1	--	0.000
TRCDF	2	5	40%	0.23	0.54	--	--	--	--	--
TRICHLOROFLUOROMETHANE	3	40	8%	0.41	0.33	--	--	3.0E-1	--	0.000
M&P-XYLENE	27	40	68%	10.08	27.05	--	--	2.0E+0	--	0.000
O-XYLENE	19	40	48%	3.03	8.12	--	--	2.0E+0	--	0.000

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

Table 6-B-28
Ranking Process for Chemicals of Potential Concern Selection
Production Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Cancer slope factor. "--" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information System (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[f]Chronic reference dose; "--" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

[g]Relative ranking score--carcinogenic.

[h]Relative ranking score--noncarcinogenic.

Table 6-B-29
Ranking Process for Chemicals of Potential Concern Selection Summary of Carcinogens
Production Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RRS-C[f]	Cumulative RRS-C
PCB-1260	7	83	8%	2.94	6.10	--	7.7E+0	0.994	0.994
GAMMA-CHLORDANE	7	43	16%	0.07	0.13	--	1.3E+0	0.004	0.997
OCDD	1	5	20%	0.00	0.00	--	1.6E+2	0.002	0.999
TCDF	3	5	60%	0.00	0.00	--	1.6E+2	0.001	1.000
BIS(2-ETHYLHEXYL)PHTHALATE	16	41	39%	1.09	1.49	--	1.4E-2	0.000	1.000
METHYLENE CHLORIDE	6	40	15%	0.27	0.01	--	7.5E-3	0.000	1.000

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Cancer slope factor. "--" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[f]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-30
Ranking Process for Chemicals of Potential Concern Selection Summary of Noncarcinogens
Production Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	RfD[e] (mg/kg-day)	RRS-N[f]	Cumulative RRS-N
PCB-1248	39	101	39%	51.91	126.62	--	3.0E-5	0.803	0.803
PCB-1254	94	104	90%	15.80	20.31	--	2.0E-5	0.193	0.997
2-NITROANILINE	8	41	20%	6.25	0.89	--	6.0E-5	0.003	0.999
GAMMA-CHLORDANE	7	43	16%	0.07	0.13	--	6.0E-5	0.000	1.000
NITROBENZENE	3	41	7%	1.25	0.14	--	5.0E-4	0.000	1.000
4-CHLOROANILINE	5	41	12%	1.32	0.64	--	4.0E-3	0.000	1.000
METHOXYCHLOR	7	43	16%	0.35	0.55	--	5.0E-3	0.000	1.000
BIS(2-ETHYLHEXYL)PHTHALATE	16	41	39%	1.09	1.49	--	2.0E-2	0.000	1.000
ETHYLBENZENE	10	40	25%	1.29	3.41	--	1.0E-1	0.000	1.000
BUTYLBENZYLPHTHALATE	13	41	32%	1.83	3.22	--	2.0E-1	0.000	1.000
CHLOROBENZENE	7	40	18%	0.20	0.28	--	2.0E-2	0.000	1.000
IRGASAN DP-300	3	26	12%	9.42	4.20	--	3.0E-1	0.000	1.000
M&P-XYLENE	27	40	68%	10.08	27.05	--	2.0E+0	0.000	1.000
DI-N-BUTYLPHTHALATE	8	41	20%	1.00	1.30	--	1.0E-1	0.000	1.000
DIBENZOFURAN	12	41	29%	1.20	0.13	--	1.0E-2	0.000	1.000
DINOSEB	3	40	8%	0.07	0.01	--	1.0E-3	0.000	1.000
3&4-METHYLPHENOL	5	16	31%	0.18	0.25	--	5.0E-2	0.000	1.000
O-XYLENE	19	40	48%	3.03	8.12	--	2.0E+0	0.000	1.000
TOLUENE	18	40	45%	0.39	0.71	--	2.0E-1	0.000	1.000
TRICHLOROFLUOROMETHANE	3	40	8%	0.41	0.33	--	3.0E-1	0.000	1.000
METHYLENE CHLORIDE	6	40	15%	0.27	0.01	--	6.0E-2	0.000	1.000
NICKEL	28	31	90%	7.77	11.62	--	2.0E-2	0.000	1.000

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

Table 6-B-30
Ranking Process for Chemicals of Potential Concern Selection Summary of Noncarcinogens
Production Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	RfD[e] (mg/kg-day)	RRS-N[f]	Cumulative RRS-N

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Chronic reference dose; "--" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

[f]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-31
Ranking Process for Chemicals of Potential Concern Selection
Production Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]
ACETONE	1	80	1%	1.409	0.053	--	NA	NA	NA	NA
ACETOPHENONE	4	84	5%	0.870	0.660	--	NA	NA	NA	NA
ALDRIN	2	86	2%	0.023	0.034	--	NA	NA	NA	NA
ANILINE	5	84	6%	0.867	0.280	--	5.7E-3	--	0.000	--
ALPHA-BHC	5	86	6%	0.018	0.018	--	6.3E+0	--	0.002	--
BETA-BHC	1	86	1%	0.019	0.028	--	NA	NA	NA	NA
DELTA-BHC	2	86	2%	0.019	0.028	--	NA	NA	NA	NA
GAMMA-BHC	9	86	10%	0.019	0.027	--	1.3E+0	3.0E-4	0.001	0.000
BIS(2-CHLOROETHYL)ETHER	2	84	2%	0.864	0.680	--	NA	NA	NA	NA
BIS(2-ETHYLHEXYL)PHTHALATE	36	84	43%	0.850	1.074	--	1.4E-2	2.0E-2	0.000	0.000
2-BUTANONE	4	80	5%	1.414	0.300	--	NA	NA	NA	NA
BUTAZOLIDIN	1	40	3%	7.858	5.200	--	NA	1.4E-1	NA	NA
BUTYLBENZYLPHTHALATE	18	84	21%	1.130	1.819	--	--	2.0E-1	--	0.000
ALPHA-CHLORDANE	2	86	2%	0.018	0.010	--	NA	NA	NA	NA
GAMMA-CHLORDANE	13	86	15%	0.037	0.070	--	1.3E+0	6.0E-5	0.002	0.000
4-CHLOROANILINE	8	84	10%	0.917	0.640	--	--	4.0E-3	--	0.000
CHLOROBENZENE	9	80	11%	0.666	0.280	--	--	2.0E-2	--	0.000
CHLOROBENZILATE	1	85	1%	0.813	0.098	--	NA	NA	NA	NA
CHLOROFORM	1	80	1%	0.664	0.034	--	NA	NA	NA	NA
2,4-D	6	48	13%	0.059	0.064	--	--	1.0E-2	--	0.000
4,4'-DDD	2	86	2%	0.026	0.041	--	NA	NA	NA	NA
4,4'-DDE	1	86	1%	0.031	0.051	--	NA	NA	NA	NA
4,4'-DDT	2	86	2%	0.039	0.055	--	NA	NA	NA	NA
DI-N-BUTYLPHTHALATE	14	84	17%	0.715	0.955	--	--	1.0E-1	--	0.000
DIBENZOFURAN	14	84	17%	0.821	0.400	--	--	1.0E-2	--	0.000
1,2-DICHLOROBENZENE	3	84	4%	0.857	0.760	--	NA	NA	NA	NA
1,4-DICHLOROBENZENE	1	84	1%	0.860	0.240	--	NA	NA	NA	NA
3,3'-DICHLOROBENZIDINE	1	84	1%	1.725	0.730	--	NA	NA	NA	NA
2,4-DICHLOROPHENOL	4	84	5%	0.971	1.253	--	NA	NA	NA	NA

Table 6-B-31
Ranking Process for Chemicals of Potential Concern Selection
Production Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]
2,6-DICHLOROPHENOL	1	84	1%	0.891	1.159	--	NA	NA	NA	NA
DIETHYLPHTHALATE	1	84	1%	0.806	0.670	--	NA	NA	NA	NA
2,4-DIMETHYLPHENOL	7	84	8%	0.885	1.150	--	--	2.0E-2	--	0.000
DIMETHYLPHTHALATE	1	84	1%	0.833	0.250	--	NA	NA	NA	NA
DINOSEB	5	78	6%	0.102	0.009	--	--	1.0E-3	--	0.000
1,4-DIOXANE	1	84	1%	2.790	3.267	--	NA	NA	NA	NA
DISULFOTON	1	84	1%	0.062	0.010	--	NA	NA	NA	NA
ENDOSULFAN SULFATE	1	86	1%	0.072	0.096	--	NA	NA	NA	NA
ENDRIN ALDEHYDE	2	86	2%	0.035	0.002	--	NA	NA	NA	NA
ETHYL PARATHION	1	84	1%	0.029	0.013	--	NA	NA	NA	NA
ETHYLBENZENE	24	80	30%	1.693	2.977	--	--	1.0E-1	--	0.000
FAMPHUR	2	84	2%	0.154	0.016	--	NA	NA	NA	NA
HEPTACHLOR	4	86	5%	0.018	0.026	--	NA	NA	NA	NA
IRGASAN DP-300	7	44	16%	24.485	42.568	--	--	3.0E-1	--	0.000
ISODRIN	2	86	2%	0.026	0.040	--	NA	NA	NA	NA
KEPONE	1	86	1%	0.073	0.015	--	NA	NA	NA	NA
METHOXYCHLOR	12	86	14%	0.208	0.311	--	--	5.0E-3	--	0.000
METHYL PARATHION	6	84	7%	0.009	0.007	--	--	2.5E-4	--	0.000
METHYLENE CHLORIDE	20	80	25%	0.791	1.402	--	7.5E-3	6.0E-2	0.000	0.000
2-METHYLPHENOL	1	84	1%	0.863	0.084	--	NA	NA	NA	NA
3&4-METHYLPHENOL	17	42	40%	0.282	0.372	--	--	5.0E-2	--	0.000
4-METHYLPHENOL	4	42	10%	1.559	2.043	--	--	5.0E-3	--	0.000
N-OCTANE	2	5	40%	3.263	8.246	--	--	--	--	--
NICKEL	49	54	91%	8.160	9.440	6.36000	--	2.0E-2	--	0.000
2-NITROANILINE	9	84	11%	4.301	4.200	--	--	6.0E-5	--	0.019
NITROBENZENE	5	84	6%	0.873	1.140	--	--	5.0E-4	--	0.001
2-NITROPHENOL	1	84	1%	0.863	0.075	--	NA	NA	NA	NA
OCDD	1	9	11%	0.000	0.000	--	1.6E+2	--	0.001	--
ORTHOPHOSPHATE	35	42	83%	10.285	14.566	--	--	--	--	--
P-PHENYLENEDIAMINE	1	84	1%	4.734	6.116	--	NA	NA	NA	NA

Table 6-B-31
Ranking Process for Chemicals of Potential Concern Selection
Production Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]
PCB-1248	39	144	27%	36.430	88.450	--	--	3.0E-5	--	0.784
PCB-1254	127	147	86%	86.390	14.780	--	--	2.0E-5	--	0.196
PCB-1260	15	126	12%	11.900	4.810	--	7.7E+0	--	0.993	--
PHENOL	4	84	5%	0.857	0.630	--	NA	NA	NA	NA
STYRENE	5	80	6%	0.664	0.049	--	--	2.0E-1	--	0.000
SULFOTEPP	1	84	1%	0.018	0.009	--	NA	NA	NA	NA
2,4,5-T	2	48	4%	0.011	0.011	--	NA	NA	NA	NA
TCDF	4	9	44%	0.000	0.000	--	1.6E+2	--	0.000	--
TETRACHLOROETHENE	1	80	1%	0.664	0.069	--	NA	NA	NA	NA
TINUVIN 327	2	40	5%	7.187	5.200	--	NA	NA	NA	NA
TOLUENE	31	80	39%	30.822	61.536	--	--	2.0E-1	--	0.000
2,4,5-TP (SILVEX)	2	48	4%	0.009	0.007	--	NA	NA	NA	NA
TRCDF	3	10	30%	0.140	0.286	--	--	--	--	--
1,2,4-TRICHLOROBENZENE	3	84	4%	0.862	0.640	--	NA	NA	NA	NA
TRICHLOROFLUOROMETHANE	3	80	4%	1.336	0.330	--	NA	NA	NA	NA
M&P-XYLENE	48	80	60%	9.443	18.270	--	--	2.0E+0	--	0.000
O-XYLENE	37	80	46%	2.609	5.198	--	--	2.0E+0	--	0.000

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Cancer slope factor. "--" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information System (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[f]Chronic reference dose; "--" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

[g]Relative ranking score--carcinogenic.

Table 6-B-31
Ranking Process for Chemicals of Potential Concern Selection
Production Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] $(mg/kg\text{-}day)^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]

[h]Relative ranking score--noncarcinogenic.

Table 6-B-32
Ranking Process for Chemicals of Potential Concern Selection Summary of Carcinogens
Production Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RRS-C[f]	Cumulative RRS-C
PCB-1260	15	126	12%	11.900	4.810	--	7.7E+0	0.993	0.993
ALPHA-BHC	5	86	6%	0.018	0.018	--	6.3E+0	0.002	0.995
GAMMA-CHLORDANE	13	86	15%	0.037	0.070	--	1.3E+0	0.002	0.997
OCDD	1	9	11%	0.000	0.000	--	1.6E+2	0.001	0.998
GAMMA-BHC	9	86	10%	0.019	0.027	--	1.3E+0	0.001	0.999
TCDF	4	9	44%	0.000	0.000	--	1.6E+2	0.000	0.999
BIS(2-ETHYLHEXYL)PHTHALATE	36	84	43%	0.850	1.074	--	1.4E-2	0.000	1.000
METHYLENE CHLORIDE	20	80	25%	0.791	1.402	--	7.5E-3	0.000	1.000
ANILINE	5	84	6%	0.867	0.280	--	5.7E-3	0.000	1.000

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Cancer slope factor. "--" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[f]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-33
Ranking Process for Chemicals of Potential Concern Selection Summary of Noncarcinogens
Production Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	RfD[e] (mg/kg-day)	RRS-N[f]	Cumulative RRS-N
PCB-1248	39	144	27%	36.430	88.450	--	3.0E-5	0.784	0.784
PCB-1254	127	147	86%	86.390	14.780	--	2.0E-5	0.196	0.980
2-NITROANILINE	9	84	11%	4.301	4.200	--	6.0E-5	0.019	0.999
NITROBENZENE	5	84	6%	0.873	1.140	--	5.0E-4	0.001	0.999
GAMMA-CHLORDANE	13	86	15%	0.037	0.070	--	6.0E-5	0.000	1.000
4-METHYLPHENOL	4	42	10%	1.559	2.043	--	5.0E-3	0.000	1.000
TOLUENE	31	80	39%	30.822	61.536	--	2.0E-1	0.000	1.000
4-CHLOROANILINE	8	84	10%	0.917	0.640	--	4.0E-3	0.000	1.000
IRGASAN DP-300	7	44	16%	24.485	42.568	--	3.0E-1	0.000	1.000
GAMMA-BHC	9	86	10%	0.019	0.027	--	3.0E-4	0.000	1.000
METHOXYCHLOR	12	86	14%	0.208	0.311	--	5.0E-3	0.000	1.000
2,4-DIMETHYLPHENOL	7	84	8%	0.885	1.150	--	2.0E-2	0.000	1.000
BIS(2-ETHYLHEXYL)PHTHALATE	36	84	43%	0.850	1.074	--	2.0E-2	0.000	1.000
DIBENZOFURAN	14	84	17%	0.821	0.400	--	1.0E-2	0.000	1.000
ETHYLBENZENE	24	80	30%	1.693	2.977	--	1.0E-1	0.000	1.000
METHYL PARATHION	6	84	7%	0.009	0.007	--	2.5E-4	0.000	1.000
METHYLENE CHLORIDE	20	80	25%	0.791	1.402	--	6.0E-2	0.000	1.000
CHLOROBENZENE	9	80	11%	0.666	0.280	--	2.0E-2	0.000	1.000
DI-N-BUTYLPHTHALATE	14	84	17%	0.715	0.955	--	1.0E-1	0.000	1.000
M&P-XYLENE	48	80	60%	9.443	18.270	--	2.0E+0	0.000	1.000
BUTYLBENZYLPHTHALATE	18	84	21%	1.130	1.819	--	2.0E-1	0.000	1.000
DINOSEB	5	78	6%	0.102	0.009	--	1.0E-3	0.000	1.000
3&4-METHYLPHENOL	17	42	40%	0.282	0.372	--	5.0E-2	0.000	1.000
2,4-D	6	48	13%	0.059	0.064	--	1.0E-2	0.000	1.000
O-XYLENE	37	80	46%	2.609	5.198	--	2.0E+0	0.000	1.000
STYRENE	5	80	6%	0.664	0.049	--	2.0E-1	0.000	1.000
NICKEL	49	54	91%	8.160	9.440	6.360	2.0E-2	0.000	1.000

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

Table 6-B-33
Ranking Process for Chemicals of Potential Concern Selection Summary of Noncarcinogens
Production Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	RfD[e] (mg/kg-day)	RRS-N[f]	Cumulative RRS-N

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Chronic reference dose; "--" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

[f]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-34
Ranking Process for Chemicals of Potential Concern Selection
Production Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]
ACETOPHENONE	14	50	28%	7.45	9.38	0.00	--	1.0E-1	--	0.000
ALDRIN	3	48	6%	0.119	0.013	--	1.7E+1	3.0E-5	0.002	0.001
ANILINE	13	50	26%	17.29	19.00	--	5.7E-3	--	0.001	--
BENZENE	4	50	8%	150.37	100.00	--	2.9E-2	--	0.025	--
BENZYL ALCOHOL	8	49	16%	12.14	18.80	--	--	3.0E-1	--	0.000
ALPHA-BHC	3	49	6%	0.10	0.14	--	6.3E+0	--	0.008	--
DELTA-BHC	3	49	6%	0.13	0.19	--	--	3.0E-4	--	0.002
GAMMA-BHC	2	48	4%	0.13	0.19	--	NA	NA	NA	NA
1,1-BIPHENYL	5	14	36%	3.53	2.10	--	--	5.0E-2	--	0.000
BIS(2-CHLOROETHOXY)METHANE	1	50	2%	5.66	1.00	--	NA	NA	NA	NA
BIS(2-CHLOROETHYL)ETHER	1	50	2%	5.69	1.80	--	NA	NA	NA	NA
BIS(2-ETHYLHEXYL)PHTHALATE	4	50	8%	5.54	2.00	--	1.4E-2	2.0E-2	0.000	0.000
2-BUTANONE	1	43	2%	1029.67	21.00	--	NA	NA	NA	NA
BUTAZOLIDIN	7	50	14%	66.83	19.00	--	--	1.4E-1	--	0.001
BUTYLBENZYLPHTHALATE	1	50	2%	5.66	0.60	--	NA	NA	NA	NA
ALPHA-CHLORDANE	1	48	2%	0.145	0.013	--	NA	NA	NA	NA
GAMMA-CHLORDANE	4	48	8%	0.20	0.29	--	1.3E+0	6.0E-5	0.003	0.017
4-CHLORO-3-METHYLPHENOL	8	49	16%	7.37	9.15	--	--	--	--	--
4-CHLOROANILINE	2	50	4%	8.71	10.00	--	NA	NA	NA	NA
CHLOROBENZENE	25	50	50%	1528.18	2556.19	--	--	2.0E-2	--	0.441
CHLOROFORM	1	50	2%	154.07	8.40	--	NA	NA	NA	NA
2-CHLOROPHENOL	12	49	24%	14.32	23.07	--	--	5.0E-3	--	0.016
2-CYCLOHEXENE-1-OL	3	14	21%	6.93	0.74	--	--	--	--	--
2-CYCLOHEXENE-1-ONE	5	14	36%	6.32	0.87	--	--	--	--	--
2,4-D	2	50	4%	0.43	0.48	--	NA	NA	NA	NA
4,4'-DDD	2	48	4%	0.15	0.20	--	NA	NA	NA	NA
4,4'-DDE	2	48	4%	0.17	0.22	--	NA	NA	NA	NA
4,4'-DDT	3	48	6%	0.21	0.02	--	3.4E-1	5.0E-4	0.000	0.000
DI-N-BUTYLPHTHALATE	6	50	12%	5.29	2.40	--	--	1.0E-1	--	0.000

Table 6-B-34
Ranking Process for Chemicals of Potential Concern Selection
Production Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]
1,2-DICHLOROBENZENE	14	50	28%	17.30	26.34	--	--	9.0E-2	--	0.001
1,3-DICHLOROBENZENE	3	50	6%	5.50	1.00	--	--	8.9E-2	--	0.000
1,4-DICHLOROBENZENE	7	50	14%	4.52	3.00	--	2.4E-2	--	0.001	--
1,1-DICHLOROETHANE	5	50	10%	153.99	4.00	--	--	1.0E-1	--	0.000
DICHLOROETHENE	2	49	4%	154.90	140.00	--	NA	NA	NA	NA
2,4-DICHLOROPHENOL	9	49	18%	63.50	112.60	--	--	3.0E-3	--	0.129
2,6-DICHLOROPHENOL	1	49	2%	5.77	5.00	--	NA	NA	NA	NA
DIELDRIN	1	47	2%	0.154	0.012	--	NA	NA	NA	NA
DIETHYLPHTHALATE	6	50	12%	5.30	1.50	--	--	8.0E-1	--	0.000
DIMETHOATE	3	50	6%	2.10	0.19	--	--	2.0E-4	--	0.003
2,4-DIMETHYLPHENOL	19	49	39%	9.17	11.43	--	--	2.0E-2	--	0.002
DINOSEB	3	49	6%	7.61	0.65	--	--	1.0E-3	--	0.002
1,4-DIOXANE	3	50	6%	2403.85	45.00	--	1.1E-2	--	0.004	--
ENDOSULFAN II	3	48	6%	0.271	0.007	--	--	6.0E-3	--	0.000
ENDOSULFAN SULFATE	1	48	2%	0.398	0.008	--	NA	NA	NA	NA
ENDRIN	1	49	2%	0.141	0.086	--	NA	NA	NA	NA
ENDRIN ALDEHYDE	3	48	6%	0.25	0.37	--	--	3.0E-4	--	0.004
ETHYL PARATHION	1	50	2%	0.39	0.37	--	NA	NA	NA	NA
ETHYLBENZENE	15	50	30%	244.60	354.24	--	--	1.0E-1	--	0.012
FAMPHUR	2	50	4%	1.09	0.27	--	NA	NA	NA	NA
FLUORANTHENE	9	50	18%	5.39	6.45	5.15	NA	NA	NA	NA
HEPTACHLOR	1	48	2%	0.11	0.15	--	NA	NA	NA	NA
1,2,3,4,6,7,8-HPCDD	1	26	4%	0.001	0.001	--	NA	NA	NA	NA
HPCDD	1	26	4%	0.001	0.001	--	NA	NA	NA	NA
1,2,3,4,6,7,8-HPCDF	1	26	4%	0.0003	0.0004	--	NA	NA	NA	NA
HPCDF	1	26	4%	0.0005	0.0004	--	NA	NA	NA	NA
1,2,3,4,7,8-HXCDD	1	26	4%	0.0005	0.0004	--	NA	NA	NA	NA
HXCDD	1	26	4%	0.0005	0.0004	--	NA	NA	NA	NA
1,2,3,4,7,8-HXCDF	1	26	4%	0.0002	0.0003	--	NA	NA	NA	NA
HXCDF	1	26	4%	0.0003	0.0003	--	NA	NA	NA	NA

Table 6-B-34
Ranking Process for Chemicals of Potential Concern Selection
Production Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]
HYDROCARBONS	10	24	42%	2.10	3.82	--	--	--	--	--
IODOMETHANE	1	49	2%	157.60	262.93	--	NA	NA	NA	NA
IRGASAN DP-300	12	50	24%	147.67	248.88	--	--	3.0E-1	--	0.003
ISODRIN	2	48	4%	0.085	0.095	--	NA	NA	NA	NA
METHYL PARATHION	1	50	2%	0.18	0.21	--	NA	NA	NA	NA
2-METHYLPHENOL	15	49	31%	44.73	72.77	--	--	5.0E-2	--	0.005
3&4-METHYLPHENOL	10	26	38%	37.31	63.11	--	--	5.0E-2	--	0.004
4-METHYLPHENOL	13	23	57%	68.41	149.84	--	--	5.0E-3	--	0.103
2-NITROANILINE	1	50	2%	28.37	12.00	--	NA	NA	NA	NA
4-NITROPHENOL	1	50	2%	28.29	8.00	--	NA	NA	NA	NA
OCDD	11	26	42%	0.0015	0.0021	--	1.6E+2	--	0.003	--
OCDF	1	26	4%	0.0007	0.0003	--	NA	NA	NA	NA
ORTHOPHOSPHATE	9	24	38%	0.18	0.33	--	--	--	--	--
PCB-1260	2	48	4%	2.89	4.31	--	NA	NA	NA	NA
PECDF	1	26	4%	0.0003	0.0001	--	NA	NA	NA	NA
PENTACHLOROPHENOL	1	48	2%	28.33	3.00	--	NA	NA	NA	NA
PHENOL	15	50	30%	32.14	52.31	--	--	6.0E-1	--	0.000
PHORATE	1	50	2%	0.39	0.06	--	NA	NA	NA	NA
NICKEL	3	50	6%	16.49	18.33	173.52	NA	NA	NA	NA
SULFOTEPP	3	50	6%	0.27	0.29	--	--	5.0E-4	--	0.002
2,3,7,8-TCDF	1	26	4%	0.0002	0.0003	--	NA	NA	NA	NA
TCDF	3	26	12%	0.002	0.005	--	1.6E+2	--	0.006	--
TETRACHLOROETHENE	2	50	4%	154.51	31.00	--	NA	NA	NA	NA
THIONAZIN	1	50	2%	1.36	0.06	--	NA	NA	NA	NA
TINUVIN 327	3	50	6%	24.32	3.00	--	--	2.5E-3	--	0.004
TKN	18	24	75%	2.85	4.60	--	--	--	--	--
TOLUENE	21	50	42%	8225.61	14062.28	--	--	2.0E-1	--	0.242
O-TOLUIDINE	1	50	2%	5.66	1.00	--	NA	NA	NA	NA
2,4,5-TP (SILVEX)	2	50	4%	0.13	0.15	--	NA	NA	NA	NA
1,2,4-TRICHLOROBENZENE	2	50	4%	5.64	3.00	--	NA	NA	NA	NA

Table 6-B-34
Ranking Process for Chemicals of Potential Concern Selection
Production Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]
1,1,1-TRICHLOROETHANE	2	50	4%	155.73	89.00	--	NA	NA	NA	NA
TRICHLOROETHENE	2	50	4%	153.98	4.00	--	NA	NA	NA	NA
VINYL CHLORIDE	4	49	8%	30.99	57.93	--	1.9E+0	--	0.947	--
M&P-XYLENE	23	50	46%	522.53	812.44	--	--	2.0E+0	--	0.001
O-XYLENE	18	50	36%	259.87	407.15	--	--	2.0E+0	--	0.001

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Cancer slope factor. "--" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information System (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[f]Chronic reference dose; "--" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

[g]Relative ranking score--carcinogenic.

[h]Relative ranking score--noncarcinogenic.

Table 6-B-35
Ranking Process for Chemicals of Potential Concern Selection Summary of Carcinogens
Production Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RRS-C[f]	Cumulative RRS-C
VINYL CHLORIDE	4	49	8%	30.99	57.93	--	1.9E+0	0.947	0.947
BENZENE	4	50	8%	150.37	100.00	--	2.9E-2	0.025	0.972
ALPHA-BHC	3	49	6%	0.10	0.14	--	6.3E+0	0.008	0.979
TCDF	3	26	12%	0.002	0.005	--	1.6E+2	0.006	0.986
1,4-DIOXANE	3	50	6%	2403.85	45.00	--	1.1E-2	0.004	0.990
GAMMA-CHLORDANE	4	48	8%	0.20	0.29	--	1.3E+0	0.003	0.993
OCDD	11	26	42%	0.0015	0.0021	--	1.6E+2	0.003	0.996
ALDRIN	3	48	6%	0.119	0.013	--	1.7E+1	0.002	0.998
ANILINE	13	50	26%	17.29	19.00	--	5.7E-3	0.001	0.999
1,4-DICHLOROBENZENE	7	50	14%	4.52	3.00	--	2.4E-2	0.001	1.000
BIS(2-ETHYLHEXYL)PHTHALATE	4	50	8%	5.54	2.00	--	1.4E-2	0.000	1.000
4,4'-DDT	3	48	6%	0.21	0.02	--	3.4E-1	0.000	1.000

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Cancer slope factor. "--" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[f]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-36
Ranking Process for Chemicals of Potential Concern Selection Summary of Noncarcinogens
Production Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	RfD[e] (mg/kg-day)	RRS-N[f]	Cumulative RRS-N
CHLOROBENZENE	25	50	50%	1528.18	2556.19	--	2.0E-2	0.441	0.441
TOLUENE	21	50	42%	8225.61	14062.28	--	2.0E-1	0.242	0.683
2,4-DICHLOROPHENOL	9	49	18%	63.50	112.60	--	3.0E-3	0.129	0.812
4-METHYLPHENOL	13	23	57%	68.41	149.84	--	5.0E-3	0.103	0.916
GAMMA-CHLORDANE	4	48	8%	0.20	0.29	--	6.0E-5	0.017	0.932
2-CHLOROPHENOL	12	49	24%	14.32	23.07	--	5.0E-3	0.016	0.948
ETHYLBENZENE	15	50	30%	244.60	354.24	--	1.0E-1	0.012	0.961
2-METHYLPHENOL	15	49	31%	44.73	72.77	--	5.0E-2	0.005	0.966
3&4-METHYLPHENOL	10	26	38%	37.31	63.11	--	5.0E-2	0.004	0.970
ENDRIN ALDEHYDE	3	48	6%	0.25	0.37	--	3.0E-4	0.004	0.974
TINUVIN 327	3	50	6%	24.32	3.00	--	2.5E-3	0.004	0.978
DIMETHOATE	3	50	6%	2.10	0.19	--	2.0E-4	0.003	0.982
IRGASAN DP-300	12	50	24%	147.67	248.88	--	3.0E-1	0.003	0.984
DINOSEB	3	49	6%	7.61	0.65	--	1.0E-3	0.002	0.987
DELTA-BHC	3	49	6%	0.13	0.19	--	3.0E-4	0.002	0.989
SULFOTEPP	3	50	6%	0.27	0.29	--	5.0E-4	0.002	0.991
2,4-DIMETHYLPHENOL	19	49	39%	9.17	11.43	--	2.0E-2	0.002	0.993
ALDRIN	3	48	6%	0.119	0.013	--	3.0E-5	0.001	0.994
M&P-XYLENE	23	50	46%	522.53	812.44	--	2.0E+0	0.001	0.996
BUTAZOLIDIN	7	50	14%	66.83	19.00	--	1.4E-1	0.001	0.997
1,2-DICHLOROBENZENE	14	50	28%	17.30	26.34	--	9.0E-2	0.001	0.998
O-XYLENE	18	50	36%	259.87	407.15	--	2.0E+0	0.001	0.999
BIS(2-ETHYLHEXYL)PHTHALATE	4	50	8%	5.54	2.00	--	2.0E-2	0.000	0.999
ACETOPHENONE	14	50	28%	7.45	9.38	--	1.0E-1	0.000	0.999
PHENOL	15	50	30%	32.14	52.31	--	6.0E-1	0.000	1.000
BENZYL ALCOHOL	8	49	16%	12.14	18.80	--	3.0E-1	0.000	1.000
4,4'-DDT	3	48	6%	0.21	0.02	--	5.0E-4	0.000	1.000
1,1-BIPHENYL	5	14	36%	3.53	2.10	--	5.0E-2	0.000	1.000
1,1-DICHLOROETHANE	5	50	10%	153.99	4.00	--	1.0E-1	0.000	1.000

Table 6-B-36
Ranking Process for Chemicals of Potential Concern Selection Summary of Noncarcinogens
Production Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	RfD[e] (mg/kg-day)	RRS-N[f]	Cumulative RRS-N
DI-N-BUTYLPHTHALATE	6	50	12%	5.29	2.40	--	1.0E-1	0.000	1.000
1,3-DICHLOROBENZENE	3	50	6%	5.50	1.00	--	8.9E-2	0.000	1.000
DIETHYLPHTHALATE	6	50	12%	5.30	1.50	--	8.0E-1	0.000	1.000
ENDOSULFAN II	3	48	6%	0.271	0.007	--	6.0E-3	0.000	1.000

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Chronic reference dose; "--" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

[f]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-37
Ranking Process for Chemicals of Potential Concern Selection
Waste Water Treatment Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]
ACETONE	4	18	22%	0.094	0.129	–	–	1.0E-1	–	0.000
ACETOPHENONE	2	18	11%	0.782	0.026	–	–	1.0E-1	–	0.000
ANILINE	4	18	22%	0.893	1.409	–	5.7E-3	–	0.000	–
ALPHA-BHC	2	18	11%	0.048	0.002	–	6.3E+0	–	0.001	–
BETA-BHC	1	18	6%	0.049	0.016	–	1.8E+0	–	0.002	–
DELTA-BHC	1	18	6%	0.049	0.026	–	–	3.0E-4	–	0.001
BIS(2-ETHYLHEXYL)PHTHALATE	5	18	28%	1.281	2.071	–	1.4E-2	2.0E-2	0.002	0.001
BUTYLBENZYLPHTHALATE	1	18	6%	0.777	0.150	–	–	2.0E-1	–	0.000
GAMMA-CHLORDANE	7	21	33%	1.611	3.192	–	1.3E+0	6.0E-5	0.232	0.695
4-CHLOROANILINE	4	18	22%	1.337	2.002	–	–	4.0E-3	–	0.007
CHLOROBENZENE	10	18	56%	1.621	3.337	–	–	2.0E-2	–	0.002
2,4-D	1	15	7%	0.050	0.065	–	–	1.0E-2	–	0.000
4,4'-DDD	4	18	22%	0.127	0.217	–	2.4E-1	–	0.003	–
4,4'-DDE	3	18	17%	0.107	0.195	–	3.4E-1	–	0.004	–
4,4'-DDT	1	18	6%	0.095	0.008	–	3.4E-1	5.0E-4	0.000	0.000
DI-N-BUTYLPHTHALATE	1	18	6%	0.795	0.100	–	–	1.0E-1	–	0.000
DI-N-OCTYLPHTHALATE	1	18	6%	0.778	0.160	–	–	2.0E-2	–	0.000
DIBENZOFURAN	2	18	11%	0.519	0.718	–	–	1.0E-2	–	0.001
2,4-DICHLOROPHENOL	9	18	50%	0.644	0.909	–	–	3.0E-3	–	0.004
DIELDRIN	4	18	22%	0.104	0.170	–	1.6E+1	5.0E-5	0.152	0.044
DIETHYLPHTHALATE	4	18	22%	0.539	0.776	–	–	8.0E-1	–	0.000
DIMETHOATE	1	18	6%	0.140	0.026	–	–	2.0E-4	–	0.002
ENDRIN ALDEHYDE	3	18	17%	0.153	0.264	–	–	3.0E-4	–	0.011
ETHYL PARATHION	2	17	12%	0.036	0.046	–	–	6.0E-3	–	0.000
ETHYLBENZENE	4	18	22%	0.276	0.551	–	–	1.0E-1	–	0.000
FAMPHUR	3	17	18%	0.169	0.077	–	–	–	–	–
HEPTACHLOR	2	18	11%	0.053	0.098	–	4.5E+0	5.0E-4	0.025	0.003
IRGASAN DP-300	6	10	60%	51.820	111.551	–	–	3.0E-1	–	0.005
METHYL PARATHION	2	17	12%	0.012	0.014	–	–	2.5E-4	–	0.001

Table 6-B-37
Ranking Process for Chemicals of Potential Concern Selection
Waste Water Treatment Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]
METHYLENE CHLORIDE	7	18	39%	0.139	0.011	--	7.5E-3	6.0E-2	0.000	0.000
2-METHYLPHENOL	1	18	6%	0.594	0.827	--	--	5.0E-2	--	0.000
3&4-METHYLPHENOL	2	9	22%	0.203	0.251	--	--	5.0E-2	--	0.000
4-METHYLPHENOL	2	9	22%	1.270	0.290	--	--	5.0E-3	--	0.001
2-NITROANILINE	2	18	11%	3.885	0.073	--	--	6.0E-5	--	0.016
ORTHOPHOSPHATE	10	10	100%	31.041	61.502	--	--	--	--	--
PCB-1254	1	18	6%	0.955	0.210	--	--	2.0E-5	--	0.137
PHENOL	3	18	17%	0.563	0.800	--	--	6.0E-1	--	0.000
PHORATE	2	17	12%	0.030	0.017	--	--	2.0E-4	--	0.001
PROPAZINE	1	10	10%	6.041	10.784	--	--	2.0E-2	--	0.007
STYRENE	1	18	6%	0.039	0.019	--	--	2.0E-1	--	0.000
SULFOTEPP	5	17	29%	0.027	0.041	--	--	5.0E-4	--	0.001
2,4,5-T	4	15	27%	0.017	0.023	--	--	1.0E-2	--	0.000
2,3,7,8-TCDF	2	6	33%	0.000	0.001	--	1.6E+4	--	0.542	--
TCDF	2	6	33%	0.002	0.004	--	1.6E+2	--	0.039	--
TRCDF	1	1	100%	1.100	1.100	--	--	1.0E-2	--	0.001
TETRACHLOROETHENE	1	18	6%	0.038	0.006	--	5.2E-2	1.0E-2	0.000	0.000
TINUVIN 327	2	9	22%	5.978	10.996	--	--	2.5E-3	--	0.057
TOLUENE	11	18	61%	1.159	2.295	--	--	2.0E-1	--	0.000
1,2,4-TRICHLOROBENZENE	1	18	6%	0.791	0.034	--	--	1.0E-2	--	0.000
M&P-XYLENE	10	18	56%	1.018	2.096	--	--	2.0E+0	--	0.000
O-XYLENE	8	18	44%	0.393	0.799	--	--	2.0E+0	--	0.000

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation

Table 6-B-37
Ranking Process for Chemicals of Potential Concern Selection
Waste Water Treatment Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] $(mg/kg\text{-}day)^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]

limit is assumed for background samples in which the compound was not detected.

[c]Cancer slope factor. "--" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information System (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[f]Chronic reference dose; "--" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitue RfDs.

[g]Relative ranking score--carcinogenic.

[h]Relative ranking score--noncarcinogenic.

Table 6-B-38
Ranking Process for Chemicals of Potential Concern Selection Summary of Carcinogens
Waste Water Treatment Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RRS-C[f]	Cumulative RRS-C
2,3,7,8-TCDF	2	6	33%	0.000	0.001	--	1.6E+4	0.542	0.542
GAMMA-CHLORDANE	7	21	33%	1.611	3.192	--	1.3E+0	0.232	0.773
DIELDRIN	4	18	22%	0.104	0.170	--	1.6E+1	0.152	0.925
TCDF	2	6	33%	0.002	0.004	--	1.6E+2	0.039	0.964
HEPTACHLOR	2	18	11%	0.053	0.098	--	4.5E+0	0.025	0.989
4,4'-DDE	3	18	17%	0.107	0.195	--	3.4E-1	0.004	0.993
4,4'-DDD	4	18	22%	0.127	0.217	--	2.4E-1	0.003	0.995
BIS(2-ETHYLHEXYL)PHTHALATE	5	18	28%	1.281	2.071	--	1.4E-2	0.002	0.997
BETA-BHC	1	18	6%	0.049	0.016	--	1.8E+0	0.002	0.999
ALPHA-BHC	2	18	11%	0.048	0.002	--	6.3E+0	0.001	0.999
ANILINE	4	18	22%	0.893	1.409	--	5.7E-3	0.000	1.000
4,4'-DDT	1	18	6%	0.095	0.008	--	3.4E-1	0.000	1.000
TETRACHLOROETHENE	1	18	6%	0.038	0.006	--	5.2E-2	0.000	1.000
METHYLENE CHLORIDE	7	18	39%	0.139	0.011	--	7.5E-3	0.000	1.000

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Cancer slope factor. "--" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[f]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-39
Ranking Process for Chemicals of Potential Concern Selection
Waste Water Treatment Area Summary of Noncarcinogens
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	RfD[e] (mg/kg-day)	RRS-N[f]	Cumulative RRS-N
GAMMA-CHLORDANE	7	21	33%	1.611	3.192	--	6.0E-5	0.695	0.695
PCB-1254	1	18	6%	0.955	0.210	--	2.0E-5	0.137	0.832
TINUVIN 327	2	9	22%	5.978	10.996	--	2.5E-3	0.057	0.890
DIELDRIN	4	18	22%	0.104	0.170	--	5.0E-5	0.044	0.934
2-NITROANILINE	2	18	11%	3.885	0.073	--	6.0E-5	0.016	0.950
ENDRIN ALDEHYDE	3	18	17%	0.153	0.264	--	3.0E-4	0.011	0.961
PROPAZINE	1	10	10%	6.041	10.784	--	2.0E-2	0.007	0.968
4-CHLOROANILINE	4	18	22%	1.337	2.002	--	4.0E-3	0.007	0.975
IRGASAN DP-300	6	10	60%	51.820	111.551	--	3.0E-1	0.005	0.980
2,4-DICHLOROPHENOL	9	18	50%	0.644	0.909	--	3.0E-3	0.004	0.984
HEPTACHLOR	2	18	11%	0.053	0.098	--	5.0E-4	0.003	0.986
CHLOROBENZENE	10	18	56%	1.621	3.337	--	2.0E-2	0.002	0.989
DIMETHOATE	1	18	6%	0.140	0.026	--	2.0E-4	0.002	0.990
TRCDF	1	1	100%	1.100	1.100	--	1.0E-2	0.001	0.992
BIS(2-ETHYLHEXYL)PHTHALATE	5	18	28%	1.281	2.071	--	2.0E-2	0.001	0.993
DELTA-BHC	1	18	6%	0.049	0.026	--	3.0E-4	0.001	0.994
PHORATE	2	17	12%	0.030	0.017	--	2.0E-4	0.001	0.995
SULFOTEPP	5	17	29%	0.027	0.041	--	5.0E-4	0.001	0.996
DIBENZOFURAN	2	18	11%	0.519	0.718	--	1.0E-2	0.001	0.997
4-METHYLPHENOL	2	9	22%	1.270	0.290	--	5.0E-3	0.001	0.998
METHYL PARATHION	2	17	12%	0.012	0.014	--	2.5E-4	0.001	0.999
2-METHYLPHENOL	1	18	6%	0.594	0.827	--	5.0E-2	0.000	0.999
4,4'-DDT	1	18	6%	0.095	0.008	--	5.0E-4	0.000	0.999
TOLUENE	11	18	61%	1.159	2.295	--	2.0E-1	0.000	0.999
DI-N-OCTYLPHTHALATE	1	18	6%	0.778	0.160	--	2.0E-2	0.000	0.999
ETHYL PARATHION	2	17	12%	0.036	0.046	--	6.0E-3	0.000	1.000
2,4-D	1	15	7%	0.050	0.065	--	1.0E-2	0.000	1.000
ETHYLBENZENE	4	18	22%	0.276	0.551	--	1.0E-1	0.000	1.000
3&4-METHYLPHENOL	2	9	22%	0.203	0.251	--	5.0E-2	0.000	1.000

Table 6-B-39
Ranking Process for Chemicals of Potential Concern Selection
Waste Water Treatment Area Summary of Noncarcinogens
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	RfD[e] (mg/kg-day)	RRS-N[f]	Cumulative RRS-N
1,2,4-TRICHLOROBENZENE	1	18	6%	0.791	0.034	--	1.0E-2	0.000	1.000
2,4,5-T	4	15	27%	0.017	0.023	--	1.0E-2	0.000	1.000
PHENOL	3	18	17%	0.563	0.800	--	6.0E-1	0.000	1.000
ACETONE	4	18	22%	0.094	0.129	--	1.0E-1	0.000	1.000
M&P-XYLENE	10	18	56%	1.018	2.096	--	2.0E+0	0.000	1.000
DI-N-BUTYLPHTHALATE	1	18	6%	0.795	0.100	--	1.0E-1	0.000	1.000
DIETHYLPHTHALATE	4	18	22%	0.539	0.776	--	8.0E-1	0.000	1.000
BUTYLBENZYLPHTHALATE	1	18	6%	0.777	0.150	--	2.0E-1	0.000	1.000
TETRACHLOROETHENE	1	18	6%	0.038	0.006	--	1.0E-2	0.000	1.000
O-XYLENE	8	18	44%	0.393	0.799	--	2.0E+0	0.000	1.000
ACETOPHENONE	2	18	11%	0.782	0.026	--	1.0E-1	0.000	1.000
METHYLENE CHLORIDE	7	18	39%	0.139	0.011	--	6.0E-2	0.000	1.000
STYRENE	1	18	6%	0.039	0.019	--	2.0E-1	0.000	1.000

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Chronic reference dose; "--" No value available. Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitue RfDs.

[f]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-40
Ranking Process for Chemicals for Potential Concern Selection
Waste Water Treatment Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]
ACETONE	9	31	29%	0.103	0.126	--	--	1.0E-1	--	0.000
ACETOPHENONE	3	31	10%	0.644	0.026	--	--	1.0E-1	--	0.000
ALDRIN	1	31	3%	0.037	0.001	--	1.7E+1	3.0E-5	NA	NA
ANILINE	9	31	29%	0.715	1.031	--	5.7E-3	--	0.000	--
ALPHA-BHC	2	31	6%	0.037	0.002	--	6.3E+0	--	0.001	--
BETA-BHC	1	31	3%	0.038	0.016	--	1.8E+0	--	NA	NA
DELTA-BHC	3	31	10%	0.039	0.026	--	--	3.0E-4	--	0.002
GAMMA-BHC	3	31	10%	0.037	0.004	--	--	3.0E-4	--	0.000
BIS(2-ETHYLHEXYL)PHTHALATE	11	31	35%	0.940	1.412	--	1.4E-2	2.0E-2	0.001	0.001
BUTYLBENZYLPHTHALATE	2	31	6%	0.642	0.150	--	--	2.0E-1	--	0.000
ALPHA-CHLORDANE	1	31	3%	0.037	0.013	--	--	6.0E-5	NA	NA
GAMMA-CHLORDANE	14	34	41%	1.228	2.200	--	1.3E+0	6.0E-5	0.200	0.643
4-CHLOROANILINE	11	31	35%	1.019	1.425	--	--	4.0E-3	--	0.006
CHLOROBENZENE	11	31	35%	0.956	1.946	--	--	2.0E-2	--	0.002
2,4-D	3	23	13%	0.057	0.067	--	--	1.0E-2	--	0.000
4,4'-DDD	4	31	13%	0.092	0.145	--	2.4E-1	--	0.002	--
4,4'-DDE	3	31	10%	0.080	0.132	--	3.4E-1	--	0.003	--
4,4'-DDT	2	31	6%	0.074	0.024	--	3.4E-1	5.0E-4	0.001	0.001
DI-N-BUTYLPHTHALATE	4	31	13%	0.649	0.410	--	--	1.0E-1	--	0.000
DI-N-OCTYLPHTHALATE	1	31	3%	0.646	0.160	--	--	2.0E-2	NA	NA
DIBENZOFURAN	3	31	10%	0.494	0.650	--	--	1.0E-2	--	0.001
2,4-DICHLOROPHENOL	15	31	48%	0.563	0.750	--	--	3.0E-3	--	0.004
DIELDRIN	6	31	19%	0.079	0.130	--	1.6E+1	5.0E-5	0.145	0.045
DIETHYLPHTHALATE	4	31	13%	0.508	0.679	--	--	8.0E-1	--	0.000
DIMETHOATE	1	31	3%	0.134	0.026	--	--	2.0E-4	NA	NA
ENDOSULFAN I	1	31	3%	0.037	0.013	--	--	6.0E-3	NA	NA
ENDRIN	1	31	3%	0.068	0.019	--	--	3.0E-4	NA	NA
ENDRIN ALDEHYDE	5	31	16%	0.138	0.220	--	--	3.0E-4	--	0.013
ETHYL PARATHION	3	30	10%	0.032	0.040	--	--	6.0E-3	--	0.000

Table 6-B-40
Ranking Process for Chemicals for Potential Concern Selection
Waste Water Treatment Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]
ETHYLBENZENE	4	31	13%	0.171	0.330	--	--	1.0E-1	--	0.000
FAMPHUR	4	30	13%	0.164	0.077	--	--	--	--	--
HEPTACHLOR	3	31	10%	0.040	0.067	--	4.5E+0	5.0E-4	0.021	0.002
IRGASAN DP-300	8	18	44%	34.983	67.284	--	--	3.0E-1	--	0.004
KEPONE	1	31	3%	0.323	0.005	--	1.8E+1	--	NA	NA
METHYL PARATHION	3	30	10%	0.010	0.012	--	--	2.5E-4	--	0.001
METHYLENE CHLORIDE	12	31	39%	0.101	0.011	--	7.5E-3	6.0E-2	0.000	0.000
2-METHYLPHENOL	1	31	3%	0.540	0.710	--	--	5.0E-2	NA	NA
3&4-METHYLPHENOL	3	16	19%	0.188	0.215	--	--	5.0E-2	--	0.000
4-METHYLPHENOL	2	15	13%	1.082	0.290	--	--	5.0E-3	--	0.001
2-NITROANILINE	2	31	6%	3.254	0.073	--	--	6.0E-5	--	0.021
ORTHOPHOSPHATE	13	16	81%	19.828	38.837	--	--	--	--	--
PCB-1254	2	31	6%	0.736	0.210	--	--	2.0E-5	--	0.184
PHENOL	4	31	13%	0.509	0.683	--	--	6.0E-1	--	0.000
PHORATE	2	30	7%	0.030	0.017	--	--	2.0E-4	--	0.001
PROPAZINE	2	18	11%	4.772	7.449	--	--	2.0E-2	--	0.007
STYRENE	2	31	6%	0.034	0.019	--	--	2.0E-1	--	0.000
SULFOTEPP	5	30	17%	0.024	0.032	--	--	5.0E-4	--	0.001
2,4,5-T	4	23	17%	0.015	0.018	--	--	1.0E-2	--	0.000
2,3,7,8-TCDF	3	7	43%	2.12E-04	5.33E-04	--	1.6E+4	--	0.581	--
TCDF	3	7	43%	1.63E-03	0.004	--	1.6E+2	--	0.044	--
TETRACHLOROETHENE	1	31	3%	0.033	0.006	--	5.2E-2	1.0E-2	NA	NA
THIONAZIN	1	30	3%	0.164	0.006	--	--	3.0E-3	NA	NA
TINUVIN 327	2	15	13%	5.220	8.204	--	--	2.5E-3	--	0.058
TOLUENE	15	31	48%	0.687	1.345	--	--	2.0E-1	--	0.000
TRCDF	2	3	67%	0.767	1.200	--	--	--	--	--
1,2,4-TRICHLOROBENZENE	1	31	3%	0.654	0.034	--	--	1.0E-2	NA	NA
M&P-XYLENE	11	31	35%	0.610	1.231	--	--	2.0E+0	--	0.000
O-XYLENE	9	31	29%	0.244	0.477	--	--	2.0E+0	--	0.000

Table 6-B-40
Ranking Process for Chemicals for Potential Concern Selection
Waste Water Treatment Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] $(mg/kg\text{-}day)^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Cancer slope factor. "--" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information System (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[f]Chronic reference dose; "--" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitue RfDs.

[g]Relative ranking score--carcinogenic.

[h]Relative ranking score--noncarcinogenic.

Table 6-B-41
Ranking Process for Chemicals for Potential Concern Selection Summary of Carcinogens
Waste Water Treatment Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RRS-C[f]	Cumulative RRS-C
2,3,7,8-TCDF	3	7	43%	2.12E-04	5.33E-04	--	1.6E+4	0.581	0.581
GAMMA-CHLORDANE	14	34	41%	1.228	2.200	--	1.3E+0	0.200	0.781
DIELDRIN	6	31	19%	0.079	0.130	--	1.6E+1	0.145	0.926
TCDF	3	7	43%	1.63E-03	0.004	--	1.6E+2	0.044	0.970
HEPTACHLOR	3	31	10%	0.040	0.067	--	4.5E+0	0.021	0.991
4,4'-DDE	3	31	10%	0.080	0.132	--	3.4E-1	0.003	0.994
4,4'-DDD	4	31	13%	0.092	0.145	--	2.4E-1	0.002	0.997
BIS(2-ETHYLHEXYL)PHTHALATE	11	31	35%	0.940	1.412	--	1.4E-2	0.001	0.998
ALPHA-BHC	2	31	6%	0.037	0.002	--	6.3E+0	0.001	0.999
4,4'-DDT	2	31	6%	0.074	0.024	--	3.4E-1	0.001	1.000
ANILINE	9	31	29%	0.715	1.031	--	5.7E-3	0.000	1.000
METHYLENE CHLORIDE	12	31	39%	0.101	0.011	--	7.5E-3	0.000	1.000

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Cancer slope factor. "--" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[f]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-42
Ranking Process for Chemicals for Potential Concern Selection Summary of Noncarcinogens
Waste Water Treatment Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	RfD[e] (mg/kg-day)	RRS-N[f]	Cumulative RRS-N
GAMMA-CHLORDANE	14	34	41%	1.228	2.200	--	6.0E-5	0.643	0.643
PCB-1254	2	31	6%	0.736	0.210	--	2.0E-5	0.184	0.827
TINUVIN 327	2	15	13%	5.220	8.204	--	2.5E-3	0.058	0.885
DIELDRIN	6	31	19%	0.079	0.130	--	5.0E-5	0.045	0.931
2-NITROANILINE	2	31	6%	3.254	0.073	--	6.0E-5	0.021	0.952
ENDRIN ALDEHYDE	5	31	16%	0.138	0.220	--	3.0E-4	0.013	0.965
PROPAZINE	2	18	11%	4.772	7.449	--	2.0E-2	0.007	0.971
4-CHLOROANILINE	11	31	35%	1.019	1.425	--	4.0E-3	0.006	0.978
2,4-DICHLOROPHENOL	15	31	48%	0.563	0.750	--	3.0E-3	0.004	0.982
IRGASAN DP-300	8	18	44%	34.983	67.284	--	3.0E-1	0.004	0.986
HEPTACHLOR	3	31	10%	0.040	0.067	--	5.0E-4	0.002	0.988
CHLOROBENZENE	11	31	35%	0.956	1.946	--	2.0E-2	0.002	0.990
DELTA-BHC	3	31	10%	0.039	0.026	--	3.0E-4	0.002	0.991
PHORATE	2	30	7%	0.030	0.017	--	2.0E-4	0.001	0.993
BIS(2-ETHYLHEXYL)PHTHALATE	11	31	35%	0.940	1.412	--	2.0E-2	0.001	0.994
DIBENZOFURAN	3	31	10%	0.494	0.650	--	1.0E-2	0.001	0.995
SULFOTEPP	5	30	17%	0.024	0.032	--	5.0E-4	0.001	0.996
4-METHYLPHENOL	2	15	13%	1.082	0.290	--	5.0E-3	0.001	0.997
4,4'-DDT	2	31	6%	0.074	0.024	--	5.0E-4	0.001	0.998
METHYL PARATHION	3	30	10%	0.010	0.012	--	2.5E-4	0.001	0.999
GAMMA-BHC	3	31	10%	0.037	0.004	--	3.0E-4	0.000	0.999
TOLUENE	15	31	48%	0.687	1.345	--	2.0E-1	0.000	0.999
2,4-D	3	23	13%	0.057	0.067	--	1.0E-2	0.000	1.000
ETHYL PARATHION	3	30	10%	0.032	0.040	--	6.0E-3	0.000	1.000
3&4-METHYLPHENOL	3	16	19%	0.188	0.215	--	5.0E-2	0.000	1.000
DI-N-BUTYLPHTHALATE	4	31	13%	0.649	0.410	--	1.0E-1	0.000	1.000
ETHYLBENZENE	4	31	13%	0.171	0.330	--	1.0E-1	0.000	1.000
2,4,5-T	4	23	17%	0.015	0.018	--	1.0E-2	0.000	1.000
ACETONE	9	31	29%	0.103	0.126	--	1.0E-1	0.000	1.000

Table 6-B-42
Ranking Process for Chemicals for Potential Concern Selection Summary of Noncarcinogens
Waste Water Treatment Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	RfD[e] (mg/kg-day)	RRS-N[f]	Cumulative RRS-N
PHENOL	4	31	13%	0.509	0.683	--	6.0E-1	0.000	1.000
DIETHYLPHTHALATE	4	31	13%	0.508	0.679	--	8.0E-1	0.000	1.000
BUTYLBENZYLPHTHALATE	2	31	6%	0.642	0.150	--	2.0E-1	0.000	1.000
M&P-XYLENE	11	31	35%	0.610	1.231	--	2.0E+0	0.000	1.000
ACETOPHENONE	3	31	10%	0.644	0.026	--	1.0E-1	0.000	1.000
O-XYLENE	9	31	29%	0.244	0.477	--	2.0E+0	0.000	1.000
METHYLENE CHLORIDE	12	31	39%	0.101	0.011	--	6.0E-2	0.000	1.000
STYRENE	2	31	6%	0.034	0.019	--	2.0E-1	0.000	1.000

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Chronic reference dose; "--" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitue RfDs.

[f]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-43
Ranking Process for Chemicals of Potential Concern Selection
Waste Water Treatment Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]
ACETOPHENONE	1	24	4%	4.867	3.000	--	--	1.0E-1	NA	NA
ALDRIN	2	24	8%	0.061	0.011	--	1.7E+1	3.0E-5	0.038	0.013
ANILINE	2	24	8%	21.283	28.593	--	5.7E-3	--	0.033	--
ALPHA-BHC	2	24	8%	0.061	0.015	--	6.3E+0	--	0.019	--
DELTA-BHC	3	24	13%	0.075	0.128	--	--	3.0E-4	--	0.015
BIS(2-ETHYLHEXYL)PHTHALATE	2	24	8%	10.250	19.493	--	1.4E-2	2.0E-2	0.055	0.035
BUTAZOLIDIN	3	24	13%	69.983	20.000	--	--	1.4E-1	--	0.036
ALPHA-CHLORDANE	1	24	4%	0.071	0.003	--	1.3E+0	6.0E-5	NA	NA
GAMMA-CHLORDANE	3	24	13%	0.103	0.192	--	1.3E+0	6.0E-5	0.050	0.115
4-CHLORO-3-METHYLPHENOL	1	24	4%	4.758	0.600	--	--	--	NA	NA
4-CHLOROANILINE	2	24	8%	6.408	6.000	--	--	4.0E-3	--	0.054
CHLOROBENZENE	4	24	17%	3.400	4.400	--	--	2.0E-2	--	0.008
CHLOROBENZILATE	1	24	4%	2.817	0.500	--	2.7E-1	2.0E-2	NA	NA
CHLOROETHANE	1	24	4%	5.500	6.357	--	--	4.0E-1	NA	NA
CHLOROFORM	3	24	13%	2.833	3.195	--	6.1E-3	1.0E-2	0.004	0.011
4,4'-DDE	1	24	4%	0.097	0.012	--	3.4E-1	--	NA	NA
4,4'-DDT	1	24	4%	0.120	0.007	--	3.4E-1	5.0E-4	NA	NA
DI-N-BUTYLPHTHALATE	2	24	8%	4.607	1.000	--	--	1.0E-1	--	0.000
DI-N-OCTYLPHTHALATE	1	24	4%	4.790	1.100	--	--	2.0E-2	NA	NA
DIBENZOFURAN	1	24	4%	4.750	0.400	--	--	1.0E-2	NA	NA
1,2-DICHLOROBENZENE	3	24	13%	4.471	2.000	--	--	9.0E-2	--	0.001
1,1-DICHLOROETHANE	4	24	17%	2.583	2.951	--	--	1.0E-1	--	0.001
2,4-DICHLOROPHENOL	4	24	17%	6.113	7.858	--	--	3.0E-3	--	0.094
DIELDRIN	3	24	13%	0.115	0.196	--	1.6E+1	5.0E-5	0.632	0.140
DIETHYLPHTHALATE	1	24	4%	4.783	1.000	--	--	8.0E-1	NA	NA
DINOSEB	1	24	4%	7.534	0.038	--	--	1.0E-3	NA	NA
ENDRIN ALDEHYDE	1	24	4%	0.148	0.277	--	--	3.0E-4	NA	NA
ETHYLBENZENE	3	24	13%	2.621	2.794	--	--	1.0E-1	--	0.001
HEPTACHLOR	1	24	4%	0.062	0.045	--	4.5E+0	5.0E-4	NA	NA

Table 6-B-43
Ranking Process for Chemicals of Potential Concern Selection
Waste Water Treatment Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]
1,2,3,4,6,7,8-HPCDF	2	12	17%	0.000	0.000	--	1.6E+3	--	0.025	--
HPCDF	2	12	17%	0.000	0.001	--	1.6E+0	--	0.000	--
IRGASAN DP-300	5	24	21%	79.625	130.922	--	--	3.0E-1	--	0.016
ISODRIN	3	24	13%	0.058	0.106	--	--	3.0E-5	--	0.126
3&4-METHYLPHENOL	1	12	8%	4.625	0.500	--	--	5.0E-2	--	0.000
4-METHYLPHENOL	2	12	17%	13.067	23.388	--	--	5.0E-3	--	0.168
N-NITROSODIMETHYLAMINE	1	24	4%	4.983	5.067	--	--	5.1E+1	NA	NA
OCDD	4	12	33%	0.001	0.001	--	1.6E+2	--	0.038	--
OCDF	2	12	17%	0.000	0.000	--	1.6E+2	--	0.006	--
ORTHOPHOSPHATE	2	12	17%	0.013	0.024	--	--	--	--	--
PHENOL	2	24	8%	9.117	14.100	--	--	6.0E-1	--	0.001
PROPAZINE	3	24	13%	45.667	60.863	--	--	2.0E-2	--	0.109
TCDD	1	12	8%	0.004	0.000	--	1.6E+3	--	0.020	--
TCDF	3	12	25%	0.001	0.001	--	1.6E+2	--	0.031	--
TKN	7	12	58%	2.694	5.187	--	--	--	--	--
TOFRANIL	3	24	13%	30.000	35.262	--	--	5.7E-2	--	0.055
TOLUENE	3	24	13%	3.650	5.616	--	--	2.0E-1	--	0.001
TRCDF	1	6	17%	1.517	4.573	--	--	--	--	--
1,1,1-TRICHLOROETHANE	2	24	8%	2.538	2.674	--	9.0E-2	--	0.048	--
TRICHLOROETHENE	1	24	4%	2.517	2.545	--	1.1E-2	6.0E-3	NA	NA
M&P-XYLENE	2	24	8%	3.792	5.407	--	--	2.0E+0	--	0.000
O-XYLENE	2	24	8%	3.000	3.646	--	--	2.0E+0	--	0.000

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation

Table 6-B-43
Ranking Process for Chemicals of Potential Concern Selection
Waste Water Treatment Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]

limit is assumed for background samples in which the compound was not detected.

[e]Cancer slope factor. "--" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information System (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[f]Chronic reference dose; "--" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitue RfDs.

[g]Relative ranking score--carcinogenic.

[h]Relative ranking score--noncarcinogenic.

Table 6-B-44
Ranking Process for Chemicals of Potential Concern Selection Summary of Carcinogens
Waste Water Treatment Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RRS-C[f]	Cumulative RRS-C
DIELDRIN	3	24	13%	0.115	0.196	--	1.6E+1	0.632	0.632
BIS(2-ETHYLHEXYL)PHTHALATE	2	24	8%	10.250	19.493	--	1.4E-2	0.055	0.687
GAMMA-CHLORDANE	3	24	13%	0.103	0.192	--	1.3E+0	0.050	0.737
1,1,1-TRICHLOROETHANE	2	24	8%	2.538	2.674	--	9.0E-2	0.048	0.785
ALDRIN	2	24	8%	0.061	0.011	--	1.7E+1	0.038	0.823
OCDD	4	12	33%	0.001	0.001	--	1.6E+2	0.038	0.861
ANILINE	2	24	8%	21.283	28.593	--	5.7E-3	0.033	0.893
TCDF	3	12	25%	0.001	0.001	--	1.6E+2	0.031	0.925
1,2,3,4,6,7,8-HPCDF	2	12	17%	0.000	0.000	--	1.6E+3	0.025	0.950
TCDD	1	12	8%	0.004	0.000	--	1.6E+3	0.020	0.970
ALPHA-BHC	2	24	8%	0.061	0.015	--	6.3E+0	0.019	0.989
OCDF	2	12	17%	0.000	0.000	--	1.6E+2	0.006	0.996
CHLOROFORM	3	24	13%	2.833	3.195	--	6.1E-3	0.004	1.000
HPCDF	2	12	17%	0.000	0.001	--	1.6E+0	0.000	1.000

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Cancer slope factor. "--" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[f]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-45
Ranking Process for Chemicals of Potential Concern Selection Summary of Noncarcinogens
Waste Water Treatment Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	RfD[e] (mg/kg-day)	RRS-N[f]	Cumulative RRS-N
4-METHYLPHENOL	2	12	17%	13.067	23.388	–	5.0E-3	0.168	0.168
DIELDRIN	3	24	13%	0.115	0.196	–	5.0E-5	0.140	0.308
ISODRIN	3	24	13%	0.058	0.106	–	3.0E-5	0.126	0.434
GAMMA-CHLORDANE	3	24	13%	0.103	0.192	–	6.0E-5	0.115	0.549
PROPAZINE	3	24	13%	45.667	60.863	–	2.0E-2	0.109	0.658
2,4-DICHLOROPHENOL	4	24	17%	6.113	7.858	–	3.0E-3	0.094	0.752
TOFRANIL	3	24	13%	30.000	35.262	–	5.7E-2	0.055	0.806
4-CHLOROANILINE	2	24	8%	6.408	6.000	–	4.0E-3	0.054	0.860
BUTAZOLIDIN	3	24	13%	69.983	20.000	–	1.4E-1	0.036	0.975
BIS(2-ETHYLHEXYL)PHTHALATE	2	24	8%	10.250	19.493	–	2.0E-2	0.035	0.895
IRGASAN DP-300	5	24	21%	79.625	130.922	–	3.0E-1	0.016	0.911
DELTA-BHC	3	24	13%	0.075	0.128	–	3.0E-4	0.015	0.926
ALDRIN	2	24	8%	0.061	0.011	–	3.0E-5	0.013	0.939
CHLOROFORM	3	24	13%	2.833	3.195	–	1.0E-2	0.011	0.987
CHLOROBENZENE	4	24	17%	3.400	4.400	–	2.0E-2	0.008	0.994
1,1-DICHLOROETHANE	4	24	17%	2.583	2.951	–	1.0E-1	0.001	0.995
TOLUENE	3	24	13%	3.650	5.616	–	2.0E-1	0.001	0.996
ETHYLBENZENE	3	24	13%	2.621	2.794	–	1.0E-1	0.001	0.997
PHENOL	2	24	8%	9.117	14.100	–	6.0E-1	0.001	0.998
1,2-DICHLOROBENZENE	3	24	13%	4.471	2.000	–	9.0E-2	0.001	0.999
DI-N-BUTYLPHTHALATE	2	24	8%	4.607	1.000	–	1.0E-1	0.000	0.999
3&4-METHYLPHENOL	1	12	8%	4.625	0.500	–	5.0E-2	0.000	1.000
M&P-XYLENE	2	24	8%	3.792	5.407	–	2.0E+0	0.000	1.000
O-XYLENE	2	24	8%	3.000	3.646	–	2.0E+0	0.000	1.000

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation

Table 6-B-45
Ranking Process for Chemicals of Potential Concern Selection Summary of Noncarcinogens
Waste Water Treatment Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	RfD[e] (mg/kg-day)	RRS-N[f]	Cumulative RRS-N

limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Chronic reference dose; "--" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

[f]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-46
Ranking Process for Chemicals of Potential Concern Selection
Warwick Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]
ALDRIN	3	33	9%	0.11	0.21	--	1.7E+1	3.0E-5	0.348	0.007
ALPHA-BHC	6	33	18%	0.09	0.18	--	6.3E+0	--	0.113	--
ALPHA-CHLORDANE	3	32	9%	0.05	0.08	--	--	6.0E-5	--	0.001
ANTIMONY	6	23	26%	2.27	5.35	0.00	--	4.0E-4	--	0.013
BERYLLIUM	31	31	100%	0.70	0.82	0.61	4.3E+0	5.0E-3	0.257	0.000
BETA-BHC	2	32	6%	0.05	0.01	--	1.8E+0	--	0.002	--
GAMMA-BHC	2	32	6%	0.05	0.00	--	1.3E+0	3.0E-4	0.000	0.000
1,1-BIPHENYL	1	2	50%	1.04	1.90	--	--	5.0E-2	--	0.000
BIS(2-CHLOROETHYL)ETHER	2	31	6%	1.38	0.43	--	1.1E+0	--	0.047	--
BIS(2-ETHYLHEXYL)PHTHALATE	14	31	45%	10.46	20.04	--	1.4E-2	2.0E-2	0.028	0.001
2-BUTANONE	2	32	6%	0.10	0.12	--	--	6.0E-1	--	0.000
BUTYLBENZYLPHTHALATE	6	31	19%	1.39	0.78	--	--	2.0E-1	--	0.000
CADMIUM	15	31	48%	1.56	2.22	0.47	--	5.0E-4	--	0.004
GAMMA-CHLORDANE	9	32	28%	0.08	0.15	--	1.3E+0	6.0E-5	0.019	0.003
4-CHLOROANILINE	10	31	32%	1.44	2.01	--	--	4.0E-3	--	0.001
CHLOROBENZENE	9	32	28%	0.41	0.67	--	--	2.0E-2	--	0.000
CHROMIUM	31	31	100%	68.15	101.04	12.65	--	5.0E-3	--	0.020
4,4'-DDD	2	32	6%	0.07	0.13	--	2.4E-1	--	0.003	--
4,4'-DDE	6	33	18%	0.08	0.15	--	3.4E-1	--	0.005	--
4,4'-DDT	9	32	28%	0.12	0.25	--	3.4E-1	5.0E-4	0.008	0.001
DELTA-BHC	3	33	9%	0.06	0.12	--	--	3.0E-4	--	0.000
DI-N-OCTYLPHTHALATE	8	31	26%	2.52	4.12	--	--	2.0E-2	--	0.000
DIBENZOFURAN	3	31	10%	1.36	0.20	--	--	1.0E-2	--	0.000
1,4-DICHLOROBENZENE	2	31	6%	1.39	0.04	--	2.4E-2	--	0.000	--
DIELDRIN	5	32	16%	0.08	0.16	--	1.6E+1	5.0E-5	0.253	0.003
DISULFOTON	3	30	10%	0.06	0.01	--	--	4.0E-5	--	0.000
ENDOSULFAN I	3	32	9%	0.05	0.02	--	--	6.0E-3	--	0.000
ENDOSULFAN SULFATE	3	32	9%	0.25	0.29	--	--	6.0E-3	--	0.000
ENDRIN	6	32	19%	0.07	0.14	--	--	3.0E-4	--	0.000
ENDRIN ALDEHYDE	5	32	16%	0.22	0.44	--	--	3.0E-4	--	0.002
ETHYL PARATHION	2	30	7%	0.04	0.01	--	--	6.0E-3	--	0.000
HEPTACHLOR EPOXIDE	7	32	22%	0.10	0.19	--	9.1E+0	1.3E-5	0.172	0.015
ISODRIN	2	32	6%	0.08	0.16	--	--	3.0E-5	--	0.005

Table 6-B-46
Ranking Process for Chemicals of Potential Concern Selection
Warwick Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]
METHOXYCHLOR	13	34	38%	110.39	231.88	--	--	5.0E-3	--	0.048
METHYLENE CHLORIDE	3	32	9%	0.24	0.03	--	7.5E-3	6.0E-2	0.000	0.000
3&4-METHYLPHENOL	1	6	17%	0.15	0.02	--	--	5.0E-2	--	0.000
2-NITROANILINE	2	31	6%	7.15	7.00	--	--	6.0E-5	--	0.120
NITROBENZENE	2	31	6%	1.48	2.13	--	--	5.0E-4	--	0.004
ORTHOPHOSPHATE	20	25	80%	4.57	6.88	--	--	--	--	--
PCB-1248	3	34	9%	6.89	15.16	--	--	3.0E-5	--	0.518
PCB-1254	15	32	47%	2.86	5.19	--	--	2.0E-5	--	0.266
PHENOL	3	31	10%	1.18	0.89	--	--	6.0E-1	--	0.000
SAFROLE	4	31	13%	2.46	4.05	--	--	2.0E-2	--	0.000
TETRACHLOROETHENE	9	32	28%	0.13	0.25	--	5.2E-2	--	0.001	--
TINUVIN 327	8	25	32%	6.34	9.27	--	--	2.5E-3	--	0.004
TOLUENE	18	32	56%	0.23	0.36	--	--	2.0E-1	--	0.000
2,4,5-TP (SILVEX)	4	31	13%	0.03	0.05	--	--	8.0E-3	--	0.000
TRICHLOROETHENE	2	32	6%	0.05	0.06	--	1.1E-2	--	0.000	--
M&P-XYLENE	8	32	25%	0.05	0.05	--	--	2.0E+0	--	0.000
O-XYLENE	4	32	13%	0.04	0.05	--	--	2.0E+0	--	0.000
ZINC	28	31	90%	2538.67	3911.97	75.94	--	3.0E-1	--	0.013

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Cancer slope factor. "--" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information System (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[f]Chronic reference dose; "--" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

[g]Relative ranking score--carcinogenic.

[h]Relative ranking score--noncarcinogenic.

Table 6-B-47
Ranking Process for Chemicals of Potential Concern Selection Summary of Carcinogens
Warwick Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RRS-C[f]	Cumulative RRS-C
ALDRIN	3	33	9%	0.11	0.21	--	1.7E+1	0.348	0.348
BERYLLIUM	31	31	100%	0.70	0.82	0.61	4.3E+0	0.257	0.605
DIELDRIN	5	32	16%	0.08	0.16	--	1.6E+1	0.253	0.859
HEPTACHLOR EPOXIDE	7	32	22%	0.10	0.19	--	9.1E+0	0.172	1.030
ALPHA-BHC	6	33	18%	0.09	0.18	--	6.3E+0	0.113	1.144
BIS(2-CHLOROETHYL)ETHER	2	31	6%	1.38	0.43	--	1.1E+0	0.047	1.190
BIS(2-ETHYLHEXYL)PHTHALATE	14	31	45%	10.46	20.04	--	1.4E-2	0.028	1.218
GAMMA-CHLORDANE	9	32	28%	0.08	0.15	--	1.3E+0	0.019	1.237
4,4'-DDT	9	32	28%	0.12	0.25	--	3.4E-1	0.008	1.245
4,4'-DDE	6	33	18%	0.08	0.15	--	3.4E-1	0.005	1.250
4,4'-DDD	2	32	6%	0.07	0.13	--	2.4E-1	0.003	1.254
BETA-BHC	2	32	6%	0.05	0.01	--	1.8E+0	0.002	1.255
TETRACHLOROETHENE	9	32	28%	0.13	0.25	--	5.2E-2	0.001	1.257
GAMMA-BHC	2	32	6%	0.05	0.00	--	1.3E+0	0.000	1.257
1,4-DICHLOROBENZENE	2	31	6%	1.39	0.04	--	2.4E-2	0.000	1.257
TRICHLOROETHENE	2	32	6%	0.05	0.06	--	1.1E-2	0.000	1.257
METHYLENE CHLORIDE	3	32	9%	0.24	0.03	--	7.5E-3	0.000	1.257

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Cancer slope factor. "--" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[f]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-48
Ranking Process for Chemicals of Potential Concern Selection Summary of Noncarcinogens
Warwick Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	RfD[e] (mg/kg-day)	RRS-N[f]	Cumulative RRS-N
PCB-1248	3	34	9%	6.89	15.16	--	3.0E-5	0.518	0.518
PCB-1254	15	32	47%	2.86	5.19	--	2.0E-5	0.266	0.785
2-NITROANILINE	2	31	6%	7.15	7.00	--	6.0E-5	0.120	0.904
METHOXYCHLOR	13	34	38%	110.39	231.88	--	5.0E-3	0.048	0.952
CHROMIUM	31	31	100%	68.15	101.04	12.65	5.0E-3	0.020	0.972
HEPTACHLOR EPOXIDE	7	32	22%	0.10	0.19	--	1.3E-5	0.015	0.987
ANTIMONY	6	23	26%	2.27	5.35	0.00	4.0E-4	0.013	1.000
ZINC	28	31	90%	2538.67	3911.97	75.94	3.0E-1	0.013	1.013
ALDRIN	3	33	9%	0.11	0.21	--	3.0E-5	0.007	1.020
ISODRIN	2	32	6%	0.08	0.16	--	3.0E-5	0.005	1.025
NITROBENZENE	2	31	6%	1.48	2.13	--	5.0E-4	0.004	1.030
CADMIUM	15	31	48%	1.56	2.22	0.47	5.0E-4	0.004	1.034
TINUVIN 327	8	25	32%	6.34	9.27	--	2.5E-3	0.004	1.038
DIELDRIN	5	32	16%	0.08	0.16	--	5.0E-5	0.003	1.041
GAMMA-CHLORDANE	9	32	28%	0.08	0.15	--	6.0E-5	0.003	1.043
ENDRIN ALDEHYDE	5	32	16%	0.22	0.44	--	3.0E-4	0.002	1.045
ALPHA-CHLORDANE	3	32	9%	0.05	0.08	--	6.0E-5	0.001	1.046
BIS(2-ETHYLHEXYL)PHTHALATE	14	31	45%	10.46	20.04	--	2.0E-2	0.001	1.047
4,4'-DDT	9	32	28%	0.12	0.25	--	5.0E-4	0.001	1.048
4-CHLOROANILINE	10	31	32%	1.44	2.01	--	4.0E-3	0.001	1.048
ENDRIN	6	32	19%	0.07	0.14	--	3.0E-4	0.000	1.049
DELTA-BHC	3	33	9%	0.06	0.12	--	3.0E-4	0.000	1.049
DI-N-OCTYLPHTHALATE	8	31	26%	2.52	4.12	--	2.0E-2	0.000	1.049
SAFROLE	4	31	13%	2.46	4.05	--	2.0E-2	0.000	1.050
DISULFOTON	3	30	10%	0.06	0.01	--	4.0E-5	0.000	1.050
ENDOSULFAN SULFATE	3	32	9%	0.25	0.29	--	6.0E-3	0.000	1.050
1,1-BIPHENYL	1	2	50%	1.04	1.90	--	5.0E-2	0.000	1.050
CHLOROBENZENE	9	32	28%	0.41	0.67	--	2.0E-2	0.000	1.050
DIBENZOFURAN	3	31	10%	1.36	0.20	--	1.0E-2	0.000	1.050

Table 6-B-48
Ranking Process for Chemicals of Potential Concern Selection Summary of Noncarcinogens
Warwick Area
Surface Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	RfD[e] (mg/kg-day)	RRS-N[f]	Cumulative RRS-N
2,4,5-TP (SILVEX)	4	31	13%	0.03	0.05	--	8.0E-3	0.000	1.050
GAMMA-BHC	2	32	6%	0.05	0.00	--	3.0E-4	0.000	1.050
BUTYLBENZYLPHTHALATE	6	31	19%	1.39	0.78	--	2.0E-1	0.000	1.050
ENDOSULFAN I	3	32	9%	0.05	0.02	--	6.0E-3	0.000	1.050
TOLUENE	18	32	56%	0.23	0.36	--	2.0E-1	0.000	1.050
PHENOL	3	31	10%	1.18	0.89	--	6.0E-1	0.000	1.050
ETHYL PARATHION	2	30	7%	0.04	0.01	--	6.0E-3	0.000	1.050
3&4-METHYLPHENOL	1	6	17%	0.15	0.02	--	5.0E-2	0.000	1.050
METHYLENE CHLORIDE	3	32	9%	0.24	0.03	--	6.0E-2	0.000	1.050
2-BUTANONE	2	32	6%	0.10	0.12	--	6.0E-1	0.000	1.050
M&P-XYLENE	8	32	25%	0.05	0.05	--	2.0E+0	0.000	1.050
O-XYLENE	4	32	13%	0.04	0.05	--	2.0E+0	0.000	1.050
BERYLLIUM	31	31	100%	0.70	0.82	0.61	5.0E-3	0.000	1.050

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Chronic reference dose; "--" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

[f]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-49
Ranking Process for Chemicals of Potential Concern Selection
Warwick Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]
ACETONE	1	52	2%	0.116	0.183	–	NA	NA	NA	NA
ALDRIN	3	53	6%	0.076	0.137	–	1.7E+1	3.0E-5	0.339	0.007
BENZENE	1	52	2%	0.041	0.034	–	NA	NA	NA	NA
ALPHA-BHC	7	53	13%	0.066	0.121	–	6.3E+0	–	0.111	–
ANTIMONY	7	30	23%	1.840	4.190	0.000	–	4.0E-4	–	0.014
BERYLLIUM	43	44	98%	0.680	0.850	0.600	4.3E+0	5.0E-3	0.326	0.000
BETA-BHC	2	52	4%	0.039	0.010	–	NA	NA	NA	NA
DELTA-BHC	4	53	8%	0.043	0.084	–	–	3.0E-4	–	0.000
GAMMA-BHC	2	52	4%	0.039	0.002	–	NA	NA	NA	NA
1,1-BIPHENYL	1	2	50%	1.038	1.900	–	–	5.0E-2	–	0.000
BIS(2-CHLOROETHYL)ETHER	2	41	5%	1.098	0.430	–	NA	NA	NA	NA
BIS(2-ETHYLHEXYL)PHTHALATE	18	41	44%	11.885	21.446	–	1.4E-2	2.0E-2	0.044	0.002
2-BUTANONE	2	52	4%	0.115	0.182	–	NA	NA	NA	NA
BUTYLBENZYLPHTHALATE	7	41	17%	1.102	0.780	–	–	2.0E-1	–	0.000
CADMIUM	17	38	45%	1.550	2.170	0.440	–	5.0E-4	–	0.006
ALPHA-CHLORDANE	6	52	12%	0.042	0.077	–	–	6.0E-5	–	0.002
GAMMA-CHLORDANE	13	52	25%	0.059	0.104	–	1.3E+0	6.0E-5	0.020	0.003
4-CHLOROANILINE	13	41	32%	1.184	1.625	–	–	4.0E-3	–	0.001
CHLOROBENZENE	12	52	23%	10.063	26.571	–	–	2.0E-2	–	0.002
CHLOROBENZILATE	1	55	2%	0.858	0.046	–	NA	NA	NA	NA
CHLOROFORM	1	52	2%	0.042	0.048	–	NA	NA	NA	NA
2-CHLOROPHENOL	1	41	2%	1.110	0.240	–	NA	NA	NA	NA
CHROMIUM	44	44	100%	61.350	90.300	10.730	–	5.0E-3	–	0.025
4,4'-DDD	4	52	8%	0.050	0.094	–	2.4E-1	–	0.003	–
4,4'-DDE	10	53	19%	0.058	0.103	–	3.4E-1	–	0.005	–
4,4'-DDT	15	52	29%	0.088	0.171	–	3.4E-1	5.0E-4	0.008	0.001
DI-N-BUTYLPHTHALATE	1	41	2%	1.097	0.057	–	NA	NA	NA	NA
DI-N-OCTYLPHTHALATE	10	41	24%	4.128	7.904	–	–	2.0E-2	–	0.001
DIBENZOFURAN	6	41	15%	1.072	0.200	–	–	1.0E-2	–	0.000
1,2-DICHLOROBENZENE	1	41	2%	1.098	0.180	–	NA	NA	NA	NA

Table 6-B-49
Ranking Process for Chemicals of Potential Concern Selection
Warwick Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]
1,4-DICHLOROBENZENE	2	41	5%	1.103	0.039	--	NA	NA	NA	NA
1,1-DICHLOROETHANE	1	52	2%	0.042	0.044	--	NA	NA	NA	NA
DIELDRIN	6	52	12%	0.063	0.114	--	1.6E+1	5.0E-5	0.266	0.003
3,3'-DIMETHYLBENZIDINE	1	41	2%	2.255	3.284	--	NA	NA	NA	NA
DINOSEB	1	41	2%	0.074	0.072	--	NA	NA	NA	NA
DIPHENYLAMINE	1	41	2%	1.108	0.140	--	NA	NA	NA	NA
DISULFOTON	3	40	8%	0.070	0.008	--	--	4.0E-5	--	0.000
ENDOSULFAN I	4	52	8%	0.039	0.018	--	--	6.0E-3	--	0.000
ENDOSULFAN II	1	52	2%	0.107	0.018	--	NA	NA	NA	NA
ENDOSULFAN SULFATE	4	52	8%	0.165	0.290	--	--	6.0E-3	--	0.000
ENDRIN	6	52	12%	0.056	0.102	--	--	3.0E-4	--	0.001
ENDRIN ALDEHYDE	6	52	12%	0.149	0.287	--	--	3.0E-4	--	0.001
ETHYL PARATHION	2	40	5%	0.034	0.006	--	NA	NA	NA	NA
ETHYLBENZENE	1	52	2%	0.041	0.027	--	NA	NA	NA	NA
HEPTACHLOR	1	52	2%	0.039	0.003	--	NA	NA	NA	NA
HEPTACHLOR EPOXIDE	8	52	15%	0.069	0.125	--	9.1E+0	1.3E-5	0.166	0.014
ISODRIN	2	52	4%	0.061	0.110	--	NA	NA	NA	NA
KEPONE	1	52	2%	0.119	0.205	--	NA	NA	NA	NA
METHOXYCHLOR	22	54	41%	105.859	199.371	--	--	5.0E-3	--	0.059
METHYLENE CHLORIDE	10	52	19%	0.180	0.264	--	7.5E-3	6.0E-2	0.000	0.000
3&4-METHYLPHENOL	2	15	13%	0.164	0.028	--	--	5.0E-2	--	0.000
N-NITROSO-DI-N-PROPYLAMINE	1	41	2%	1.073	0.170	--	NA	NA	NA	NA
2-NITROANILINE	3	41	7%	5.682	7.000	--	--	6.0E-5	--	0.173
NITROBENZENE	2	41	5%	1.172	1.678	--	NA	NA	NA	NA
OCDD	2	8	25%	0.001	0.002	--	1.6E+2	--	0.035	--
ORTHOPHOSPHATE	21	26	81%	4.415	6.650	--	--	--	--	--
PCB-1248	3	54	6%	4.466	9.655	--	--	3.0E-5	--	0.477
PCB-1254	17	52	33%	1.896	3.347	--	--	2.0E-5	--	0.248
PHENACETIN	1	41	2%	1.120	1.000	--	NA	NA	NA	NA
PHENOL	4	41	10%	1.067	1.533	--	--	6.0E-1	--	0.000

Table 6-B-49
Ranking Process for Chemicals of Potential Concern Selection
Warwick Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]
PHORATE	1	40	3%	0.035	0.010	--	NA	NA	NA	NA
PROPAZINE	1	26	4%	8.027	11.657	--	NA	NA	NA	NA
SAFROLE	4	41	10%	1.918	3.132	--	--	2.0E-2	--	0.000
SULFOTEPP	1	41	2%	0.022	0.004	--	NA	NA	NA	NA
2,4,5-T	1	38	3%	0.013	0.010	--	NA	NA	NA	NA
TETRACHLOROETHENE	14	52	27%	0.123	0.224	--	5.2E-2	1.0E-2	0.002	0.000
THIONAZIN	1	40	3%	0.179	0.006	--	NA	NA	NA	NA
TINUVIN 327	8	26	31%	6.199	9.017	--	--	2.5E-3	--	0.005
TOLUENE	23	52	44%	2.070	5.305	--	--	2.0E-1	--	0.000
2,4,5-TP (SILVEX)	4	38	11%	0.029	0.046	--	--	8.0E-3	--	0.000
TRICHLOROETHENE	2	52	4%	0.043	0.057	--	NA	NA	NA	NA
M&P-XYLENE	12	52	23%	0.244	0.599	--	--	2.0E+0	--	0.000
O-XYLENE	5	52	10%	0.160	0.377	--	--	2.0E+0	--	0.000
ZINC	41	44	93%	2000.61	3016.350	75.940	--	3.0E-1	--	0.014

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Cancer slope factor. "--" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information System (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[f]Chronic reference dose; "--" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

[g]Relative ranking score--carcinogenic.

[h]Relative ranking score--noncarcinogenic.

Table 6-B-50
Ranking Process for Chemicals of Potential Concern Selection Summary of Carcinogens
Warwick Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RRS-C[f]	Cumulative RRS-C
ALDRIN	3	53	6%	0.076	0.137	--	1.7E+1	0.339	0.339
BERYLLIUM	43	44	98%	0.680	0.850	0.600	4.3E+0	0.326	0.665
DIELDRIN	6	52	12%	0.063	0.114	--	1.6E+1	0.266	0.931
HEPTACHLOR EPOXIDE	8	52	15%	0.069	0.125	--	9.1E+0	0.166	1.098
ALPHA-BHC	7	53	13%	0.066	0.121	--	6.3E+0	0.111	1.209
BIS(2-ETHYLHEXYL)PHTHALATE	18	41	44%	11.885	21.446	--	1.4E-2	0.044	1.253
OCDD	2	8	25%	0.001	0.002	--	1.6E+2	0.035	1.287
GAMMA-CHLORDANE	13	52	25%	0.059	0.104	--	1.3E+0	0.020	1.307
4,4'-DDT	15	52	29%	0.088	0.171	--	3.4E-1	0.008	1.316
4,4'-DDE	10	53	19%	0.058	0.103	--	3.4E-1	0.005	1.321
4,4'-DDD	4	52	8%	0.050	0.094	--	2.4E-1	0.003	1.324
TETRACHLOROETHENE	14	52	27%	0.123	0.224	--	5.2E-2	0.002	1.326
METHYLENE CHLORIDE	10	52	19%	0.180	0.264	--	7.5E-3	0.000	1.326

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Cancer slope factor. "--" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[f]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-51
Ranking Process for Chemicals of Potential Concern Selection Summary of Noncarcinogens
Warwick Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	RfD[e] (mg/kg-day)	RRS-N[f]	Cumulative RRS-N
PCB-1248	3	54	6%	4.466	9.655	--	3.0E-5	0.477	0.477
PCB-1254	17	52	33%	1.896	3.347	--	2.0E-5	0.248	0.725
2-NITROANILINE	3	41	7%	5.682	7.000	--	6.0E-5	0.173	0.898
METHOXYCHLOR	22	54	41%	105.859	199.371	--	5.0E-3	0.059	0.957
CHROMIUM	44	44	100%	61.350	90.300	10.730	5.0E-3	0.025	0.982
HEPTACHLOR EPOXIDE	8	52	15%	0.069	0.125	--	1.3E-5	0.014	0.997
ANTIMONY	7	30	23%	1.840	4.190	0.000	4.0E-4	0.014	1.011
ZINC	41	44	93%	2000.61	3016.350	75.940	3.0E-1	0.014	1.025
ALDRIN	3	53	6%	0.076	0.137	--	3.0E-5	0.007	1.031
CADMIUM	17	38	45%	1.550	2.170	0.440	5.0E-4	0.006	1.037
TINUVIN 327	8	26	31%	6.199	9.017	--	2.5E-3	0.005	1.043
DIELDRIN	6	52	12%	0.063	0.114	--	5.0E-5	0.003	1.046
GAMMA-CHLORDANE	13	52	25%	0.059	0.104	--	6.0E-5	0.003	1.049
CHLOROBENZENE	12	52	23%	10.063	26.571	--	2.0E-2	0.002	1.051
ALPHA-CHLORDANE	6	52	12%	0.042	0.077	--	6.0E-5	0.002	1.053
BIS(2-ETHYLHEXYL)PHTHALATE	18	41	44%	11.885	21.446	--	2.0E-2	0.002	1.054
ENDRIN ALDEHYDE	6	52	12%	0.149	0.287	--	3.0E-4	0.001	1.056
4-CHLOROANILINE	13	41	32%	1.184	1.625	--	4.0E-3	0.001	1.056
DI-N-OCTYLPHTHALATE	10	41	24%	4.128	7.904	--	2.0E-2	0.001	1.057
4,4'-DDT	15	52	29%	0.088	0.171	--	5.0E-4	0.001	1.057
ENDRIN	6	52	12%	0.056	0.102	--	3.0E-4	0.001	1.058
DELTA-BHC	4	53	8%	0.043	0.084	--	3.0E-4	0.000	1.058
DISULFOTON	3	40	8%	0.070	0.008	--	4.0E-5	0.000	1.059
SAFROLE	4	41	10%	1.918	3.132	--	2.0E-2	0.000	1.059
ENDOSULFAN SULFATE	4	52	8%	0.165	0.290	--	6.0E-3	0.000	1.059
1,1-BIPHENYL	1	2	50%	1.038	1.900	--	5.0E-2	0.000	1.059
TOLUENE	23	52	44%	2.070	5.305	--	2.0E-1	0.000	1.059
TETRACHLOROETHENE	14	52	27%	0.123	0.224	--	1.0E-2	0.000	1.059
DIBENZOFURAN	6	41	15%	1.072	0.200	--	1.0E-2	0.000	1.059

Table 6-B-51
Ranking Process for Chemicals of Potential Concern Selection Summary of Noncarcinogens
Warwick Area
Combined Soil

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	RfD[e] (mg/kg-day)	RRS-N[f]	Cumulative RRS-N
2,4,5-TP (SILVEX)	4	38	11%	0.029	0.046	--	8.0E-3	0.000	1.059
METHYLENE CHLORIDE	10	52	19%	0.180	0.264	--	6.0E-2	0.000	1.059
BUTYLBENZYLPHTHALATE	7	41	17%	1.102	0.780	--	2.0E-1	0.000	1.059
ENDOSULFAN I	4	52	8%	0.039	0.018	--	6.0E-3	0.000	1.059
PHENOL	4	41	10%	1.067	1.533	--	6.0E-1	0.000	1.059
3&4-METHYLPHENOL	2	15	13%	0.164	0.028	--	5.0E-2	0.000	1.059
M&P-XYLENE	12	52	23%	0.244	0.599	--	2.0E+0	0.000	1.059
O-XYLENE	5	52	10%	0.160	0.377	--	2.0E+0	0.000	1.059
BERYLLIUM	43	44	98%	0.680	0.850	0.600	5.0E-3	0.000	1.059

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Chronic reference dose; "--" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

[f]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-52
Ranking Process for Chemicals of Potential Concern Selection
Warwick Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]
ACETOPHENONE	1	27	4%	4.822	1.400	--	NA	NA	NA	NA
ALDRIN	3	27	11%	0.024	0.029	--	1.7E+1	3.0E-5	0.127	0.031
ANILINE	2	27	7%	17.519	7.000	--	5.7E-3	--	0.010	--
BENZENE	2	27	7%	4.833	7.082	--	2.9E-2	--	0.052	--
BERYLLIUM	1	27	4%	2.040	2.290	0.00000	NA	NA	NA	NA
ALPHA-BHC	2	27	7%	0.021	0.024	--	6.3E+0	NA	0.039	--
DELTA-BHC	1	27	4%	0.020	0.017	--	NA	NA	NA	NA
GAMMA-BHC	2	27	7%	0.025	0.033	--	1.3E+0	3.0E-4	0.011	0.003
BIS(2-CHLOROETHYL)ETHER	4	27	15%	4.433	2.000	--	1.1E+0	--	0.562	--
CADMIUM	2	27	7%	3.370	4.440	0.00000	--	5.0E-4	--	0.207
GAMMA-CHLORDANE	2	27	7%	0.025	0.031	--	1.3E+0	6.0E-5	0.010	0.016
4-CHLOROANILINE	4	27	15%	7.322	8.630	--	--	4.0E-3	--	0.068
CHLOROBENZENE	7	27	26%	221.59	449.07	--	--	2.0E-2	--	0.709
2-CHLOROPHENOL	5	27	19%	5.022	5.479	--	--	5.0E-3	--	0.035
CHROMIUM	1	27	4%	5.270	5.730	8.01000	--	5.0E-4	--	0.207
4,4'-DDT	1	27	4%	0.039	0.023	--	NA	NA	NA	NA
DI-N-BUTYLPHTHALATE	1	27	4%	4.796	0.700	--	NA	NA	NA	NA
DI-N-OCTYLPHTHALATE	2	27	7%	4.659	1.000	--	--	2.0E-2	--	0.002
1,1-DICHLOROETHANE	2	27	7%	3.285	4.100	--	--	1.0E-1	--	0.001
DIETHYLPHTHALATE	2	27	7%	4.696	2.300	--	--	8.0E-1	--	0.000
DIMETHOATE	1	27	4%	2.769	0.440	--	NA	NA	NA	NA
DINOSEB	1	26	4%	7.139	0.040	--	NA	NA	NA	NA
ETHYLBENZENE	1	27	4%	3.248	2.700	--	NA	NA	NA	NA
HYDROCARBONS	6	14	43%	0.475	0.708	--	--	--	--	--
OCDD	2	16	13%	0.0008	0.0010	--	1.6E+2	--	0.040	--
ORTHOPHOSPHATE	3	9	33%	0.050	0.103	--	--	--	--	--
2,2'-OXYBIS(1-CHLOROPROPANE)	3	27	11%	5.967	7.426	--	--	--	--	--
PECDD	1	16	6%	0.0003	0.0004	--	7.8E+2	--	0.082	--
PHENOL	2	27	7%	4.759	3.000	--	--	6.0E-1	--	0.000

Table 6-B-52
Ranking Process for Chemicals of Potential Concern Selection
Warwick Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] $(mg/kg\text{-}day)^{-1}$	RfD[f] (mg/kg-day)	RRS-C[g]	RRS-N[h]
PROPAZINE	6	27	22%	29.019	35.000	--	--	2.0E-2	--	0.055
2,4,5-T	1	27	4%	0.175	0.110	--	NA	NA	NA	NA
TETRACHLOROETHENE	2	27	7%	3.515	4.446	--	5.2E-2	1.0E-2	0.059	0.014
TINUVIN 327	4	15	27%	25.220	4.000	--	--	2.5E-3	--	0.051
TKN	5	10	50%	1.150	1.940	--	--	--	--	--
TOLUENE	2	27	7%	5.111	7.463	--	--	2.0E-1	--	0.001
O-TOLUIDINE	1	27	4%	4.993	5.066	--	NA	NA	NA	NA
1,1,1-TRICHLOROETHANE	5	27	19%	5.604	8.041	--	--	9.0E-2	--	0.003
TRICHLOROETHENE	3	27	11%	3.163	2.100	--	1.1E-2	6.0E-3	0.006	0.011
M&P-XYLENE	1	27	4%	465.715	1.800	--	NA	NA	NA	NA
O-XYLENE	1	27	4%	3.178	0.800	--	NA	NA	NA	NA
ZINC	12	27	44%	221.700	365.630	40.58000	--	3.0E-1	--	0.028

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Cancer slope factor. "--" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information System (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[f]Chronic reference dose; "--" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitute RfDs.

[g]Relative ranking score--carcinogenic.

[h]Relative ranking score--noncarcinogenic.

Table 6-B-53
Ranking Process for Chemicals of Potential Concern Selection Summary of Carcinogens
Warwick Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	CSF[e] (mg/kg-day)$^{-1}$	RRS-C[f]	Cumulative RRS-C
BIS(2-CHLOROETHYL)ETHER	4	27	15%	4.433	2.000	--	1.1E+0	0.562	0.562
ALDRIN	3	27	11%	0.024	0.029	--	1.7E+1	0.127	0.689
PECDD	1	16	6%	0.0003	0.0004	--	7.8E+2	0.082	0.772
TETRACHLOROETHENE	2	27	7%	3.515	4.446	--	5.2E-2	0.059	0.831
BENZENE	2	27	7%	4.833	7.082	--	2.9E-2	0.052	0.883
OCDD	2	16	13%	0.0008	0.0010	--	1.6E+2	0.040	0.924
ALPHA-BHC	2	27	7%	0.021	0.024	--	6.3E+0	0.039	0.963
GAMMA-BHC	2	27	7%	0.025	0.033	--	1.3E+0	0.011	0.974
GAMMA-CHLORDANE	2	27	7%	0.025	0.031	--	1.3E+0	0.010	0.984
ANILINE	2	27	7%	17.519	7.000	--	5.7E-3	0.010	0.994
TRICHLOROETHENE	3	27	11%	3.163	2.100	--	1.1E-2	0.006	1.000

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Cancer slope factor. "--" means "no value available" or for noncarcinogens "not applicable". Source: Integrated Risk Information system (IRIS) and USEPA's Health Effects Assessment Summary Tables (HEAST).

[f]Relative ranking score--carcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Table 6-B-54
Ranking Process for Chemicals of Potential Concern Selection Summary of Noncarcinogens
Warwick Area
Groundwater

Detected Compound[a]	No. of Detects	No. of Samples	Freq. of Detection (%)	Mean Conc.[b] (mg/kg)	RME Conc.[c] (mg/kg)	Bkg Conc.[d] (mg/kg)	RfD[e] (mg/kg-day)	RRS-N[f]	Cumulative RRS-N
CHLOROBENZENE	7	27	26%	221.59	449.07	--	2.0E-2	0.709	0.709
CADMIUM	2	27	7%	3.370	4.440	0.000	5.0E-4	0.207	0.916
CHROMIUM	1	27	4%	5.270	5.730	8.010	5.0E-4	0.207	1.123
4-CHLOROANILINE	4	27	15%	7.322	8.630	--	4.0E-3	0.068	1.191
PROPAZINE	6	27	22%	29.019	35.000	--	2.0E-2	0.055	1.246
TINUVIN 327	4	15	27%	25.220	4.000	--	2.5E-3	0.051	1.297
2-CHLOROPHENOL	5	27	19%	5.022	5.479	--	5.0E-3	0.035	1.331
ALDRIN	3	27	11%	0.024	0.029	--	3.0E-5	0.031	1.362
ZINC	12	27	44%	221.700	365.630	40.580	3.0E-1	0.028	1.390
GAMMA-CHLORDANE	2	27	7%	0.025	0.031	--	6.0E-5	0.016	1.406
TETRACHLOROETHENE	2	27	7%	3.515	4.446	--	1.0E-2	0.014	1.420
TRICHLOROETHENE	3	27	11%	3.163	2.100	--	6.0E-3	0.011	1.431
GAMMA-BHC	2	27	7%	0.025	0.033	--	3.0E-4	0.003	1.435
1,1,1-TRICHLOROETHANE	5	27	19%	5.604	8.041	--	9.0E-2	0.003	1.438
DI-N-OCTYLPHTHALATE	2	27	7%	4.659	1.000	--	2.0E-2	0.002	1.439
1,1-DICHLOROETHANE	2	27	7%	3.285	4.100	--	1.0E-1	0.001	1.441
TOLUENE	2	27	7%	5.111	7.463	--	2.0E-1	0.001	1.442
PHENOL	2	27	7%	4.759	3.000	--	6.0E-1	0.000	1.442
DIETHYLPHTHALATE	2	27	7%	4.696	2.300	--	8.0E-1	0.000	1.442

[a]Shading indicates chemicals of potential concern (COPCs) for the Public Health Risk Assessment.

[b]Mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected).

[c]Reasonable Maximum Exposure (RME) concentration is the 95% upper confidence limit (UCL) of the mean of detected concentrations and sample detection/quantitation limits (one-half the sample detection/quantitation limit is assumed for samples in which the compound was not detected), unless the 95% UCL is greater than the maximum detected concentration. Then the maximum detected concentration is used as the RME concentration.

[d]95% UCL of the mean of detected concentrations and sample detection/quantitation limits in background surface soil samples of PAHs and inorganics. One-half the sample detection/quantitation limit is assumed for background samples in which the compound was not detected.

[e]Chronic reference dose; "--" means "no value available". Source: IRIS and HEAST. When not available on IRIS or HEAST, other sources were used to derive substitue RfDs.

[f]Relative ranking score--noncarcinogenic. "NA" indicates that the compound was detected in less than 5% of the samples and was not further assessed.

Appendix 6-C

Exposure Assessment Methodologies and Results

6-C-1 Introduction

Exposure to the Chemicals of Potential Concern (COPC) was characterized using USEPA's *Risk Assessment Guidance for Superfund Human Health Evaluation Manual* (HHEM) methodology (USEPA, 1989). Many other resources were used in the exposure assessment and are cited where appropriate in the text and tables of this appendix. Three hypothetical exposure scenarios were evaluated: an on-site resident at the Warwick and Waste Water Treatment Areas; and an on-site worker at the Production Area; and a canoeist scenario for each of the those areas.

Soil is the only medium of interest in the on-site worker and on-site resident scenarios. Exposure is considered via the ingestion, dermal absorption, and inhalation routes. Air concentrations were modeled from the soil concentrations and consider COPC associated with fugitive dust and gaseous emissions. The soil-to-air transport model is included as Appendix 6-D to the PHERE.

The effects of constituents leaching from surface and subsurface soils to groundwater, followed by discharge to the Pawtuxet River, are considered for the canoeist scenario. The potential concentrations of COPC in river water which might result from soils via leaching was modeled for each of the three site areas. The modeling to evaluate leaching from soil to river water was performed by Woodward-Clyde Consultants. The river water model assumptions and results are included as Appendix E to the PHERE.

6-C-2 Methodologies

According to HHEM methodology, exposure is estimated as a daily intake (IN) in milligrams of chemical per kilogram of body weight (mg/kg-day) of the exposed receptor. The IN may result from exposure via ingestion, dermal absorption, and/or inhalation. Because exposure for the resident scenario includes both childhood and adult exposures, a time-weighted approach is taken using separate exposure input values for childhood and adult stages of life. The equation used for exposure via ingestion of a chemical associated with contaminated soil for the on-site worker scenario is as follows:

$$IN_{Ing} = \frac{(CS \times IR_s \times CF \times FS \times EF \times ED)}{(BW \times AT)}$$

Where:

IN_{Ing}	=	Daily intake via ingestion
CS	=	Concentration of chemical in soil (the lesser of the maximum detected concentration or the 95th percent upper confidence limit of the mean)
IR_S	=	Soil ingestion rate
CF	=	Conversion factor
FS	=	Fraction originating from contaminated source
EF	=	Exposure frequency
ED	=	Exposure duration
BW	=	Body weight
AT	=	Averaging time for exposure

The exposure via soil ingestion pathway for the resident and general worker scenarios is nearly indentical to that used for the on-site worker:

$$IN_{Ing} = \frac{(CS \times IR_S \times CF \times FS \times EF \times ED \times TF)}{(BW \times AT)}$$

Where:

TF = Fraction of time spent in contaminated area (general worker--fraction of the workday spent outdoors; resident--fraction of the day spent at home)

(Other variables are as previously described.)

The exposure equation for the soil ingestion pathway for the resident scenario includes both childhood and adult exposures. Thus, a time-weighted approach is taken, using separate values for childhood and adult exposure.

In both the on-site worker and on-site resident scenarios, exposure via dermal absorption was calculated using the following equation:

$$IN_{Der} = \frac{(CS \times CF \times SA_s \times FS \times AF \times ABS \times EF \times ED)}{(BW \times AT)}$$

Where:

IN_{Der} = Daily intake--dermally absorbed dose
SA_s = Skin surface area available for contact with soils
AF = Soil-to-skin adherence factor
ABS = Soil absorption fraction
(Other variables are as previously described.)

The equation used to calculate exposure via dermal absorption for the general worker scenario is as follows:

$$IN_{Der} = \frac{(CS \times CF \times SA_S \times FS \times AF \times ABS \times EF \times ED \times TF)}{(BW \times AT)}$$

The IN_{Der} represents the dose absorbed by the body via the dermal pathway. As described in the risk characterization (see Appendix 6-G of the PHERE), reference doses (RfDs) and cancer slope factors (CSFs) are used to evaluate the risk associated with the calculated IN values. RfDs and CSFs are developed for the ingestion exposure route and are based on the ingested dose (IN_{Ing}). The gastrointestinally absorbed dose may be less than the IN_{Ing}. There are no RfD or CSF values based on the dermal absorption route. Instead, the oral RfD (RfD) or CSF (CSF_o) is used. Because the IN_{Der} is an absorbed dose, the RfD and CSF_o must be adjusted so that the IN_{Der} may be evaluated using the RfD or CSF_o. This is accomplished by dividing the RfD or CSF_o by the gastrointestinal absorption fraction (GAF). The resulting dermal RfD (RfD_d) or CSF (CSF_d) is used during the risk characterization (Appendix 6-G). This method of adjusting the RfD or CSF_o to the fraction that is gastrointestinally absorbed is consistent with the approach described in the HHEM.

Inhalation exposure for the on-site worker and on-site resident scenarios were calculated using the following equation:

$$IN_{Inh} = \frac{(CA \times FS \times EF \times ED \times IhR \times ET)}{(BW \times AT)}$$

Where:

CA = Modeled concentration of chemical in the air. Model is described in Appendix D of the PHRA. Modeled concentrations are based on the CS values described above.

IN_{Inh} = Daily intake via inhalation

ET = Exposure time

IhR = Inhalation rate

(Other variables are as previously described)

The canoeist scenario includes river water pathways for incidental ingestion and dermal absorption. The following equation was used to calculate exposure via incidental ingestion of river water:

$$IN_{Ing} = \frac{(CW \times IR_w \times EF \times ED)}{(BW \times AT)}$$

Where:

CW = Modeled chemical concentration in river water (Appendix E of the PHRA)

IR_w = Water ingestion rate

(Other variables are as previously described)

Exposure via dermal contact with river water was calculated as follows for the canoeing scenario:

$$IN_{Der.} = \frac{(DA_{ev} \times EF \times ED \times SA_w)}{(BW \times AT)}$$

Where:

DA_{ev} = Dermally absorbed dose per event (chemical- and scenario-specific)

SA_w = Body surface area in contact with river water

(Other variables are as previously described)

The DA_{ev} was calculated using the methodology and chemical-specific data found in USEPA's *Dermal Exposure Assessment: Principals and Applications* (DEAPA) (USEPA, 1992). As shown in Table 6-5 of the PHERE, an exposure time (ET) of two hours was used for the canoeist scenario. This ET value was used to calculate the DA_{ev} values. A steady-state absorption equation recommended in the DEAPA was used to calculate the DH_{ev} if the chemical-specific absorption was predicted to reach steady state, based on the "T" value in the DEAPA. If the chemical was not predicted to reach steady state within two hours, the nonsteady-state absorption equation from the DEAPA was used. The GAF value was used to estimate RfD_ds and CSF_ds in the dermal exposure pathway for river water, as described above for soils.

6-C-3 Exposure Parameters

The USEPA (HHEM) recommends that a combination of upper-bound and average values be used in the exposure calculations. The exposure point concentrations used are upper-bound estimates (upper 95th percentile confidence limit on the mean) as described in Section 6.4.2 of the PHERE. The other exposure parameters used to estimate chemical intakes are summarized in Table 6-4 of the PHRA text. These exposure parameters have been discussed previously with USEPA Region I and are described in the following paragraphs. Chemical-specific values, such as dermal soil absorption factors (ABSs), gastrointestinal absorption factors (GAFs), and values associated with dermal exposure to chemicals in water are presented in Tables 6-C-1 through 6-C-4 of this appendix .

6-C-3.1 Resident Scenario

Under the on-site resident scenario, exposure is assumed to occur over a 30-year period. It is assumed that 6 years are spent in early childhood (ages 1 through 6). The remaining 24 years of exposure are assumed to be spent as an adult. This 30-year exposure duration (ED) is a default value recommended by the USEPA (1991a) and represents an upper-bound estimate of the length of time residents stay in one area. The distinction is made between early childhood and adult because the level of exposure experienced relative to body weight by a very young child is generally considered to be substantially greater than is experienced by an adult, particularly with regard to the ingestion of soil; such a difference is not believed to exist between an older child and an adult. The averaging time (AT) for exposure to noncarcinogenic chemicals is 10,950 days

(8,760 days for adults and 2,190 days for children). This is equal to the ED. The AT for exposure to carcinogenic chemicals is 25,550 days. This is equal to the average lifetime of a receptor (70 years).

As recommended by the USEPA (1991a) it is assumed that a 70 kilogram (kg) adult ingests 100 milligrams (mg) of soil per day and a 15 kg child ingests 200 mg of soil per day. It is further assumed that 100% of this ingested soil comes from the contaminated area (FS=1.0) for the portion of time (17 hours) that an individual is assumed to spend at home each day (TF = 0.71). It is probable that a person consumes soil at a greater rate outside the home because 50% of the time that a person spends at home is while sleeping. Presumably, soil ingestion is minimal during sleep. Also, the FS value does not factor in soil associated with ingested food. For these reasons, an FS equal to 1.0 is conservative.

An adult inhalation rate (IhR) of 0.6 m^3 per hour (USEPA, 1991b) is assumed. This is the average IhR for men and women engaged in light activity. Light activity includes most domestic work, personal care, hobbies, and conducting minor indoor repairs and home improvements. This value represents an average value for the part of the day spent at home, of which about 50% is spent sleeping. Inhalation rates will vary with activity; less during periods of rest (watching television, reading, sleeping), more during periods of higher activity (heavy cleaning, climbing stairs, exercising). An inhalation rate of 0.3 m^3 per hour was assumed for children based on recommendations from the International Commission on Radiation Protection (1976).

An exposure time (ET) of 17 hours per day was assigned even though the USEPA (1990a) estimates that on average men and women spend 15.4 hours per day at home. It was assumed that young children will not be home alone, therefore, the ET for children is the same as that for adults.

Parameters specific to exposure via dermal absorption are the skin surface area available for contact (SA_s), soil-to-skin adherence factor (AF), and soil absorption fraction (ABS). The USEPA (1992) recommended average SA_s of 5% of the total body surface area was used for this assessment for the Rhode Island winter, 25% for summer, and 10% for spring and fall. A total body surface area of 20,000cm^2 for an adult and 8,000 cm^2 for a young child were used for the PHERE, as recommended by USEPA Region I.

For the soil-to-skin adherence factor (AF), all of the studies cited in the current USEPA dermal

exposure assessment guidance (USEPA, 1992), are based on adherence to hands. As the guidance states, because hands generally have much greater contact with soil than do other parts of the body, AF values based on adherence to hands may overestimate the average adherence of soil to the entire exposed skin area. This is particularly true under this scenario where during the spring and fall most of the selected SA_s would be covered by clothing. A conservative AF of 1.0 was selected as strongly advocated by Region I. An AF of 0.2 was recommended by Region I for a central tendency value.

The ABS for the dermal pathway is chemical specific. ABS values for the COPC are presented in Table 6-C-1. These values represent upper-bound estimates of potential dermal absorption. Chemical-specific GAF values are also shown in Table 6-C-1. As described in Section 6-C-2, the GAF is used to convert the RfD to an RfD_d. The GAFs presented in Table 6-C-1 are generally the only values available for this parameter.

An exposure frequency (EF) of 350 days per year was used for inhalation exposures. This EF assumes that a resident will spend 15 days away from home on vacations, holidays, and weekend trips. This value does not take into account the potential reduction in air emissions resulting from snow cover and frozen ground in the winter. An EF of 230 days per year was used for exposure to soil (soil ingestion, dermal contact). This value assumes 15 days are spend away from home each year and that residents are not exposed to soil during 120 winter days per year when cold weather will cause a reduction in outdoor activities, an increase in the amount of clothing worn (thus decreasing dermal contact and hand to mouth soil transfer), and reducing the availability of soil due to snow cover and frozen ground. It is noted that the USEPA dermal guidance suggests that a typical EF value for an adult who gardens one or two days per week during the warmer months is approximately 40 days/year (USEPA, 1992). If this is the case, then an EF of 230 days severely overestimates exposure.

6-C-3.2 Worker Scenarios

The City of Cranston plans for the Production Area to be used for vehicle parking, storage of snow removal equipment, and the storing and loading of road salt and sand. Two worker scenarios were developed for evaluation. The first (on-site worker) was specific to the parking and storage facility use in which it was assumed that a worker would be outside all day in salt and sand movement activities every work day during the 4 month Rhode Island winter. This is an overestimate of time outside and more than allows for other outside site activity during the

warmer months. The second scenario (general worker) is a more general occupational description with year around exposure. A reasonable maximum exposure for this open, paved area is a grounds keeper (although there are no grounds to deep) and maintenance worker performing periodic maintenance on the asphalt pavement. Exposure is assumed to occur over a 25-year period in both scenarios. This 25-year ED is a default value recommended by the USEPA (1991a) and represents an upper-bound estimate of the length of time workers remain at one job. A standard 8-hour workday was assumed for the on-site worker ET, and 1.5 hours/day for the general worker. The averaging time for exposure to noncarcinogenic chemicals is 9,125 days. This is equal to the exposure duration. The averaging time for exposure to carcinogenic chemicals is 25,550 days. This is equal to the assumed average lifetime of a receptor (70 years).

As recommended by the USEPA (1991a), it is assumed that for the on-site worker, a 70-kg adult ingests 50 mg of soil per workday. The 50 mg of soil per workday is based on commercial and industrial workers (USEPA, 1991). It is further assumed that 100% of this ingested soil comes from the contaminated area (FS=1.0). Together, these input values form the assumption that the on-site worker will spend his/her entire workday in the contaminated area. This is likely to be an overestimate, since a worker probably will, on average, spend less than the entire workday in this area. Although the PHERE addresses the site in its current state, the Production Area will be covered with asphalt for its proposed use by the City of Cranston. This will virtually eliminate direct contact with the soil. The same exposure assumptions are made for the general worker, except that an FT value of 0.19 was used to prorate exposure to the fraction of the workday spent outside (1.5 hours outside divided by an 8-hour workday).

For inhalation exposures, an adult worker inhalation rate (IhR) of 1.4 m^3 per hour is assumed (USEPA, 1990a). This is the average IhR for men and women engaged in moderate activity. Moderate activity includes such things as heavy cleaning and climbing stairs.

The source SA_s and AF values used for the resident scenario were used in the worker scenarios for estimating potential dermal and ingestion exposures. For the general worker, an FT value of 0.19 was used to prorate exposure to the fraction of the workday spent outside (1.5 hours outside divided by an 8-hour workday). The ABS and GAF are chemical-specific. ABS values for the COPCs and background chemicals are presented in Table 6-C-1. These values represent upper-bound estimates of potential dermal absorption. Chemical-specific GAF values are also shown in Table 6-C-1. As described in Section 6-C-2, the GAF was used to convert the RfD to an RfD_d. The GAFs presented in Table 6-C-1 are generally the only values available for this parameter.

An on-site worker EF of 80 days per year was used for all three routes of exposure. This represents five workdays per week for 17 winter weeks, minus five holidays, vacation days, and sick days during this period. These assumptions do not address the fact that exposure will be limited in the winter by snow cover, frozen ground, and heavy clothing. A general worker EF of 250 days per year was used for inhalation exposure. Ingestion and dermal exposure for the winter months (85 work days) was regarded as negligible due to snow cover, frozen ground, and heavy clothing.

6-C-3.3 Canoeist Scenario

Under the recreational canoeist scenario, exposure is assumed to occur over a 30-year period. This 30-year ED represents an upper-bound estimate of the length of time residents stay in one area (USEPA, 1991a). The AT for exposure to noncarcinogenic chemicals is 10,950 days. This is equal to the exposure duration. The averaging time for exposure to carcinogenic chemicals is 25,550 days. This is equal to the average lifetime of a receptor (70 years).

It is assumed that a 70 kg adult visits the river one day per month during the non-winter months of the year (eight events/year) and spends two hours canoeing in the vicinity of the Site each time. It is further assumed that the canoeist may ingest a small amount of water (5 ml/event) splashed into the canoe. The hands and/or feet of the canoeist may also get wet due to water splashed into the canoe. Therefore, it is assumed that the total surface of an adult's hands and feet (2,300 cm^2) is available for dermal exposure to chemicals during each two-hour canoe outing.

The DA_{ev} (Section 6-C-2) for chemicals absorbed from water is chemical specific. The chemical-specific data used in the exposure calculations are from the DEAPA. These chemical-specific values, modeled surface water concentrations (From Appendix 6-E of the PHERE), and the calculated DA_{ev} values are shown for the three Site areas in Tables 6-C-2 through 6-C-4.

6-C-4 Results

As discussed in Appendix B of the PHRA, COPCs were selected separately for each Site area. The level of exposure associated with each COPC, measured in mg/kg-day, was estimated

The level of exposure associated with each COPC, measured in mg/kg-day, was estimated under each exposure scenario. Because the exposure assumptions differ somewhat, exposure levels of noncancer and cancer effects were calculated separately. Tables 6-C-5 through 6-C-7 summarize the exposure results of the three Site areas.

Exposure results are combined with the appropriate criteria identified during the toxicity assessment (Section 6.4.3 of the PHERE) to quantitatively characterize risks. The values shown in Tables 6-C-5 through 6-C-7 are carried into the risk characterization, included as Appendix 6-G. The values shown for the inhalation pathway include the combined contributions associated with fugitive dust and volatilization from soil to air.

Table 6-C-1
Dermal Soil Absorption Factors (ABSs) and Gastrointestinal Absorption Factors (GAFs) for the Chemicals of Potential Concern (COPCs) and Background Chemicals

CHEMICAL	ABS[a]	GAF[b]
COPCs		
Aldrin	0.1	0.85[c]
Beryllium	0.001[d]	0.001
gamma-Chlordane	0.1	0.85[c]
Chlorobenzene	0.2	0.9[e]
bis(2-Chloroethyl)ether	0.1	0.85[c]
Dieldrin	0.1	0.85[c]
bis(2-Ethylhexyl)phthalate	0.1	0.85[c]
2-Nitroaniline	0.1	0.9[e]
PCB 1248	0.06[f]	1.0[f]
PCB 1254	0.06[f]	1.0[f]
PCB 1260	0.06[f]	1.0[f]
2,3,7,8-TCDF	0.03[g]	0.85[c]
Tinuvin 327	0.1	0.85[c]
Toluene	0.2	1.0
Vinyl chloride	0.2	0.9
Background		
Antimony	0.001	0.1[h]
Arsenic	0.001	0.98
Benzo(a)pyrene	0.05[i]	0.5
Beryllium	0.001	0.001
Chromium	0.001	0.05
Dibenz(a,h)anthracene	0.05[j]	0.5[j]
Manganese	0.001	0.1[h]
Vanadium	0.001	0.1[h]

a. Except where otherwise noted, soil absorption values were selected based on the recommendations of Ryan et al. (1987). Ryan et al. Recommend a range of 0.1 to 0.25 for volatile organics and 0.01 to 0.1 for semivolatiles. The PHRA uses a value of 0.2 for volatiles and 0.1 for semivolatiles, unless otherwise noted.

b. Source: Jones and Owen (1989), unless otherwise noted.

c. No chemical-specific value could be found. Value shown is the lowest listed in Jones and Owen (1989) for organic compounds other than polycyclic aromatic compounds.

Table 6-C-1
Dermal Soil Absorption Factors (ABSs) and Gastrointestinal Absorption Factors (GAFs) for the Chemicals of Potential Concern (COPCs) and Background Chemicals

d. No chemical-specific value could be found. Value shown is the average absorption of cadmium. the only inorganic for which a soil dermal absorption coefficient could be found (USEPA, 1992). If the relative dermal absorption efficiencies of cadmium and beryllium are similar to their relative oral absorption efficients (0.06 and 0.001, respectively - Jones and Owen, 1989), then the use of this dermal value for beryllium is an overestimate and adds conservativeness to the exposure estimation.

e. No chemical-specific value could be found. Value shown is the lowest listed in Jones and Owen for substituted benzene compounds.

f. This value was requested by USEPA Region I. Source: USEPA, 1995a.

g. No chemical-specific value could be found. Value shown is the upperbound of the current USEPA-recommended ABS value range for 2,3,7,8-TCDD of 0.001 to 0.03 (USEPA, 1992).

h. No value listed in Jones and Owen (1989). Assumed value is typical for inorganics listed by Jones and Owen including barium, lead, and mercury.

I. Assumed based on studies referenced in the USEPA dermal exposure guidance (USEPA, 1992).

j. Assumed to be the same as benzo(a)pyrene.

Table 6-C-2

Dermal Absorption From River Water Calculation of Dermally Absorbed Dose Per Event[a]

Canoeist Scenario

Production Area

Chemical	Kp[b] (cm/hr)	CW (ug/l)	CF (ug/l)/ (mg/cm3)	CWc (mg/cm3)	tau[b] (hr)	ET (hr)	T*[b] (hr)	B[b]	DAnss (mg/cm2-event)	DAss (mg/cm2-event)	DAev (mg/cm2-event)
Noncarcinogens											
PCB 1254[c]	7.1E-01	1.41E-03	1E-06	1.41E-09	1.4E+01	2.0E+00	6.6E+01	5.2E+02	1.5E-08	8.4E-08	1.46E-08
PCB 1248	7.1E-01	7.58E-03	1E-06	7.58E-09	1.4E+01	2.0E+00	6.6E+01	5.2E+02	7.9E-08	4.5E-07	7.87E-08
gamma-Chlordane	5.2E-02	1.32E-04	1E-06	1.32E-10	2.8E+01	2.0E+00	1.3E+02	3.5E+01	1.4E-10	1.1E-09	1.42E-10
2-Nitroaniline[d]	2.2E-03	4.83E+00	1E-06	4.83E-06	3.2E-01	2.0E+00	7.8E-01	7.9E-04	2.3E-08	2.8E-08	2.80E-08
Chlorobenzene	4.1E-02	1.16E+00	1E-06	1.16E-06	4.3E-01	2.0E+00	1.0E+00	6.9E-02	1.2E-07	1.4E-07	1.35E-07
Toluene	4.5E-02	9.90E+00	1E-06	9.90E-06	3.2E-01	2.0E+00	7.7E-01	5.4E-02	9.9E-07	1.2E-06	1.16E-06
Carcinogens											
PCB 1260[c]	7.1E-01	1.33E-05	1E-06	1.33E-11	1.4E+01	2.0E+00	6.6E+01	5.2E+02	1.4E-10	7.9E-10	1.38E-10
gamma-Chlordane	5.2E-02	1.32E-04	1E-06	1.32E-10	2.8E+01	2.0E+00	1.3E+02	3.5E+01	1.4E-10	1.1E-09	1.42E-10
Vinyl chloride	7.3E-03	2.63E-02	1E-06	2.63E-08	2.1E-01	2.0E+00	5.1E-01	2.3E-03	3.4E-10	4.6E-10	4.64E-10

a. Developed from: USEPA, 1991, "Dermal Exposure Assessment: Principles and Applications - Interim Report," Office of Health and Environmental Assessment, Washington, D.C., pp. 5-47 - 5-52.

b. From Table 5-8 of the document referenced in "a".

c. Not listed in Table 5-8 of the document listed in "a"; values for hexachlorobiphenyl were used.

d. Not listed in Table 5-8 of the document listed in "a"; values for aniline were used.

Table 6-C-3

Dermal Absorption From River Water Calculation of Dermally Absorbed Dose Per Event[a]

Canoeist Scenario

Waste Water Treatment Area

Chemical	Kp[b] (cm/hr)	CW (ug/l)	CF (ug/l)/ (mg/cm3)	CWc (mg/cm3)	tau[b] (hr)	ET (hr)	T*[b] (hr)	B[b]	DAnss (mg/cm2-event)	DAss (mg/cm2-event)	DAev (mg/cm2-event)
Noncarcinogens											
PCB 1254	7.1E-01	2.84E-06	1E-06	2.84E-12	1.4E+01	2.0E+00	6.6E+01	5.2E+02	2.9E-11	1.7E-10	2.9E-11
Dieldrin	1.6E-02	9.09E-05	1E-06	9.09E-11	1.8E+01	2.0E+00	9.4E+01	3.6E+00	2.4E-11	1.3E-10	2.4E-11
Tinuvin 327	1.4E+00	4.91E-04	1E-06	4.91E-10	8.1E+00	2.0E+00	3.8E+01	6.3E+02	7.6E-09	3.3E-08	7.6E-09
bis(2-Ethylhexyl)phthalate	4.8E-03	8.63E-03	1E-06	8.63E-09	2.0E+00	2.0E+00	4.7E+00	3.0E-02	2.3E-10	2.6E-10	2.3E-10
gamma-Chlordane	5.2E-02	8.63E-05	1E-06	8.63E-11	2.8E+01	2.0E+00	1.3E+02	3.5E+01	9.3E-11	7.4E-10	9.3E-11
Carcinogens											
2,3,7,8-TCDF[d]	1.4E+00	7.72E-09	1E-06	7.72E-15	8.1E+00	2.0E+00	3.8E+01	6.3E+02	1.2E-13	5.2E-13	1.2E-13
Dieldrin	1.6E-02	9.09E-05	1E-06	9.09E-11	1.8E+01	2.0E+00	9.4E+01	3.6E+00	2.4E-11	1.3E-10	2.4E-11
gamma-Chlordane	5.2E-02	8.63E-05	1E-06	8.63E-11	2.8E+01	2.0E+00	1.3E+02	3.5E+01	9.3E-11	7.4E-10	9.3E-11
bis(2-Ethylhexyl)phthalate	4.8E-03	8.63E-03	1E-06	8.63E-09	2.0E+00	2.0E+00	4.7E+00	3.0E-02	2.3E-10	2.6E-10	2.3E-10

a. Developed from: USEPA, 1991, "Dermal Exposure Assessment: Principles and Applications - Interim Report," Office of Health and Environmental Assessment, Washington, D.C., pp. 5-47 - 5-52.

b. From Table 5-8 of the document referenced in "a".

c. Not listed in Table 5-8; values for aniline were used.

d. Not listed in Table 5-8; values for tetrachlorodibenzodioxin were used.

Table 6-C-4

Dermal Absorption From River Water Calculation of Dermally Absorbed Dose Per Event[a]
Canoeist Scenario
Warwick Area

Chemical	Kp[b] (cm/hr)	CW (ug/l)	CF (ug/l)/ (mg/cm3)	CWc (mg/cm3)	tau[b] (hr)	ET (hr)	T*[b] (hr)	B[b]	DAnss (mg/cm2-event)	DAss (mg/cm2-event)	DAev (mg/cm2-event)
Noncarcinogens											
PCB 1254	7.1E-01	9.70E-05	1E-06	9.7E-11	1.4E+01	2.0E+00	6.6E+01	5.2E+02	1.0E-09	5.8E-09	1.0E-09
PCB 1248	7.1E-01	2.12E-04	1E-06	2.12E-10	1.4E+01	2.0E+00	6.6E+01	5.2E+02	2.2E-09	1.3E-08	2.2E-09
Aldrin	1.6E-03	3.88E-03	1E-06	3.88E-09	1.5E+01	2.0E+00	3.6E+01	1.0E-01	9.4E-11	2.3E-10	9.4E-11
Beryllium	1.0E-03	4.77E-03	1E-06	4.77E-09		2.0E+00			0.0E+00	9.5E-12	9.5E-12
Dieldrin	1.6E-02	4.61E-05	1E-06	4.61E-11	1.8E+01	2.0E+00	9.4E+01	3.6E+00	1.2E-11	6.8E-11	1.2E-11
Chlorobenzene	4.1E-02	6.11E-01	1E-06	6.11E-07	4.1E-02	2.0E+00	1.0E+00	6.9E-02	2.0E-08	4.9E-08	4.9E-08
2-Nitroaniline	2.2E-03	1.69E+01	1E-06	0.0000169	3.2E-01	2.0E+00	7.8E-01	7.9E-04	8.2E-08	9.8E-08	9.8E-08
Carcinogens											
Aldrin	1.6E-03	3.88E-03	1E-06	3.88E-09	1.5E+01	2.0E+00	3.6E+01	1.0E-01	9.4E-11	2.3E-10	9.4E-11
Beryllium	1.0E-03	4.77E-03	1E-06	4.77E-09		2.0E+00			0.0E+00	9.5E-12	9.5E-12
Dieldrin	1.6E-02	4.61E-05	1E-06	4.61E-11	1.8E+01	2.0E+00	9.4E+01	3.6E+00	1.2E-11	6.8E-11	1.2E-11
bis(2-Chlorethyl) ether	2.1E-03	2.72E-03	1E-06	2.72E-09	6.5E-01	2.0E+00	1.6E+00	1.9E-03	1.8E-11	1.9E-11	1.9E-11

a. Developed from: USEPA, 1991, "Dermal Exposure Assessment: Principles and Applications - Interim Report," Office of Health and Environmental Assessment, Washington, D.C., pp. 5-47 - 5-52.

b. From Table 5-8 of the document referenced in "a".

c. Not listed in Table 5-8 of the document listed in "a". The values for tetrachlorodibenzodioxin (TCDD) were substituted. This is a conservative assumption, as the values listed for TCDD result in a higher rate of absorption than all or nearly all other compounds listed in Table 5-8.

Table 6-C-5
Exposure Intake Summary
Production Area

Noncancer Effects

Chemical	On-Site Worker			General Worker			Canoeist	
	Ingestion (mg/kg-day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)	Ingestion (mg/kg-day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)	Ingestion (mg/kg-day)	Dermal (mg/kg-day)
PCB 1248	2.0×10^{-5}	2.4×10^{-5}	5.5×10^{-7}	8.3×10^{-6}	3.0×10^{-5}	3.3×10^{-7}	1.2×10^{-11}	5.7×10^{-8}
PCB 1254	3.1×10^{-6}	3.8×10^{-6}	2.4×10^{-8}	1.3×10^{-6}	4.7×10^{-6}	1.4×10^{-8}	2.2×10^{-12}	1.1×10^{-8}
2-Nitroaniline	1.4×10^{-7}	2.8×10^{-7}	8.5×10^{-8}	5.8×10^{-8}	3.5×10^{-7}	5.0×10^{-8}	7.6×10^{-9}	2.0×10^{-8}
Chlorobenzene	4.4×10^{-8}	1.8×10^{-7}	1.7×10^{-7}	1.8×10^{-8}	2.2×10^{-7}	10×10^{-8}	1.8×10^{-9}	1.0×10^{-7}
Toluene	1.1×10^{-7}	4.5×10^{-7}	5.7×10^{-5}	4.6×10^{-8}	5.5×10^{-7}	3.3×10^{-5}	1.6×10^{-8}	8.6×10^{-7}
gamma-Chlordane	2.0×10^{-8}	4.1×10^{-8}	1.8×10^{-10}	8.5×10^{-9}	5.1×10^{-8}	1.0×10^{-10}	2.1×10^{-13}	1.0×10^{-10}

Cancer Effects

Chemical	On-Site Worker			General Worker			Canoeist	
	Ingestion (mg/kg-day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)	Ingestion (mg/kg-day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)	Ingestion (mg/kg-day)	Dermal (mg/kg-day)
PCB 1260	3.4×10^{-7}	4.1×10^{-7}	1.8×10^{-9}	1.4×10^{-7}	5.1×10^{-7}	1.2×10^{-7}	8.9×10^{-15}	4.3×10^{-11}
gamma-Chlordane	7.3×10^{-9}	1.5×10^{-8}	6.2×10^{-11}	3.0×10^{-9}	1.8×10^{-8}	3.7×10^{-11}	8.9×10^{-14}	4.3×10^{-11}
Vinyl chloride	0	0	0	0	0	0	1.8×10^{-11}	1.4×10^{-10}
Total PCBs	8.1×10^{-7}	9.7×10^{-8}	2.1×10^{-7}	3.4×10^{-6}	1.2×10^{-5}	1.2×10^{-7}	6.0×10^{-12}	2.9×10^{-8}

Table 6-C-6
Exposure Intake Summary
Waste Water Treatment Area

Noncancer Effects

Chemical	On-Site Resident--Adult			On-Site Resident--Child			Canoeist	
	Ingestion (mg/kg- day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)	Ingestion (mg/kg-day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)	Ingestion (mg/kg-day)	Dermal (mg/kg-day)
gamma-Chlordane	2.0×10^{-6}	8.6×10^{-6}	2.2×10^{-8}	1.9×10^{-5}	1.3×10^{-5}	5.2×10^{-8}	1.3×10^{-13}	6.7×10^{-11}
PCB 1254	1.3×10^{-7}	3.4×10^{-7}	1.4×10^{-9}	1.3×10^{-6}	5.3×10^{-7}	3.2×10^{-9}	4.4×10^{-15}	2.1×10^{-11}
Tinuvin 327	7.0×10^{-6}	3.0×10^{-5}	1.2×10^{-8}	6.6×10^{-5}	4.6×10^{-5}	2.7×10^{-8}	7.7×10^{-13}	5.5×10^{-9}
Dieldrin	1.1×10^{-7}	4.6×10^{-7}	1.4×10^{-9}	1.0×10^{-6}	7.1×10^{-7}	3.2×10^{-9}	1.4×10^{-13}	1.7×10^{-11}
bis(2-Ethylhexyl)phthalate	1.3×10^{-6}	5.7×10^{-6}	3.0×10^{-9}	1.3×10^{-5}	8.8×10^{-6}	7.0×10^{-9}	1.4×10^{-11}	1.7×10^{-10}

Cancer Effects

Chemical	On-Site Resident--Adult			On-Site Resident--Child			Canoeist	
	Ingestion (mg/kg- day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)	Ingestion (mg/kg-day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)	Ingestion (mg/kg-day)	Dermal (mg/kg-day)
2,3,7,8-TCDF	1.4×10^{-10}	1.7×10^{-10}	3.7×10^{-12}	3.2×10^{-10}	6.7×10^{-11}	2.1×10^{-12}	5.1×10^{-18}	3.7×10^{-14}
Dieldrin	3.7×10^{-8}	1.6×10^{-7}	4.6×10^{-10}	8.7×10^{-8}	6.1×10^{-8}	2.8×10^{-10}	6.1×10^{-14}	7.4×10^{-12}
bis(2-Ethylhexyl)phthalate	4.6×10^{-7}	1.9×10^{-6}	1.0×10^{-9}	1.1×10^{-6}	7.6×10^{-7}	6.0×10^{-10}	5.8×10^{-12}	7.1×10^{-11}
gamma-Chlordane	7.0×10^{-7}	3.0×10^{-6}	7.6×10^{-9}	1.6×10^{-6}	1.2×10^{-6}	4.4×10^{-9}	5.8×10^{-14}	2.9×10^{-11}
Total PCBs	4.6×10^{-8}	1.2×10^{-7}	4.7×10^{-10}	1.1×10^{-7}	4.5×10^{-8}	2.8×10^{-10}	1.9×10^{-15}	9.0×10^{-12}

Table 6-C-7
Exposure Intake Summary
Warwick Area

Noncancer Effects

Chemical	On-Site Resident--Adult			On-Site Resident--Child			Canoeist	
	Ingestion (mg/kg- day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)	Ingestion (mg/kg-day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)	Ingestion (mg/kg-day)	Dermal (mg/kg-day)
PCB 1248	9.6×10^{-6}	2.4×10^{-5}	2.2×10^{-7}	8.9×10^{-5}	3.8×10^{-5}	5.2×10^{-7}	3.3×10^{-13}	1.6×10^{-9}
PCB 1254	3.3×10^{-6}	8.4×10^{-6}	2.0×10^{-8}	3.1×10^{-5}	1.3×10^{-5}	4.7×10^{-8}	1.5×10^{-13}	7.2×10^{-10}
Chlorobenzene	4.3×10^{-7}	3.6×10^{-6}	6.0×10^{-5}	4.0×10^{-6}	5.6×10^{-6}	1.4×10^{-4}	9.6×10^{-10}	3.5×10^{-8}
2-Nitroaniline	4.5×10^{-6}	1.9×10^{-5}	5.2×10^{-7}	4.2×10^{-5}	2.9×10^{-5}	1.2×10^{-6}	2.7×10^{-8}	7.1×10^{-8}
Aldrin	1.3×10^{-7}	5.7×10^{-7}	1.0×10^{-9}	1.3×10^{-6}	8.8×10^{-7}	2.4×10^{-9}	6.1×10^{-12}	6.8×10^{-11}
Beryllium	5.2×10^{-7}	2.2×10^{-8}	4.1×10^{-10}	5.6×10^{-6}	3.9×10^{-8}	1.1×10^{-9}	7.5×10^{-12}	6.8×10^{-12}
Dieldrin	1.0×10^{-7}	4.3×10^{-7}	1.0×10^{-9}	9.5×10^{-7}	6.7×10^{-7}	2.4×10^{-9}	7.2×10^{-14}	8.6×10^{-12}

Cancer Effects

Chemical	On-Site Resident--Adult			On-Site Resident--Child			Canoeist	
	Ingestion (mg/kg- day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)	Ingestion (mg/kg-day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)	Ingestion (mg/kg-day)	Dermal (mg/kg-day)
Aldrin	4.6×10^{-8}	1.9×10^{-7}	3.6×10^{-10}	1.1×10^{-7}	7.6×10^{-8}	2.1×10^{-10}	2.6×10^{-12}	2.9×10^{-11}
Beryllium	1.8×10^{-7}	7.6×10^{-9}	1.4×10^{-10}	4.8×10^{-7}	3.4×10^{-9}	9.3×10^{-11}	3.2×10^{-12}	2.9×10^{-12}
Dieldrin	3.5×10^{-8}	1.5×10^{-7}	3.5×10^{-10}	8.2×10^{-8}	5.8×10^{-8}	2.1×10^{-10}	3.1×10^{-14}	3.7×10^{-12}
bis(2-Chloroethyl)ether	9.4×10^{-8}	4.0×10^{-7}	7.3×10^{-11}	2.2×10^{-7}	1.6×10^{-7}	4.3×10^{-11}	1.8×10^{-12}	5.9×10^{-12}
Total PCBs	3.9×10^{-6}	1.0×10^{-5}	8.3×10^{-8}	9.2×10^{-6}	3.9×10^{-6}	4.8×10^{-8}	2.1×10^{-13}	9.9×10^{-10}

Table 6-C-8
Exposure Intake Summary
Production Area--Background Compounds

Noncancer Effects

Chemical	On-Site Worker			General Worker		
	Ingestion (mg/kg- day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)	Ingestion (mg/kg-day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)
Arsenic	2.4×10^{-6}	4.7×10^{-8}	2.1×10^{-9}	9.8×10^{-7}	5.9×10^{-8}	1.2×10^{-9}
Chromium	2.2×10^{-6}	4.4×10^{-8}	1.9×10^{-9}	9.1×10^{-7}	5.5×10^{-8}	1.1×10^{-9}
Vanadium	3.3×10^{-6}	6.6×10^{-8}	2.9×10^{-9}	1.4×10^{-6}	8.2×10^{-8}	1.7×10^{-9}

Cancer Effects

Chemical	On-Site Worker			General Worker		
	Ingestion (mg/kg- day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)	Ingestion (mg/kg-day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)
Arsenic	8.4×10^{-7}	1.7×10^{-8}	7.3×10^{-10}	3.5×10^{-7}	2.1×10^{-8}	4.3×10^{-10}
Benzo(a)pyrene	9.5×10^{-8}	9.5×10^{-8}	8.4×10^{-11}	4.0×10^{-8}	1.2×10^{-7}	4.9×10^{-11}
Chromium	--[a]	--[a]	6.8×10^{-10}	--[a]	--[a]	4.0×10^{-10}
Dibenz(a,h)anthracene	3.8×10^{-8}	3.8×10^{-8}	3.3×10^{-11}	1.6×10^{-8}	4.7×10^{-8}	1.9×10^{-11}

a. Chromium is regarded as carcinogenic only via inhalation.

Table 6-C-9
Exposure Intake Summary
Waste Water Treatment Area--Background Compounds

Noncancer Effects

Chemical	On-Site Resident--Adult			On-Site Resident--Child		
	Ingestion (mg/kg- day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)	Ingestion (mg/kg-day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)
Arsenic	4.9×10^{-6}	2.1×10^{-7}	8.1×10^{-9}	4.6×10^{-5}	3.2×10^{-7}	1.9×10^{-8}
Beryllium	3.3×10^{-7}	1.4×10^{-8}	5.4×10^{-10}	3.1×10^{-6}	2.2×10^{-8}	1.3×10^{-9}
Chromium	7.7×10^{-6}	3.2×10^{-7}	1.3×10^{-9}	7.1×10^{-5}	5.0×10^{-7}	2.9×10^{-8}
Manganese	1.7×10^{-4}	7.4×10^{-6}	2.9×10^{-7}	1.6×10^{-3}	1.2×10^{-5}	6.7×10^{-7}
Vanadium	7.7×10^{-6}	3.2×10^{-7}	1.3×10^{-8}	7.1×10^{-5}	5.0×10^{-7}	2.9×10^{-8}

Cancer Effects

Chemical	On-Site Resident--Adult			On-Site Resident--Child		
	Ingestion (mg/kg- day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)	Ingestion (mg/kg-day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)
Arsenic	1.7×10^{-6}	7.1×10^{-8}	2.8×10^{-9}	3.9×10^{-6}	2.8×10^{-8}	1.6×10^{-9}
Benzo(a)pyrene	3.3×10^{-7}	6.9×10^{-7}	5.4×10^{-10}	7.7×10^{-7}	2.7×10^{-7}	3.2×10^{-10}
Beryllium	1.1×10^{-7}	4.8×10^{-9}	1.9×10^{-9}	2.7×10^{-7}	1.9×10^{-9}	1.1×10^{-10}
Chromium	--[a]	--[a]	4.3×10^{-9}	--[a]	--[a]	2.5×10^{-9}
Dibenz(a,h)anthracene	6.6×10^{-7}	1.4×10^{-6}	1.1×10^{-9}	1.5×10^{-8}	5.4×10^{-7}	6.3×10^{-10}

a. Chromium is regarded as carcinogenic only vial inhalation.

Table 6-C-10
Exposure Intake Summary
Warwick Area--Background Compounds

Noncancer Effects

Chemical	On-Site Resident--Adult			On-Site Resident--Child		
	Ingestion (mg/kg- day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)	Ingestion (mg/kg-day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)
Antimony	3.4×10^{-6}	1.5×10^{-7}	2.7×10^{-9}	3.2×10^{-5}	2.3×10^{-7}	6.3×10^{-9}
Arsenic	6.4×10^{-6}	2.7×10^{-7}	5.0×10^{-9}	6.0×10^{-5}	4.2×10^{-7}	1.2×10^{-8}
Manganese	1.6×10^{-4}	6.6×10^{-6}	1.2×10^{-7}	1.5×10^{-3}	1.0×10^{-5}	2.9×10^{-7}

Cancer Effects

Chemical	On-Site Resident--Adult			On-Site Resident--Child		
	Ingestion (mg/kg- day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)	Ingestion (mg/kg-day)	Dermal (mg/kg-day)	Inhalation (mg/kg-day)
Arsenic	2.2×10^{-6}	9.3×10^{-8}	1.7×10^{-9}	5.1×10^{-6}	3.6×10^{-8}	1.0×10^{-9}
Benzo(a)pyrene	3.5×10^{-7}	7.4×10^{-7}	2.8×10^{-10}	8.2×10^{-7}	2.9×10^{-7}	1.6×10^{-10}

Appendix 6-D

Air Transport Analysis

6-D1.0 Introduction

An air transport analysis of contaminants of potential concern from the Ciba-Geigy Cranston, Rhode Island Site (the Site) was conducted to support the public health risk assessment (PHRA). This appendix presents a description of the methodology, data base, assumptions, and models used in the analysis.

The objective of the air transport analysis is to predict the maximum ground-level concentrations of the contaminants potentially released to the atmosphere from the Site. These predicted concentrations are representative of maximum long-term, on-site exposures associated with the potential land uses described in the risk assessment scenarios for hypothetical residents and hypothetical workers (See Section 6.4.2.2 of the PHRA).

The analysis was conducted following guidelines established by the U.S. Environmental Protection Agency (USEPA) (USEPA, 1986a; 1987a; 1992a; 1993). A regulatory guideline air dispersion model was used to predict the maximum ground-level concentrations (USEPA, 1992b). Regulatory guidelines were also employed to predict air emission rates of each contaminant of potential concern (COPC) (USEPA, 1992a).

The air dispersion model accounts for the dilution and dispersion of contaminants from an emission source to a receptor considering site meteorological conditions. Site-specific data were used in the predictions of air emission rates and air concentrations whenever available. When site-specific data were not available, conservative assumptions were made so that health risks associated with the air pathway would not be underestimated.

6-D2.0 Site Description

The Site has been separated into three distinct areas for the purpose of investigating the magnitude and extent of possible chemical contamination. These areas are designated as:

- the Production Area,
- the Waste Water Treatment Area, and
- the Warwick Area.

These areas are mostly covered by vegetation, concrete, and asphalt. A soil boring survey has been conducted for the areas and a number of different organic and inorganic chemical compounds have been detected in the soil samples. The aerial extent of assumed chemical contamination and the amount of soil cover (Houlday, 1994) for each designated area are provided in Table 6- D1.

6-D3.0 Air Emissions Sources and Chemicals of Potential Concern

The Production, Warwick, and Waste Water Treatment Areas are a potential sources of gaseous emissions due to the evaporation of volatile and semivolatile compounds from the subsurface soil. Each area is also a potential source of wind blown dust contaminated by volatile, semivolatile, and nonvolatile compounds, including inhalable particulate matter (particle diameters < 10 μm; referred to as PM-10). The definition of volatile, semivolatile, and nonvolatile compounds and examples of the types of contaminants in each category are listed in Table 6-D2 (USEPA, 1990).

Separate COPC were selected for each of the Site areas (refer to Section 6.4.1 of the PHRA). A list of COPC for each area along with their classification as volatile, semivolatile, or nonvolatile is provided in Table 6-D3. The relevant physical and chemical properties of the COPC are provided in Table 6-D4.

6-D4.0 Predicting Air Concentrations

A screening-type air dispersion modeling analysis was performed to predict long-term concentrations due to the area sources associated with the Site. The USEPA's Industrial Source Complex Short-Term (ISCST2) model (1992b) was used in the analysis. The ISCST2 model is a regulatory guideline air dispersion model and is designated as the preferred model for predicting concentrations from complicated sources such as area sources (USEPA, 1986a; 1987a; 1993). The model is based on the Gaussian plume equations to predict concentrations from continuous

sources. For this analysis, it was assumed that emissions of COPC do not undergo any chemical reactions in the atmosphere and that no removal processes, such as wet or dry deposition, act on the plume during its transport from the source.

The ISCST2 model can perform multiple source short-term concentration predictions on square area sources. Data required to run the model include source characteristics, meteorology, and receptor grid locations.

The ISCST2 model was run to predict concentrations representative of maximum long-term on-site exposures associated with the potential land uses described in the risk assessment scenarios for hypothetical residents and hypothetical workers. This was accomplished by assuming each area of the Site is configured as a square with a receptor located in the center of the square. The ISCST2 model is not capable of predicting concentrations within an area source. Therefore, each designated area of the Site was subdivided so that the receptor was located at the edge of four square emission sources as illustrated in Figure 6-D1 for the Production Area. Similar source configurations were used in the ISCST2 model to predict concentrations representative of receptors located in the center of the Warwick Area.

The characteristics of the area sources required as input to the ISCST2 model include emission rates, location coordinates, emission release height above ground, and the length of a side of a square area. Concentration predictions are directly proportional to the emission rate entered in the ISCST2 model. To simplify the air dispersion modeling analysis, a unit emission rate of 1 $\mu g/s\text{-}m^2$ was used in the ISCST2 model for each of the four sources representing the Production Area and the Warwick Area. By inputting a unit emission rate of 1 $\mu g/s\text{-}m^2$, the results obtained from the ISCST2 model are unit concentrations (i.e., $\mu g/m^3$ per $\mu g/s\text{-}m^2$).

Compound-specific concentrations can then be determined based on the unit concentrations times the compound-specific area source emission rates. The prediction of area source emission rates is described in the next section. A summary of the source characteristics used for modeling each designated area of the Site is presented in Table 6-D5.

Meteorology required as input to the ISCST2 model include wind speed, wind direction, and Pasquill atmospheric stability category. Ambient air temperature and mixing height values are also required, but these parameters have an insignificant effect on concentration predictions for the Cranston Site. Meteorology representative of annual average conditions were used in the air

dispersion modeling so that the concentration predictions are representative of annual averages. Annual meteorology is characterized by neutral (D) stability and a mean wind speed of 4.74 m/s (Bair, 1992). The mean annual wind speed is based on measurements made by the National Weather Service in Providence. A worst-case wind direction was determined for each source configuration by varying the wind direction in 10° increments and selecting the highest concentration prediction.

The receptor grid locations input to the ISCST2 model were at ground-level in the center of each designated area of the Cranston site.

The unit concentrations predicted by the ISCST2 model are presented in Table 6-D6.

6-D5.0 Predicting Air Emission Rates

6-D5.1 Introduction

The current methodologies recommended by the USEPA for predicting emissions to the atmosphere from a contaminated site are contained in *Guideline for Predictive Baseline Emissions Estimation Procedures for Superfund Sites*, (USEPA 1992a). This document contains procedures for estimating:

A) Gaseous emissions from subsurface soils;

B) Gaseous emissions from nonaerated surface impoundments and contaminants in solution pooled at soil surfaces;

C) Volatile nonmethane organic compound emissions from codisposal landfills (i.e., toxic wastes in combination with municipal or sanitary wastes);

D) Free-phase volatile contaminants directly exposed to the atmosphere; and

E) Solid and semivolatile compounds emitted as particulate matter.

Only items A and E are applicable to the Site. Gaseous emissions may be released due to the evaporation of volatile and semivolatile contaminants in the subsurface soil (Item A). In

addition, volatile, semivolatile, and nonvolatile contaminants may be released as constituents of particulate matter emissions due to wind erosion of exposed soil surfaces (Item E). The emission rate models recommended by the USEPA (1992a) predict air emission rates as a function of contaminant concentration and contaminant physical and chemical properties within the soil. The modeling methodology, data, and assumptions used to predict contaminant air emission rates are described in the following.

6-D5.2 Air Emissions from Subsurface Soils

Preferably, soil gas measurements are used to predict the air release potential of volatile and semivolatile contaminants from subsurface soils. In the absence of soil gas measurements, soil bulk concentrations can be used for predicting the air release potential of contaminants. For the Site, soil gas measurements have not been made, whereas soil bulk concentrations have been determined from an on-site soil boring survey.

The first step in determining air emission rates based on soil bulk concentrations (C_{soil}) is to determine if free-phase volatile and semivolatile contaminants exist in the soil vadose zone as a liquid-phase waste layer or discrete film. The vadose zone is that region above the water table or saturated zone of the subsurface soil. Free-phase contaminants in the vadose zone are indicated if C_{soil} is greater than the saturation concentration (C_{sat}). Under the alternative scenario, where C_{soil} is less than C_{sat}, all contaminants are assumed to be fully incorporated in the vadose zone soil matrix (i.e., in solution with the available soil moisture and adsorbed to the soil particles). It is further assumed for this scenario that no discrete waste layers or films were evident in the soil samples. An illustration of the two scenarios is given in Figure D2.

Separate procedures are required to calculate air emission rates for free-phase contaminants ($C_{soil} > C_{sat}$) and fully incorporated contaminants ($C_{soil} < C_{sat}$) in the soil vadose zone.

6-D5.2.1 Saturation Concentration Calculations

The USEPA (1992a) provides an equation for calculating C_{sat} as a function of the soil/water partition coefficient (K_d in l/kg or ml/g); the solubility of the contaminant in water (s in mg/l-water); and the soil moisture content (θ_m in l-water/kg-soil or ml-water/g-soil):

$$C_{sat} = (K_d \times s \times n_m) + (s \times \theta_m) \qquad \text{(6-D1)}$$

where n_m is the soil moisture content expressed as a weight fraction (kg-water/kg-soil). The parameter values used as input to Equation (6-D1) for the Site are presented in the following discussion.

Values of K_d were estimated based on the following equation (USEPA, 1992a):

$$K_d = K_{oc} \times f_{oc} \tag{6-D2}$$

were K_{oc} is the soil/water partition coefficient (l/kg or ml/g) and f_{oc} is the fraction of organic content in the soil (mg/mg). The default value of f_{oc} is 0.02 (USEPA, 1992a). Values of K_{oc} for each COPC are provided in Table D4.

As indicated in Table D4, K_{oc} values for 2-nitroaniline and aniline were not found in the literature. For these COPCs, K_{oc} values were calculated based on the octanol/water partition coefficient, K_{ow} (l/kg or ml/g), using the following equation recommended by the USEPA (1992a):

$$K_{oc} = 10^{0.544 \log K_{ow} + 1.377} \tag{6-D3}$$

This equation is based on a wide variety of contaminants, mostly pesticides. Table 6-D4 provides the log K_{ow} values used in the equation.

Also provided in Table 6-D4 is the solubility of each COPC in water. Site-specific data on the moisture content of the soils were not readily available. A typical value of 20% moisture content for loam (Wanielista, 1990) was used in the analysis. Equivalent values of n_m and θ_m are 0.2 kg/kg and 0.2 l/kg, respectively.

Table 6-D7 presents a summary of the worksheet for calculating C_{sat} values for each volatile and semivolatile COPC in each designated area. Also provided in the table are C_{soil} values obtained from the on-site soil boring survey. The table allows for the ready comparison of C_{soil} and C_{sat} values. For all volatile and semivolatile COPCs at the Site, the soil bulk concentrations are less

than the saturation concentrations. This indicates that the COPC are fully incorporated in the vadose zone soil matrix. The procedures required to calculate air emission rates for this scenario are described in the following.

6-D5.2.2 Air Emission Rate Calculations for $C_{soil} < C_{sat}$

The results of the soil boring survey indicate that the volatile and semivolatile COPC at the Site are fully incorporated in the vadose zone soil matrix. The USEPA (1992a) provides an equation for predicting air emission rates from contaminated subsurface soil when the measured soil bulk concentrations are less than the saturated concentration. The average air emission rate (E in g/s) of a component for a specific exposure time [t in second(s)] is a function of the exposed surface area (A in cm^2); the effective diffusivity of the component in air (D_e in cm^2/s); the soil porosity (ϵ); the soil/air partition coefficient (K_{as} in g/cm^3); and the soil bulk concentration (C_{soil}) of the component:

$$E = \frac{2AD_e \epsilon K_{as} C_{soil}}{\sqrt{\pi \alpha t}} \qquad \text{(6-D4)}$$

An estimate of the exposed surface areas of the Site is provided in Table 6-D1. The effective diffusivity of the component is calculated based on the component's diffusion coefficient in air (D in cm^2/s) and the soil porosity (USEPA, 1992a):

$$D_e = D\epsilon^{0.33}. \qquad \text{(6-D5)}$$

Diffusion coefficients in air for each COPC are provided in Table 6-D4. The air diffusion coefficients of *gamma*-chlordane, 2,3,7,8-TCDF, and 2,4-dichlorophenol were calculated using the USEPA's CHEM7 chemical compound property processor (1991). When the soil is wet more often than dry, it is appropriate to use the air-filled soil porosity (P_a) in Equations (6-D4) and (6-D5) to determine emission rates and effective diffusivities (USEPA, 1992a). The air-filled soil porosity is calculated by:

$$P_a = P_t - \theta_m \beta \qquad \text{(6-D6)}$$

where P_t is the total soil porosity (dimensionless), θ_m is the soil moisture content (ml/g), and β is the soil bulk density (g/cm^3). The total soil porosity for the Cranston site is 0.42 based on soil

boring measurements. A typical soil moisture content for loam is 0.2 ml/g (Wanielista, 1990). The default value for β is 1.5 g/cm^3 (USEPA, 1992a). Using these values in Equation (6-D6) yields an air-filled soil porosity of 0.12.

The soil/air partition coefficient of a component is calculated based on the component's soil/water partition coefficient [refer to Equation (6-D2)] and Henry's Law constant (H in atm-m^3/mole) (USEPA, 1992a):

$$K_{as} = \frac{H}{K_d} \times 41 \qquad \text{(6-D7)}$$

where 41 is a conversion factor to change H to dimensionless form. Values of K_d and H for each COPC are provided in Table 6-D4.

Soil bulk concentrations are provided in Table 6-D7. The parameter α is a function of the effective diffusivity, soil porosity, particle density (ρ in g/cm^3), and the soil/air partition coefficient (USEPA, 1992a):

$$\alpha = \frac{D_e \epsilon}{\epsilon + \rho(1-\epsilon)/K_{as}} \qquad \text{(6-D8)}$$

The default value for particle density is 2.65 g/cm^3 (USEPA, 1992a). Values of D_e and K_{as} were calculated using Equations (6-D5) and (6-D7), respectively. The air-filled soil porosity is 0.12.

Exposure time (t in Equation 6-D4) is assumed to be 30 years (USEPA, 1992a) which is equivalent to 9 x 10^8 seconds.

Table 6-D8 presents a summary of the worksheet for predicting air emission rates of volatile and semivolatile COPC from assumed contaminated subsurface soil where $C_{soil} < C_{sat}$. Air emission rates are provided in units of g/s and g/s-m^2. The latter emission rates are area source emission rates which are needed for input to the air dispersion model.

6-D5.3 Air Emissions from Wind Erosion of Exposed Soil Surfaces

Although the Site is substantially covered by vegetation, concrete, and asphalt, it does not

contain 100% unbroken soil cover. An estimate of the fraction of soil cover of th Site is given in Table 6-D1 (Woodward-Clyde Consultants, 1994). As the worst-case, it is assumed that the exposed soil surfaces do not contain any hardened crust. Therefore, there is a potential for wind erosion of exposed soil surfaces.

Currently there are two methodologies recommended by the USEPA (1992a) for predicting volatile, semivolatile, and nonvolatile contaminant emissions as constituents of particulate matter due to wind erosion of exposed soil surfaces: 1) the unlimited reservoir model, and 2) the limited reservoir model. The appropriate model is selected based on the threshold friction velocity (u_t). The threshold friction velocity is the minimum wind speed needed to suspend erodible soil particles. The lower the threshold friction velocity, the higher the potential for soil erosion by the wind. If the threshold friction velocity (corrected for nonerodible elements) is less than or equal to 0.75 cm/s, then the soil is classified as having unlimited erosion potential and the unlimited reservoir model should be used. If the threshold friction velocity (corrected for nonerodible elements) is greater than 0.75 cm/s, then the soil is classified as having limited erosion potential and the limited reservoir model should be used.

6-D5.3.1 Determining the Threshold Friction Velocity

The threshold friction velocity is determined from an empirical relationship of the mode of the surface soil aggregate size distribution. The aggregate size distribution mode is the particle size containing the highest percentage of material from a representative surface soil sample. Size distribution data of surface soil samples for the Site are available from the soil boring survey. The data are summarized as particle sizes (mm) for which 10%, 50%, 60%, and 90% of the soil sample is finer. These data are plotted in Figures 6-D3 and 6-D4 for the Production Area and the Warwick Area, respectively.

The data were analyzed to determine the mode of the distribution for size ranges recommend by the USEPA (1992a): > 4 mm; 2 to 4 mm; 1 to 2 mm; 0.5 to 1 mm; 0.25 to 0.5 mm; and < 0.25 mm. The data indicate that most of the surface soil samples are made up of particles with sizes less than 0.25 mm. The mode in the aggregate distribution lies between 0 and 0.25 mm. The aggregate size distribution mode is taken to be 0.125 mm.

The threshold friction velocity is determined from the empirical relationship with the aggregate size distribution mode as given in Figure 6-D5. The appropriate value of u_t is 27.5 cm/s for an

aggregate size distribution mode of 0.125 mm. A factor (C_f) is used to correct for the nonerodible elements (e.g., stones, clumps of grass, etc.) in the surface soil. Where site-specific data are not available to determine an appropriate value of C_f, a conservative default value of 1.5 is recommended by the USEPA (1992a). The corrected threshold friction velocity (u_t^*) is:

$$\begin{aligned} u_t^* &= u_t^f \times C_f \\ u_t^* &= 27.5 \text{ cm/s} \times 1.5 \\ u_t^* &= 41.25 \text{ cm/s} \end{aligned} \qquad \text{(6-D9)}$$

Since u_t^* is less than 75 cm/s, the unlimited reservoir model was selected to predict contaminant emission rates as constituents of particulate matter due to wind erosion of exposed soil surfaces.

6-D5.3.2 The Unlimited Reservoir Model

The annual average emission rate (E_{10} in g/s-m^2) for each contaminant emitted as inhalable particulate matter from wind erodible surface soil is predicted using the following equation (USEPA, 1992a):

$$E_{10} = 0.00001(1-V)\left(\frac{\overline{u}}{u_t}\right)^3 F(x)\ C_{surf}* \qquad \text{(6-D10)}$$

where V is the fraction of assumed contaminated surface with continuous vegetative cover; u is the mean annual wind speed at 10 m anemometer height (m/s); u_t is the equivalent threshold value of wind speed at 7 m anemometer height (m/s); F(x) is an empirical function of the unlimited reservoir model; and C_{surf} is the fractional percent by weight of the component from bulk samples of surface soil.

An estimate of the fraction of assumed contaminated surface with continuous soil cover for each of the designated areas of the Site is given in Table 6-D1. The mean annual wind speed at 10 m anemometer height is 4.74 m/s based on measurements made by the National Weather Service in Providence, Rhode Island (Bair, 1992). The equivalent threshold value of wind speed at 7 m anemometer height is calculated based on the following equation provided by the USEPA (1992a):

$$u_t = 18.1 \frac{u_t^*}{100}$$
$$u_t = 18.1 \frac{0.4125 m/s}{100}$$
$$u_t = 5.10 m/s$$

(6-D11)

The value of function F(x) is 1.65 based on the curve presented in Figure 6-D7 (USEPA, 1992a) where:

$$x = 0.886 \frac{u_t}{u}$$
$$x = 0.886 \frac{5.10 m/s}{4.74 m/s}$$
$$x = 0.953$$

(6-D12)

The concentrations of each volatile, semivolatile, and nonvolatile COPC in the surface soils for each designated area of the Site are presented in Table 6-D9. These values were obtained from the on-site soil boring survey. Table 6-D9 also presents a summary of the worksheet for predicting air emission rates of each COPC emitted as inhalable particulate matter from wind erodible surface soil.

6-D6.0 Uncertainties in the Air Transport Analysis

Atmospheric dispersion models are reasonably reliable in predicting the magnitude of the highest concentrations occurring at some time at some location within a given area of interest. The USEPA (1986a) reports errors in the highest predicted concentrations of 10 to 40 percent to be typical. To offset the inherent uncertainties in the air transport analysis, a number of conservative assumptions were made that led to overestimation of the maximum concentrations.

Annual meteorology was characterized as a single event of neutral atmospheric stability, a mean

wind speed of 4.74 m/s, and a constant worst-case wind direction for each designated area. These assumptions will tend to overestimate actual maximum long-term concentrations because they do not account for the highly variable meteorological conditions that will occur at the Site over a long period of time.

Several assumptions were made in predicting air emission rates that will tend to overestimate actual maximum long-term concentrations. First, the aeral extent of assumed chemical contamination was overstated. In addition, the concentrations of COPC were overstated by assuming the 95th percent upper confidence limit of the mean concentrations in site soils are distributed throughout the assumed contaminated area. It was also assumed that the exposed soil surfaces do not contain any hardened crust. This assumption tends to overestimate the amount of contaminants released from the site as particulate matter due to wind erosion of exposed soil surfaces.

6-D7.0 Results of the Air Transport Analysis

The results of the air transport analysis are summarized in Table 6-D10. This table provides the predicted ambient air concentrations of each COPC for each of the designated areas of the Site. The predicted ambient air concentrations are representative of the maximum long-term on-site exposures associated with the potential land uses described in the risk assessment scenarios for hypothetical residents and hypothetical workers.

The results presented in Table 6-D10 are based on the unit concentrations obtained from the USEPA's ISCST2 model (refer to Table 6-D6) multiplied by the appropriate area source emission rate (refer to Tables 6-D8 and 6-D9). Each designated area of the Site is a potential source of gaseous emissions due to the evaporation of volatile and semivolatile compounds from the subsurface soil and wind blown dust contaminated by volatile, semivolatile, and nonvolatile compounds. The ambient concentration consists of the contributions due to these two emission release mechanisms. Table 6-D10 summarizes the individual concentration components as well as the combined ambient concentrations.

6-D8.0 References

Bair, Frank E. (Editor), 1992, *Weather of U.S. Cities*, Fourth Edition, Gale Research, Inc., Detroit, Michigan.

Electronic Handbook Publishers, Inc., 1994, *Electronic Handbook of Risk Assessment Values*, Bellvue, Washington.

USEPA, 1986a, *Guideline on Air Quality Models*, Revised, EPA-450/2-78-027R, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina 27711.

USEPA, 1986b, *Superfund Public Health Evaluation Manual*, EPA-540/1-86-060, Office of Emergency Remedial Response, Washington, DC.

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USEPA, 1992b, *User's Guide for the Industrial Source Complex (ISC2) Dispersion Models; Volume I - User Instructions*, EPA-450/4-92-008a, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina 27711.

USEPA, 1993a, *Supplement B to the Guideline on Air Quality Models*, Revised, EPA-450/2-78-027R, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina 27711.

USEPA, 1993b, *Air/Superfund National Technical Guidance Study Series - Estimation of Air Impacts for Thermal Desorption Units Used at Superfund Sites*, EPA-451/R-93-005, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina.

USEPA, 1994, *Risk Reduction Engineering Laboratory (RREL) Treatability Database*, contained on the Alternative Treatment Technology Information Center (ATTIC) Bulletin Board System.

Wanielista, Martin, 1990, *Hydrology and Water Quality Control*, John Wiley & Sons, Inc., New York, New York.

Woodward-Clyde Consultants, 1994, personal communication from Mark Houlday (WCC) to Tom Marshall (PTRL), based on on-site observations by WWC personnel.

TABLE 6-D-1

Areas of Potential Contamination.

Area Designation	Area		Fraction of Soil Cover[a]
	(ft^2)	(m^2)	
Production	140,000	13,000	0.90
Warwick	35,000	3,250	0.90
Waste Water Treatment	75,000	6,970	0.80

[a](Woodward-Clyde Consultants, 1994).

TABLE 6-D-2

Volatile, Semi-Volatile, and Non-Volatile Compounds.

Volatile Compounds (> 1 mmHg vapor pressure at 25 °C)

- All monochlorinated solvents. Also trichloroethylene, trichloroethane, tetrachloroethane.
- Most simple aromatic solvents: e.g., benzene, xylene, toluene, and ethylbenzene.
- Most alkanes up to decane (C_{10})
- Inorganic gases: e.g., hydrogen sulfide, chlorine, and sulfur dioxide.

Semi-Volatile Compounds (10^{-7} to 1 mmHg vapor pressure at 25 °C)

- Most polychlorinated biphenyls, dichlorobenzenes, aniline, nitroaniline, and phthalates.
- Most pesticides: e.g., dieldrin, toxaphene, and parathion.
- Most complex alkanes: dodecane and octadecane.
- Most polynuclear aromatics: e.g., napthalene, phenanthrene, and benz(a)anthrecene.
- Mercury.

Non-Volatile Compounds or Particulate Matter (<10^{-7} mmHg vapor pressure at 25 °C)

- Larger polynuclear aromatics: e.g., chrysene.
- Metals: e.g., lead and chromium.
- Other inorganic compounds: e.g., asbestos, arsenic, and cyanides.

TABLE 6-D-3

List of Chemicals of Potential Concern (COPC)

Area	Chemical	CAS Number	Vapor Pressure (mmHg)	Classification
Production	PCB 1260	11096-82-5	1.10E-05	Semi-Volatile
	gamma-Chlordane	57-74-9	1.00E-05	Semi-Volatile
	Vinyl Chloride	75-01-4	2.66E+03	Volatile
	PCB 1254	11097-69-1	4.30E-05	Semi-Volatile
	PCB 1248	12672-29-6	1.80E-04	Semi-Volatile
	2-Nitroaniline	88-74-4	3.00E-03	Semi-Volatile
	Chlorobenzene	108-90-7	1.18E+01	Volatile
	Toluene	108-88-3	3.00E+01	Volatile
	Arsenic	7440-38-2	0.00E+00	Non-Volatile
	Benzo(a)pyrene	50-32-8	5.68E-04	Semi-Volatile
	Dibenz(a,h)anthracene	53-70-3	5.20E-11	Non-Volatile
	Vanadium	7440-62-2	0.00E+00	Non-Volatile
	Chromium	7440-47-3	0.00E+00	Non-Volatile
Warwick	Aldrin	309-00-2	6.00E-06	Semi-Volatile
	Beryllium	7440-41-7	0.00E+00	Non-Volatile
	Dieldrin	60-57-1	1.78E-07	Semi-Volatile
	bis(2-Chloroethyl)ether	111-44-4	7.10E-01	Semi-Volatile
	PCB 1248	12672-29-6	1.80E-04	Semi-Volatile
	PCB 1254	11097-69-1	4.30E-05	Semi-Volatile
	2-Nitroaniline	88-74-4	3.00E-03	Semi-Volatile
	Chlorobenzene	108-90-7	1.18E+01	Volatile
	Arsenic	7440-38-2	0.00E+00	Non-Volatile
	Benzo(a)pyrene	50-32-8	5.68E-04	Semi-Volatile
	Antimony	7440-36-0	0.00E+00	Non-Volatile
	Manganese	7439-96-5	0.00E+00	Non-Volatile
Waste Water Treatment	2,3,7,8-TCDF*	1746-01-6	1.70E-06	Semi-Volatile
	gamma-Chlordane	57-74-9	1.00E-05	Semi-Volatile
	Dieldrin	60-57-1	1.78E-07	Semi-Volatile
	bis(2-Ethylhexyl)phthalate	117-81-7	2.53E-03	Semi-Volatile
	PCB 1254	11097-69-1	4.30E-05	Semi-Volatile
	Tinuvin 327	not avail.	8.00E-09	Non-Volatile
	Arsenic	7440-38-2	0.00E+00	Non-Volatile
	Benzo(a)pyrene	50-32-8	5.68E-04	Semi-Volatile
	Dibenz(a,h)anthracene	53-70-3	5.20E-11	Non-Volatile
	Beryllium	7440-41-7	0.00E+00	Non-Volatile
	Chromium	7440-47-3	0.00E+00	Non-Volatile
	Manganese	7439-96-5	0.00E+00	Non-Volatile
	Vanadium	7440-62-2	0.00E+00	Non-Volatile

*Values for 2,3,7,8-TCDD (Dioxin).

TABLE 6-D-4

Chemical Properties

Chemical		CAS Number	MW Molecular Weight (g/g-mole)	p Vapor Pressure (mmHg)	D Diffusion Coefficient in Air (cm^2/s)		H Henry's Law Constant ($atm\text{-}m^3/mole$)	s Solubility in Water (mg/l-water)		K_{oc} Organic Carbon Partition Coeff. (ml/g)	
PCB 1260	(1)	11096-82-5	372.00	1.10E-05	0.04909		2.50E-04	1.44E-02		6700000	
gamma-Chlordane	(2)	57-74-9	410.00	1.00E-05	0.04500	(1)	3.67E-05	9.30E-01		140000	(4)
Vinyl Chloride	(2)	75-01-4	62.50	2.66E+03	0.09000		8.60E-02	1.10E+00		57	(4)
PCB 1254	(1)	11091-69-1	328.00	4.30E-05	0.05251		2.00E-04	4.10E-02		2140000	
PCB 1248	(1)	12672-29-6	288.00	1.80E-04	0.05498		4.40E-04	2.00E-01		277000	
2-Nitroaniline	(3)	88-74-4	138.14	3.00E-03	0.07300		5.00E-07	1.47E+03	(1)	38	(1)
Chlorobenzene	(2)	108-90-7	112.56	1.18E+01	0.07300		3.93E-03	4.88E+02		330	(4)
Toluene	(2)	108-88-3	92.14	3.00E+01	0.08700		6.68E-03	5.15E+02		300	(4)
Arsenic	(4)	7440-38-2	75.00	0.00E+00	0.00000		0.00E+00	0.00E+00		0	
Benzo(a)pyrene	(1)	50-32-8	252.32	5.68E-04	0.04300		1.38E-09	3.00E-03		5500000	(4)
Dibenz(a,h)anthracene	(1)	53-70-3	278.36	5.20E-11	0.04200		3.81E-08	1.50E-03		3300000	(4)
Vanadium	(4)	7440-62-2	51.00	0.00E+00	0.00000		0.00E+00	0.00E+00		0	
Chromium	(4)	7440-47-3	52.00	0.00E+00	0.00000		0.00E+00	0.00E+00		0	
Aldrine	(4)	309-00-2	365.00	6.00E-06	0.04744	(1)	1.60E-05	1.80E-01		96000	
Beryllium	(4)	7440-41-7	9.00	0.00E+00	0.00000		0.00E+00	0.00E+00		0	
Dieldrin	(4)	60-57-1	381.00	1.78E-07	0.04875	(1)	4.58E-07	1.95E-01		1700	
bis(2-Chloroethyl)ether	(4)	111-44-4	143.00	7.10E-01	0.07212	(1)	1.31E-05	1.02E+04		13.9	
2,3,7,8-TCDF	(4),(5)	1746-01-6	322.00	1.70E-06	0.05303	(1)	3.60E-03	2.00E-04		3300000	
bis(2-Ethylhexyl)Phthalate	(2)	117-81-7	390.56	2.53E-03	0.03542	(1)	1.38E-07	3.50E-01		87400	(1)
Tinuvin 327	(6)	not avail.	357.90	8.00E-09	not avail.		not avail.	1.00E-04		43800	
Antimony	(4)	7440-36-0	122.00	0.00E+00	0.00000		0.00E+00	0.00E+00		0	
Manganese	(4)	7439-96-5	55.00	0.00E+00	0.00000		0.00E+00	0.00E+00		0	

References:

(1) Electronic Handbook Publishers, Inc., 1994, "Electronic Handbook of Risk Assessment Values", Bellevue, Washington.

(2) U.S. EPA, 1993b, "Air/Superfund National Technical Guidance Study Series - Estimation of Air Impacts for Thermal Desorption Units Used at Superfund Sites", EPA-451/R-93-005, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina.

(3) U.S. EPA, 1987b, "Hazardous Waste Treatment, Storage, and Disposal Facilities (TSDF) - Air Emission Models", EPA-450/3-97-026, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina.

(4) U.S. EPA, 1986b, "Superfund Public Health Evaluation Manual (SPHEM)", EPA-540/1-86-060, Office of Emergency Remedial Response, Washington, DC.

(5) Values for 2,3,7,8-TCDD (Dioxin).

(6) Ciba-Geigy.

(7) Calculated value based on the equation $K_{oc} = 10^{0.544 \log K_{ow} + 1.37}$ (U.S. EPA, 1992a) where $\log K_{ow} = 6.00$ (Ciba-Geigy).

TABLE 6-D-5

Source Characteristics Used in the ISCST2 Model

Designated Area (Source No.)	Emission Rate ($\mu g/s\text{-}m^2$)	Location Coordinates		Emission Release Height Above Ground (m)	Length of a Side of a Square Area (m)
		X (m)	Y (m)		
Production Area					
Source No. 1	1.00	-56.9	0.0	0.0	56.9
Source No. 2	1.00	0.0	0.0	0.0	56.9
Source No. 3	1.00	0.0	-56.9	0.0	56.9
Source No. 4	1.00	-56.9	-56.9	0.0	56.9
Warwick Area					
Source No. 1	1.00	-28.5	0.0	0.0	28.5
Source No. 2	1.00	0.0	0.0	0.0	28.5
Source No. 3	1.00	0.0	-28.5	0.0	28.5
Source No. 4	1.00	-28.5	-28.5	0.0	28.5
Waste Water Treatment Area					
Source No. 1	1.00	-41.5	0.0	0.0	41.5
Source No. 2	1.00	0.0	0.0	0.0	41.5
Source No. 3	1.00	0.0	-41.5	0.0	41.5
Source No. 4	1.00	-41.5	-41.5	0.0	41.5

TABLE 6-D-6

SCST2 Model Results

Designated Area of the Cranston Site	Unit Concentrations ($\mu g/m^3$ per $\mu g/s\text{-}m^2$)
Production Area	2.94176
Warwick Area	2.68840
Waste Water Treatment Area	2.82327

TABLE 6-D-7
C_{sat} Calculation Worksheet for Volatile and Semi-Volatile Contaminants

Contaminant Production Area	CAS Number	K_d (ml/g)	K_{oc} (ml/g)	f_{oc} (mg/mg)	s (mg/l-water)	n_m (kg/kg)	q_m (l/kg)	C_{sat} (mg/kg)	C_{soil} (mg/kg)
PCB 1260	11096-82-5	134000	6700000	0.02	1.44E-02	0.2	0.2	386	4.8
gamma-Chlordane	57-74-9	2800	140000	0.02	9.30E-01	0.2	0.2	521	0.070
Vinyl Chloride	75-01-4	1.1	57	0.02	1.10E+00	0.2	0.2	0.47	ND
PCB 1254	11091-69-1	42800	2140000	0.02	4.10E-02	0.2	0.2	351	15
PCB 1248	12672-29-6	5540	277000	0.02	2.00E-01	0.2	0.2	222	88
2-Nitroaniline	88-74-4	0.76	38	0.02	1.47E+03	0.2	0.2	517	4.2
Chlorobenzene	108-90-7	6.6	330	0.02	4.88E+02	0.2	0.2	742	0.28
Toluene	108-88-3	6.0	300	0.02	5.15E+02	0.2	0.2	721	62
Arsenic	7440-38-2	compound is non-volatile, C_{sat} calculations are not applicable							12
Benzo(a)pyrene	50-32-8	110000	5500000	0.02	3.00E-03	0.2	0.2	66	1.1
Dibenz(a,h)anthracene	53-70-3	compound is non-volatile, C_{sat} calculations are not applicable							0.68
Vanadium	7440-62-2	compound is non-volatile, C_{sat} calculations are not applicable							16
Chromium	7440-47-3	compound is non-volatile, C_{sat} calculations are not applicable							12
Warwick Area									
Aldrin	309-00-2	1920	96000	0.02	1.80E-01	0.2	0.2	69	0.14
Beryllium	7440-41-7	compound is non-volatile, C_{sat} calculations are not applicable							0.83
Dieldrin	60-57-1	34	1700	0.02	1.95E-01	0.2	0.2	1.4	0.11
bis(2-Chloroethyl)ether	111-44-4	0.28	14	0.02	1.02E+04	0.2	0.2	2607	ND
PCB 1248	12672-29-6	5540	277000	0.02	2.00E-01	0.2	0.2	222	9.7
PCB 1254	11091-69-1	42800	2140000	0.02	4.10E-02	0.2	0.2	351	3.3
2-Nitroaniline	88-74-4	0.76	38	0.02	1.47E+03	0.2	0.2	517	7.0
Chlorobenzene	108-90-7	6.6	330	0.02	4.91E+02	0.2	0.2	746	27
Arsenic	7440-38-2	compound is non-volatile, C_{sat} calculations are not applicable							8.9
Benzo(a)pyrene	50-32-8	110000	5500000	0.02	3.00E-03	0.2	0.2	66	1.3
Antimony	7440-36-0	compound is non-volatile, C_{sat} calculations are not applicable							4.2
Manganese	7439-96-5	compound is non-volatile, C_{sat} calculations are not applicable							254
Waste Water Treatment Area									
2,3,7,8-TCDF	1746-01-6	66000	3300000	0.02	2.00E-04	0.2	0.2	2.6	0.00053
gamma-Chlordane	57-74-9	2800	140000	0.02	9.30E-01	0.2	0.2	521	2.2
Dieldrin	60-57-1	34	1700	0.02	1.95E-01	0.2	0.2	1.4	0.13

Contaminant Production Area	CAS Number	K_d (ml/g)	K_{oc} (ml/g)	f_{oc} (mg/mg)	s (mg/l-water)	n_m (kg/kg)	q_m (l/kg)	C_{sat} (mg/kg)	C_{soil} (mg/kg)
bis(2-Ethylhexyl)phthalate	117-81-7	1748	87400	0.02	3.50E-01	0.2	0.2	122	1.4
PCB 1254	11091-69-1	42800	2140000	0.02	4.10E-02	0.2	0.2	351	0.21
Tinuvin 327	not available	compound is non-volatile, C_{sat} calculations are not applicable							8.2
Arsenic	7440-38-2	compound is non-volatile, C_{sat} calculations are not applicable							6.4
Benzo(a)pyrene	50-32-8	110000	5500000	0.02	3.00E-03	0.2	0.2	66	1.1
Dibenz(a,h)anthracene	53-70-3	compound is non-volatile, C_{sat} calculations are not applicable							1.9
Beryllium	7440-41-7	compound is non-volatile, C_{sat} calculations are not applicable							0.56
Chromium	7440-47-3	compound is non-volatile, C_{sat} calculations are not applicable							10
Manganese	7439-96-5	compound is non-volatile, C_{sat} calculations are not applicable							228
Vanadium	7440-62-2	compound is non-volatile, C_{sat} calculations are not applicable							10

TABLE 6-D-8

Air Emission Rate Calculation Worksheet of Volatile and Semi-Volatile COPCs from Assumed Contaminated Subsurface Soil ($C_{soil} < C_{sat}$)

Contaminant	CAS Number	C_{sat} (mg/kg)	C_{soil} (mg/kg)	A (cm^2)	D_a (cm^2/s)	D (cm^2/s)	e	K_{as} (g/cm^3)	H (atm-m^3/mole)	K_d (ml/g)	a	t (s)	E (g/s)	E (g/s-m^2)
Production Area														
PCB 1260	11096-82-5	386	4.8	1.30E+08	0.0244	0.0491	0.120	7.65E-08	2.50E-04	134000	9.60E-11	9.07E+08	5.34E-07	4.11E-11
gamma-Chlordane	57-74-9	521	0.070	1.30E+08	0.0224	0.0450	0.120	5.37E-07	3.67E-05	2800	6.18E-10	9.07E+08	1.98E-08	1.52E-12
Vinyl Chloride	75-01-4	0.47	ND	1.30E+08	0.0447	0.0900	0.120	3.09E+00	8.60E-02	1.1	6.14E-03	9.07E+08	0.00E+00	0.00E+00
PCB 1254	11091-69-1	351	15	1.30E+08	0.0261	0.0525	0.120	1.92E-07	2.00E-04	42800	2.57E-10	9.07E+08	2.73E-06	2.10E-10
PCB 1248	12672-29-6	222	88	1.30E+08	0.0273	0.0550	0.120	3.26E-06	4.40E-04	5540	4.58E-09	9.07E+08	6.76E-05	5.20E-09
2-Nitroaniline	88-74-4	517	4.2	1.30E+08	0.0363	0.0730	0.120	2.70E-05	5.00E-07	0.76	5.03E-08	9.07E+08	1.07E-05	8.23E-10
Chlorobenzene	108-90-7	742	0.28	1.30E+08	0.0363	0.0730	0.120	2.44E-02	3.93E-03	6.6	4.55E-05	9.07E+08	2.15E-05	1.65E-09
Toluene	108-88-3	721	62	1.30E+08	0.0432	0.0870	0.120	4.56E-02	6.68E-03	6.0	1.01E-04	9.07E+08	7.10E-03	5.46E-07
Arsenic	7440-38-2	non-volatile	12	compound is non-volatile, gaseous emission rate calculations are not applicable									non-volatile	non-volatile
Benzo(a)pyrene	50-32-8	66	1.1	1.30E+08	0.0214	0.0430	0.120	5.14E-13	1.38E-09	110000	5.65E-16	9.07E+08	2.97E-10	2.29E-14
Dibenz(a,h)anthracene	53-70-3	non-volatile	0.68	compound is non-volatile, gaseous emission rate calculations are not applicable									non-volatile	non-volatile
Vanadium	7440-62-2	non-volatile	16	compound is non-volatile, gaseous emission rate calculations are not applicable									non-volatile	non-volatile
Chromium	7440-47-3	non-volatile	12	compound is non-volatile, gaseous emission rate calculations are not applicable									non-volatile	non-volatile
Warwick Area														
Aldrin	309-00-2	69	0.14	3.25E+07	0.0236	0.0474	0.120	3.42E-07	1.60E-05	1920	4.14E-10	9.07E+08	8.09E-09	2.49E-12
Beryllium	7440-41-7	non-volatile	0.83	compound is non-volatile, gaseous emission rate calculations are not applicable									non-volatile	non-volatile
Dieldrin	60-57-1	1.4	0.11	3.25E+07	0.0242	0.0488	0.120	5.52E-07	4.58E-07	34	6.88E-10	9.07E+08	8.19E-09	2.52E-12
bis(2-Chloroethyl)ether	111-44-4	2607	ND	3.25E+07	0.0358	0.0721	0.120	1.93E-03	1.31E-05	0.28	3.56E-06	9.07E+08	0.00E+00	0.00E+00
PCB 1248	12672-29-6	222	9.7	3.25E+07	0.0273	0.0550	0.120	3.26E-06	4.40E-04	5540	4.58E-09	9.07E+08	1.86E-06	5.73E-10
PCB 1254	11091-69-1	351	3.3	3.25E+07	0.0261	0.0525	0.120	1.92E-03	2.00E-04	42800	2.57E-10	9.07E+08	1.50E-07	4.62E-11
2-Nitroaniline	88-74-4	517	7.0	3.25E+07	0.0363	0.0730	0.120	2.70E-05	5.00E-07	0.76	5.03E-08	9.07E+08	4.46E-06	1.37E-09
Chlorobenzene	108-90-7	746	27	3.25E+07	0.0363	0.0730	0.120	2.44E-02	3.93E-03	6.6	4.55E-05	9.07E+08	5.18E-04	1.59E-07
Arsenic	7440-38-2	non-volatile	8.9	compound is non-volatile, gaseous emission rate calculations are not applicable									non-volatile	non-volatile
Benzo(a)pyrene	50-32-8	66	1.3	3.25E+07	0.0214	0.0430	0.120	5.14E-13	1.38E-09	110000	5.65E-16	9.07E+08	8.78E-11	2.70E-14
Antimony	7440-36-0	non-volatile	4.2	compound is non-volatile, gaseous emission rate calculations are not applicable									non-volatile	non-volatile
Manganese	7439-96-5	non-volatile	254	compound is non-volatile, gaseous emission rate calculations are not applicable									non-volatile	non-volatile

TABLE 6-D-8 (continued)
Air Emission Rate Calculation Worksheet of Volatile and Semi-Volatile COPCs from Assumed Contaminated Subsurface Soil ($C_{soil} < C_{sat}$)

Contaminant	CAS Number	C_{sat} (mg/kg)	C_{soil} (mg/kg)	A (cm^2)	D_e (cm^2/s)	D (cm^2/s)	ε	K_{as} (g/cm^3)	H (atm-m^3/mole)	K_d (ml/g)	α	t (s)	E (g/s)	E (g/s-m^2)
Waste Water Treatment														
2,3,7,8-TCDF	1746-01-6	2.6	0.00053	6.97E+07	0.0263	0.0530	0.120	2.24E-06	3.60E-03	66000	3.03E-09	9.07E+08	1.78E-10	2.55E-14
gamma-Chlordane	57-74-9	521	2.2	6.97E+07	0.0224	0.0450	0.120	5.37E-07	3.67E-05	2800	6.18E-10	9.07E+08	3.33E-07	4.78E-11
Dieldrin	60-57-1	1.4	0.13	6.97E+07	0.0242	0.0488	0.120	5.52E-07	4.58E-07	34	6.88E-10	9.07E+08	2.08E-08	2.98E-12
bis(2-Ethylhexyl)phthalate	117-81-7	122	1.4	6.97E+07	0.0176	0.0354	0.120	3.24E-09	1.38E-07	1748	2.93E-12	9.07E+08	1.46E-08	2.09E-12
PCB 1254	11091-69-1	351	0.21	6.97E+07	0.0261	0.0525	0.120	1.92E-07	2.00E-04	42800	2.57E-10	9.07E+08	2.05E-08	2.94E-12
Tinuvin 327	not avail.	non-volatile	8.2	compound is non-volatile, gaseous emission rate calculations are not applicable									non-volatile	non-volatile
Arsenic	7440-38-2	non-volatile	6.4	compound is non-volatile, gaseous emission rate calculations are not applicable									non-volatile	non-volatile
Benzo(a)pyrene	50-32-8	66	1.1	6.97E+07	0.0214	0.0430	0.120	5.14E-13	1.38E-09	110000	5.65E-16	9.07E+08	1.59E-10	2.29E-14
Dibenz(a,h)anthracene	53-70-3	non-volatile	1.9	compound is non-volatile, gaseous emission rate calculations are not applicable									non-volatile	non-volatile
Beryllium	7440-41-7	non-volatile	0.56	compound is non-volatile, gaseous emission rate calculations are not applicable									non-volatile	non-volatile
Chromium	7440-47-3	non-volatile	10	compound is non-volatile, gaseous emission rate calculations are not applicable									non-volatile	non-volatile
Manganese	7439-96-5	non-volatile	228	compound is non-volatile, gaseous emission rate calculations are not applicable									non-volatile	non-volatile
Vanadium	7440-62-2	non-volatile	10	compound is non-volatile, gaseous emission rate calculations are not applicable									non-volatile	non-volatile

TABLE 6-D-9
Air Emission Rate Calculation Worksheet of Volatile, Semi-Volatile, and Non-Volatile COPCs from Wind Erodible Surface Soils

Contaminant	CAS Number	V	u (m/s)	u_t (m/s)	F(x)	C_{soil} (mg/kg)	E_{10} (g/s-m^2)
Production Area							
PM-10	-	0.90	4.74	5.10	1.65	-	1.32E-06
PCB 1260	11096-82-5	0.90	4.74	5.10	1.65	6.1	8.08E-12
gamma-Chlordane	57-74-9	0.90	4.74	5.10	1.65	0.13	1.72E-13
Vinyl Chloride	75-01-4	0.90	4.74	5.10	1.65	ND	0.00E+00
PCB 1254	11091-69-1	0.90	4.74	5.10	1.65	20	2.65E-11
PCB 1248	12672-29-6	0.90	4.74	5.10	1.65	127	1.68E-10
2-Nitroaniline	88-74-4	0.90	4.74	5.10	1.65	0.89	1.18E-12
Chlorobenzene	108-90-7	0.90	4.74	5.10	1.65	0.28	3.71E-13
Toluene	108-88-3	0.90	4.74	5.10	1.65	0.71	9.41E-13
Arsenic	7440-38-2	0.90	4.74	5.10	1.65	15	1.99E-11
Benzo(a)pyrene	50-32-8	0.90	4.74	5.10	1.65	1.7	2.25E-12
Dibenz(a,h)anthracene	53-70-3	0.90	4.74	5.10	1.65	0.68	9.01E-13
Vanadium	7440-62-2	0.90	4.74	5.10	1.65	21	2.78E-11
Chromium	7440-47-3	0.90	4.74	5.10	1.65	14	1.85E-11
Warwick Area							
PM-10	-	0.90	4.74	5.10	1.65	-	1.32E-06
Aldrin	309-00-2	0.90	4.74	5.10	1.65	0.21	2.78E-13
Beryllium	7440-41-7	0.90	4.74	5.10	1.65	0.82	1.09E-12
Dieldrin	60-57-1	0.90	4.74	5.10	1.65	0.16	2.12E-13
bis(2-Chloroethyl)ether	111-44-4	0.90	4.74	5.10	1.65	0.43	5.70E-13
PCB 1248	12672-29-6	0.90	4.74	5.10	1.65	15	1.99E-11
PCB 1254	11091-69-1	0.90	4.74	5.10	1.65	5.2	6.89E-12
2-Nitroaniline	88-74-4	0.90	4.74	5.10	1.65	7.0	9.27E-12
Chlorobenzene	108-90-7	0.90	4.74	5.10	1.65	0.67	8.88E-13
Arsenic	7440-38-2	0.90	4.74	5.10	1.65	10	1.32E-11
Benzo(a)pyrene	50-32-8	0.90	4.74	5.10	1.65	1.6	2.12E-12
Antimony	7440-36-0	0.90	4.74	5.10	1.65	5.4	7.15E-12
Manganese	7439-96-5	0.90	4.74	5.10	1.65	245	3.25E-10
Waste Water Treatment Area							
PM-10	-	0.80	4.74	5.10	1.65	-	2.65E-06
2,3,7,8-TCDF	1746-01-6	0.80	4.74	5.10	1.65	0.00062	1.64E-15
gamma-Chlordane	57-74-9	0.80	4.74	5.10	1.65	3.2	8.48E-12
Dieldrin	60-57-1	0.80	4.74	5.10	1.65	0.17	4.50E-13
bis(2-Ethylhexyl)phthalate	117-81-7	0.80	4.74	5.10	1.65	2.1	5.56E-12
PCB 1254	11091-69-1	0.80	4.74	5.10	1.65	0.21	5.56E-13
Tinuvin 327	not avail.	0.80	4.74	5.10	1.65	11	2.91E-11
Arsenic	7440-38-2	0.80	4.74	5.10	1.65	7.7	2.04E-11
Benzo(a)pyrene	50-32-8	0.80	4.74	5.10	1.65	1.5	3.97E-12
Dibenz(a,h)anthracene	53-70-3	0.80	4.74	5.10	1.65	3.0	7.95E-12
Beryllium	7440-41-7	0.80	4.74	5.10	1.65	0.52	1.38E-12
Chromium	7440-47-3	0.80	4.74	5.10	1.65	12	3.18E-11
Manganese	7439-96-5	0.80	4.74	5.10	1.65	273	7.23E-10
Vanadium	7440-62-2	0.80	4.74	5.10	1.65	12	3.18E-11

TABLE 6-D-10
Results of the Air Transport Analysis

Contaminant	CAS Number	Predicted Air Concentration (µg/m³)		
		Gaseous Emissions from Subsurface Soils	Wind Blown Dust from Surface Soils	Ambient
Production Area				
PM-10	-	non-volatile	3.90E+00	3.90E+00
PCB 1260	11096-82-5	1.21E-04	2.38E-05	1.45E-04
gamma-Chlordane	57-74-9	4.47E-06	5.07E-07	4.98E-06
Vinyl Chloride	75-01-4	0.00E+00	0.00E+00	0.00E+00
PCB 1254	11091-69-1	6.18E-04	7.79E-05	6.96E-04
PCB 1248	12672-29-6	1.53E-02	4.95E-04	1.58E-02
2-Nitroaniline	88-74-4	2.42E-03	3.47E-06	2.43E-03
Chlorobenzene	108-90-7	4.86E-03	1.09E-06	4.86E-03
Toluene	108-88-3	1.61E+00	2.77E-06	1.61E+00
Arsenic	7440-38-2	non-volatile	5.85E-05	5.85E-05
Benzo(a)pyrene	50-32-8	6.72E-08	6.62E-06	6.69E-06
Dibenz(a,h)anthracene	53-70-3	non-volatile	2.65E-06	2.65E-06
Vanadium	7440-62-2	non-volatile	8.18E-05	8.18E-05
Chromium	7440-47-3	non-volatile	5.46E-05	5.46E-05
Warwick Area				
PM-10	-	non-volatile	3.56E+00	3.56E+00
Aldrin	309-00-2	6.69E-06	7.48E-07	7.44E-06
Beryllium	7440-41-7	non-volatile	2.92E-06	2.92E-06
Dieldrin	60-57-1	6.78E-06	5.70E-07	7.35E-06
bis(2-Chloroethyl)ether	111-44-4	0.00E+00	1.53E-06	1.53E-06
PCB 1248	12672-29-6	1.54E-03	5.34E-05	1.59E-03
PCB 1254	11091-69-1	1.24E-04	1.85E-05	1.43E-04
2-Nitroaniline	88-74-4	3.69E-03	2.49E-05	3.71E-03
Chlorobenzene	108-90-7	4.28E-01	2.39E-06	4.28E-01
Arsenic	7440-38-2	non-volatile	3.56E-05	3.56E-05
Benzo(a)pyrene	50-32-8	7.26E-08	5.70E-06	5.77E-06
Antimony	7440-36-0	non-volatile	1.92E-05	1.92E-05
Manganese	7439-96-5	non-volatile	8.73E-04	8.73E-04
Waste Water Treatment Area				
PM-10	-	non-volatile	7.48E+00	7.48E+00
2,3,7,8-TCDF	1746-01-6	7.20E-08	4.64E-09	7.66E-08
gamma-Chlordane	57-74-9	1.35E-04	2.39E-05	1.59E-04
Dieldrin	60-57-1	8.41E-06	1.27E-06	9.68E-06
bis(2-Ethylhexyl)phthalate	117-81-7	5.91E-06	1.57E-05	2.16E-05
PCB 1254	11091-69-1	8.31E-06	1.57E-06	9.88E-06
Tinuvin 327	not avail.	non-volatile	8.23E-05	8.23E-05
Arsenic	7440-38-2	non-volatile	5.76E-05	5.76E-05
Benzo(a)pyrene	50-32-8	6.45E-08	1.12E-05	1.13E-05
Dibenz(a,h)anthracene	53-70-3	non-volatile	2.24E-05	2.24E-05
Beryllium	7440-41-7	non-volatile	3.89E-06	3.89E-06
Chromium	7440-47-3	non-volatile	8.98E-05	8.98E-05
Manganese	7439-96-5	non-volatile	2.04E-03	2.04E-03
Vanadium	7440-62-2	non-volatile	8.98E-05	8.98E-05

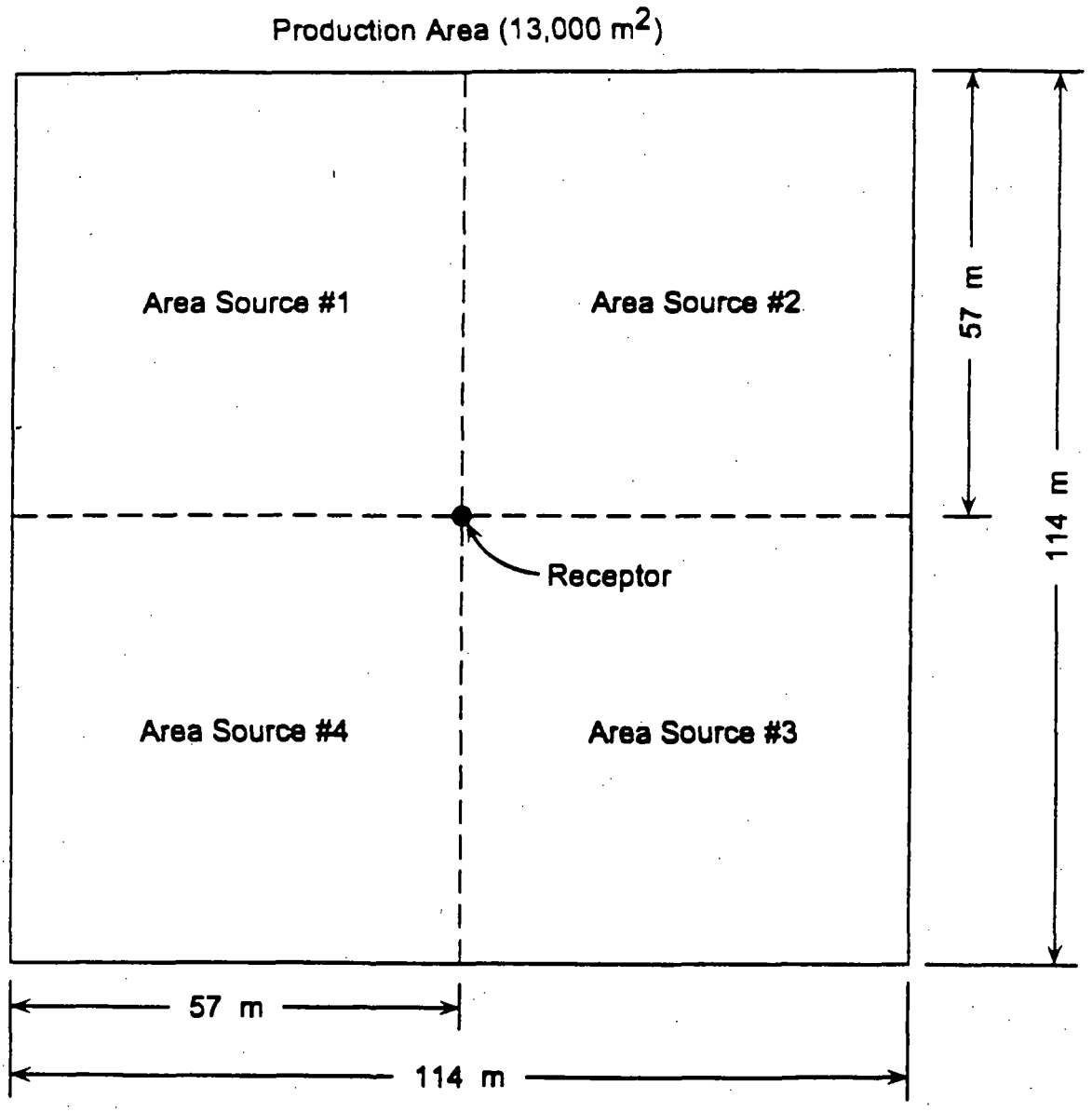
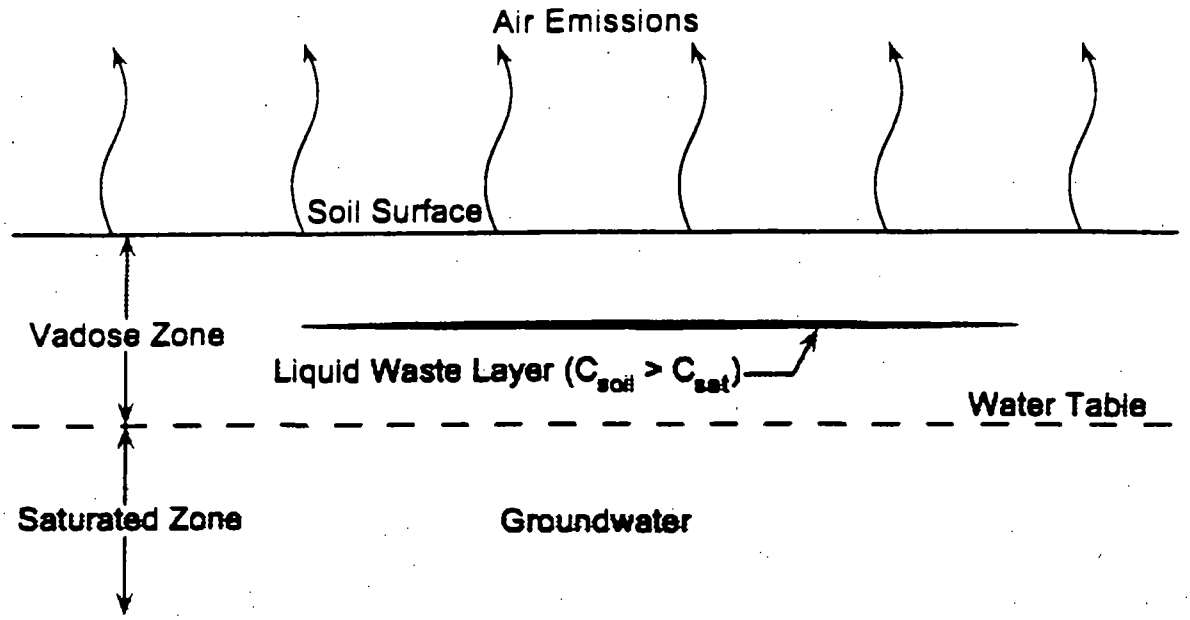


Figure 6-D-1. Area Source Configuration Used in the Air Dispersion Modeling



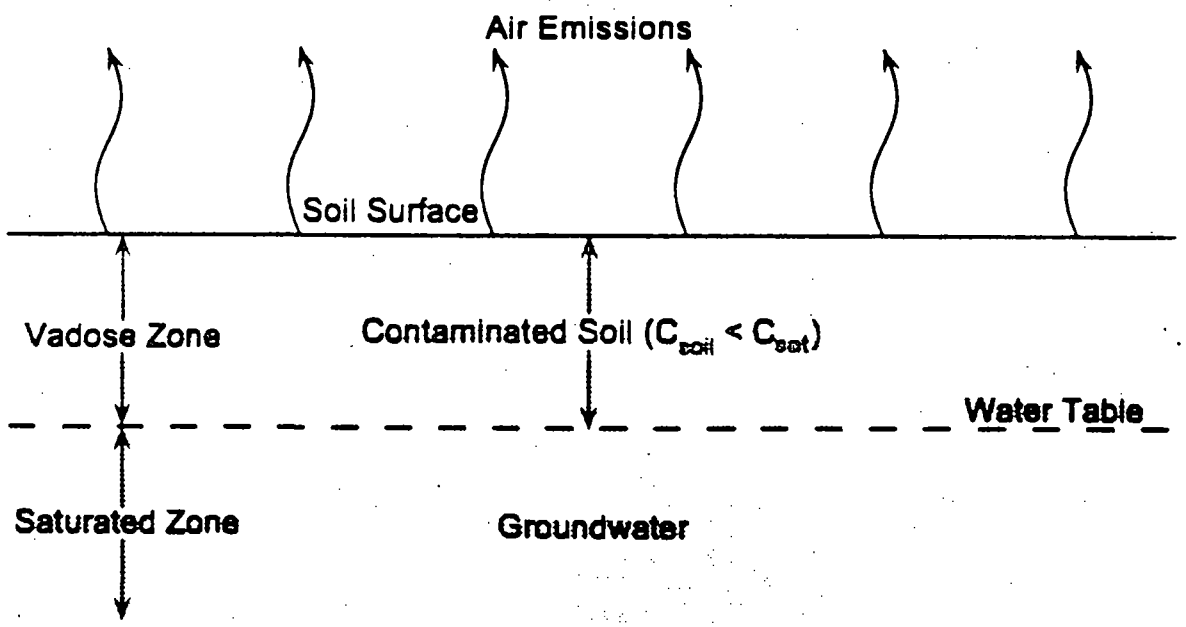


Figure 6-D-2. Gaseous Air Emissions From Contaminated Subsurface Soil.

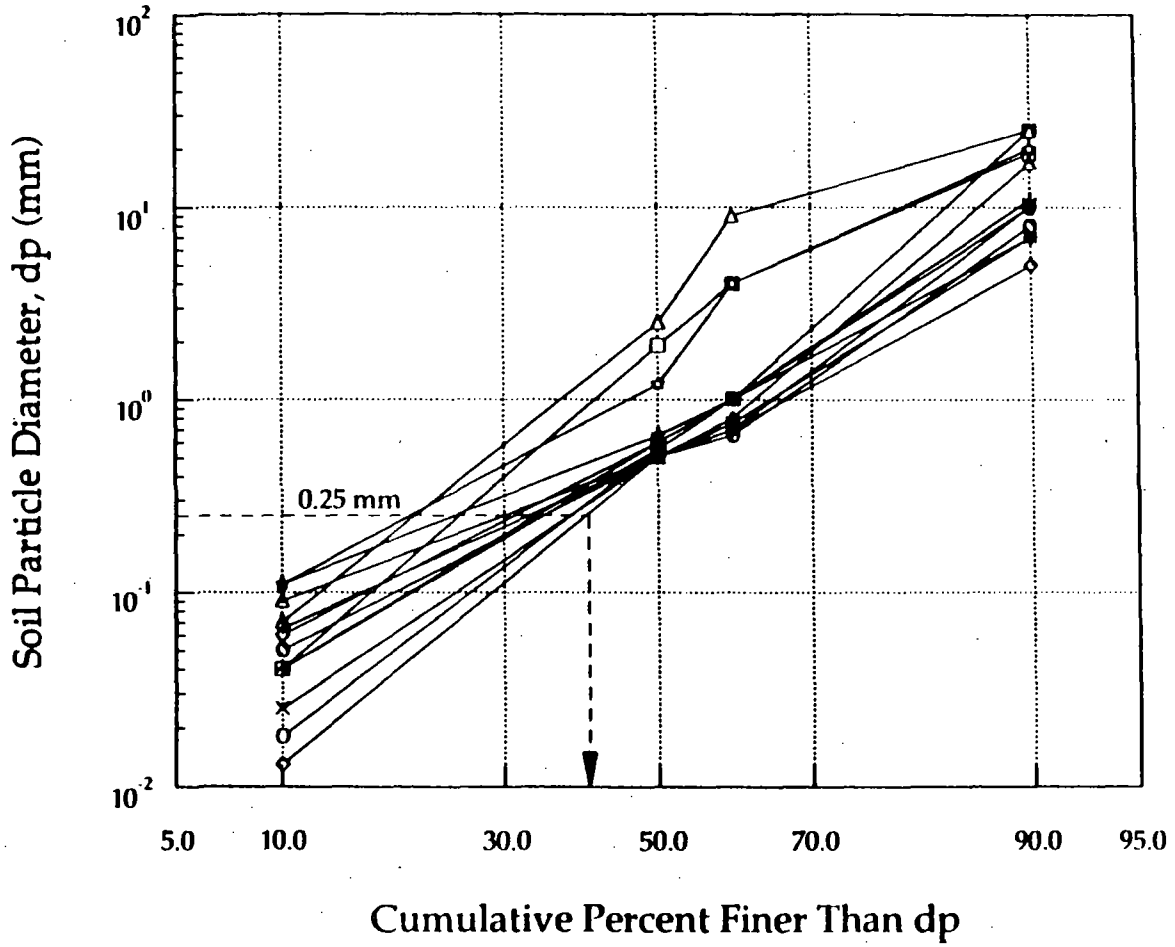


Figure 6-D-3. Soil Particle Size Distribution for the Production Area.

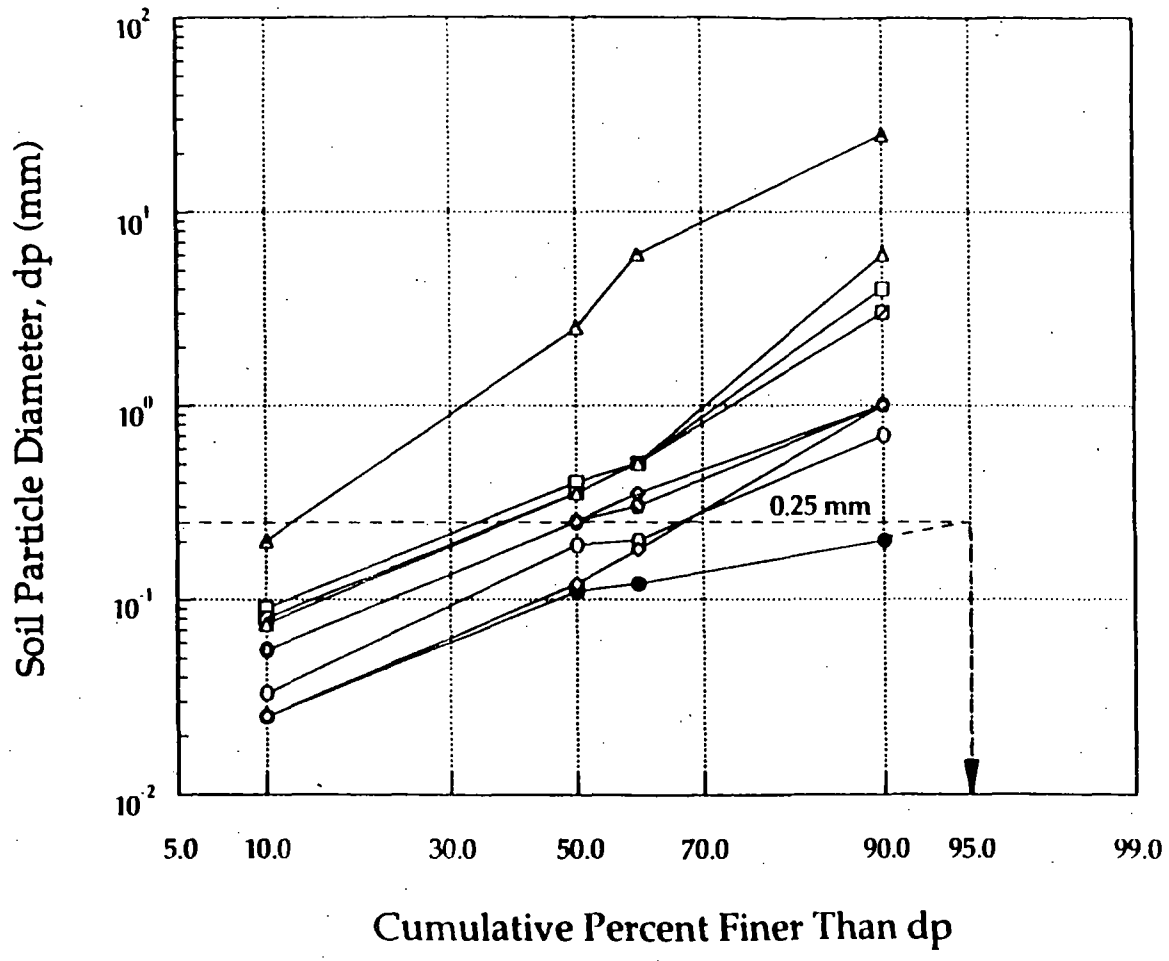


Figure 6-D-4. Soil particle size distribution for the Warwick Area.

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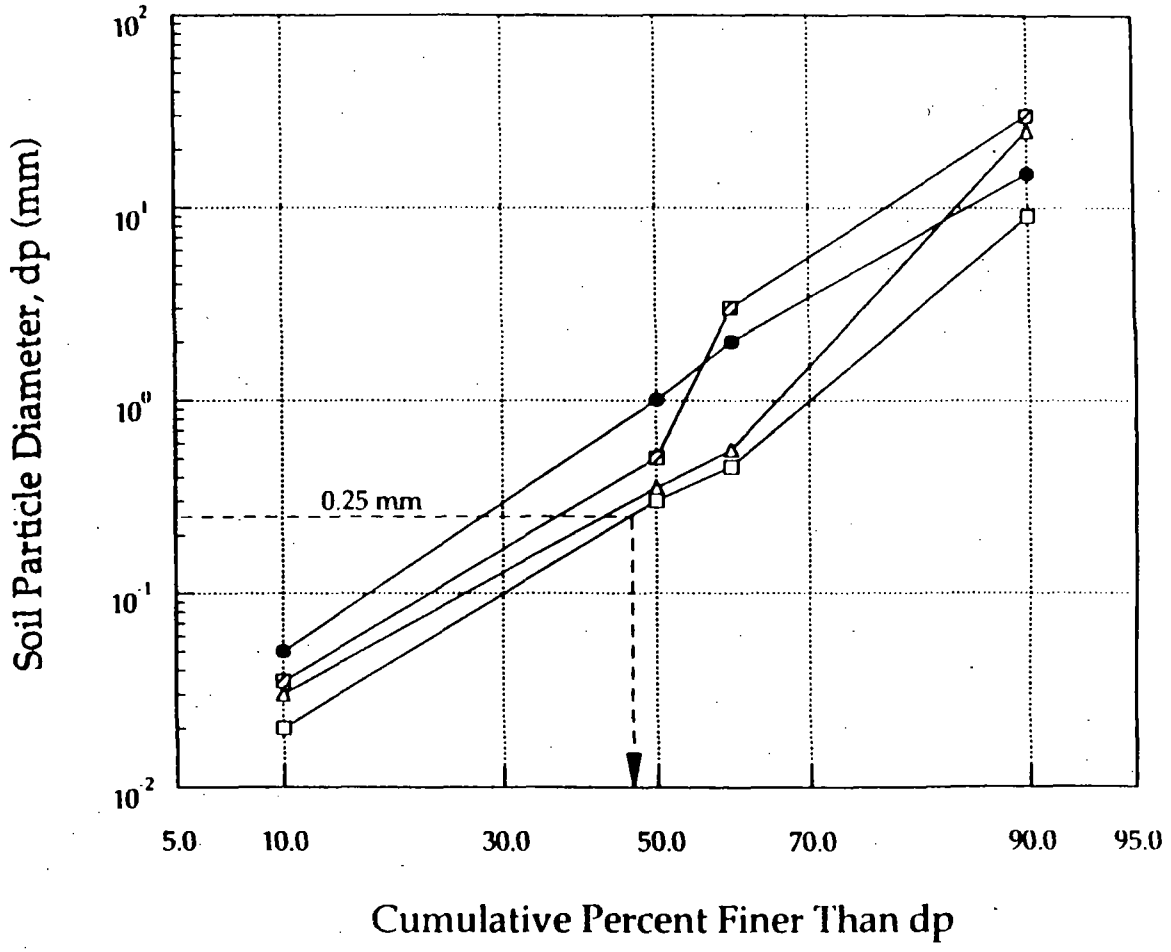


Figure 6-D-5. Soil particle size distribution for the Waste Water Treatment Area.

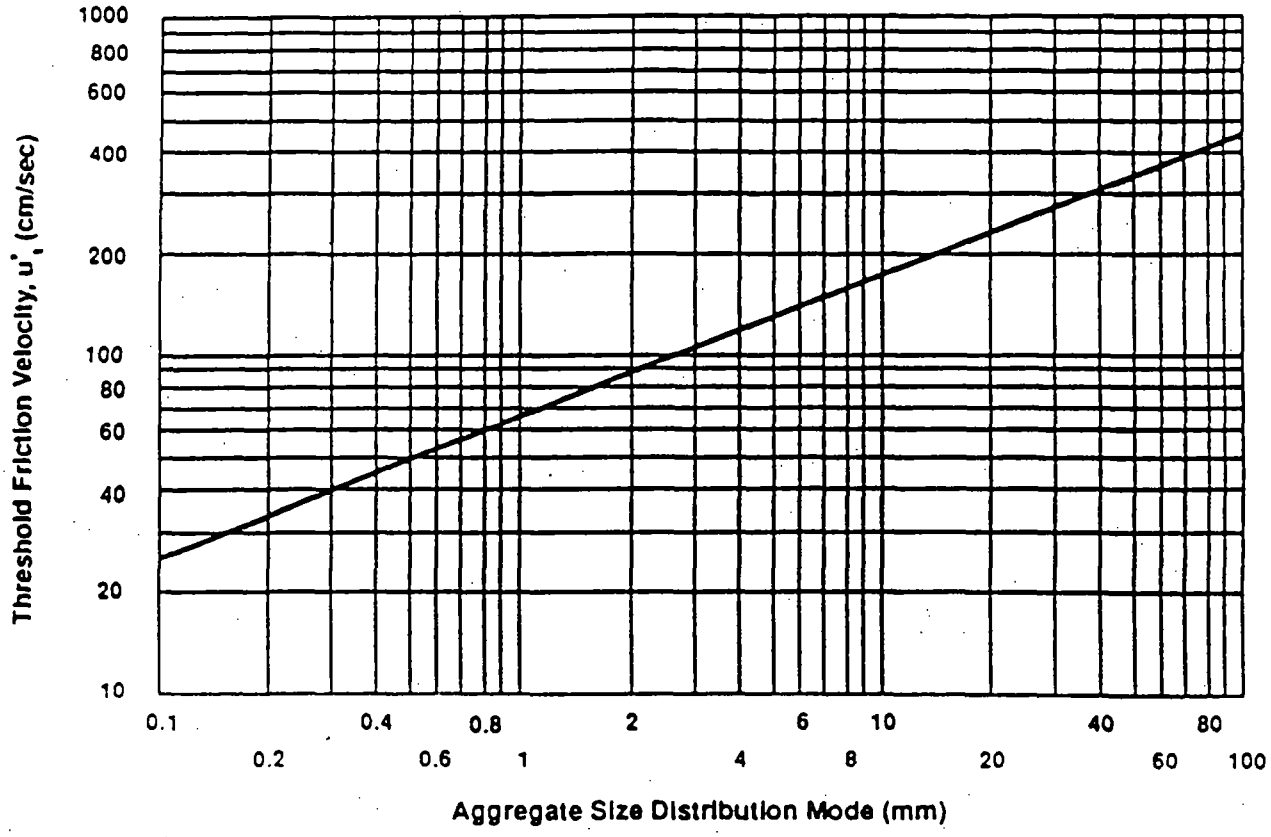
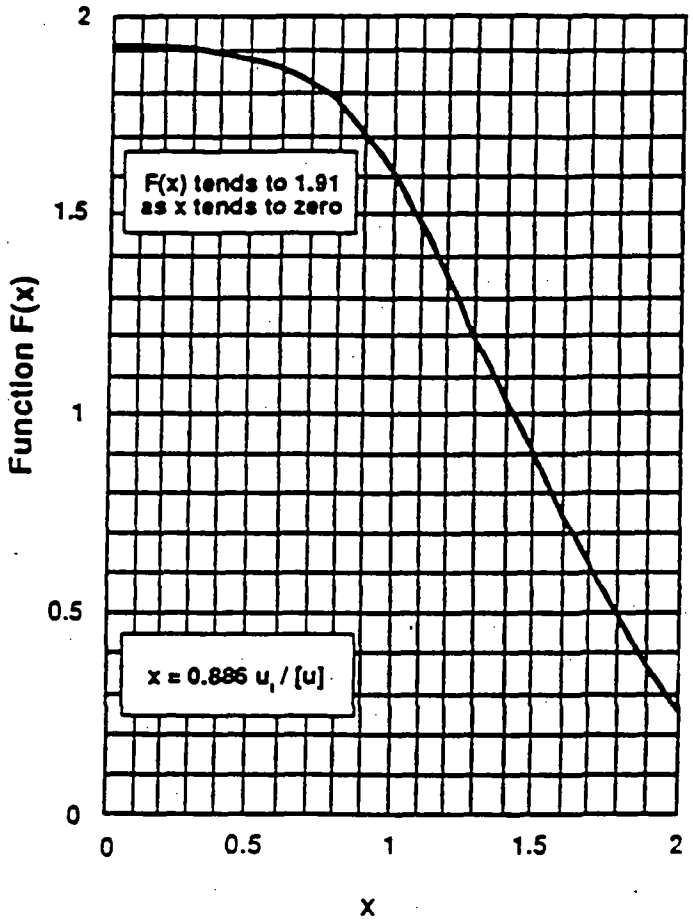


Figure 6-D-6. Threshold Friction Velocity Versus Aggregate Size Distribution.



NOTE: If $x > 2$,
$F(x) = 0.18\,(8x^3 + 12x)\,e^{-x^2}$

Figure 6-D-7. Function Curve Used in the Unlimited Reservoir Model.

ATTACHMENT A.

ISCST2 Model Run Printout

```
CO STARTING
CO TITLEONE Ciba-Geigy  Cranston, RI Site
CO MODELOPT DFAULT CONC RURAL
CO AVERTIME 1
CO POLLUTID PM-10
CO RUNORNOT RUN
CO FINISHED

SO STARTING
**                                  X       Y       Z
**              ID       TYPE      (m)     (m)     (m)
**           --------  ------    -----   -----   ----
SO LOCATION  PRODUCT1   AREA       0.      0.      0.
             PRODUCT2   AREA       0.    -56.9     0.
             PRODUCT3   AREA     -56.9   -56.9     0.
             PRODUCT4   AREA     -56.9     0.      0.

SO LOCATION  WARWICK1   AREA       0.      0.      0.
             WARWICK2   AREA       0.    -28.5     0.
             WARWICK3   AREA     -28.5   -28.5     0.
             WARWICK4   AREA     -28.5     0.      0.

SO LOCATION  WWTREAT1   AREA       0.      0.      0.
             WWTREAT2   AREA       0.    -41.5     0.
             WWTREAT3   AREA     -41.5   -41.5     0.
             WWTREAT4   AREA     -41.5     0.      0.

** Area Source            QS        HS    XO
** Parameters:         (g/s-m2)    (m)   (m)
**                    ----------   ---   ----
SO SRCPARAM  PRODUCT1 0.00000100    0.   56.9
             PRODUCT2 0.00000100    0.   56.9
             PRODUCT3 0.00000100    0.   56.9
             PRODUCT4 0.00000100    0.   56.9

SO SRCPARAM  WARWICK1 0.00000100    0.   28.5
             WARWICK2 0.00000100    0.   28.5
             WARWICK3 0.00000100    0.   28.5
             WARWICK4 0.00000100    0.   28.5

SO SRCPARAM  WWTREAT1 0.00000100    0.   41.5
             WWTREAT2 0.00000100    0.   41.5
             WWTREAT3 0.00000100    0.   41.5
             WWTREAT4 0.00000100    0.   41.5

SO SRCGROUP PRODUCT PRODUCT1-PRODUCT4
            WARWICK WARWICK1-WARWICK4
            WWTREAT WWTREAT1-WWTREAT4

SO FINISHED

RE STARTING
**                X       Y
**               (m)     (m)
**             ------  -----
**
RE DISCCART       0.      0.
RE FINISHED

ME STARTING
ME INPUTFIL CRANSTON.MET
ME ANEMHGHT 10.
ME SURFDATA 99999 1994 CRANSTON, RI
ME UAIRDATA 99999 1994 CRANSTON, RI
ME FINISHED

OU STARTING
OU RECTABLE ALLAVE FIRST
OU MAXTABLE ALLAVE 50
OU FINISHED

**********************************
*** SETUP Finishes Successfully ***
**********************************
```

*** MODELING OPTIONS USED: CONC RURAL FLAT DFAULT

*** MODEL SETUP OPTIONS SUMMARY ***

. . . . . .

**Model Is Setup For Calculation of Average CONCentration Values.

**Model Uses RURAL Dispersion.

**Model Uses Regulatory DEFAULT Options:
1. Final Plume Rise.
2. Stack-tip Downwash.
3. Buoyancy-induced Dispersion.
4. Use Calms Processing Routine.
5. Not Use Missing Data Processing Routine.
6. Default Wind Profile Exponents.
7. Default Vertical Potential Temperature Gradients.
8. "Upper Bound" Values for Supersquat Buildings.
9. No Exponential Decay for RURAL Mode

**Model Assumes Receptors on FLAT Terrain.

**Model Assumes No FLAGPOLE Receptor Heights.

**Model Calculates 1 Short Term Average(s) of: 1-HR

**This Run Includes: 12 Source(s); 3 Source Group(s); and 1 Receptor(s)

**The Model Assumes A Pollutant Type of: PM-10

**Model Set To Continue RUNning After the Setup Testing.

**Output Options Selected:
Model Outputs Tables of Highest Short Term Values by Receptor (RECTABLE Keyword)
Model Outputs Tables of Overall Maximum Short Term Values (MAXTABLE Keyword)

**NOTE: The Following Flags May Appear Following CONC Values: c for Calm Hours
m for Missing Hours
b for Both Calm and Missing Hours

**Misc. Inputs: Anem. Hgt. (m) = 10.00 ; Decay Coef. = 0.0000 ; Rot. Angle = 0.0
Emission Units = GRAMS/SEC ; Emission Rate Unit Factor = 0.10000E+07
Output Units = MICROGRAMS/M**3

**Input Runstream File: CRANSTON.DAT ; **Output Print File: CRANSTON.OUT

*** MODELING OPTIONS USED: CONC RURAL FLAT DFAULT

*** AREA SOURCE DATA ***

SOURCE ID	NUMBER PART. CATS.	EMISSION RATE (GRAMS/SEC /METER**2)	COORD (SW CORNER) X (METERS)	Y (METERS)	BASE ELEV. (METERS)	RELEASE HEIGHT (METERS)	WIDTH OF AREA (METERS)	EMISSION RATE SCALAR VARY BY
PRODUCT1	0	0.10000E-05	0.0	0.0	0.0	0.00	56.90	
PRODUCT2	0	0.10000E-05	0.0	-56.9	0.0	0.00	56.90	
PRODUCT3	0	0.10000E-05	-56.9	-56.9	0.0	0.00	56.90	
PRODUCT4	0	0.10000E-05	-56.9	0.0	0.0	0.00	56.90	
WARWICK1	0	0.10000E-05	0.0	0.0	0.0	0.00	28.50	
WARWICK2	0	0.10000E-05	0.0	-28.5	0.0	0.00	28.50	
WARWICK3	0	0.10000E-05	-28.5	-28.5	0.0	0.00	28.50	
WARWICK4	0	0.10000E-05	-28.5	0.0	0.0	0.00	28.50	
WWTREAT1	0	0.10000E-05	0.0	0.0	0.0	0.00	41.50	
WWTREAT2	0	0.10000E-05	0.0	-41.5	0.0	0.00	41.50	
WWTREAT3	0	0.10000E-05	-41.5	-41.5	0.0	0.00	41.50	
WWTREAT4	0	0.10000E-05	-41.5	0.0	0.0	0.00	41.50	

*** MODELING OPTIONS USED: CONC RURAL FLAT DFAULT

*** SOURCE IDs DEFINING SOURCE GROUPS ***

GROUP ID	SOURCE IDs
PRODUCT	PRODUCT1, PRODUCT2, PRODUCT3, PRODUCT4,
WARWICK	WARWICK1, WARWICK2, WARWICK3, WARWICK4,
WWTREAT	WWTREAT1, WWTREAT2, WWTREAT3, WWTREAT4,

*** MODELING OPTIONS USED: CONC RURAL FLAT DFAULT

*** DISCRETE CARTESIAN RECEPTORS ***
(X-COORD, Y-COORD, ZELEV, ZFLAG)
(METERS)

(0.0, 0.0, 0.0, 0.0); ********

*** MODELING OPTIONS USED: CONC RURAL FLAT DFAULT

*** METEOROLOGICAL DAYS SELECTED FOR PROCESSING ***
(1=YES; 0=NO)

```
1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1
1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1
1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1
1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1
1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1
1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1
1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1 1 1 1 1
1 1 1 1 1 1 1 1 1 1   1 1 1 1 1 1
```

NOTE: METEOROLOGICAL DATA ACTUALLY PROCESSED WILL ALSO DEPEND ON WHAT IS INCLUDED IN THE DATA FILE.

*** UPPER BOUND OF FIRST THROUGH FIFTH WIND SPEED CATEGORIES ***
(METERS/SEC)

1.54, 3.09, 5.14, 8.23, 10.80,

*** WIND PROFILE EXPONENTS ***

STABILITY CATEGORY	WIND SPEED CATEGORY 1	2	3	4	5	6
A	.70000E-01	.70000E-01	.70000E-01	.70000E-01	.70000E-01	.70000E-01
B	.70000E-01	.70000E-01	.70000E-01	.70000E-01	.70000E-01	.70000E-01
C	.10000E+00	.10000E+00	.10000E+00	.10000E+00	.10000E+00	.10000E+00
D	.15000E+00	.15000E+00	.15000E+00	.15000E+00	.15000E+00	.15000E+00
E	.35000E+00	.35000E+00	.35000E+00	.35000E+00	.35000E+00	.35000E+00
F	.55000E+00	.55000E+00	.55000E+00	.55000E+00	.55000E+00	.55000E+00

*** VERTICAL POTENTIAL TEMPERATURE GRADIENTS ***
(DEGREES KELVIN PER METER)

STABILITY CATEGORY	WIND SPEED CATEGORY 1	2	3	4	5	6
A	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00
B	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00
C	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00
D	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00	.00000E+00
E	.20000E-01	.20000E-01	.20000E-01	.20000E-01	.20000E-01	.20000E-01
F	.35000E-01	.35000E-01	.35000E-01	.35000E-01	.35000E-01	.35000E-01

*** MODELING OPTIONS USED: CONC RURAL FLAT DFAULT

*** THE FIRST 6 HOURS OF METEOROLOGICAL DATA ***

FILE: CRANSTON.MET FORMAT: (4I2,2F9.4,F6.1,I2,2F7.1)
SURFACE STATION NO.: 99999 UPPER AIR STATION NO.: 99999
NAME: CRANSTON, NAME: CRANSTON,
YEAR: 1994 YEAR: 1994

YEAR	MONTH	DAY	HOUR	FLOW VECTOR	SPEED (M/S)	TEMP (K)	STAB CLASS	MIXING HEIGHT (M) RURAL	MIXING HEIGHT (M) URBAN
94	1	1	1	360.0	4.74	283.0	4	1520.0	1520.0
94	1	1	2	10.0	4.74	283.0	4	1520.0	1520.0
94	1	1	3	20.0	4.74	283.0	4	1520.0	1520.0
94	1	1	4	30.0	4.74	283.0	4	1520.0	1520.0
94	1	1	5	40.0	4.74	283.0	4	1520.0	1520.0
94	1	1	6	45.0	4.74	283.0	4	1520.0	1520.0

*** NOTES: STABILITY CLASS 1=A, 2=B, 3=C, 4=D, 5=E AND 6=F.
FLOW VECTOR IS DIRECTION TOWARD WHICH WIND IS BLOWING.

*** MODELING OPTIONS USED: CONC RURAL FLAT DFAULT

*** THE 1ST HIGHEST 1-HR AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: PRODUCT ***
INCLUDING SOURCE(S): PRODUCT1, PRODUCT2, PRODUCT3, PRODUCT4,

*** DISCRETE CARTESIAN RECEPTOR POINTS ***

** CONC OF PM-10 IN MICROGRAMS/M**3 **

X-COORD (M)	Y-COORD (M)	CONC	(YYMMDDHH)	X-COORD (M)	Y-COORD (M)	CONC	(YYMMDDHH)
0.00	0.00	2.94176	(94010102)	**********	**********	**********	**********

*** MODELING OPTIONS USED: CONC RURAL FLAT DFAULT

*** THE 1ST HIGHEST 1-HR AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: WARWICK ***
INCLUDING SOURCE(S): WARWICK1, WARWICK2, WARWICK3, WARWICK4,

*** DISCRETE CARTESIAN RECEPTOR POINTS ***

** CONC OF PM-10 IN MICROGRAMS/M**3 **

X-COORD (M)	Y-COORD (M)	CONC	(YYMMDDHH)	X-COORD (M)	Y-COORD (M)	CONC	(YYMMDDHH)
0.00	0.00	2.68840	(94010102)				

*** MODELING OPTIONS USED: CONC RURAL FLAT DFAULT

*** THE 1ST HIGHEST 1-HR AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: WWTREAT ***
INCLUDING SOURCE(S): WWTREAT1, WWTREAT2, WWTREAT3, WWTREAT4,

*** DISCRETE CARTESIAN RECEPTOR POINTS ***

** CONC OF PM-10 IN MICROGRAMS/M**3 **

X-COORD (M)	Y-COORD (M)	CONC	(YYMMDDHH)	X-COORD (M)	Y-COORD (M)	CONC	(YYMMDDHH)
0.00	0.00	2.82327	(94010102)				

*** MODELING OPTIONS USED: CONC RURAL FLAT DFAULT

*** THE MAXIMUM 50 1-HR AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: PRODUCT ***
INCLUDING SOURCE(S): PRODUCT1, PRODUCT2, PRODUCT3, PRODUCT4,

** CONC OF PM-10 IN MICROGRAMS/M**3 **

RANK	CONC	(YYMMDDHH)	AT	RECEPTOR (XR,YR)		OF TYPE	RANK	CONC	(YYMMDDHH)	AT	RECEPTOR (XR,YR)		OF TYPE
1.	2.94176	(94010102)	AT (	0.00,	0.00)	DC	26.	0.00000	(0)	AT (	0.00,	0.00)	
2.	2.79475	(94010103)	AT (	0.00,	0.00)	DC	27.	0.00000	(0)	AT (	0.00,	0.00)	
3.	2.70293	(94010104)	AT (	0.00,	0.00)	DC	28.	0.00000	(0)	AT (	0.00,	0.00)	
4.	2.65875	(94010105)	AT (	0.00,	0.00)	DC	29.	0.00000	(0)	AT (	0.00,	0.00)	
5.	2.65331	(94010106)	AT (	0.00,	0.00)	DC	30.	0.00000	(0)	AT (	0.00,	0.00)	
6.	0.00000	(0)	AT (	0.00,	0.00)		31.	0.00000	(0)	AT (	0.00,	0.00)	
7.	0.00000	(0)	AT (	0.00,	0.00)		32.	0.00000	(0)	AT (	0.00,	0.00)	
8.	0.00000	(0)	AT (	0.00,	0.00)		33.	0.00000	(0)	AT (	0.00,	0.00)	
9.	0.00000	(0)	AT (	0.00,	0.00)		34.	0.00000	(0)	AT (	0.00,	0.00)	
10.	0.00000	(0)	AT (	0.00,	0.00)		35.	0.00000	(0)	AT (	0.00,	0.00)	
11.	0.00000	(0)	AT (	0.00,	0.00)		36.	0.00000	(0)	AT (	0.00,	0.00)	
12.	0.00000	(0)	AT (	0.00,	0.00)		37.	0.00000	(0)	AT (	0.00,	0.00)	
13.	0.00000	(0)	AT (	0.00,	0.00)		38.	0.00000	(0)	AT (	0.00,	0.00)	
14.	0.00000	(0)	AT (	0.00,	0.00)		39.	0.00000	(0)	AT (	0.00,	0.00)	
15.	0.00000	(0)	AT (	0.00,	0.00)		40.	0.00000	(0)	AT (	0.00,	0.00)	
16.	0.00000	(0)	AT (	0.00,	0.00)		41.	0.00000	(0)	AT (	0.00,	0.00)	
17.	0.00000	(0)	AT (	0.00,	0.00)		42.	0.00000	(0)	AT (	0.00,	0.00)	
18.	0.00000	(0)	AT (	0.00,	0.00)		43.	0.00000	(0)	AT (	0.00,	0.00)	
19.	0.00000	(0)	AT (	0.00,	0.00)		44.	0.00000	(0)	AT (	0.00,	0.00)	
20.	0.00000	(0)	AT (	0.00,	0.00)		45.	0.00000	(0)	AT (	0.00,	0.00)	
21.	0.00000	(0)	AT (	0.00,	0.00)		46.	0.00000	(0)	AT (	0.00,	0.00)	
22.	0.00000	(0)	AT (	0.00,	0.00)		47.	0.00000	(0)	AT (	0.00,	0.00)	
23.	0.00000	(0)	AT (	0.00,	0.00)		48.	0.00000	(0)	AT (	0.00,	0.00)	
24.	0.00000	(0)	AT (	0.00,	0.00)		49.	0.00000	(0)	AT (	0.00,	0.00)	
25.	0.00000	(0)	AT (	0.00,	0.00)		50.	0.00000	(0)	AT (	0.00,	0.00)	

*** RECEPTOR TYPES: GC = GRIDCART
GP = GRIDPOLR
DC = DISCCART
DP = DISCPOLR
BD = BOUNDARY

*** MODELING OPTIONS USED: CONC RURAL FLAT DFAULT

*** THE MAXIMUM 50 1-HR AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: WARWICK ***
INCLUDING SOURCE(S): WARWICK1, WARWICK2, WARWICK3, WARWICK4,

** CONC OF PM-10 IN MICROGRAMS/M**3 **

RANK	CONC	(YYMMDDHH)	AT	RECEPTOR (XR,YR)		OF TYPE	RANK	CONC	(YYMMDDHH)	AT	RECEPTOR (XR,YR)		OF TYPE
1.	2.68840	(94010102)	AT (	0.00,	0.00)	DC	26.	0.00000	(0)	AT (	0.00,	0.00)	
2.	2.55405	(94010103)	AT (	0.00,	0.00)	DC	27.	0.00000	(0)	AT (	0.00,	0.00)	
3.	2.47014	(94010104)	AT (	0.00,	0.00)	DC	28.	0.00000	(0)	AT (	0.00,	0.00)	
4.	2.42977	(94010105)	AT (	0.00,	0.00)	DC	29.	0.00000	(0)	AT (	0.00,	0.00)	
5.	2.42479	(94010106)	AT (	0.00,	0.00)	DC	30.	0.00000	(0)	AT (	0.00,	0.00)	
6.	0.00000	(0)	AT (	0.00,	0.00)		31.	0.00000	(0)	AT (	0.00,	0.00)	
7.	0.00000	(0)	AT (	0.00,	0.00)		32.	0.00000	(0)	AT (	0.00,	0.00)	
8.	0.00000	(0)	AT (	0.00,	0.00)		33.	0.00000	(0)	AT (	0.00,	0.00)	
9.	0.00000	(0)	AT (	0.00,	0.00)		34.	0.00000	(0)	AT (	0.00,	0.00)	
10.	0.00000	(0)	AT (	0.00,	0.00)		35.	0.00000	(0)	AT (	0.00,	0.00)	
11.	0.00000	(0)	AT (	0.00,	0.00)		36.	0.00000	(0)	AT (	0.00,	0.00)	
12.	0.00000	(0)	AT (	0.00,	0.00)		37.	0.00000	(0)	AT (	0.00,	0.00)	
13.	0.00000	(0)	AT (	0.00,	0.00)		38.	0.00000	(0)	AT (	0.00,	0.00)	
14.	0.00000	(0)	AT (	0.00,	0.00)		39.	0.00000	(0)	AT (	0.00,	0.00)	
15.	0.00000	(0)	AT (	0.00,	0.00)		40.	0.00000	(0)	AT (	0.00,	0.00)	
16.	0.00000	(0)	AT (	0.00,	0.00)		41.	0.00000	(0)	AT (	0.00,	0.00)	
17.	0.00000	(0)	AT (	0.00,	0.00)		42.	0.00000	(0)	AT (	0.00,	0.00)	
18.	0.00000	(0)	AT (	0.00,	0.00)		43.	0.00000	(0)	AT (	0.00,	0.00)	
19.	0.00000	(0)	AT (	0.00,	0.00)		44.	0.00000	(0)	AT (	0.00,	0.00)	
20.	0.00000	(0)	AT (	0.00,	0.00)		45.	0.00000	(0)	AT (	0.00,	0.00)	
21.	0.00000	(0)	AT (	0.00,	0.00)		46.	0.00000	(0)	AT (	0.00,	0.00)	
22.	0.00000	(0)	AT (	0.00,	0.00)		47.	0.00000	(0)	AT (	0.00,	0.00)	
23.	0.00000	(0)	AT (	0.00,	0.00)		48.	0.00000	(0)	AT (	0.00,	0.00)	
24.	0.00000	(0)	AT (	0.00,	0.00)		49.	0.00000	(0)	AT (	0.00,	0.00)	
25.	0.00000	(0)	AT (	0.00,	0.00)		50.	0.00000	(0)	AT (	0.00,	0.00)	

*** RECEPTOR TYPES: GC = GRIDCART
GP = GRIDPOLR
DC = DISCCART
DP = DISCPOLR
BD = BOUNDARY

*** MODELING OPTIONS USED: CONC RURAL FLAT DFAULT

*** THE MAXIMUM 50 1-HR AVERAGE CONCENTRATION VALUES FOR SOURCE GROUP: WWTREAT ***
INCLUDING SOURCE(S): WWTREAT1, WWTREAT2, WWTREAT3, WWTREAT4,

** CONC OF PM-10 IN MICROGRAMS/M**3 **

RANK	CONC	(YYMMDDHH)	AT	RECEPTOR (XR,YR)		OF TYPE	RANK	CONC	(YYMMDDHH)	AT	RECEPTOR (XR,YR)		OF TYPE
1.	2.82327	(94010102)	AT (	0.00,	0.00)	DC	26.	0.00000	(0)	AT (	0.00,	0.00)	
2.	2.68218	(94010103)	AT (	0.00,	0.00)	DC	27.	0.00000	(0)	AT (	0.00,	0.00)	
3.	2.59407	(94010104)	AT (	0.00,	0.00)	DC	28.	0.00000	(0)	AT (	0.00,	0.00)	
4.	2.55166	(94010105)	AT (	0.00,	0.00)	DC	29.	0.00000	(0)	AT (	0.00,	0.00)	
5.	2.54644	(94010106)	AT (	0.00,	0.00)	DC	30.	0.00000	(0)	AT (	0.00,	0.00)	
6.	0.00000	(0)	AT (	0.00,	0.00)		31.	0.00000	(0)	AT (	0.00,	0.00)	
7.	0.00000	(0)	AT (	0.00,	0.00)		32.	0.00000	(0)	AT (	0.00,	0.00)	
8.	0.00000	(0)	AT (	0.00,	0.00)		33.	0.00000	(0)	AT (	0.00,	0.00)	
9.	0.00000	(0)	AT (	0.00,	0.00)		34.	0.00000	(0)	AT (	0.00,	0.00)	
10.	0.00000	(0)	AT (	0.00,	0.00)		35.	0.00000	(0)	AT (	0.00,	0.00)	
11.	0.00000	(0)	AT (	0.00,	0.00)		36.	0.00000	(0)	AT (	0.00,	0.00)	
12.	0.00000	(0)	AT (	0.00,	0.00)		37.	0.00000	(0)	AT (	0.00,	0.00)	
13.	0.00000	(0)	AT (	0.00,	0.00)		38.	0.00000	(0)	AT (	0.00,	0.00)	
14.	0.00000	(0)	AT (	0.00,	0.00)		39.	0.00000	(0)	AT (	0.00,	0.00)	
15.	0.00000	(0)	AT (	0.00,	0.00)		40.	0.00000	(0)	AT (	0.00,	0.00)	
16.	0.00000	(0)	AT (	0.00,	0.00)		41.	0.00000	(0)	AT (	0.00,	0.00)	
17.	0.00000	(0)	AT (	0.00,	0.00)		42.	0.00000	(0)	AT (	0.00,	0.00)	
18.	0.00000	(0)	AT (	0.00,	0.00)		43.	0.00000	(0)	AT (	0.00,	0.00)	
19.	0.00000	(0)	AT (	0.00,	0.00)		44.	0.00000	(0)	AT (	0.00,	0.00)	
20.	0.00000	(0)	AT (	0.00,	0.00)		45.	0.00000	(0)	AT (	0.00,	0.00)	
21.	0.00000	(0)	AT (	0.00,	0.00)		46.	0.00000	(0)	AT (	0.00,	0.00)	
22.	0.00000	(0)	AT (	0.00,	0.00)		47.	0.00000	(0)	AT (	0.00,	0.00)	
23.	0.00000	(0)	AT (	0.00,	0.00)		48.	0.00000	(0)	AT (	0.00,	0.00)	
24.	0.00000	(0)	AT (	0.00,	0.00)		49.	0.00000	(0)	AT (	0.00,	0.00)	
25.	0.00000	(0)	AT (	0.00,	0.00)		50.	0.00000	(0)	AT (	0.00,	0.00)	

*** RECEPTOR TYPES: GC = GRIDCART
GP = GRIDPOLR
DC = DISCCART
DP = DISCPOLR
BD = BOUNDARY

*** MODELING OPTIONS USED: CONC RURAL FLAT DFAULT

*** THE SUMMARY OF HIGHEST 1-HR RESULTS ***

** CONC OF PM-10 IN MICROGRAMS/M**3 **

GROUP ID		AVERAGE CONC	DATE (YYMMDDHH)	RECEPTOR (XR, YR, ZELEV, ZFLAG)				OF TYPE	NETWORK GRID-ID
PRODUCT HIGH	1ST HIGH VALUE IS	2.94176	ON 94010102: AT (	0.00,	0.00,	0.00,	0.00)	DC	
WARWICK HIGH	1ST HIGH VALUE IS	2.68840	ON 94010102: AT (	0.00,	0.00,	0.00,	0.00)	DC	
WWTREAT HIGH	1ST HIGH VALUE IS	2.82327	ON 94010102: AT (	0.00,	0.00,	0.00,	0.00)	DC	

*** RECEPTOR TYPES: GC = GRIDCART
GP = GRIDPOLR
DC = DISCCART
DP = DISCPOLR
BD = BOUNDARY

*** MODELING OPTIONS USED: CONC RURAL FLAT DFAULT

*** Message Summary For ISC2 Model Execution ***

--------- Summary of Total Messages --------

A Total of 0 Fatal Error Message(s)
A Total of 0 Warning Message(s)
A Total of 0 Informational Message(s)

******** FATAL ERROR MESSAGES ********
*** NONE ***

******** WARNING MESSAGES ********
*** NONE ***

****
*** ISCST2 Finishes Successfully ***
****

Appendix 6-E

Estimated Surface Water Concentrations of Site Contamination from Groundwater Discharges

Woodward-Clyde
Engineering & sciences applied to the earth & its environment

July 6, 1995
87X4660

Mr. Thomas Marshall
PTRL Environmental Services
Four Oaks Center
1942 Oak Ridge Turnpike
Oak Ridge, TN 37830

Re: Estimated Surface Water Concentrations of Site Contaminants From Groundwater Discharges - Former Ciba-Geigy Facility, Cranston, Rhode Island

This letter presents a summary of the methodology used and results obtained in estimating surface water concentrations of selected site contaminants resulting from groundwater discharges into the Pawtuxet River. These calculations were made in order to assist PTRL Environmental Services in performing a human health risk assessment (for human contact with water from the Pawtuxet River). This letter contains estimates of the surface water contaminant concentrations discharging to the river from the Production Area, Waste Water Treatment Area, and Warwick Area.

This letter includes three sections: Methods and Assumptions, Input Values, and Results of the Calculations.

METHODS AND ASSUMPTIONS

This section outlines in detail the methods used to calculate surface water (river) concentrations of site contaminants, given soil and/or groundwater concentrations of these contaminants in areas adjacent to the river. Several methods were evaluated including simple partitioning of contaminants, use of existing groundwater concentrations as surface water contaminant concentrations, soil/water equilibrium models, and detailed fate and transport modeling. The Summers Model, a particular soil/water equilibrium model, was used to determine groundwater concentrations. It was selected for its simplicity, availability of required input data, relative accuracy of the results obtained, and acceptance by USEPA. The groundwater concentrations from the Summers Model are combined with a flow net analysis of groundwater flow to the river and a dilution equation to determine river concentrations. The modeling approach and assumptions used in selecting model input values are described below.

The Summers Model (Summers, et. al. 1980) uses soil/water partitioning coefficients, adsorbed soil concentrations, and infiltration through areas of contamination to determine the groundwater concentrations for each contaminant. Areas on the site having concentrations above a cetrain threshold value are delineated for each contaminant. A certain percentage of measured site precipitation is assumed to result in infiltration. The rate of infiltration is governed by the unsaturated vertical hydraulic conductivity. This infiltrating water moves through the soil in the unsaturated zone, eventually reaching the water table, where it becomes mixed with the groundwater. As the water moves through the unsaturated zone, the contaminants adsorbed to the unsaturated soil particles partition into the infiltrating water according to contaminant-

Wayne Office
P.O. Box 290 • 201 Willowbrook Boulevard • Wayne, New Jersey 07470
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dependant partitioning coefficients, K_d. This contaminated infiltration reaches the water table and is diluted into the groundwater. The volumetric flow rate of the contaminated water is determined by multiplying the infiltration rate times the area of contamination for each contaminant.

Data obtained from the Summers model are then used as input to a dilution equation to predict the concentration of the contaminants in the surface water. Contribution of the groundwater to flow in the river is calculated by use of a flow net analysis, as shown in Attachment A.

The sequence of calculations in the Summers Model and dilution equations are as follows:

1) Receipt of list of chemicals of potential concern for river risk analysis from PTRL. The complete list by area is presented below.

Table 1: Chemicals of Potential Concern for Human Health Risk Assessment (from PTRL)

PRODUCTION AREA	WASTEWATER TREATMENT AREA	WARWICK AREA
2-Nitroaniline	2,3,7,8-TCDF	2-Nitroaniline
Chlorobenzene	*bis*(2-Ethylhexyl)phthalate	Aldrin
gamma-Chlordane	Dieldrin	Beryllium
PCB 1248	gamma-Chlordane	*bis*(2-Chloroethyl)ether
PCB 1254	PCB 1254	Chlorobenzene
PCB 1260	Tinuvin 327	Dieldrin
Toluene		PCB 1248
Vinyl Chloride		PCB 1254

2) Calculation of areal extent of contamination of each listed contaminant in each area was performed by contouring regions around known measurement points. This process was carried out using the SitePlanner program on a Sun workstation. Non-detect measurements were represented by a value equal to one-half the non-detect value, and kreiging was the numerical method used to solve for contours (see attached figures). Contour thresholds were taken as the mean concentration values from the PTRL analysis of concentrations. Non-detect values in the analysis were represented by one-half the non-detect measurement. Since this process tends to over-represent the mean concentration, this approach is a conservative method of estimating contamination areas. An assumption in this approach is that measured data accurately reflect the true extent

of site contamination. It also assumes that concentrations below the threshold value do not contribute to contamination in the aquifer.

3) Estimation of vertical infiltration rate into the aquifer. The infiltration rate is controlled by the lesser of the percentage of precipitation that is assumed to infiltrate into the ground, or the vertical unsaturated hydraulic conductivity. An assumption is that infiltration is constant across the site and that aquifer materials are homogeneous and uniform.

4) Prediction of groundwater contaminant concentrations that result from partitioning of contaminants from the soil into the infiltrating water. This is a two-step calculation as follows:

Step A: Determination of the concentration of the contaminants in the infiltrating water as a result of partitioning from the soil using the following formula (USEPA, 1989):

$$C_p = C_s/K_d$$

where

C_p = concentration of the contaminant in the infiltrating water
C_s = concentration of the constituent in soil from PTRL data*
K_d = distribution coefficient for chemical of potential concern

K_d is calculated from $K_d = K_{oc}(F_{oc})$ where K_{oc} is the published organic carbon partitioning coefficient of the chemical (from Montgomery and Welkom, 1990) and F_{oc} is the organic carbon content of the soil (estimated to range from about 0.5% for the Sand/Fill unit to 1% for soils that are high in organic content, such as those in the Production Area which were subject to historic sewage discharges). The use of the above equation assumes that desorption is spacially homogeneous and uniform across the site.

Step B: Prediction of the concentrations of contaminants in groundwater (C_{gw}) after concentrations in infiltrating water (C_p) have reached the water table and have been diluted, using the following formula (Summers, et. al. 1980):

$$C_{gw} = \frac{(Q_p)(C_p)}{(Q_p + Q_{gw})}$$

where

*From PTRL letter dated March 31, 1995. The concentrations represent the lesser of the maximum detected concentration and the 95th percentile upper confidence limit of the mean concentration. Non-detect (ND) values were used in the data set as one half the non-detect value for the purposes of statistical analysis. If non-detect values are high relative to the maximum detected concentration, the 95th percentile upper confidence limit greatly overpredicts the mean and the maximum detected concentration is used.

$Q_p = q_z(A_p)$, the volumetric infiltration rate into the aquifer
$q_z = v_z(e)$, the Darcy flow in the downward direction
v_z = the groundwater seepage velocity
e = the void fraction
A_p = the plan areal extent of contaminated soil
C_p = concentration of contaminant in infiltrating water
Q_{gw} = the volumetric flow rate of groundwater from the specific area into the Pawtuxet River (see Attachment A)

This relationship implies that mixing in the groundwater is instantaneous and uniform across the section of the aquifer through which the contamination travels. While this may not well aproximate a transient system or one in which discharge is from a small portion of the aquifer, for the steady state system in this problem, where the entire cross-section of the aquifer discharges into the river, it is a valid assumption. Another assumption of this equation is that all of the water in the aquifer flows into the river. Since the Pawtuxet River is a local groundwater divide, this is a reasonable assumption.

5) Comparison of calculated groundwater contaminant concentrations to statistically determined concentration values from analysis of measured site groundwater data by PTRL.** To ensure a conservative approach to modeling the contamination contribution to the river, the maximum of these two values was selected as the value for succeeding calculations.

6) Prediction of the concentrations of the chemicals of potential concern when they enter the Pawtuxet River based on the following dilution calculation:

$$C_{sw} = \frac{(Q_{gw})(C_{gw})}{(Q_{gw} + Q_{sw})}$$

where

C_{sw} = the river concentration of the contaminant at the point of discharge from the groundwater aquifer into the Pawtuxet River
C_{gw} = the concentrations of contaminants in the groundwater
Q_{gw} = the volumetric flow rate of groundwater from the specific area into the Pawtuxet River (see Attachment A)
Q_{sw} = the flow rate of the Pawtuxet River

The equation assumes that contaminants reaching the river will become instaneously

**From PTRL letter dated March 31, 1995. The concentrations represent the lesser of the maximum detected concentration and the 95th percentile upper confidence limit of the mean concentration. Non-detect values were represented in the data set as one-half the non-detect value for the purposes of statistical analysis. If non-detect values are high relative to the maximum detected concentration, the 95th percentile upper confidence limit greatly overpredicts the mean and maximum detected concentration is used.

mixed throughout the river cross-section. While this may not be a good assumption for a point discharge, since the groundwater is discharging to the river all along the cross-section and along the reach, mixing will invariably result.

INPUT VALUES

The following values were selected for input in the Summers Model and dilution equation. Table 2 lists the input variables by specific area. A list of contaminant-specific input values and a summary of results is presented in Table 3 for the lower range of F_{oc} and Q_{sw}, representing a low soil organic carbon content and a low river flow rate, and in Table 4 for the upper range of F_{oc} and Q_{sw}, representing a higher soil organic carbon content and a higher river flow rate.

Table 2: Area-specific input values

	v_z ft/d (a)	e (b)	q_z ft/d	Q_{gs} ft3/d (c)	Q_{sw} ft3/d (d)	F_{oc} (e)
Production Area	2.83	0.3	0.85	15,000	1.12E7 to 3.3E7	0.005 to 0.01
Waste Water Treatment Area	2.83	0.3	0.85	15,000	1.12E7 to 3.3E7	0.005 to 0.01
Warwick Area	2.83	0.3	0.85	45,000	1.12E7 to 3.3E7	0.005 to 0.01

(a) Taken from the Phase I RCRA Facility Investigation (RFI) vertical hydraulic conductivity tests (Phase IA Report, November, 1991). The value is the mean result.
(b) Estimated based on the soil properties. This selection was consistent with summary of values in Freeze and Cherry, 1979.
(c) Determined from the aquifer test data and the calculations provided in Attachment A for the Production Area. The discharge for the Warwick and Wastewater Treatment Area was determined based on the relative size of the discharge area along the river relative to that of the Production Area.
(d) Based on the Phase II Pawtuxet River Proposal (January 1992). A range of flow rates was used for sensitivity analysis.

RESULTS OF THE CALCULATIONS

The results of the calculations are shown in Table 3 and Table 4. The results show that in the Production Area, the highest estimated surface water concentrations for the chemicals of potential concern are those of toluene and of 2-nitroaniline. In the Warwick area, the estimated surface water calculations are highest for chlorobenzene and for 2-nitroaniline. Concentrations in the Waste Water Treatment Area are generally lower than in the other areas.

This modeling approach provides a rough estimate of concentrations of the chemicals of potential concern that could possibly enter the Pawtuxet River. The accuracy of these results is dependant upon the assumptions made in estimating model input parameters and in selecting the simplified analytical model. In general, the approach represents a conservative model of soil to groundwater to surface water transport of chemical contaminantion in an unconfined aquifer system.

Please call if any additional information is required.

Sincerely,

Margaret Schwartz
Staff Engineer

Gordon Jamieson
Chief Hydrogeologist

cc: Dr. Barry Berdahl, CHMM, Ciba-Geigy Corporation
Mark Houlday, Project Manager, WCC
Bob Cebula, Assistant Project Scientist, WCC

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Table 3: Estimations of Surface Water Concentrations of Contaminants from Groundwater Discharges (Low Flow/Low Kd)
Former CIBA-GEIGY Facility, Cranston, Rhode Island

Chemicals of Potential Concern	Koc mL/g (1)	Kd mL/g (2)	Ap ft^2 (3)	Cs Subsurface Soil Concentration (ug/kg) (4)	Cp - Impact to Groundwater Concentration (ug/l) (5)	Predicted Contaminant Concentration in Groundwater (ug/L) (6)	PTRL Groundwater Concentration (ug/l) (7)	Cgw - Selected Groundwater Concentration (ug/L) (8)	Csw - Predicted Contaminant Concentration in Surface Water (ug/L) (9)
Production Area									
2-Nitroaniline	2.66E+01	0.13	36356	4200	3.16E+04	2.13E+04	ND	2.13E+04	2.83E+01
Chlorobenzene	1.15E+02	0.58	14984	280	4.87E+02	2.23E+02	2.56E+03	2.56E+03	3.41E+00
gamma-Chlordane	5.50E+05	2750.00	39940	70	2.55E-02	1.76E-02	2.90E-01	2.90E-01	3.87E-04
PCB 1248	4.36E+05	2180.00	84010	88000	4.04E+01	3.34E+01	ND	3.34E+01	4.45E-02
PCB 1254	4.07E+05	2035.00	94069	15000	7.37E+00	6.21E+00	ND	6.21E+00	8.28E-03
PCB 1260	2.63E+06	13150.00	3366	4800	3.65E-01	5.84E-02	ND	5.84E-02	7.79E-05
Toluene	1.32E+02	0.66	15287	62000	9.39E+04	4.36E+04	1.41E+04	4.36E+04	5.81E+01
Vinyl Chloride	2.45E+00	0.01	ND	ND	NA	NA	5.80E+01	5.80E+01	7.74E-02
Waste Water Treatment Area									
2,3,7,8-TCDF*	4.57E+06	22850.00	0	0.53	2.32E-05	NA	1.70E-05	1.70E-05	2.27E-08
bis(2-Ethylhexyl)phthalate	1.00E+05	500.00	1525	1400	2.80E+00	2.22E-01	1.90E+01	1.90E+01	2.53E-02
Dieldrin	2.07E+04	103.50	2441	130	1.26E+00	1.52E-01	2.00E-01	2.00E-01	2.67E-04
gamma-Chlordane	5.50E+05	2750.00	1040	2200	8.00E-01	4.45E-02	1.90E-01	1.90E-01	2.53E-04
PCB 1254	4.07E+05	2035.00	2436	210	1.03E-01	1.25E-02	ND	1.25E-02	1.67E-05
Tinuvin 327**	4.40E+04	220.00	1087	8200	3.73E+01	2.16E+00	ND	2.16E+00	2.88E-03
Warwick Area									
2-Nitroaniline	2.66E+01	0.13	47176	7000	5.26E+04	2.48E+04	ND	2.48E+04	9.89E+01
Aldrin	4.07E+02	2.04	4789	140	6.88E+01	5.70E+00	2.90E-02	5.70E+00	2.27E-02
Beryllium***	NA	NA	5856	830	NA	NA	3.50E+00	3.50E+00	1.40E-02
bis(2-Chloroethyl)ether	1.41E+01	0.07	4114	ND	NA	NA	2.00E+00	2.00E+00	7.98E-03
Chlorobenzene	1.15E+02	0.58	860	27000	4.70E+04	7.50E+02	4.49E+02	7.50E+02	2.99E+00
Dieldrin	2.07E+04	103.50	3608	110	1.06E+00	6.77E-02	ND	6.77E-02	2.70E-04
PCB 1248	4.36E+05	2180.00	3992	9700	4.45E+00	3.12E-01	ND	3.12E-01	1.24E-03
PCB 1254	4.07E+05	2035.00	5108	3300	1.62E+00	1.43E-01	ND	1.43E-01	5.69E-04

Notes:

1. Koc (organic carbon partitioning coefficient) values from Montgomery and Welkom, Groundwater Chemicals Desk Reference, Lewis Publishers, Chelsea, MI, 1990
2. Kd (the distribution coefficient) = Koc x Foc (organic carbon content of the soil estimated to range from 0.5 to 1%. 0.05% is the low Kd estimate.)
3. The area containing contaminated soil (Ap), was determined using the SitePlanner program with the existing database of soil analytical data. Non-detect values were represented by one-half the non-detect level.
4. 95% upper confidence limit of the mean combined soil concentration or the highest detected concentration (whichever is lower) - data from PTRL
5. Impact to groundwater concentration, Cp = Cs / Kd
6. Contaminant concentration in groundwater, Cgw = (Qp)(Cp)/(Qp + Qgw) (Summers, et. al., 1980) where:
7. 95% upper confidence limit of the mean groundwater concentration or the highest detected concentration (whichever is lower) - data from PTRL
8. The selected groundwater concentration is the higher of the Predicted Groundwater Concentration or the PTRL Mean Groundwater Concentration values
9. Csw = (Cgw)(Qgw)/(Qsw + Qgw)

Qsw = the flow rate of the Pawtuxet River which ranges from 130 to 382 cubic feet/second (The 130 cubic feet/second is the low flow value)

NA - not applicable

ND - not detected

* Koc for TCDD was used as an approximation for the Koc for TCDF as per PTRL; Not detected in Waste Wate Treatment Area groundwater.
As per PTRL, for modeling purposes it is assumed that TCDF is present in groundwater at one-half its sample quantitation limit (0.0034 ug/L), and that 2,3,7,8-TCDF comprises 10 percent of this concentration.

** Koc for Tinuvin 327 estimated by PTRL

*** Beryllium is an inorganic metal and therefore does not partition between soil and water in the same manner as do organic compounds. Beryllium measured groundwater concentration (at one location) was used as a representative groundwater concentration.

Table 4: Estimations of Surface Water Concentrations of Contaminants from Groundwater Discharges (High Flow/High Kd)
Former CIBA-GEIGY Facility, Cranston, Rhode Island

Chemicals of Potential Concern	Koc mL/g (1)	Kd mL/g (2)	Ap ft^2 (3)	Cs Subsurface Soil Concentration (ug/kg) (4)	Cp - Impact to Groundwater Concentration (ug/l) (5)	Predicted Contaminant Concentration in Groundwater (ug/L) (6)	PTRL Groundwater Concentration (ug/l) (7)	Cgw - Selected Groundwater Concentration (ug/L) (8)	Csw - Predicted Contaminant Concentration in Surface Water (ug/L) (9)
Production Area									
2-Nitroaniline	2.66E+01	0.27	36356	4200	1.58E+04	1.06E+04	ND	1.06E+04	4.83E+00
Chlorobenzene	1.15E+02	1.15	14984	280	2.43E+02	1.12E+02	2.56E+03	2.56E+03	1.16E+00
gamma-Chlordane	5.50E+05	5500.00	39940	70	1.27E-02	8.82E-03	2.90E-01	2.90E-01	1.32E-04
PCB 1248	4.36E+05	4360.00	84010	88000	2.02E+01	1.67E+01	ND	1.67E+01	7.58E-03
PCB 1254	4.07E+05	4070.00	94069	15000	3.69E+00	3.10E+00	ND	3.10E+00	1.41E-03
PCB 1260	2.63E+06	26300.00	3366	4800	1.83E-01	2.92E-02	ND	2.92E-02	1.33E-05
Toluene	1.32E+02	1.32	15287	62000	4.70E+04	2.18E+04	1.41E+04	2.18E+04	9.90E+00
Vinyl Chloride	2.45E+00	0.02	ND	ND	NA	NA	5.80E+01	5.80E+01	2.63E-02
Waste Water Treatment Area									
2,3,7,8-TCDF*	4.57E+06	45700.00	0	0.53	1.16E-05	NA	1.70E-05	1.70E-05	7.72E-09
bis(2-Ethylhexyl)phthalate	1.00E+05	1000.00	1525	1400	1.40E+00	1.11E-01	1.90E+01	1.90E+01	8.63E-03
Dieldrin	2.07E+04	207.00	2441	130	6.28E-01	7.62E-02	2.00E-01	2.00E-01	9.09E-05
gamma-Chlordane	5.50E+05	5500.00	1040	2200	4.00E-01	2.22E-02	1.90E-01	1.90E-01	8.63E-05
PCB 1254	4.07E+05	4070.00	2436	210	5.16E-02	6.25E-03	ND	6.25E-03	2.84E-06
Tinuvin 327**	4.40E+04	440.00	1087	8200	1.86E+01	1.08E+00	ND	1.08E+00	4.91E-04
Warwick Area									
2-Nitroaniline	2.66E+01	0.27	47176	7000	2.63E+04	1.24E+04	ND	1.24E+04	1.69E+01
Aldrin	4.07E+02	4.07	4789	140	3.44E+01	2.85E+00	2.90E-02	2.85E+00	3.88E-03
Beryllium***	NA	NA	5856	830	NA	NA	3.50E+00	3.50E+00	4.77E-03
bis(2-Chloroethyl)ether	1.41E+01	0.14	4114	ND	NA	NA	2.00E+00	2.00E+00	2.72E-03
Chlorobenzene	1.15E+02	1.15	860	27000	2.35E+04	3.75E+02	4.49E+02	4.49E+02	6.11E-01
Dieldrin	2.07E+04	207.00	3606	110	5.31E-01	3.38E-02	ND	3.38E-02	4.61E-05
PCB 1248	4.36E+05	4360.00	3992	9700	2.22E+00	1.56E-01	ND	1.56E-01	2.12E-04
PCB 1254	4.07E+05	4070.00	5108	3300	8.11E-01	7.13E-02	ND	7.13E-02	9.70E-05

Notes:

1. Koc (organic carbon partitioning coefficient) values from Montgomery and Welkom, Groundwater Chemicals Desk Reference, Lewis Publishers, Chelsea, MI, 1990
2. Kd (the distribution coefficient) = Koc x Foc (organic carbon content of the soil estimated to range from 0.5 to 1%. 0.05% is the low Kd estimate.)
3. The area containing contaminated soil (Ap), was determined using the SitePlanner program with the existing database of soil analytical data. Non-detect values were represented by one-half the non-detect level.
4. 95% upper confidence limit of the mean combined soil concentration or the highest detected concentration (whichever is lower) - data from PTRL
5. Impact to groundwater concentration, Cp = Cs / Kd
6. Contaminant concentration in groundwater, Cgw = (Qp)(Cp)/(Qp + Qgw) (Summers, et. al., 1980) where:
7. 95% upper confidence limit of the mean groundwater concentration or the highest detected concentration (whichever is lower) - data from PTRL
8. The selected groundwater concentration is the higher of the Predicted Groundwater Concentration or the PTRL Mean Groundwater Concentration values
9. Csw = (Cgw)(Qgw)/(Qsw + Qgw)

   Qsw = the flow rate of the Pawtuxet River which ranges from 130 to 382 cubic feet/second (The 130 cubic feet/second is the low flow value)

NA - not applicable

ND - not detected

* Koc for TCDD was used as an approximation for the Koc for TCDF as per PTRL; Not detected in Waste Wate Treatment Area groundwater.
As per PTRL, for modeling purposes it is assumed that TCDF is present in groundwater at one-half its sample quantitation limit (0.0034 ug/L), and that 2,3,7,8-TCDF comprises 10 percent of this concentration.

** Koc for Tinuvin 327 estimated by PTRL

*** Beryllium is an inorganic metal and therefore does not partition between soil and water in the same manner as do organic compounds. Beryllium measured groundwater concentration (at one location) was used as a representative groundwater concentration.

Freeze, R.A and J. A. Cherry, *Groundwater*, 1979, Prentice-Hall, Englewood Cliffs, NJ.

Montgomery and Welkom, *Groundwater Chemicals Desk Reference,* 1990, Lewis Publishers, Chelsea, MI.

Summers, K.S., Gherini and C. Chen, Tetra Tech Inc., *Methodology to evaluate the potential for groundwater contamination from geothermal fluid release*, EPA-600/7-80-117, 1980, as modified by EPA Region IV.

USEPA, *Determining soil response action levels based on potential contaminant migration to groundwater: a compendium of examples*, EPA/540/2-89/057, October 1989.

Estimate of Groundwater Flow Volume into the Pawtuxet River using Flow Net Analysis

MEMORANDUM

To: Ed Garland, Hydroqual

CC: Marc Houlday

From: Thomas R. Pisciotta

Date: April 20, 1995

Subject: Ciba-Geigy Site, Cranston, RI - Estimate of Flow Volume into the Pawtuxet River

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As you requested for the Pawtuxet River study at the Ciba -Geigy facility in Cranston, Rhode Island, we have developed an estimate of the volume of groundwater that flows into the river from the Production Area groundwater. This estimate is based on a flow net analysis. Flow nets, graphical representations of equipotential and groundwater flow lines, are the most common method for estimating groundwater discharge volumes under steady-state conditions.

A flow net analysis was used in the Phase I Interim Report (November 20,1991) to estimate the discharge rate of groundwater from the overburden aquifer in the Production Area to the Pawtuxet River. This estimate (350 ft^3/day or 2600 gallons/day) was based on inaccurate aquifer characteristic data. As such, the Phase I Interim Report discharge estimate is considered invalid. The data from the Stabilization Aquifer Testing Program which took place in the fall of 1992 is representative of the site conditions and is used in this analysis.

The stabilization aquifer testing program showed that there is considerable variability in the properties of the overburden aquifer in the Production Area of the Site. The Gravelly Sand and deeper Fine Sand units that are present in much of the eastern half of the Production Area had higher transmissivities than the Sand/Fill and deeper Fine Sand units that are present in the western half of the Production Area. Also, the test well in the eastern portion of the Production Area (RC-1) had a higher yield than the test well in the western portion of the Production Area (RC-2). Figure 2-2 (attached, from the Stabilization Investigation Report and Design Concepts Proposal, May 3, 1993) shows the locations of these and other wells and the approximate extent of the Gravelly Sand unit

Due to the differences in the properties of the overburden units in the Production Area, two flow net cross-sections were used to estimate the total discharge into the river. The flow nets are Cross-Sectional Flow Diagram A-A' (attached, Figure 4-4 of the Phase I Interim Report) and

Cross-Sectional Flow Diagram D-D' (attached, Figure 2-14 of the Stabilization Investigation Report and Design Concepts Proposal). Flow sectional diagram A-A' represents a discharge area that is is 220 feet wide at the river. The discharge area represented by flow sectional diagram D-D' is 250 feet wide at the river. These discharge areas are represented on Figure 2-2 (attached).

The following flow net analysis was used to calculate the discharge into the Pawtuxet River from the four different stratigraphic units in the two flow sections representing the Production Area:

$$Q = (mKH/n)(dm/ds)$$

where:

Q = flow net discharge rate
m = number of flow tubes
K = hydraulic conductivity
H = head drop across the region of flow
n = number of divisions of head in flow net
dm/ds = ratio of vertical to horizontal scales of flow net

From the stabilization aquifer tests, transmissivity and storativity data for each of the four units previously mentioned were generated. The transmissivities (T) values of each unit (as listed on Table 2-12 of the Stabilization Investigation Report and Design Concepts Proposal) were used to calculate the hydraulic conductivity (K) of the unit according to the following equation :

$$T = Kb$$

where b is the saturated thickness of the unit.

The results of the flow net analysis are as follows:

1) The Gravelly Sand unit in the Eastern Portion of the Production Area (in Section A-A'):

$T = 1.22\ ft^2/min$, $b = 7$ ft, therefore: $K = 017 ft/min = 245$ ft/day
$m = 1$
$n = 5$
$H = 5$ ft
$dm/ds = 10$ ft/100 ft $= 1/10$

$Q = (1\ [245\ ft/day]\ 5\ ft/5)(1/10) = 24.5\ ft^3/day/ft$ along river

Therefore, the groundwater flow from the Gravelly Sand unit of Section A-A' into the river is 24.5 $ft^3/day/ft$ along the river times 220 feet of river frontage = 5390 ft^3/day. Since the Gravelly Sand unit represents about 42% of the upper flow tube in Section A-A', the total flow from this unit is 42% of 5390 ft^3/day or **2246 ft^3/day.**

2) The Fine Sand unit in the Eastern Portion of the Production Area (in Section A-A'):

$T = 1.68\ ft^2/min$, $b = 45$ ft, therefore: $K = 0.04$ ft.min = 58 ft/day
$m = 3$
$n = 6$
$H = 6$ ft
$dm/ds = 10\ ft/100\ ft = 1/10$
$Q = (1\ [58\ ft/day]\ 6\ ft/6)(1/10) = 17.4\ ft^3/day/ft$ along river

Since the upper flow tube of Section A-A' is 42% Gravelly Sand which is accounted for in 1), the remaining 58% is Fine Sand. Subtracting 42% of the flow estimated from the upper flow tube reduces the total flow from the Fine Sand unit to 15.0 $ft^3/day/ft$ along the river.

Therefore, the groundwater flow from the Fine sand unit in Section A-A' into the river is 15.0 $ft^3/day/ft$ along the river times 220 feet of river frontage = **3300 ft^3/day.**

3) The Sand/Fill unit in the Western Portion of the Production Area (in Section D-D'):

$T = 0.91\ ft^2/min$, $b = 8$ ft, therefore: $K = 0.11$ ft/min = 158.40 ft/day
$m = 1$
$n = 2$
$H = 2$ ft
$dm/ds = 10\ ft/50\ ft = 1/5$
$Q = (1\ [158.4\ ft/day]\ 2\ ft/2)(1/5) = 31.68\ ft^3/day/ft$ along river

Therefore, the groundwater flow from the Sand/Fill unit of Section D-D' into the river is 31.68 $ft^3/day/ft$ along the river times 250 feet of river frontage = **7920 ft^3/day.**

4) The Fine Sand unit in the Western Portion of the Production Area (in Section D-D'):

$T = 0.27\ ft^2/min$, $b = 37$ ft, therefore: $K = 0.007$ ft/min $= 10.1$ ft/day
$m = 2$
$n = 2$
$H = 3$ ft
$dm/ds = 10$ ft/50 ft $= 1/5$
$Q = (2\ [10.1\ ft/day]\ 3\ ft/2)(1/5) = 6.06\ ft^3/day/ft$ along river

Therefore, the groundwater flow from the Fine Sand unit in Section D-D' into the river is 6.06 ft^3/day/ft along the river times 250 feet of river frontage = **1515 ft^3/day.**

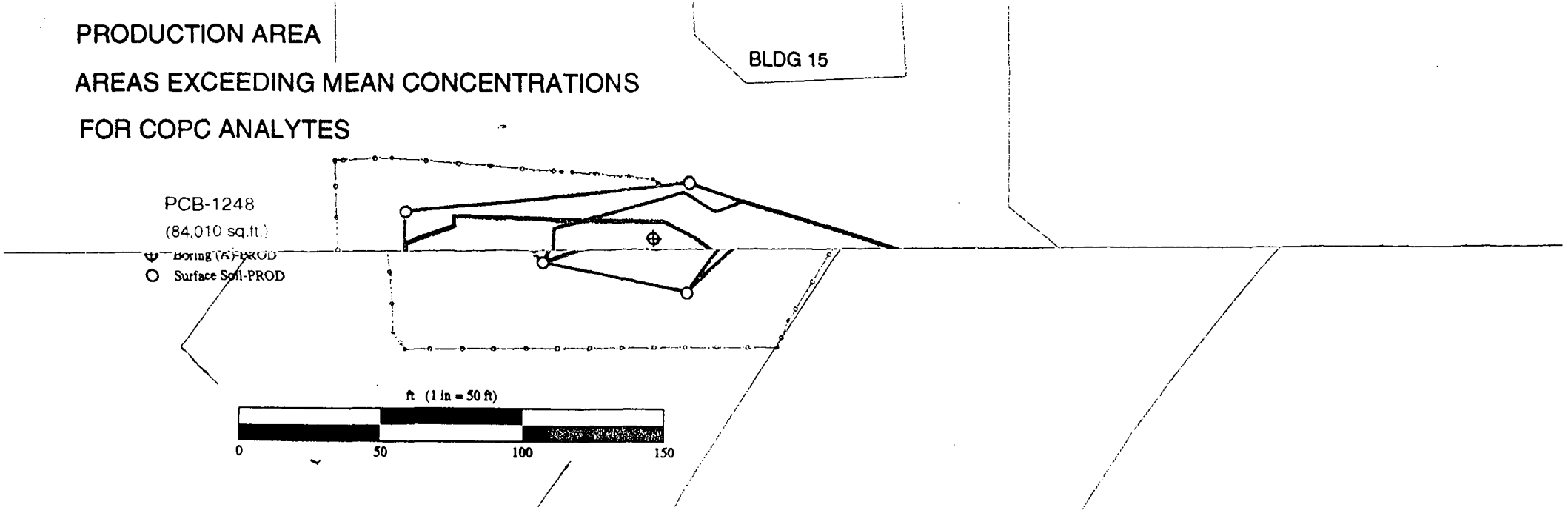
The total flow from the overburden groundwater of the Production Area into the Pawtuxet River is obtained by adding the discharges of each of the four units of the two sections. This total is 14,981 ft^3/day or 112,058 gallons/day (15,000 ft^3/day or 112,000 gallons/day rounded).

Figures Delineating Areal Extent of Contamination above Threshold Level

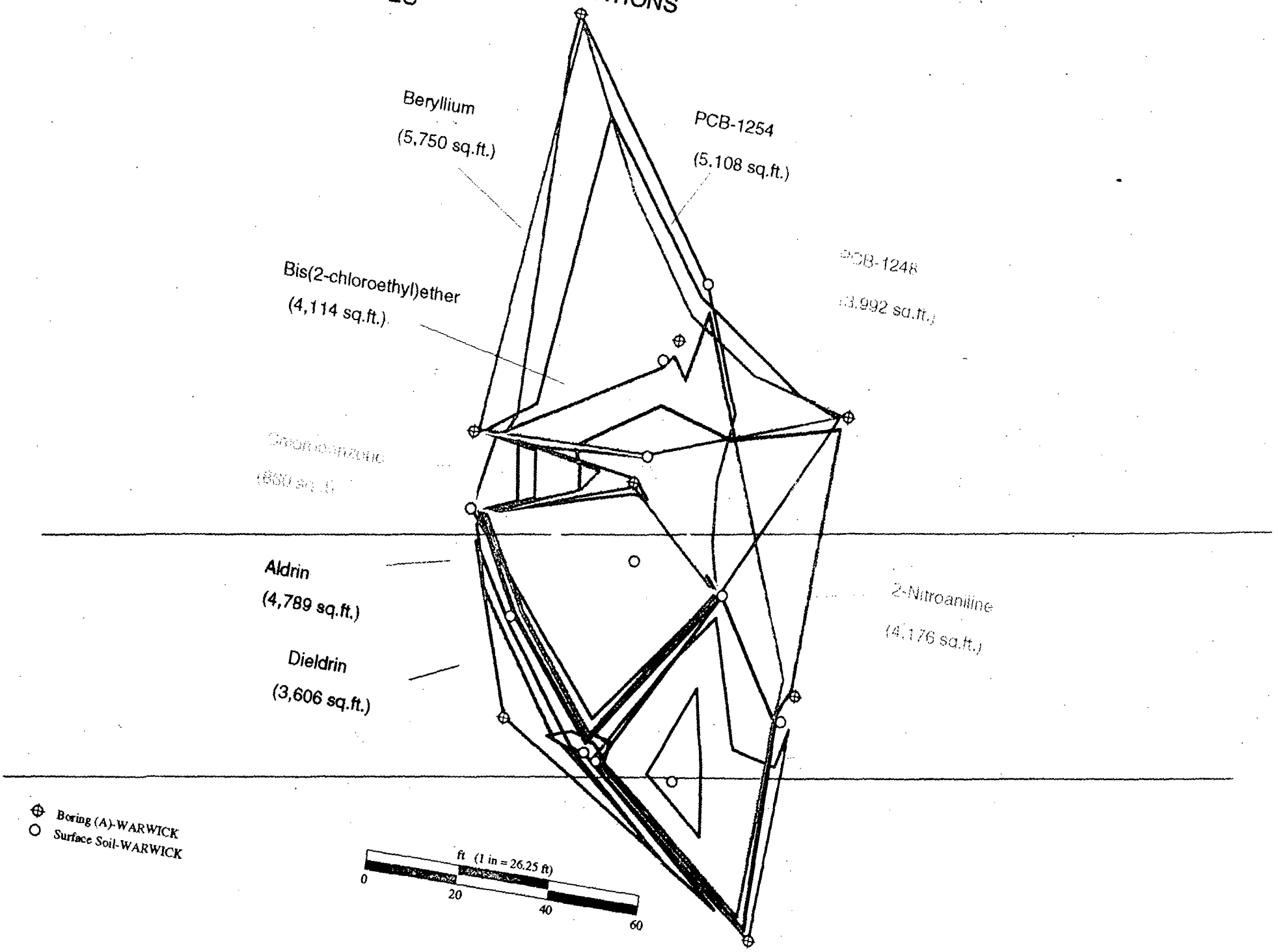
PRODUCTION AREA

AREAS EXCEEDING MEAN CONCENTRATIONS

FOR COPC ANALYTES



WARWICK AREA SWMU-5
AREAS EXCEEDING MEAN CONCENTRATIONS
FOR COPC ANALYTES

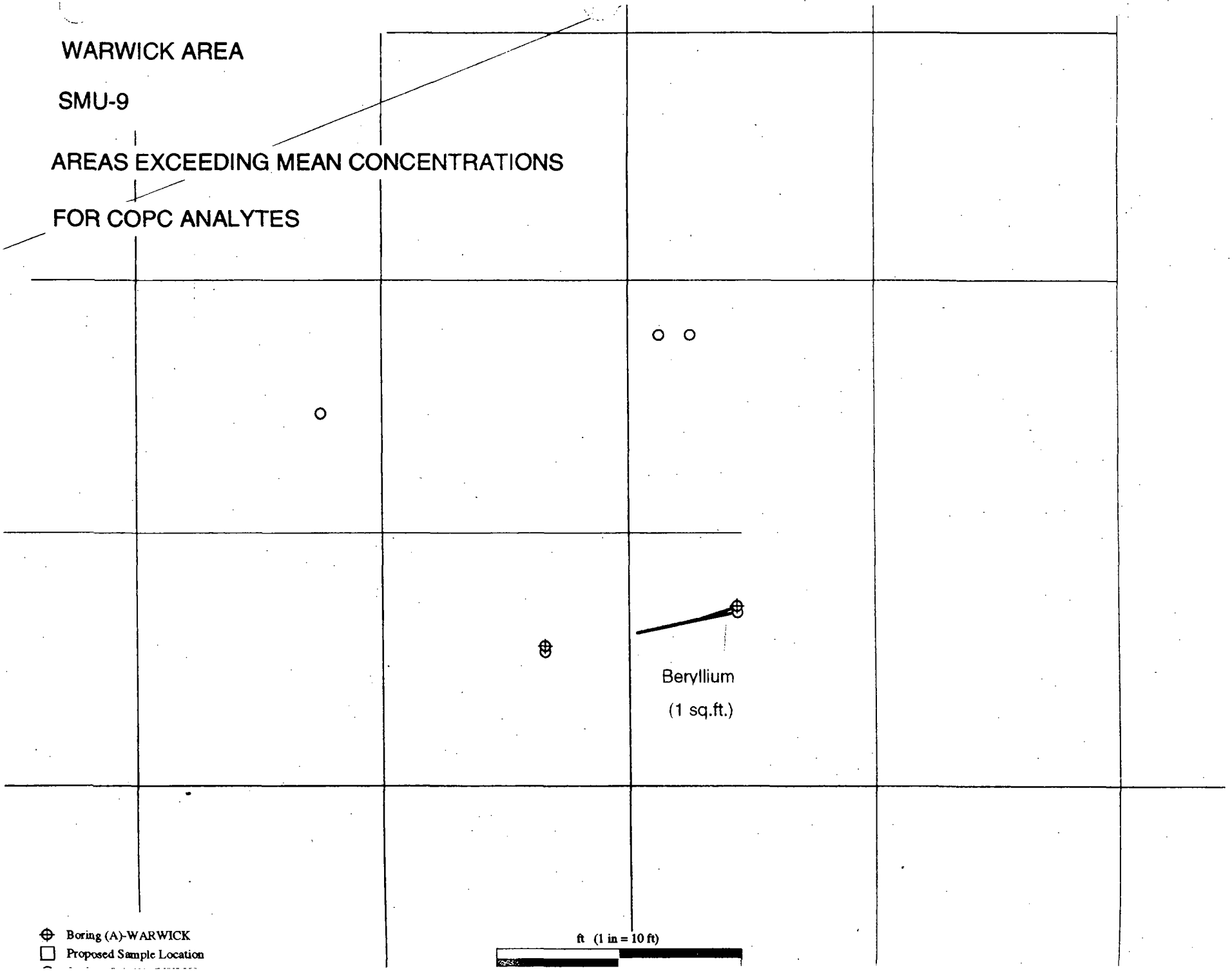


WARWICK AREA

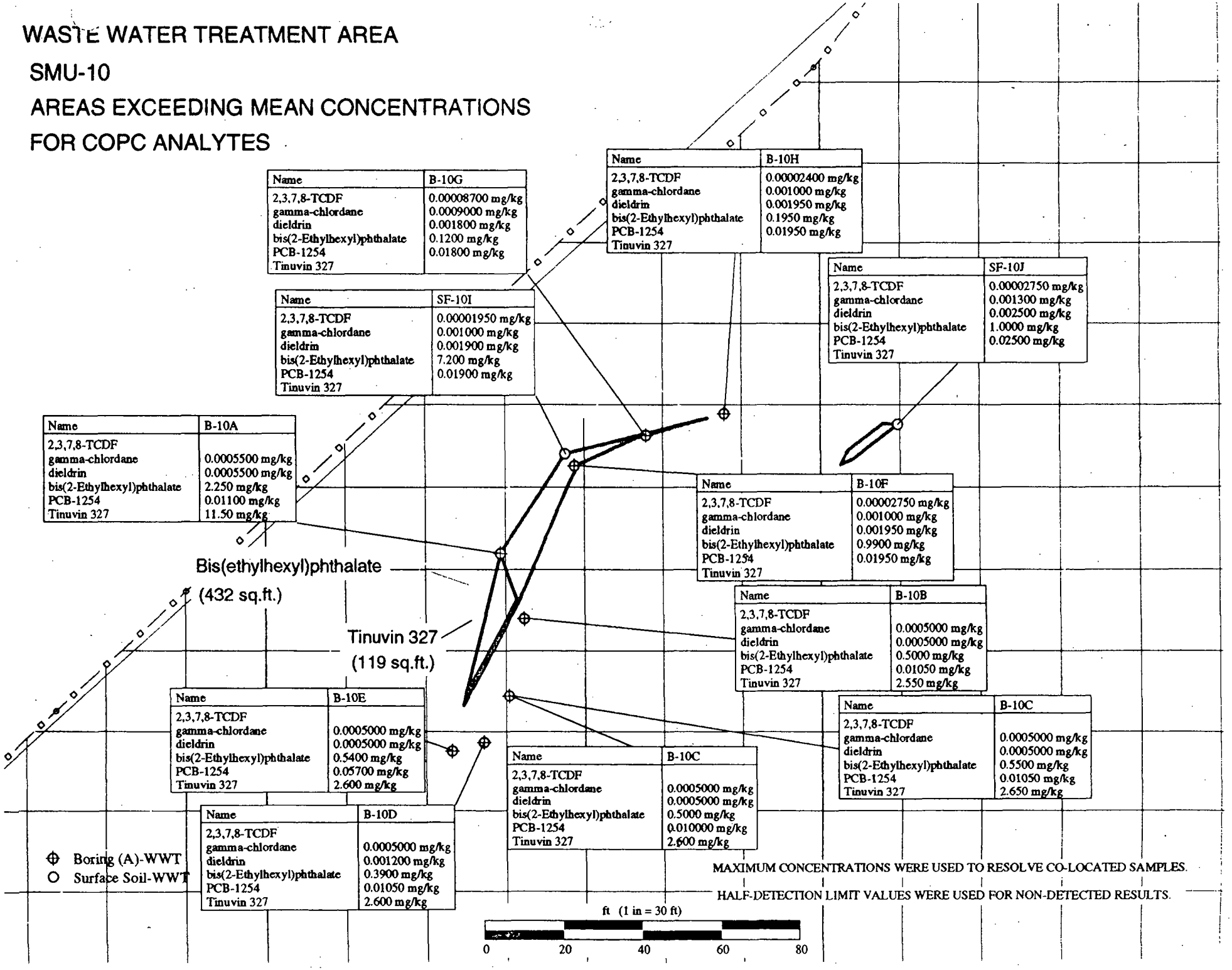
SMU-9

AREAS EXCEEDING MEAN CONCENTRATIONS

FOR COPC ANALYTES



WASTE WATER TREATMENT AREA
SMU-10
AREAS EXCEEDING MEAN CONCENTRATIONS FOR COPC ANALYTES



Name	B-10G
2,3,7,8-TCDF	0.00008700 mg/kg
gamma-chlordane	0.0009000 mg/kg
dieldrin	0.001800 mg/kg
bis(2-Ethylhexyl)phthalate	0.1200 mg/kg
PCB-1254	0.01800 mg/kg
Tinuvin 327	

Name	B-10H
2,3,7,8-TCDF	0.00002400 mg/kg
gamma-chlordane	0.001000 mg/kg
dieldrin	0.001950 mg/kg
bis(2-Ethylhexyl)phthalate	0.1950 mg/kg
PCB-1254	0.01950 mg/kg
Tinuvin 327	

Name	SF-10I
2,3,7,8-TCDF	0.00001950 mg/kg
gamma-chlordane	0.001000 mg/kg
dieldrin	0.001900 mg/kg
bis(2-Ethylhexyl)phthalate	7.200 mg/kg
PCB-1254	0.01900 mg/kg
Tinuvin 327	

Name	SF-10J
2,3,7,8-TCDF	0.00002750 mg/kg
gamma-chlordane	0.001300 mg/kg
dieldrin	0.002500 mg/kg
bis(2-Ethylhexyl)phthalate	1.0000 mg/kg
PCB-1254	0.02500 mg/kg
Tinuvin 327	

Name	B-10A
2,3,7,8-TCDF	
gamma-chlordane	0.0005500 mg/kg
dieldrin	0.0005500 mg/kg
bis(2-Ethylhexyl)phthalate	2.250 mg/kg
PCB-1254	0.01100 mg/kg
Tinuvin 327	11.50 mg/kg

Name	B-10F
2,3,7,8-TCDF	0.00002750 mg/kg
gamma-chlordane	0.001000 mg/kg
dieldrin	0.001950 mg/kg
bis(2-Ethylhexyl)phthalate	0.9900 mg/kg
PCB-1254	0.01950 mg/kg
Tinuvin 327	

Name	B-10B
2,3,7,8-TCDF	
gamma-chlordane	0.0005000 mg/kg
dieldrin	0.0005000 mg/kg
bis(2-Ethylhexyl)phthalate	0.5000 mg/kg
PCB-1254	0.01050 mg/kg
Tinuvin 327	2.550 mg/kg

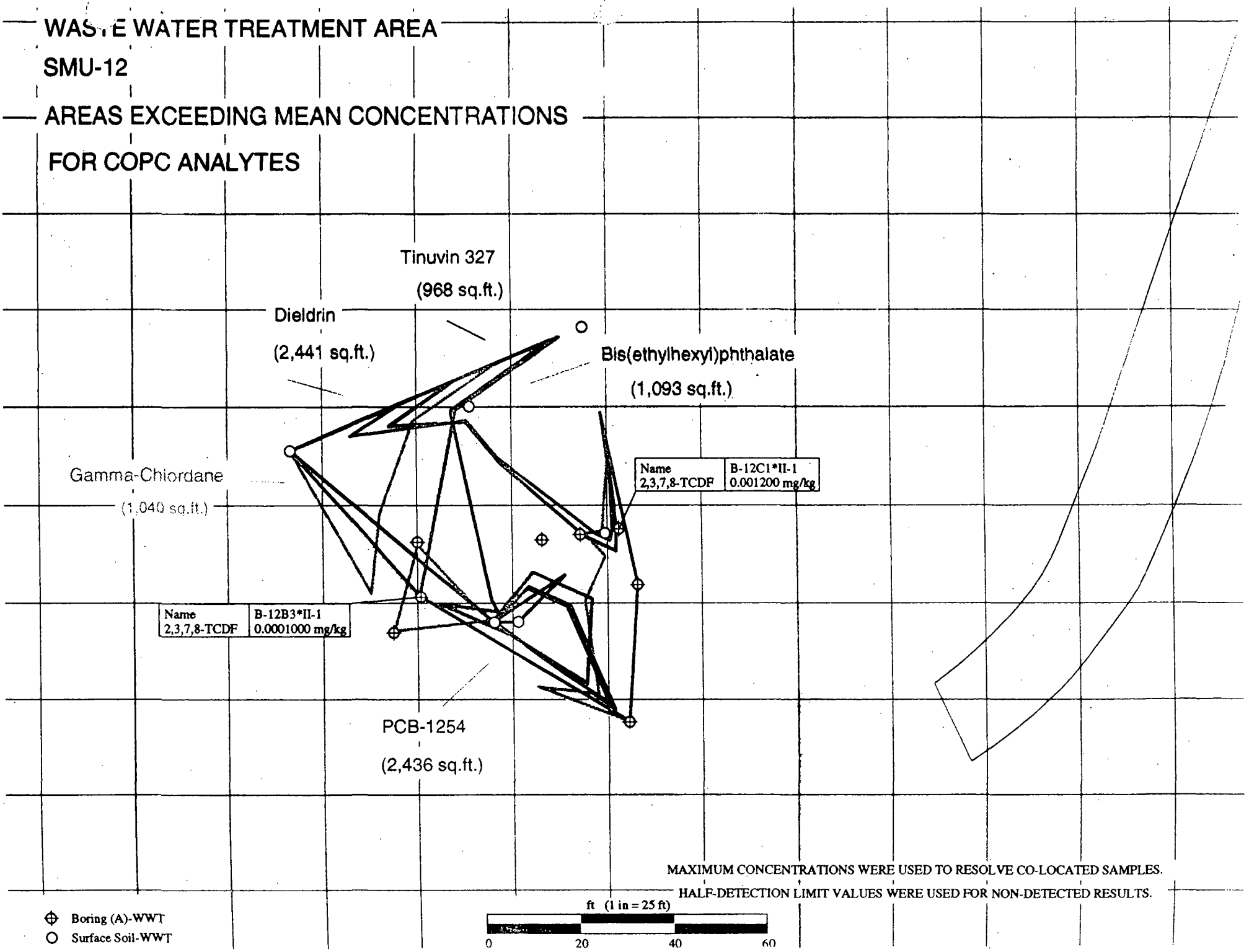
Name	B-10E
2,3,7,8-TCDF	
gamma-chlordane	0.0005000 mg/kg
dieldrin	0.0005000 mg/kg
bis(2-Ethylhexyl)phthalate	0.5400 mg/kg
PCB-1254	0.05700 mg/kg
Tinuvin 327	2.600 mg/kg

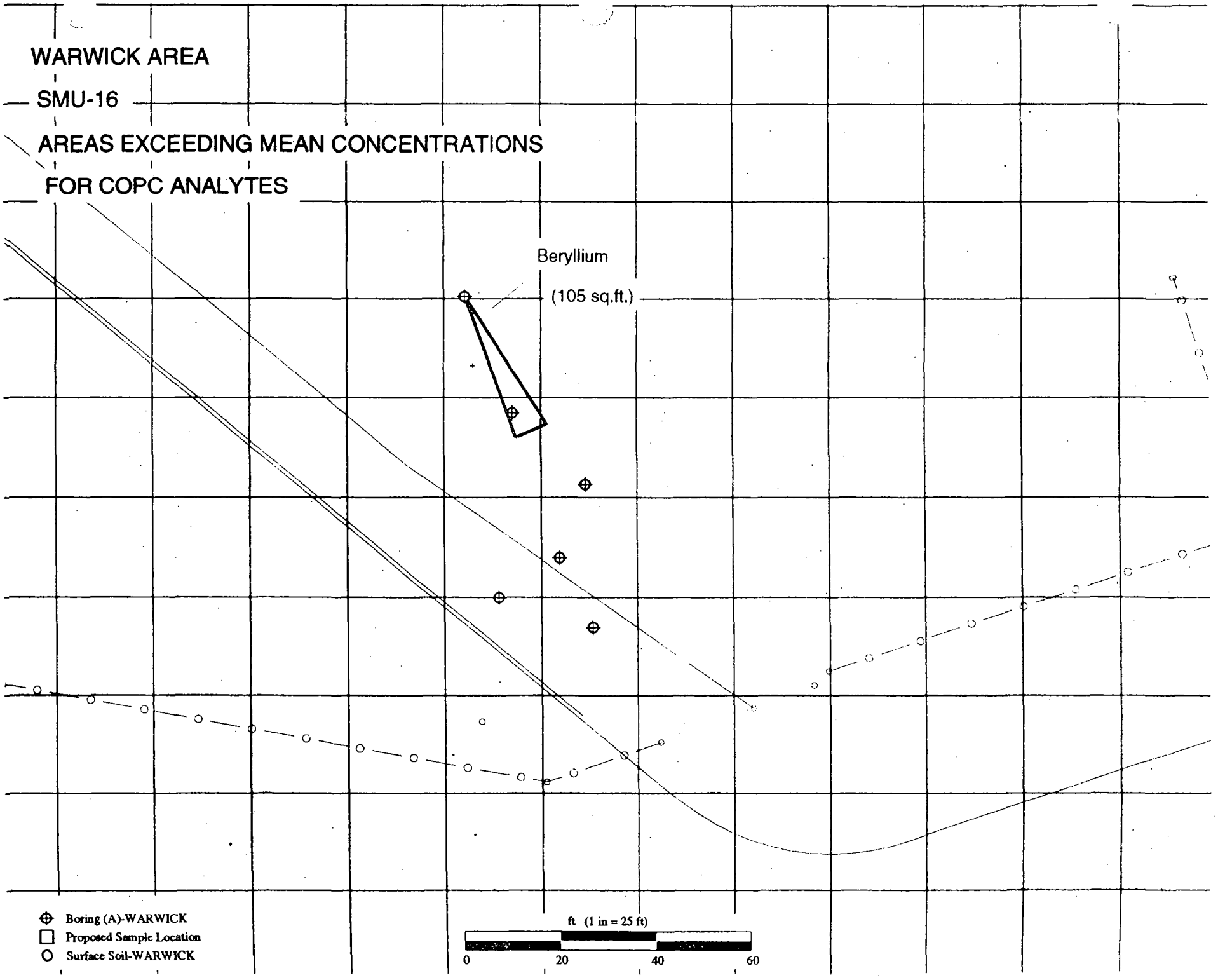
Name	B-10C
2,3,7,8-TCDF	
gamma-chlordane	0.0005000 mg/kg
dieldrin	0.0005000 mg/kg
bis(2-Ethylhexyl)phthalate	0.5500 mg/kg
PCB-1254	0.01050 mg/kg
Tinuvin 327	2.650 mg/kg

Name	B-10C
2,3,7,8-TCDF	
gamma-chlordane	0.0005000 mg/kg
dieldrin	0.0005000 mg/kg
bis(2-Ethylhexyl)phthalate	0.5000 mg/kg
PCB-1254	0.010000 mg/kg
Tinuvin 327	2.600 mg/kg

Name	B-10D
2,3,7,8-TCDF	
gamma-chlordane	0.0005000 mg/kg
dieldrin	0.001200 mg/kg
bis(2-Ethylhexyl)phthalate	0.3900 mg/kg
PCB-1254	0.01050 mg/kg
Tinuvin 327	2.600 mg/kg

WASTE WATER TREATMENT AREA
SMU-12
AREAS EXCEEDING MEAN CONCENTRATIONS FOR COPC ANALYTES




WARWICK AREA
SMU-16
AREAS EXCEEDING MEAN CONCENTRATIONS
FOR COPC ANALYTES
Beryllium
(105 sq.ft.)
Boring (A)-WARWICK
Proposed Sample Location
Surface Soil-WARWICK
ft (1 in = 25 ft)
0
20
40
60

Appendix 6-F

IRIS Toxicity Printouts

Aldrin

1 - IRIS
NAME - Aldrin
RN - 309-00-2
IRSN - 127
DATE - 930701
UPDT - 07/01/93. 3 fields
STAT - Oral RfD Assessment (RDO) on-line 03/01/88
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) on-line 07/01/93
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 09/30/87 CAR Carcinogenicity section added
IRH - 03/01/88 CARO Confidence statement revised
IRH - 12/01/88 CARO Corrected slope factor in text
IRH - 09/01/89 CAREV Ditraglia reference changed to Ditraglia et al.
IRH - 09/01/89 CAREV Deichmann reference changed to Deichmann et al.
IRH - 09/01/89 CARO Body weight for mice corrected to kg
IRH - 09/01/89 REFS Bibliography on-lineIRH - 01/01/91 CAR Text edited
IRH - 01/01/91 CARI Inhalation slope factor removed (global change)
IRH - 01/01/92 RDO Secondary contact changed
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 07/01/93 CARDR Secondary contact's phone number changed
RLEN - 22408
SY - Aldrex
SY - Aldrin
SY - Aldrite
SY - Aldrosol
SY - 1,4:5,8-Dimethanonaphthalene, 1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-
SY - Hexahydro-, (1 alpha, 4 alpha, 4a beta, 5 alpha, 8 alpha, 8a beta)-
SY - 1,4:5,8-Dimethanonaphthalene,
1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-Hexahydro-
SY - Drinox
SY - ENT 15,949
SY - 1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-Hexahydro-1,4,5,8-Dimethanonapht
halene
SY - 1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-Hexahydro-1,4-endo-exo-5,8-
SY - Dimethanonaphthalene
SY - 1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-Hexahydro-exo-1,4-endo-5,8-
SY - Dimethanonaphthalene
SY - Hexachlorohexahydro-endo-exo-Dimethanonaphthalene
SY - HHDN
SY - NCI-C00044
SY - Octalene
SY - Seedrin

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Liver toxicity	NOAEL: none	1000	1	3E-5
		mg/kg/day		
Rat Chronic Feeding	LOAEL: 0.5 ppm diet			

Study (0.025 mg/kg/day)

Fitzhugh et al., 1964

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*Conversion Factors: 1 ppm = 0.05 mg/kg/day (assumed rat food consumption)

---

o ORAL RFD STUDIES :

Fitzhugh, O.G., A.A. Nelson, and M.L. Quaife. 1964. Chronic oral toxicity of aldrin and dieldrin in rats and dogs. Food Cosmet. Toxicol. 2: 551-562.

Groups of 24 rats (12/sex) were fed aldrin in the diet at levels of 0, 0.5, 2, 10, 50, 100, or 150 ppm for 2 years. Liver lesions characteristic of chlorinated insecticide poisoning were observed at dose levels of 0.5 ppm and greater. These lesions were characterized by enlarged centrilobular hepatic cells, with increased cytoplasmic oxyphilia, and peripheral migration of basophilic granules. A statistically significant increase in liver-to-body weight ratio was observed at all dose levels. Kidney lesions occurred at the highest dose levels. Survival was markedly decreased at dose levels of 50 ppm and greater.

Additional data are fairly supportive. Effect and no-effect levels are similar (to those found for rats) for liver effects in dogs after 15 months' exposure to aldrin in the diet. Liver effects were observed at slightly higher doses in several other subchronic-to-chronic rat and dog studies. Short-term exposure to higher doses resulted in mortality for a number of species.

---

o ORAL RFD UNCERTAINTY :

UF – The composite UF of 1000 encompasses the uncertainty of extrapolation from animals to humans, the uncertainty in the range of human sensitivities, and an additional uncertainty because the RfD is based on a LOAEL rather than a NOAEL.

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o ORAL RFD MODIFYING FACTOR :

MF – None

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o ORAL RFD COMMENTS :

None.

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o ORAL RFD CONFIDENCE :

Study – Medium
Data Base – Medium
RfD – Medium

The principal study, designed as a carcinogenesis bioassay, is strong in histopathologic analysis but lacks other toxicologic parameters, and is therefore rated medium. The data base is fairly extensive, and generally supportive, but is rated medium because of the lack of NOELs for some studies.

Also, no chronic data exist for the dog, which may be a more sensitive species than the rat. Medium confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

U S EPA. 1982. Toxicity-Based Protective Ambient Water Levels for Various Carcinogens. Environmental Criteria and Assessment Office, Cincinnati, OH. ECAO-CIN-431. Internal review draft.

The RfD has been reviewed internally by ECAO-Cin.

---

o REVIEW DATES : 12/18/85
o VERIFICATION DATE : 12/18/85
o EPA CONTACTS :

Michael L. Dourson / OHEA – (513)569-7533

Moiz Mumtaz / OHEA – (513)569-7553

RDI - NO DATA
CAREV-
o CLASSIFICATION : B2; probable human carcinogen
o BASIS FOR CLASSIFICATION : Orally administered aldrin produced significant increases in tumor responses in three different strains of mice in both males and females. Tumor induction has been observed for structurally related chemicals, including dieldrin, a metabolite.
o HUMAN CARCINOGENICITY DATA :

Inadequate. Two studies of workers exposed to aldrin and dieldrin (a metabolite of aldrin) did not find these workers to have an excess risk of cancer. Both studies, however, were limited in their ability to detect an excess of deaths from cancer. Van Raalte (1977) observed two cases of cancer (gastric and lymphosarcoma) among 166 pesticide manufacturing workers exposed 4 to 19 years and followed from 15 to 20 years. Exposure was not quantified, and workers were also exposed to other organochlorine pesticides (endrin and telodrin). A small number of workers was studied, the mean age of the cohort (47.7 years) was low, the number of expected deaths was not calculated, and the duration of exposure and of latency was relatively short.

In a retrospective mortality study, Ditraglia et al. (1981) reported no increased incidence of deaths from cancer among 1155 organochlorine pesticide manufacturing workers (31 observed vs. 37.8 expected, SMR=82). This result was not statistically significant. Workers were employed for 6 or more months and followed for 13 or more years (24,939 person-years). Workers with no exposure (for example, office workers) were included in the cohort. Vital status was not known for 112 (10%) of the workers, and these workers were assumed to be alive; therefore, additional deaths may have occurred but were not observed. Exposure was not quantified and workers were also exposed to

other chemicals and pesticides (including endrin). An increased incidence of deaths from cancer was seen at several specific sites: esophagus (2 deaths observed, SMR=235), rectum (3, SMR=242), liver (2, SMR=225), and lymphatic and hematopoietic system (6, SMR=147); but these site-specific incidences were not statistically significant.

---

o ANIMAL CARCINOGENICITY DATA :

Sufficient. Davis and Fitzhugh (1962) fed a group of 215 male and female C3HeB/Fe mice a dietary mixture containing 10 ppm aldrin for up to 2 years. The control group consisted of 217 mice. The aldrin-treated mice died 2 months earlier than controls. Intercurrent disease, pneumonia, and intestinal parasitism may have influenced the long-term survival rate. A statistically significant increase of hepatomas was reported in the treated animals as compared with controls. An independent reevaluation of the liver lesions showed most of the hepatomas to be liver carcinomas (Epstein, 1975). In a follow-up study, Davis (1965) administered aldrin at 0 or 10 ppm in the diet to 100 male and 100 female C3H mice for 2 years. The incidence of hepatic hyperplasia and benign hepatomas in the aldrin group was approximately double that of controls, whereas the number of hepatic carcinomas was about the same.

Neither study provided a detailed pathologic examination or data separated by sex.

Aldrin (95% pure) was administered in the diet to 50 male and 50 female B6C3FI mice at TWA doses of 4 and 8 ppm or 3 and 6 ppm. Treatment was for 80 weeks, and animals were observed for an additional 10 to 13 weeks (NCI, 1978). In male mice, there was a significant dose-related increase in hepatocellular carcinomas when compared with matched or pooled controls.

Treon and Cleveland (1955) administered aldrin in the diet to 40 Carworth rats/sex at concentrations of 2.5, 12.5, or 25 ppm for a period of 2 years. Forty animals/sex served as controls. Mortality of the treated rats was greater than controls, with 50% surviving in the 2.5 and 12.5 ppm groups and 40% surviving in the 25 ppm group at the end of the experiment. Cleveland (1966) reported that no apparent treatment-related tumors were present in the above study. Deichmann et al. (1970) fed 50 male and 50 female Osborne-Mendel rats aldrin (95% pure) at final concentrations of 20, 30, or 50 ppm for 31 months. Controls consisted of 100 rats/sex. There was no evidence of carcinogenic response in male or female rats fed aldrin. The NCI (1978) fed 50 Osborne-Mendel rats/sex aldrin (95% pure) at 30 or 60 ppm. Male rats were treated 111 to 113 weeks and followed for 37 to 38 weeks of observation, and female rats were treated for 80 weeks and followed for 32 to 33 weeks of observation. Aldrin produced no significant effect on the mortality of the rats of either sex. The tumors observed awere randomly distributed, with no apparent relationship to aldrin treatment. Four additional bioassays observed no carcinogenic effect of aldrin in rats, but were considered inadequate for carcinogenicity assessment.

---

o SUPPORTING DATA :

Aldrin causes chromosomal aberrations in mouse, rat, and human cells (Georgian, 1974) and unscheduled DNA synthesis in rats (Probst et al., 1981) and humans (Rocchi et al., 1980) cells. Aldrin does not cause reverse

mutations in S. typhimurium, E. coli, or S. marcesans, or mitotic gene conversion in S. cerevisiae (Fahrig, 1974).

Five compounds structurally related to aldrin—dieldrin, chlordane, heptachlor, heptachlor epoxide, and chlorendic acid—have induced malignant liver tumors in mice. Chlorendic acid has also induced liver tumors in rats.

CARO -
o CLASSIFICATION : B2; probable human carcinogen
o BASIS FOR CLASSIFICATION : Orally administered aldrin produced significant increases in tumor responses in three different strains of mice in both males and females. Tumor induction has been observed for structurally related chemicals, including dieldrin, a metabolite.
o ORAL SLOPE FACTOR : 1.7E+1 per (mg/kg)/day
o DRINKING WATER UNIT RISK : 4.9E-4 per (ug/L)
o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk
o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	2E-1 ug/L
E-5 (1 in 100,000)	2E-2 ug/L
E-6 (1 in 1,000,000)	2E-3 ug/L

---

o ORAL DOSE-RESPONSE DATA :

Tumor Type – liver carcinoma
Test Animals – mouse/C3H (Davis); mouse/B6C3F1, male (NCI)
Route – diet
Reference – Davis, 1965 (see table); NCI, 1978

Administered Dose (ppm)	Human Equivalent Dose (mg/kg-day)	Tumor Incidence	Reference
females			
0	0	2/53	Davis, 1965
10	0.104	72/85	reevaluated
males			by Reuber
0	0	22/73	(cited in
10	0.104	75/91	Epstein, 1975)
0	0	3/20	NCI, 1978
4	0.04	16/49	
8	0.08	25/45	

---

o ADDITIONAL COMMENTS :

Body weights for mice were assumed to be 0.03 kg for purposes of dose conversion. The above data sets were used for calculation of the following slope factors: 2.3E+1 per (mg/kg)/day for female C3H mice, 1.8E+1 per (mg/kg)/day for male C3H mice, and 1.2E+1 per (mg/kg)/day for male B6C3F1 mice. No strain or sex specificity was noted in the studies, since aldrin treatment induced liver tumors in all mouse strains tested. A geometric mean of 1.7E+1 per (mg/kg)/day was thus chosen for the quantitative estimate, since all three slope factors were very similar.

The unit risk should not be used if the water concentration exceeds 20 ug/L, since above this concentration the unit risk may not be appropriate.

---

o DISCUSSION OF CONFIDENCE :

Adequate numbers of animals were treated for a large proportion of their lifetime. The route of treatment was appropriate. Slope factors calculated from three data sets from two independent assays were within a factor of 2. A slope factor for dieldrin, a major metabolite of aldrin, was determined to be 1.6E+1, essentially identical to that of aldrin.

CARI -

o CLASSIFICATION : B2; probable human carcinogen

o BASIS FOR CLASSIFICATION : Orally administered aldrin produced significant increases in tumor responses in three different strains of mice in both males and females. Tumor induction has been observed for structurally related chemicals, including dieldrin, a metabolite.

o INHALATION UNIT RISK : 4.9E-3 per (ug/cu.m)

o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk

o RISK/AIR CONCENTRATIONS :

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	2E-2 ug/cu.m
E-5 (1 in 100,000)	2E-3 ug/cu.m
E-6 (1 in 1,000,000)	2E-4 ug/cu.m

---

o INHALATION DOSE-RESPONSE DATA :

The unit risk was calculated from the oral data presented in CARO.

---

o ADDITIONAL COMMENTS :

The unit risk should not be used if the air concentration exceeds 2 ug/cu.m, since above this concentration the unit risk may not be appropriate.

---

o DISCUSSION OF CONFIDENCE :

See CARO.

CARDR-
o CARCINOGENICITY SOURCE :

Source Document – U.S. EPA, 1986

The values in the 1986 Carcinogenicity Assessment for Aldrin/Dieldrin have been reviewed by the Carcinogen Assessment Group.
DOCUMENT

---

o REVIEW DATES : 03/22/87
o VERIFICATION DATE : 03/22/87
o EPA CONTACTS :

Dharm V. Singh / OHEA – (202)260-5889

Jim Cogliano / OHEA – (202)260-3814

HAONE- NO DATA

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA - NO DATA

WQCHU-

Water and Fish Consumption – 7.4E-5 ug/L

Fish Consumption Only – 7.9E-5 ug/L

Considers technological or economic feasibility? – NO

Discussion – For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. However, zero may not be attainable at this time, so the recommended criteria

represents a E-6 estimated incremental increase of cancer risk over a lifetime.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 3.0E+0 ug/L
Chronic -- None

Marine:

Acute -- 1.3E+0 ug/L
Chronic -- None

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute tests on a variety of species. Requirements and methods are covered in the reference to the Federal Register.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

No data available

MCL -

No data available

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

Status -- Listed (Final, 1991)

Discussion -- "Unregulated" contaminants are those contaminants for which EPA establishes a monitoring requirement but which do not have an associated final MCLG, MCL, or treatment technique. EPA may regulate these contaminants in the future.

Monitoring requirement -- All systems to be monitored unless a vulnerability assessment determines the system is not vulnerable.

Analytical methodology -- Microextraction/gas chromatography (EPA 505); electron-
capture/gas chromatography (EPA 508); gas chromatographic/mass spectrometry (EPA 525).

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

SMCL - NO DATA

FISTD-

Status -- Issued (1986)

Reference -- Aldrin Pesticide Registration Standard. December, 1986 (NTIS No. PB-87-183778).

EPA Contact -- Registration Branch / OPP
(703)557-7760 / FTS 557-7760

FIREV-

Action -- Cancellations issued prior to RPAR/special review process (1974)

Considers technological or economic feasibility? -- YES

Summary of regulatory action -- All uses canceled except those in the following list: 1) subsurface ground insertion for termite control, 2) dipping of nonfood roots and tops, 3) moth-proofing by manufacturing processes in a closed system. Accelerated Decision of the Chief Administrative Law Judge (5/27/75) and the order Declining Review of the Accelerated Decision of the Administrative Law Judge issued by the Chief Judicial Officer (6/30/75); criterion of concern: carcinogenicity, bio-accumulation, wildlife hazard and other chronic effects.

Reference – 39 FR 37246 (10/18/74)

EPA Contact – Special Review Branch / OPP
(703)557-7400 / FTS 557-7400

CERC -

Value (status) – 1 pound (Final, 1989)

Considers technological or economic feasibility? – NO

Discussion – The RQ for aldrin is 1 pound, based on its aquatic toxicity and its potential carcinogenicity. The available data, as established under the CWA Section 311 (40 CFR 117.3), indicate the aquatic 96-hour Median Threshold Limit for aldrin is less than 0.1 ppm. This corresponds to an RQ of 1 pound. In addition, aldrin has been identified as a potential carcinogen and assigned a hazard ranking of high, based on a potency factor of 180.00/mg/kg/day and weight-of-evidence group B2, which also corresponds to an RQ of 1 pound.

Reference – 54 FR 33418 (08/14/89)

EPA Contact – RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

SARA - NO DATA

RCRA -

Status – Listed

Reference – 52 FR 25942 (07/09/87)

EPA Contact – RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - Fitzhugh, O.G., A.A. Nelson, and M.L. Quaife. 1964. Chronic oral toxicity of aldrin and dieldrin in rats and dogs. Food Cosmet. Toxicol. 2: 551-562.

OREF - U.S. EPA. 1982. Toxicity-Based Protective Ambient Water Levels for Various Carcinogens. Environmental Criteria and Assessment Office, Cincinnati, OH. ECAO-CIN-431. Internal review draft.
IREF - None
CREF - Cleveland, F.P. 1966. A summary of work on aldrin and dieldrin toxicity at the Kettering Laboratory. Arch. Environ. Health. 13: 195.
CREF - Davis, K.J. 1965. Pathology report on mice fed dieldrin, aldrin, heptachlor, or heptachlor epoxide for two years. Internal FDA memorandum to Dr. A.J. Lehrman, July 19.
CREF - Davis, K.J. and O.G. Fitzhugh. 1962. Tumorigenic potential of aldrin and dieldrin for mice. Toxicol. Appl. Pharmacol. 4: 187-189.
CREF - Deichmann, W.B., W.E. McDonald, E. Blum, et al. 1970. Tumorigenicity of aldrin, dieldrin and endrin in the albino rat. Ind. Med. 39(10): 426-434.
CREF - Ditraglia, D., D.P. Brown, T. Namekata and N. Iverson. 1981. Mortality study of workers employed at organochlorine pesticide manufacturing plants. Scand. J. Environ. Health. 7(suppl 4): 140-146.
CREF - Epstein, S.S. 1975. The carcinogenicity of dieldrin. Part 1. Sci. Total Environ. 4: 1-52.
CREF - Fahrig, R. 1974. Comparative mutagenicity with pesticides. IARC Publ. (U.N.) 10: 161-181.
CREF - Georgian, L. 1975. The comparative cytogenic effects of aldrin and phosphamidon. Mutat. Res. 31: 103-108.
CREF - NCI (National Cancer Institute). 1978. Bioassays of aldrin and dieldrin for possible carcinogenicity. DHEW Publication No. (NIH) 78-821. NCI Carcinogenesis Tech. Rep. Ser. No. 21. NCI-C6-TR-21.
CREF - Probst, G.S., R.E. McMahon, L.W. Hill, D.Z. Thompson, J.K. Epp and S.B. Neal. 1981. Chemically-induced unscheduled DNA synthesis in primary rat hepatocyte cultures: A comparison with bacterial mutagenicity using 218 chemicals. Environ. Mutagen. 3: 11-32.
CREF - Rocchi, P., P. Perocco, W. Alberghini, A. Fini and G. Prodi. 1980. Effect of pesticides on scheduled and unscheduled DNA synthesis of rat thymocytes and human lymphocytes. Arch. Toxicol. 45: 101-108.
CREF - Treon, J.F. and F.P. Cleveland. 1955. Toxicity of certain chlorinated hydrocarbon insecticides for laboratory animals, with special reference to aldrin and dieldrin. Agric. Food Chem. 3: 402-408.
CREF - U.S. EPA. 1986. Carcinogenicity Assessment of Aldrin and Dieldrin. Prepared by the Office of Health and Environmental Assessment, Carcinogen Assessment Group, Washington, DC, for the Hazard Evaluation Division, Office of Pesticides and Toxic Substances, Office of Pesticide Programs, Washington, DC.
CREF - Van Raalte, H.G.S. 1977. Human experience with dieldrin in perspective. Ecotoxicol. Environ. Safety. 1: 203-210.
HAREF- None

[IRIS] SS 5 /cf?
USER:

Antimony

2 - IRIS
NAME - Antimony
RN - 7440-36-0
IRSN - 5
DATE - 920120
UPDT - 01/20/92, 52 fields
STAT - Oral RfD Assessment (RDO) on-line 02/01/91
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) no data
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 08/01/89 RDO Gross et al. (1955) citation clarified
IRH - 08/01/89 REFS Bibliography on-line
IRH - 02/01/91 RDO Conversion factor revised
IRH - 02/01/91 RDO Text added
IRH - 02/01/91 RDO Paragraph 2 added
IRH - 02/01/91 OREF Dunn, 1928 and Monier-Williams, 1934 added
IRH - 01/01/92 RDO Primary contact changed
IRH - 01/01/92 EXSR Regulatory actions updated
RLEN - 13562
SY - Antimony
SY - ANTIMONY BLACK
SY - ANTIMONY POWDER
SY - ANTIMONY, REGULUS
SY - ANTYMON
SY - C.I. 77050
SY - STIBIUM
SY - UN 2871

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Longevity, blood glucose, and cholesterol	NOEL: none LOAEL: 0.35 mg/kg bw/day	1000	1	4E-4 mg/kg/day

Rat Chronic Oral Bioassay

Schroeder et al., 1970

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*Conversion Factors: 5 mg/L (5 ppm) given as 0.350 mg/kg/day in the discussion section of the critical study

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o ORAL RFD STUDIES :

Schroeder, H.A., M. Mitchner and A.P. Nasor. 1970. Zirconium, niobium, antimony, vanadium and lead in rats: Life term studies. J. Nutrition. 100: 59-66.

An experimental group of 50 male and 50 female rats was administered 5 ppm potassium antimony tartrate in water. Over the period of study, growth

rates of treated animals were not affected, but male rats survived 106 and females 107 fewer days than did controls at median lifespans. Nonfasting blood glucose levels were decreased in treated males, and cholesterol levels were altered in both sexes. Since there was only one level of antimony administered, a NOEL was not established in this study. A decrease in mean heart weight for the males was noted. No increase in tumors was seen as a result of treatment. Although not precisely stated, the concentration of 5 ppm antimony was expressed as an exposure of 0.35 mg/kg/day by the authors.

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o ORAL RFD UNCERTAINTY :

UF = 1000. An uncertainty factor of 1000 (10 for interspecies conversion, 10 to protect sensitive individuals, and 10 because the effect level was a LOAEL and no NOEL was established) was applied to the LOAEL of 0.35 mg/kg bw/day.

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o ORAL RFD MODIFYING FACTOR :

MF = 1

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o ORAL RFD COMMENTS :

In a similar study (Kanisawa and Schroeder, 1969), groups of CD-1 mice (54/sex) were given potassium antimony tartrate in drinking water at 0 or 5 mg/L (5 ppm) for 540 days (18 months). Lifespans were significantly reduced in both males and females, but the degree of antimony toxicity was less severe in mice than rats. Bradley and Fredrick (1941) and Browning (1969) reported disturbances in glucose and cholesterol metabolism in rats ingesting 5 mg/L antimony, but no signs of injury to the heart were observed in rats receiving doses up to 100 mg/kg/day. Substantially higher doses of antimony trioxide were tolerated by rats in studies by Sunagawa (1981) and Gross et al. (1955a,b), suggesting a NOAEL of 500 mg/kg, but these studies are of inadequate duration to assess adverse effects on toxicity.

Seventy people became acutely ill after drinking lemonade containing 0.013% antimony (Dunn, 1928 and Monier-Williams, 1934). The lemonade had been prepared and left overnight in buckets coated with an enamel containing 2.88% antimony trioxide. Fifty-six people were taken to the hospital with burning stomach pains, colic, nausea and vomiting. Most recovered within 3 hours, but in some cases recovery was not complete for several days. It is estimated that a person consuming 300 mL of lemonade would have received a dose of approximately 36 mg antimony, or approximately 0.5 mg/kg for a 70-kg adult.

According to U.S. EPA (1980), multimedia antimony exposures are essentially negligible by comparison to occupational exposures at which discrete clinical health effects have been observed. Myocardial effects are among the best-characterized human health effects associated with antimony exposure. Studies by Brieger et al. (1954) suggest an inhalation NOEL for myocardial damage to be approximately 0.5 mg/cu.m. This exposure is approximately equivalent to an oral reference dose of 0.003 mg/kg bw/day (i.e., 0.5 mg/cu.m x 10 cu.m/day x 0.5 / 1.0 x 5 days/7 days / 70 kg / 10). Parallel studies in rats and rabbits resulted in observation of EKG alterations following exposure to 3.1-5.6 mg/cu.m. There are, however, no adequate data on oral exposure to antimony which permit reasonable estimate of

no effect levels regarding heart damage.

One study (Belyaeva, 1967) indicated that women workers exposed in an antimony plant experienced a greater incidence of spontaneous abortions than did a control group of nonexposed working women. A high rate of premature deliveries among women workers in antimony smelting and processing was also observed (Aiello, 1955).

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o ORAL RFD CONFIDENCE :

Study: Low
Data Base: Low
RfD: Low

Confidence in the chosen study is rated as low because only one species was used, only one dose level was used, no NOEL was determined, and gross pathology and histopathology were not well described. Confidence in the data base is low due to lack of adequate oral exposure investigations. Low confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

U.S. EPA. 1980. Ambient Water Quality Criteria Document for Antimony. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards, Washington, DC. EPA-440/5-80-020. NTIS PB 81-117319.

The ADI in the 1980 Ambient Water Quality Criteria Document was extensively reviewed by the Agency and was reviewed by the public.

U.S. EPA. 1985. Health and Environmental Effects Profile for Antimony Oxides. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.

Limited peer review and extensive Agency-wide review, 1985.

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o REVIEW DATES : 11/06/85
o VERIFICATION DATE : 11/06/85
o EPA CONTACTS :

Harlal Choudhury / ORD -- (513)569-7553 / FTS 684-7553

Michael L. Dourson / ORD -- (513)569-7544 / FTS 684-7544

RDI - NO DATA
CAREV- NO DATA
CARO - NO DATA
CARI - NO DATA

CARDR- NO DATA

HAONE- NO DATA

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA - NO DATA

WQCHU-

Water and Fish Consumption: 1.46E+2 ug/L

Fish Consumption Only: 4.5E+4 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The WQC of 1.46E+2 ug/L is based on consumption of contaminated aquatic organisms and water. A WQC of 4.5E+4 ug/L has also been established based
on consumption of contaminated aquatic organisms alone.

Reference -- 45 FR 79315 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 8.8E+1 ug/L
Chronic -- 3.0E+1 ug/L

Marine:

Acute -- 1.5E+3 ug/L
Chronic -- 5.0E+2 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute and chronic tests on a variety of species. Requirements and methods are covered in the reference to the Federal Register. The proposed values are based on studies of antimony (III).

Reference -- 55 FR 19986 (05/14/89)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value -- 0.003 mg/L (Proposed, 1990)

Considers technological or economic feasibility? -- NO

Discussion -- EPA is proposing to regulate antimony based on its potential adverse effects (decreased longevity and altered blood cholesterol and glucose) reported in a lifetime oral exposure study in rats. The MCLG is based upon a DWEL of 0.015 mg/L and an assumed drinking water contribution of 20 percent.

Reference -- 55 FR 30370 (07/25/90)

EPA Contact -- Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value -- 0.01 mg/L (Proposed, 1990)

Considers technological or economic feasibility? -- YES

Discussion -- EPA proposes an MCL of 0.01 mg/L based upon a PQL of 10x the MDL. EPA also proposes as an alternative option an MCL of 0.005 based on a PQL of 5x the MDL.

Monitoring requirements -- Ground water systems every 3 years; surface water systems annually; will allow monitoring at up to 10-year intervals after the system completes 3 rounds of sampling at <50% of the MCL.

Analytical methodology -- Atomic absorption/furnace technique (EPA 204.2; SM 304); ICP mass spectrometry (EPA 200.8); hydride-atomic absorption spectrometry (ASTM D-3697): PQL= 0.01 / 0.005 mg/L.

Best available technology -- Coagulation/filtration; reverse osmosis.

Reference -- 55 FR 30370 (07/25/90)

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

Status -- Listed (Final, 1991)

Discussion -- "Unregulated" contaminants are those contaminants for which EPA establishes a monitoring requirement but which do not have an associated final MCLG, MCL, or treatment technique. EPA may regulate these contaminants in the future.

Monitoring requirement -- All systems to be monitored unless a vulnerability assessment determines the system is not vulnerable.

Analytical methodology -- Atomic absorption/furnace technique (EPA 502.2; SM 304); inductively coupled plasma (EPA 200.8).

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

SMCL - NO DATA

FISTD- NO DATA

FIREV- NO DATA

CERC -

Value (status) -- 5000 lbs (Final, 1986)

Considers technological or economic feasibility? -- NO

Discussion -- No data have been found to permit the ranking of this hazardous substance. The available data for acute hazards may lie above the upper limit for the 5000-pound RQ, but since it is a designated hazardous substance, the largest assignable RQ is 5000 pounds. This chemical is currently being assessed for chronic toxicity and is subject to change in future rulemaking.

Reference -- 51 FR 34534 (09/29/86); 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

SARA - NO DATA

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - Aiello, G. 1955. Pathology of antimony. Folia Med. (Naples). 38: 100. (Ital.)

OREF - Belyaeva, A.P. 1967. The effect of antimony on reproduction. Gig. Truda Prof. Zabol. 11: 32.

OREF - Bradley, W.R. and W.G. Frederick. 1941. The toxicity of antimony--animal studies. Ind. Med. 10 Ind. Hyg. Sec. 2: 15-22.

OREF - Brieger, H., C.W. Semisch, III, J. Stasney and D.A. Platnek. 1954. Industrial antimony poisoning. Ind. Med. Surg. 23: 521.

OREF - Browning, E. 1969. Antimony. In: Toxicity of Industrial Metals, 2nd ed. Appleton-Century-Craft, New York. p. 23-38.

OREF - Dunn, J.T. 1928. A curious case of antimony poisoning. Analyst. 53: 532-533.

OREF - Gross, P., J.H.V. Brown, M.L. Westrick, R.P.Srsic, N.L. Butler and T.F. Hatch. 1955a. A toxicological study of calcium halophosphate phospher and antimony trioxide. I. Acute and chronic toxicity and some. pharmacological aspects. Arch. Ind. Health. 11: 473-479.

OREF - Gross, P., M.L. Westrick, J.H.V. Brown, R.P. Srsic, H.H. Schrenk and T.F. Hatch. 1955b. Toxicologic study of calcium halophosphate phosphors and antimony trioxide. II. Pulmonary studies. Arch. Ind. Health. 11: 479-486.

OREF - Kanisawa, M. and H.A. Schroeder. 1969. Life term studies on the effect of trace elements on spontaneous tumor in mice and rats. Cancer Res. 29: 892-895.

OREF - Monier-Williams, G.W. 1934. Antimony in enamelled hollow-ware. Report on Public Health and Medical Subjects, No. 73, Ministry of Health, London. p. 18. (Cited in U.S. EPA, 1985)

OREF - Schroeder, H.A., M. Mitchner and A.P. Nasor. 1970. Zirconium, niobium, antimony, vanadium and lead in rats: Life term studies. J. Nutr.

100(1): 59-68.
OREF - Sunagawa, S. 1981. Experimental studies on antimony poisoning. Igaku Kenkyu. 51(3): 129-142. (Jap.) (CA 096/080942D)
OREF - U.S. EPA. 1980. Ambient Water Quality Criteria Document for Antimony. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Air Quality Planning and Standards, Washington, DC. EPA 440/5-80-020.
OREF - U.S. EPA. 1985. Health and Environmental Effects Profile for Antimony Oxides. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.
IREF - None
CREF - None
HAREF- None

Arsenic

1 - IRIS
NAME - Arsenic, inorganic
RN - 7440-38-2
IRSN - 272
DATE - 950703
UPDT - 07/03/95, 2 fields
STAT - Oral RfD Assessment (RDO) on-line 02/01/93
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) on-line 07/01/95
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 06/30/88 CARO Revised last paragraph
IRH - 06/30/88 CARI Inhalation slope factor changed
IRH - 06/30/88 CARI Paragraph 2 added
IRH - 09/07/88 CARO Major text changes
IRH - 12/01/88 CAREV Mabuchi et al. citation year corrected
IRH - 12/01/88 CAREV Pershagen et al. citation year corrected
IRH - 09/01/89 CARI Citations added to anacondor smelter
IRH - 09/01/89 REFS Bibliography on-line
IRH - 06/01/90 CAREV 2nd & 3rd paragraph - Text revised
IRH - 06/01/90 CAREV Text corrected
IRH - 06/01/90 CARI Inhalation slope factor removed (format change)
IRH - 06/01/90 RCRA EPA contact changed
IRH - 06/01/90 CREF References added
IRH - 12/01/90 CARO Changed slope factor to "unit risk", 2nd para, 1st sen
IRH - 02/01/91 CARI Text edited
IRH - 09/01/91 RDO Oral RfD summary now on-line
IRH - 09/01/91 RDO Oral RfD bibliography added
IRH - 10/01/91 RDO Conversion factor text clarified
IRH - 10/01/91 MCLG MCLG noted as pending change
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 08/01/92 CAR Note added to indicate text in oral quant. estimate
IRH - 10/01/92 CREF Missing reference added to bibliography
IRH - 02/01/93 RDO Citations added to second paragraph
IRH - 02/01/93 OREF References added to bibliography
IRH - 03/01/93 OREF Corrections to references
IRH - 03/01/94 CARDR Work group review date added
IRH - 06/01/94 CAR Carcinogen assessment noted as pending change
IRH - 01/01/95 CAR Pending change note revised
IRH - 01/01/95 CARO Dates and document no. added to oral quant. estimate
IRH - 06/01/95 CAR Carcinogenicity assessment replaced
IRH - 06/01/95 CREF Carcinogenicity references replaced
IRH - 07/01/95 CARDR Documentation year corrected; review statement revised
IRH - 07/01/95 CREF U.S. EPA, 1994 corrected to 1993
RLEN - 60157
SY - Arsenic
SY - Arsenic, inorganic
SY - gray-arsenic

RDO -
o ORAL RFD SUMMARY :

NOTE: There was not a clear consensus among Agency scientists on the oral RfD. Applying the Agency's RfD methodology, strong scientific arguments can

be made for various values within a factor of 2 or 3 of the currently recommended RfD value, i.e., 0.1 to 0.8 ug/kg/day. It should be noted, however, that the RfD methodology, by definition, yields a number with inherent uncertainty spanning perhaps an order of magnitude. New data that possibly impact on the recommended RfD for arsenic will be evaluated by the Work Group as it becomes available. Risk managers should recognize the considerable flexibility afforded them in formulating regulatory decisions when uncertainty and lack of clear consensus are taken into account.

Critical Effect	Experimental Doses*	UF	MF	RfD
Hyperpigmentation, keratosis and possible vascular complications	NOAEL: 0.009 mg/L converted to 0.0008 mg/kg-day	3	1	3E-4 mg/kg-day
Human chronic oral exposure	LOAEL: 0.17 mg/L converted to 0.014 mg/kg-day			
Tseng, 1977; Tseng et al., 1968				

*Conversion Factors: NOAEL was based on an arithmetic mean of 0.009 mg/L in a range of arsenic concentration of 0.001 to 0.017 mg/L. This NOAEL also included estimation of arsenic from food. Since experimental data were missing, arsenic concentrations in sweet potatoes and rice were estimated as 0.002 mg/day. Other assumptions included consumption of 4.5 L water/day and 55 kg bw (Abernathy et al., 1989). NOAEL = [(0.009 mg/L x 4.5 L/day) + 0.002 mg/day] / 55 kg = 0.0008 mg/kg-day. The LOAEL dose was estimated using the same assumptions as the NOAEL starting with an arithmetic mean water concentration from Tseng (1977) of 0.17 mg/L. LOAEL = [(0.17 mg/L x 4.5 L/day) + 0.002 mg/day] / 55 kg = 0.014 mg/kg-day.

o ORAL RFD STUDIES :

Tseng, W.P. 1977. Effects and dose-response relationships of skin cancer and blackfoot disease with arsenic. Environ. Health Perspect. 19: 109-119.

Tseng, W.P., H.M. Chu, S.W. How, J.M. Fong, C.S. Lin and S. Yeh. 1968. Prevalence of skin cancer in an endemic area of chronic arsenicism in Taiwan. J. Natl. Cancer Inst. 40: 453-463.

The data reported in Tseng (1977) show an increased incidence of blackfoot disease that increases with age and dose. Blackfoot disease is a significant adverse effect. The prevalences (males and females combined) at the low dose are 4.6 per 1000 for the 20-39 year group, 10.5 per 1000 for the 40-59 year group, and 20.3 per 1000 for the >60 year group. Moreover, the prevalence of blackfoot disease in each age group increases with increasing dose. However, a recent report indicates that it may not be strictly due to arsenic exposure (Lu, 1990). The data in Tseng et al. (1968) also show increased incidences of hyperpigmentation and keratosis with age. The overall prevalences of hyperpigmentation and keratosis in the exposed groups are 184 and 71 per 1000, respectively. The text states that the incidence increases with dose, but data for the individual doses are not shown. These data show that the skin

lesions are the more sensitive endpoint. The low dose in the Tseng (1977) study is considered a LOAEL.

The control group described in Tseng et al. (1968; Table 3) shows no evidence of skin lesions and presumably blackfoot disease, although this latter point is not explicitly stated. This group is considered a NOAEL.

The arithmetic mean of the arsenic concentration in the wells used by the individuals in the NOAEL group is 9 ug/L (range: 1-17 ug/L) (Abernathy et al., 1989). The arithmetic mean of the arsenic concentration in the wells used by the individuals in the LOAEL group is 170 ug/L (Tseng, 1977; Figure 4). Using estimates provided by Abernathy et al. (1989), the NOAEL and LOAEL doses for both food and water are as follows: LOAEL - [170 ug/L x 4.5 L/day + 2 ug/day (contribution of food)] x (1/55 kg) = 14 ug/kg/day; NOAEL - [9 ug/L x 4.5 L/day + 2 ug/day (contribution of food)] x (1/55 kg) = 0.8 ug/kg/day.

Although the control group contained 2552 individuals, only 957 (approximately 38%) were older than 20, and only 431 (approximately 17%) were older than 40. The incidence of skin lesions increases sharply in individuals above 20; the incidence of blackfoot disease increases sharply in individuals above 40 (Tseng, 1968; Figures 5, 6 and 7). This study is less powerful than it appears at first glance. However, it is certainly the most powerful study available on arsenic exposure to people.

This study shows an increase in skin lesions, 22% (64/296) at the high dose vs. 2.2% (7/318) at the low dose. The average arsenic concentration in the wells at the high dose is 410 ug/L and at the low dose is 5 ug/L (Cebrian et al., 1983; Figure 2 and Table 1) or 7 ug/L (cited in the abstract). The average water consumption is 3.5 L/day for males and 2.5 L/day for females. There were about an equal number of males and females in the study. For the dose estimates given below we therefore assume an average of 3 L/day. No data are given on the arsenic exposure from food or the body weight of the participants (we therefore assume 55 kg). The paper states that exposure times are directly related to chronological age in 75% of the cases. Approximately 35% of the participants in the study are more than 20 years old (Figure 1).

Exposure estimates (water only) are: high dose - 410 ug/L x 3 L/day x (1/55 kg) = 22 ug/kg/day; low dose - 5-7 ug/L x 3 L/day x (1/55 kg) = 0.3-0.4 ug/kg/day.

The high-dose group shows a clear increase in skin lesions and is therefore designated a LOAEL. There is some question whether the low dose is a NOAEL or a LOAEL since there is no way of knowing what the incidence of skin lesions would be in a group where the exposure to arsenic is zero. The 2.2% incidence of skin lesions in the low-dose group is higher than that reported in the Tseng et al. (1968) control group, but the dose is lower (0.4 vs. 0.8 ug/kg/day).

The Southwick et al. (1983) study shows a marginally increased incidence of a variety of skin lesions (palmar and plantar keratosis, diffuse palmar or plantar hyperkeratosis, diffuse pigmentation, and arterial insufficiency) in the individuals exposed to arsenic. The incidences are 2.9% (3/105) in the control group and 6.3% (9/144) in the exposed group. There is a slight, but

not statistically significant increase in the percent of exposed individuals that have abnormal nerve conduction (8/67 vs. 13/83, or 12% vs. 16% (Southwick et al., 1983; Table 8). The investigators excluded all individuals older than 47 from the nerve conduction portion of the study. These are the individuals most likely to have the longest exposure to arsenic.

Although neither the increased incidence of skin lesions nor the increase in abnormal nerve conduction is statistically significant, these effects may be biologically significant because the same abnormalities occur at higher doses in other studies. The number of subjects in this study was insufficient to establish statistical significance.

Table 3 (Southwick et al., 1983) shows the annual arsenic exposure from drinking water. No data are given on arsenic exposure from food or the body weight (assume 70 kg). Exposure times are not clearly defined, but are >5 years, and dose groups are ranges of exposure.

Exposure estimates (water only) are: dosed group - 152.4 mg/year x 1 year/365 days x (1/70) kg = 6 ug/kg/day; control group - 24.2 mg/year x year/365 days x (1/70) kg = 0.9 ug/kg/day.

Again because there are no data for a group not exposed to arsenic, there is some question if the control group is a NOAEL or a LOAEL. The incidence of skin lesions in this group is about the same as in the low-dose group from the Cebrian et al. (1983) study; the incidence of abnormal nerve conduction in the control group is higher than that from the low-dose group in the Hindmarsh et al. (1977) study described below. The control dose is comparable to the dose to the control group in the Tseng et al. (1968) and Hindmarsh et al. (1977) studies. The dosed group may or may not be a LOAEL, since it is does not report statisically significant effects when compared to the control.

This study shows an increased incidence of abnormal clinical findings and abnormal electromyographic findings with increasing dose of arsenic (Hindmarsh et al., 1977; Tables III and VI). However, the sample size is extremely small. Percentages of abnormal clinical signs possibly attributed to As were 10, 16, and 40% at the low, mid and high doses, respectively. Abnormal EMG were 0, 17 and 53% in the same three groups.

The exact doses are not given in the Hindmarsh et al. (1977) paper; however, some well data are reported in Table V. The arithmetic mean of the arsenic concentration in the high-dose and mid-dose wells is 680 and 70 ug/L, respectively. Figure 1 (Hindmarsh et al., 1977) shows that the average arsenic concentration of the low-dose wells is about 25 ug/L. No data are given on arsenic exposure from food. We assume daily water consumption of 2 liters and body weight of 70 kg. Exposure times are not clearly stated.

Exposure estimates (water only) are: low - 25 ug/L x 2 L/day x (1/70) kg = 0.7 ug/kg/day; mid - 70 ug/L x 2 L/day x (1/70) kg = 2 ug/kg/day; high - 680 ug/L x 2 L/day x (1/70) kg = 19 ug/kg/day.

The low dose is a no-effect level for abnormal EMG findings. However, because there is no information on the background incidence of abnormal clinical findings in a population with zero exposure to arsenic, there is no way of knowing if the low dose is a no-effect level or another marginal effect level

for abnormal clinical findings. The low dose is comparable to the dose received by the control group in the Tseng (1977) and Southwick et al. (1983) studies.

The responses at the mid dose do not show a statistically significant increase but are part of a statistically significant trend and are biologically significant. This dose is an equivocal NOAEL/LOAEL. The high dose is a clear LOAEL for both responses.

As discussed previously there is no way of knowing whether the low doses in the Cebrian et al. (1983), Southwick et al. (1983) and Hindmarsh et al. (1977) studies are NOAELs for skin lesions and/or abnormal nerve conduction. However, because the next higher dose in the Southwick and Hindmarsh studies only shows marginal effects at doses 3-7 times higher, the Agency feels comfortable in assigning the low doses in these studies as NOAELs.

The Tseng (1977) and Tseng et al. (1968) studies are therefore considered superior for the purposes of developing an RfD and show a NOAEL for a sensitive endpoint. Even discounting the people <20 years of age, the control group consisted of 957 people that had a lengthy exposure to arsenic with no evidence of skin lesions.

The following is a summary of the defined doses in mg/kg-day from the principal and supporting studies:

1) Tseng (1977): NOAEL = 8E-4; LOAEL = 1.4E-2

2) Cebrian et al. (1983): NOAEL = 4E-4; LOAEL = 2.2E-2

3) Southwick et al. (1983): NOAEL = 9E-4; LOAEL = none (equivocal effects at 6E-3)

4) Hindmarsh et al., 1977: NOAEL = 7E-4; LOAEL = 1.9E-2 (equivocal effects at 2E-3)

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o ORAL RFD UNCERTAINTY :

UF -- The UF of 3 is to account for both the lack of data to preclude reproductive toxicity as a critical effect and to account for some uncertainty in whether the NOAEL of the critical study accounts for all sensitive individuals.

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o ORAL RFD MODIFYING FACTOR :

MF -- None

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o ORAL RFD COMMENTS :

Ferm and Carpenter (1968) produced malformations in 15-day hamster fetuses via intravenous injections of sodium arsenate into pregnant dams on day 8 of gestation at dose levels of 15, 17.5, or 20 mg/kg bw. Exencephaly, encephaloceles, skeletal defects and genitourinary systems defects were produced. These and other terata were produced in mice and rats all at levels around 20 mg/kg bw. Minimal effects or no effects on fetal development have

been observed in studies on chronic oral exposure of pregnant rats or mice to relatively low levels of arsenic via drinking water (Schroeder and Mitchner, 1971). Nadeenko et al. (1978) reported that intubation of rats with arsenic solution at a dose level of 25 ug/kg/day for a period of 7 months, including pregnancy, produced no significant embryotoxic effects and only infrequent slight expansion of ventricles of the cerebrum, renal pelves and urinary bladder. Hood et al. (1977) reported that very high single oral doses of arsenate solutions (120 mg/kg) to pregnant mice were necessary to cause prenatal fetal toxicity, while multiple doses of 60 mg/kg on 3 days had little effect.

Extensive human pharmacokinetic, metabolic, enzymic and long-term information is known about arsenic and its metabolism. Valentine et al. (1987) established that human blood arsenic levels did not increase until daily water ingestion of arsenic exceeded approximately 250 ug/day (approximately 120 ug of arsenic/L. Methylated species of arsenic are successively 1 order of magnitude less toxic and less teratogenic (Marcus and Rispin, 1988). Some evidence suggests that inorganic arsenic is an essential nutrient in goats, chicks, minipigs and rats (NRC, 1989). No comparable data are available for humans.

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o ORAL RFD CONFIDENCE :

Study -- Medium
Data Base -- Medium
RfD -- Medium

Confidence in the chosen study is considered medium. An extremely large number of people were included in the assessment (>40,000) but the doses were not well-characterized and other contaminants were present. The supporting human toxicity data base is extensive but somewhat flawed. Problems exist with all of the epidemiological studies. For example, the Tseng studies do not look at potential exposure from food or other source. A similar criticism can be made of the Cebrian et al. (1983) study. The U.S. studies are too small in number to resolve several issues. However, the data base does support the choice of NOAEL. It garners medium confidence. Medium confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

Source Document -- This assessment is not presented in any existing U.S. EPA document.

This analysis has been reviewed by EPA's Risk Assessment Council on 11/15/90. This assessment was discussed by the Risk Assessment Council of EPA on 11/15/90 and verified through a series of meetings during the 1st, 2nd and 3rd quarters of FY91.

Other EPA Documentation -- U.S. EPA, 1984, 1988

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o REVIEW DATES : 03/24/88, 05/25/88, 03/21/89, 09/19/89, 08/22/90, 09/20/90
o VERIFICATION DATE : 11/15/90
o EPA CONTACTS :

Charles Abernathy / OST -- (202)260-5374

Michael Dourson / OHEA -- (513)569-7533

RDI - NO DATA
CAREV-
o CLASSIFICATION : A; human carcinogen
o BASIS FOR CLASSIFICATION : based on sufficient evidence from human data. An increased lung cancer mortality was observed in multiple human populations exposed primarily through inhalation. Also, increased mortality from multiple internal organ cancers (liver, kidney, lung, and bladder) and an increased incidence of skin cancer were observed in populations consuming drinking water high in inorganic arsenic.
o HUMAN CARCINOGENICITY DATA :

Sufficient. Studies of smelter worker populations (Tacoma, WA; Magma, UT; Anaconda, MT; Ronnskar, Sweden; Saganoseki-Machii, Japan) have all found an association between occupational arsenic exposure and lung cancer mortality (Enterline and Marsh, 1982; Lee-Feldstein, 1983; Axelson et al., 1978; Tokudome and Kuratsune, 1976; Rencher et al., 1977). Both proportionate mortality and cohort studies of pesticide manufacturing workers have shown an excess of lung cancer deaths among exposed persons (Ott et al., 1974; Mabuchi et al., 1979). One study of a population residing near a pesticide manufacturing plant revealed that these residents were also at an excess risk of lung cancer (Matanoski et al., 1981). Case reports of arsenical pesticide applicators have also corroborated an association between arsenic exposure and lung cancer (Roth, 1958).

A cross-sectional study of 40,000 Taiwanese exposed to arsenic in drinking water found significant excess skin cancer prevalence by comparison to 7500 residents of Taiwan and Matsu who consumed relatively arsenic-free water (Tseng et al., 1968; Tseng, 1977). Although this study demonstrated an association between arsenic exposure and development of skin cancer, it has several weaknesses and uncertainties, including poor nutritional status of the exposed populations, their genetic susceptibility, and their exposure to inorganic arsenic from non-water sources, that limit the study's usefulness in risk estimation. Dietary inorganic arsenic was not considered nor was the potential confounding by contaminants other than arsenic in drinking water. There may have been bias of examiners in the original study since no skin cancer or preneoplastic lesions were seen in 7500 controls; prevalence rates rather than mortality rates are the endpoint; and furthermore there is concern of the applicability of extrapolating data from Taiwanese to the U.S. population because of different background rates of cancer, possibly genetically determined, and differences in diet other than arsenic (e.g., low protein and fat and high carbohydrate) (U.S. EPA, 1988).

A prevalence study of skin lesions was conducted in two towns in Mexico,

one with 296 persons exposed to drinking water with 0.4 mg/L arsenic and a similar group with exposure at 0.005 mg/L. The more exposed group had an increased incidence of palmar keratosis, skin hyperpigmentation and hypopigmentation, and four skin cancers (histologically unconfirmed) (Cebrian et al. (1983). The association between skin cancer and arsenic is weak because of the small number of cases, small cohort size, and short duration follow-up; also there was no unexposed group in either town. No excess skin cancer incidence has been observed in U.S. residents consuming relatively high levels of arsenic in drinking water but the numbers of exposed persons were low (Morton et al., 1976; Southwick et al., 1981). Therapeutic use of Fowler's solution (potassium arsenite) has also been associated with development of skin cancer and hyperkeratosis (Sommers and McManus, 1953; Fierz, 1965); several case reports implicate exposure to Fowler's solution in skin cancer development (U.S. EPA, 1988).

Several follow-up studies of the Taiwanese population exposed to inorganic arsenic in drinking water showed an increase in fatal internal organ cancers as well as an increase in skin cancer. Chen et al. (1985) found that the standard mortality ratios (SMR) and cumulative mortality rates for cancers of the bladder, kidney, skin, lung and liver were significantly greater in the Blackfoot disease endemic area of Taiwan when compared with the age adjusted rates for the general population of Taiwan. Blackfoot disease (BFD, an endemic peripheral artery disease) and these cancers were all associated with high levels of arsenic in drinking water. In the endemic area, SMRs were greater in villages that used only artesian well water (high in arsenic) compared with villages that partially or completely used surface well water (low in arsenic). However, dose-response data were not developed (Chen et al. 1985).

A retrospective case-control study showed a significant association between duration of consuming high-arsenic well water and cancers of the liver, lung and bladder (Chen et al., 1986). In this study, cancer deaths in the Blackfoot disease endemic area between January 1980 and December 1982 were chosen for the case group. About 90% of the 86 lung cancers and 95 bladder cancers in the registry were histologically or cytologically confirmed and over 70% of the liver cancers were confirmed by biopsy or -fetoprotein presence with a positive liver x-ray image. Only confirmed cancer cases were included in the study. A control group of 400 persons living in the same area was frequency-matched with cases by age and sex. Standardized questionnaires of the cases (by proxy) and controls determined the history of artesian well water use, socioeconomic variables, disease history, dietary habits, and lifestyle. For the cancer cases, the age-sex adjusted odds ratios were increased for bladder (3.90), lung (3.39), and liver (2.67) cancer for persons who had used artesian well water for 40 or more years when compared with controls who had never used artesian well water. Similarly, in a 15-year study of a cohort of 789 patients of Blackfoot disease, an increased mortality from cancers of the liver, lung, bladder and kidney was seen among BFD patients when compared with the general population in the endemic area or when compared with the general population of Taiwan. Multiple logistic regression analysis to adjust for other risk factors including cigarette smoking did not markedly affect the exposure-response relationships or odds ratios (Chen et al., 1988).

A significant dose-response relationship was found between arsenic levels

in artesian well water in 42 villages in the southwestern Taiwan and age-adjusted mortality rates from cancers at all sites, cancers of the bladder, kidney, skin, lung, liver and prostate (Wu et al., 1989). An ecological study of cancer mortality rates and arsenic levels in drinking water in 314 townships in Taiwan also corroborated the association between arsenic levels and mortality from the internal cancers (Chen and Wang, 1990).

Chen et al.(1992) conducted a recent analysis of cancer mortality data from the arsenic-exposed population to compare risk of various internal cancers and compare risk between males and females. The study area and population have been described by Wu et al. (1989). It is limited to 42 southwestern coastal villages where residents have used water high in arsenic from deep artesian wells for more than 70 years. Arsenic levels in drinking water ranged from 0.010 to 1.752 ppm. The study population had 898,806 person-years of observation and 202 liver cancer, 304 lung cancer, 202 bladder cancer and 64 kidney cancer deaths. The study population was stratified into four groups according to median arsenic level in well water (<0.10 ppm, 0.10-0.29 ppm, 0.30-0.59 ppm and 60+ ppm), and also stratified into four age groups (<30 years, 30-49 years, 50-69 years and 70+ years). Mortality rates were found to increase significantly with age for all cancers and significant dose-response relationships were observed between arsenic level and mortality from cancer of the liver, lung, bladder and kidney in most age groups of both males and females. The data generated by Chen et al. (1992) provide evidence for an association of the levels of arsenic in drinking water and duration of exposure with the rate of mortality from cancers of the liver, lung, bladder, and kidney. Dose-response relationships are clearly shown by the tabulated data (Tables II-V of Chen et al., 1992). Previous studies summarized in U.S. EPA (1988) showed a similar association in the same Taiwanese population with the prevalence of skin cancers (which are often non-fatal). Bates et al. (1992) and Smith et al. (1992) have recently reviewed and evaluated the evidence for arsenic ingestion and internal cancers.

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o ANIMAL CARCINOGENICITY DATA :

Inadequate. There has not been consistent demonstration of carcinogenicity in test animals for various chemical forms of arsenic administered by different routes to several species (IARC, 1980). Furst (1983) has cited or reviewed animal carcinogenicity testing studies of nine inorganic arsenic compounds in over nine strains of mice, five strains of rats, in dogs, rabbits, swine and chickens. Testing was by the oral, dermal, inhalation, and parenteral routes. All oxidation states of arsenic were tested. No study demonstrated that inorganic arsenic was carcinogenic in animals. Dimethylarsonic acid (DMA), the end metabolite predominant in humans and animals, has been tested for carcinogenicity in two strains of mice and was not found positive (Innes et al., 1969); however, this was a screening study and no data were provided. The meaning of non-positive data for carcinogenicity of inorganic arsenic is uncertain, the mechanism of action in causing human cancer is not known, and rodents may not be a good model for arsenic carcinogenicity testing. There are some data to indicate that arsenic may produce animal lung tumors if retention time in the lung can be increased (Pershagen et al., 1982, 1984).

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o SUPPORTING DATA :

A retrospective cohort mortality study was conducted on 478 British patients treated between 1945-1969 with Fowler's solution (potassium arsenite). The mean duration of treatment was 8.9 months and the average total oral consumption of arsenic was about 1890 mg (daily dose x duration). In 1980, 139 deaths had occurred. No excess deaths from internal cancers were seen after this 20-year follow-up. Three bladder cancer deaths were observed (1.19 expected, SMR 2.5) (Cuzick et al., 1982). A recent follow-up (Cuzick et al., 1992) indicated no increased mortality from all cancers but a significant excess from bladder cancer (5 cases observed/1.6 expected; SMR of 3.07). A subset of the original cohort (143 persons) had been examined by a dermatologist in 1970 for signs of arsenicism (palmar keratosis). In 1990, there were 80 deaths in the subcohort and 11 deaths from internal cancers. All 11 subjects had skin signs (keratosis-10, hyperpigmentation-5 and skin cancer-3). A case-control study of the prevalence of palmar keratoses in 69 bladder cancer patients, 66 lung cancer patients and 218 hospital controls (Cuzick et al., 1984), indicated an association between skin keratosis (as an indicator of arsenic exposure) and lung and bladder cancer. Above the age of 50, 87% of bladder cancer patients and 71% of lung cancer patients but only 36% of controls had one or more keratoses. Several case reports implicate internal cancers with arsenic ingestion or specifically with use of Fowler's solution but the associations are tentative (U.S. EPA, 1988).

Sodium arsenate has been shown to transform Syrian hamster embryo cells (Dipaolo and Casto, 1979) and to produce sister chromatid-exchange in DON cells, CHO cells, and human peripheral lymphocytes exposed in vitro (Wan et al., 1982; Ohno et al., 1982; Larramendy et al., 1981; Andersen, 1983; Crossen, 1983). Jacobson-Kram and Montalbano (1985) have reviewed the mutagenicity of inorganic arsenic and concluded that inorganic arsenic is inactive or very weak for induction of gene mutations in vitro but it is clastogenic with trivalent arsenic being an order of magnitude more potent than pentavalent arsenic.

Both the pentavalent and trivalent forms of inorganic arsenic are found in drinking water. In both animals and humans, arsenate (As+5) is reduced to arsenite (As+3) and the trivalent form is methylated to give the metabolites mononomethylarsinic acid (MMA) and dimethylarsonic acid (DMA) (Vahter and Marafante, 1988). The genotoxicity of arsenate (As+5) and arsenite (As+3) and the two methylated metabolites, MMA and DMA were compared in the thymidine kinase forward mutation assay in mouse lymphoma cells (Harrington-Brock et al. 1993; Moore et al., 1995, in press). Sodium arsenite (+3) and sodium arsenate (+5) were mutagenic at concentration of 1-2 ug/mL and 10-14 ug/mL, respectively, whereas MMA and DMA were significantly less potent, requiring 2.5-5 mg/mL and 10 mg/mL, respectively, to induce a genotoxic response. Based on small colony size the mutations induced were judged chromosomal rather than point mutations. The authors have previously shown that for chemicals having clastogenic activity (i.e., cause chromosomal mutations), the mutated cells grow more slowly than cells with single gene mutations and this results in small colony size. In the mouse lymphoma assay, chromosomal abberations were seen at approximately the same arsenic levels as TK forward mutations. Arsenate, arsenite and MMA were considered clastogenic but the abberation response with DMA was insufficient to consider it a clastogen. Since arsenic exerts its genotoxicity by causing chromosomal mutations, it has been suggested by the above authors that it may act in a latter stage of carcinogenesis as a progressor, rather than as a classical initiator or

promotor (Moore et al., 1994). A finding which supports this process is that arsenate (8-16 uM) and arsenite (3 uM) have been shown to induce 2-10 fold amplification of the dihydrofolate reductase gene in culture in methotrexate resistant 3T6 mouse cells (Lee et al., 1988). Although the mechanism of induction in rodent cells is not known, gene amplification of oncogenes is observed in many human tumors. Inorganic arsenic has not been shown to mutate bacterial strains, it produces preferential killing of repair deficient strains (Rossman, 1981). Sodium arsenite (As+3) induces DNA-strand breaks which are associated with DNA-protein crosslinks in cultured human fibroblasts at 3 mM but not 10 mM (Dong and Luo, 1993) and it appears that arsenite inhibits the DNA repair process by inhibiting both excision and ligation (Jha et al., 1992; Lee-Chen et al., 1993).

The inhibitory effect of arsenite on strand-break rejoining during DNA repair was found to be reduced by adding glutathione to cell cultures (Huang et al., 1993). The cytotoxic effects of sodium arsenite in Chinese hamster ovary cells also has also found to correlate with the intracellular glutathione levels (Lee et al., 1989).

In vivo studies in rodents have shown that oral exposure of rats to arsenate (As+5) for 2-3 weeks resulted in major chromosomal abnormalities in bone marrow (Datta et al., 1986) and exposure of mice to As (+3) in drinking water for 4 weeks (250 mg As/L as arsenic trioxide) caused chromosomal aberrations in bone marrow cells but not spermatogonia (Poma et al., 1987); micronuclei in bone marrow cells were also induced by intraperitoneal dosing of mice with arsenate (DeKnudt et al., 1986; Tinwell et al., 1991). Chromosomal aberrations and sister chromatid exchange have been seen in patients exposed to arsenic from treatment with Fowler's solution (Burgdorf et al., 1977) and subjects exposed occupationally (Beckman et al., 1977) but no increase in either endpoint was seen in lymphocytes of subjects exposed to arsenic in drinking water (Vig et al., 1984).

CARO -

o CLASSIFICATION : A; human carcinogen

o BASIS FOR CLASSIFICATION : based on sufficient evidence from human data. An increased lung cancer mortality was observed in multiple human populations exposed primarily through inhalation. Also, increased mortality from multiple internal organ cancers (liver, kidney, lung, and bladder) and an increased incidence of skin cancer were observed in populations consuming drinking water high in inorganic arsenic.

o ORAL SLOPE FACTOR : 1.5E+0 per (mg/kg)/day

o DRINKING WATER UNIT RISK : 5E-5 per (ug/L)

o DOSE EXTRAPOLATION METHOD : Time- and dose-related formulation of the multistage

o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	2E+0 ug/L
E-5 (1 in 100,000)	2E-1 ug/L
E-6 (1 in 100,000)	2E-2 ug/L

o ORAL DOSE-RESPONSE DATA :

The Risk Assessment Forum has completed a reassessment of the carcinogenicity risk associated with ingestion of inorganic arsenic (U.S. EPA, 1988). The data provided in Tseng et al., 1968 and Tseng, 1977 on about 40,000 persons exposed to arsenic in drinking water and 7500 relatively unexposed controls were used to develop dose-response data. The number of persons at risk over three dose intervals and four exposure durations, for males and females separately, were estimated from the reported prevalence rates as percentages. It was assumed that the Taiwanese persons had a constant exposure from birth, and that males consumed 3.5 L drinking water/day and females consumed 2.0 L/day. Doses were converted to equivalent doses for U.S. males and females based on differences in body weights and differences in water consumption and it was assumed that skin cancer risk in the U.S. population would be similar to the Taiwanese population. The multistage model with time was used to predict dose-specific and age-specific skin cancer prevalance rates associated with ingestion of inorganic arsenic; both linear and quadratic model fitting of the data were conducted. The maximum likelihood estimate (MLE) of skin cancer risk for a 70 kg person drinking 2 L of water per day ranged from 1E-3 to 2E-3 for an arsenic intake of 1 ug/kg/day. Expressed as a single value, the cancer unit risk for drinking water is 5E-5 per (ug/L). Details of the assessment are in U.S. EPA (1988).

Dose response data have not been developed for internal cancers for the Taiwanese population. The data of Chen et al. (1992) are considered inadequate at present.

o ADDITIONAL COMMENTS :

None.

o DISCUSSION OF CONFIDENCE :

This assessment is based on prevalence of skin cancer rather than mortality because the types of skin cancer studied are not normally fatal. However, competing mortality from Blackfoot disease in the endemic area of Taiwan would cause the risk of skin cancer to be underestimated. Other sources of inorganic arsenic, in particular those in food sources have not been considered because of lack of reliable information. There is also uncertainty on the amount of water consumed/day by Taiwanese males (3.5 L or 4.5 L) and the temporal variability of arsenic concentrations in specific wells was not known. The concentrations of arsenic in the wells was measured in the early 1960s and varied between 0.01 and 1.82 ppm. For many villages 2 to 5 analyses were conducted on well water and for other villages only one analysis was performed; ranges of values were not provided. Since tap water was supplied to many areas after 1966, the arsenic-containing wells were only used in dry periods. Because of the study design, particular wells used by those developing skin cancer could not be identified and arsenic intake could

not be assigned except by village. Several uncertainties in exposure measurement reliability existed and subsequent analysis of drinking water found fluorescent substances in water that are possible confounders or caused synergistic effects. Uncertainties have been discussed in detail in U.S. EPA (1988). Uncertainties in exposure measurement can affect the outcome of dose-response estimation.

CARI -
o CLASSIFICATION : A; human carcinogen
o BASIS FOR CLASSIFICATION : based on sufficient evidence from human data. An increased lung cancer mortality was observed in multiple human populations exposed primarily through inhalation. Also, increased mortality from multiple internal organ cancers (liver, kidney, lung, and bladder) and an increased incidence of skin cancer were observed in populations consuming drinking water high in inorganic arsenic.
o INHALATION UNIT RISK : 4.3E-3 per (ug/cu.m)
o DOSE EXTRAPOLATION METHOD : absolute-risk linear model
o RISK/AIR CONCENTRATIONS :

Air concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	2E-2 per (ug/cu.m)
E-5 (1 in 100,000)	2E-3 per (ug/cu.m)
E-6 (1 in 1,000,000)	2E-4 per (ug/cu.m)

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o INHALATION DOSE-RESPONSE DATA :

Tumor Type -- lung cancer
Test Animals -- human, male
Route -- inhalation, occupational exposure
Reference -- Brown and Chu, 1983a,b,c; Lee-Feldstein, 1983; Higgins, 1982; Enterline and Marsh, 1982

Ambient Unit Risk Estimates (per (ug/cu.m)

Exposure Source	Study	Unit Risk	Geometric Mean Unit Risk	Final Estimates Unit Risk
Anaconda smelter	Brown and Chu, 1983a,b,c	1.25E-3		
	Lee-Feldstein, 1983	2.80E-3	2.56E-3	
	Higgins, 1982; Higgins et al., 1982; Welch et al., 1982	4.90E-3		4.29E-3
ASARCO	Enterline and	7.6E-3	6.81E-3	7.19E-3

smelter Marsh, 1982

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o ADDITIONAL COMMENTS :

A geometric mean was obtained for data sets obtained with distinct exposed populations (U.S. EPA, 1984). The final estimate is the geometric mean of those two values. It was assumed that the increase in age-specific mortality rate of lung cancer was a function only of cumulative exposures.

The unit risk should not be used if the air concentration exceeds 2 ug/cu.m, since above this concentration the unit risk may not be appropriate.

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o DISCUSSION OF CONFIDENCE :

Overall a large study population was observed. Exposure assessments included air measurements for the Anaconda smelter and both air measurements and urinary arsenic for the ASARCO smelter. Observed lung cancer incidence was significantly increased over expected values. The range of the estimates derived from data from two different exposure areas was within a factor of 6.

CARDR-
o CARCINOGENICITY SOURCE :

U.S. EPA. 1984, 1988, 1993

A draft of the 1984 Health Assessment Document for Inorganic Arsenic was independently reviewed in public session by the Environmental Health Committee of the U.S. EPA Science Advisory Board on September 22-23, 1983. A draft of the 1988 Special Report on Ingested Inorganic Arsenic; Skin Cancer; Nutritional Essentiality was externally peer reviewed at a two-day workshop of scientific experts on December 2-3, 1986. A draft of the Drinking Water Criteria Document for Arsenic was reviewed by the Drinking Water Committee of the U.S. EPA Science Advisory Board on March 10, 1993. The comments from these reviews were evaluated and considered in the revision and finalization of these reports.
DOCUMENT

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o REVIEW DATES : 01/13/88, 12/07/89, 02/03/94
o VERIFICATION DATE : 02/03/94
o EPA CONTACTS :

Herman Gibb / NCEA -- (202)260-7315

Charles Abernathy / OST -- (202)260-5374

HAONE- NO DATA

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA - NO DATA

WQCHU-

Water and Fish Consumption -- 2.2E-3 ug/L

Fish Consumption Only -- 1.75E-2 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. However, zero may not be attainable at this time, so the recommended criteria represents a E-6 estimated incremental increase of cancer risk over a lifetime.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 3.6E+2 ug/L (Arsenic III)
Chronic -- 1.9E+2 ug/L (Arsenic III)

Marine:

Acute -- 6.9E+1 ug/L (Arsenic III)
Chronic -- 3.6E+1 ug/L (Arsenic III)

Considers technological or economic feasibility? -- NO

Discussion -- The criteria given are for Arsenic III. Much less data are available on the effects of Arsenic V to aquatic organisms, but the toxicity seems to be less. A complete discussion may be found in the referenced notice.

Reference -- 50 FR 30784 (07/29/85)

EPA Contact -- Criteria and Standards Division / OWRS

(202)260-1315 / FTS 260-1315

MCLG -

Value (status) -- 0.05 mg/L (Proposed, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- An MCLG of 0.05 mg/L for arsenic is proposed based on the current MCL of 0.05 mg/L. Even though arsenic is potentially carcinogenic in humans by inhalation and ingestion, its potential essential nutrient value was considered in determination of an MCLG. The basis for this evaluation is nutritional requirements by NAS (NAS, 1983, Vol. 5, Drinking Water and Health, National Academy of Sciences Press, Washington, DC.)

Reference -- 50 FR 46936 (11/13/85)

EPA Contact -- Health and Ecological Criteria Division / OST / (202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value (status) -- 0.05 mg/L (Interim, 1980)

Considers technological or economic feasibility? -- YES

Discussion -- As an interim measure the U.S. EPA is using the value previously derived by the Public Health Service.

Monitoring requirements -- Ground water systems every three years; surface water systems annually.

Analytical methodology -- Atomic absorption/furnace technique (EPA 206.2; SM 304); atomic absorption/gaseous hydride (EPA 206.3; SM 303E; ASTM D-2972-78B)

Best available technology -- No data available.

Reference -- 45 FR 57332 (08/27/80); 50 FR 46936 (11/13/85)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

SMCL - NO DATA

FISTD-

Status -- Issued (1988)

Reference -- Arsenic, Chromium and Chromated Arsenical Compounds Pesticide Registration Standard. June, 1988. [NTIS# PB89-102842]

EPA Contact -- Registration Branch / OPP
(703)557-7760 / FTS 557-7760

FIREV-

Action -- Final regulatory decision - PD4 (1988)

Considers technological or economic feasibility? -- NO

Summary of regulatory action -- Cancellation of specified non-wood uses. Registrant of lead arsenate voluntarily canceled 09/87. Registrant of calcium arsenate voluntarily canceled 02/14/89. Use of sodium arsenate as ant bait canceled on 07/26/89. Criterion of concern: oncogenicity, mutagenicity and teratogenicity. Previous actions: 1) Voluntary cancellation of sodium arsenite (1978). Voluntary cancellation of two products. Criterion of concern: oncogenicity, mutagenicity and teratogenicity; 2) PD4 (1984). Requires label changes for wood use including a restricted use classification.

Criterion of concern: oncogenicity, mutagenicity and teratogenicity; 3) Voluntary cancellation of copper arsenate (1977). Criterion of concern: oncogenicity.

Reference -- 53 FR 24787 (06/30/88); 43 FR 48267 (10/18/78); 42 FR 18422 (04/07/77); 49 FR 28666 (07/13/84) [NTIS# PB84-241538]; 49 FR 43772 (10/31/84); 50 FR 4269 (01/30/85)

EPA Contact -- Special Review Branch / OPP
(703)557-7400 / FTS 557-7400

CERC -

Value (status) -- 1 pound (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The 1-pound RQ for arsenic is based on its potential carcinogenicity. Available data indicate a hazard ranking of high based on a potency factor of 142.31/mg/kg/day and a weight-of-evidence group A, which corresponds to an RQ of 1 pound. Evidence found in "Water-Related Environmental Fate of 129 Priority Pollutants" (EPA 440/4-79-029a) also indicates that this material, or a constituent of this material, is bioaccumulated to toxic levels in the tissue of aquatic and marine organisms, and has the potential to concentrate in the food chain. Reporting of releases of massive forms of this hazardous substance is not required if the diameter of the pieces released exceeds 100 micrometers (0.004 inches).

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

SARA - NO DATA

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

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HAREF- None

Benzo(a)pyrene

1 - IRIS
NAME - Benzo[a]pyrene (BaP)
RN - 50-32-8
IRSN - 133
DATE - 941102
UPDT - 11/02/94, 1 field
STAT - Oral RfD Assessment (RDO) no data
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) on-line 11/01/94
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 08/01/89 REFS Bibliography on-line
IRH - 01/01/92 CAR Carcinogen assessment noted as pending change
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 04/01/92 CAR Summary revised; oral quantitative section added
IRH - 04/01/92 CREF Carcinogen assessment references revised
IRH - 05/01/92 CARDR Work group review and verification date corrected
IRH - 07/01/92 CAR Text revised in NOTE
IRH - 07/01/92 CARO Range of slope factors corrected
IRH - 07/01/92 CARO Slope factor and risks corrected
IRH - 07/01/92 CARO Data table heading corrected
IRH - 07/01/92 CARO Slope factor corrected; last paragraph
IRH - 07/01/92 CARDR Secondary contact changed
IRH - 09/01/93 CAR Carcinogenicity assessment noted as pending change
IRH - 09/01/93 CARDR Work group review date added
IRH - 12/01/93 CREF Reference revised - U.S. EPA, 1991b
IRH - 02/01/94 CARDR Primary contact's phone number changed
IRH - 03/01/94 CAR Pending change note removed; no change
IRH - 03/01/94 CARDR Work group review date added
IRH - 07/01/94 CARDR Work group review date added
IRH - 11/01/94 CARO Slope factor clarified; changed O to "0"
RLEN - 25760
SY - BaP
SY - Benzo[a]pyrene
SY - BENZO(d,e,f)CHRYSENE
SY - 3,4-BENZOPIRENE
SY - 3,4-BENZOPYRENE
SY - 6,7-BENZOPYRENE
SY - BENZO(a)PYRENE
SY - 3,4-BENZPYREN
SY - 3,4-BENZPYRENE
SY - 3,4-BENZ(a)PYRENE
SY - BENZ(a)PYRENE
SY - 3,4-BENZYPYRENE
SY - BP
SY - 3,4-BP
SY - B(a)P
SY - RCRA WASTE NUMBER U022

RDO - NO DATA

RDI - NO DATA
CAREV-
o CLASSIFICATION : B2; probable human carcinogen

o BASIS FOR CLASSIFICATION : Human data specifically linking benzo[a]pyrene (BAP) to a carcinogenic effect are lacking. There are, however, multiple animal studies in many species demonstrating BAP to be carcinogenic following administration by numerous routes. BAP has produced positive results in numerous genotoxicity assays. NOTE: At the June 1992 CRAVE Work Group meeting, a revised risk estimate for benzo[a]pyrene was verified (see Additional Comments for Oral Exposure). This section provides information on three aspects of the carcinogenic risk assessment for the agent in question; the U.S. EPA classification, and quantitative estimates of risk from oral exposure and from inhalation exposure. The classification reflects a weight-of-evidence judgment of the likelihood that the agent is a human carcinogen. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000 or 1 in 1,000,000. The Carcinogenicity Background Document provides details on the rationale and methods used to derive the carcinogenicity values found in IRIS. Users are referred to the Oral Reference Dose (RfD) and Reference Concentration (RfC) sections for information on long-term toxic effects other than carcinogenicity.

o HUMAN CARCINOGENICITY DATA :

Inadequate. Lung cancer has been shown to be induced in humans by various mixtures of polycyclic aromatic hydrocarbons known to contain BAP including cigarette smoke, roofing tar and coke oven emissions. It is not possible, however, to conclude from this information that BAP is the responsible agent.

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o ANIMAL CARCINOGENICITY DATA :

Sufficient. The animal data consist of dietary, gavage, inhalation, intratracheal instillation, dermal and subcutaneous studies in numerous strains of at least four species of rodents and several primates. Repeated BAP administration has been associated with increased incidences of total tumors and of tumors at the site of exposure. Distant site tumors have also been observed after BAP administration by various routes. BAP is frequently used as a positive control in carcinogenicity bioassays.

BAP administered in the diet or by gavage to mice, rats and hamsters has produced increased incidences of stomach tumors. Neal and Rigdon (1967) fed BAP (purity not reported) at concentrations of 0, 1, 10, 20, 30, 40, 45, 50, 100 and 250 ppm in the diets of male and female CFW-Swiss mice. The age of the mice ranged from 17-180 days old and the treatment time from 1-197 days; the size of the treated groups ranged from 9 to 73. There were 289 mice (number of mice/sex not stated) in the control group. No forestomach tumors were reported in the 0-, 1- and 10-ppm dose groups. The incidence of forestomach tumors in the 20-, 30-, 40-, 45-, 50-, 100- and 250-ppm dose groups were 1/23, 0/37, 1/40, 4/40, 23/34, 19/23 and 66/73, respectively. The authors felt that the increasing tumor incidences were related to both the concentration and the number of doses administered. Historical control forestomach tumor data are not available for CFW-Swiss strain mice. In historical control data from a related mouse strain, SWR/J Swill, the forestomach tumor incidence rate was 2/268 and 1/402 for males and females; respectively (Rabstein et al., 1973).

Brune et al., (1981) fed 0.15 mg/kg BAP (reported to be "highly pure") in the diet of 32 Sprague-Dawley rats/sex/group either every 9th day or 5 times/week. These treatments resulted in annual average doses of 6 or 39 mg/kg, respectively. An untreated group of 32 rats/sex served as the control. Rats were treated until moribund or dead; survival was similar in all groups. Histologic examinations were performed on each rat. The combined incidence of tumors of the forestomach, esophagus and larynx was 3/64, 3/64 and 10/64 in the control group, the group fed BAP every 9th day and the group fed BAP 5 times/week, respectively. A trend analysis showed a statistically significant tendancy for the proportion of animals with tumors of the forestomach, esophagus or larynx to increase steadily with dose (Knauf and Rice, 1992).

As part of the same study, Brune et al. (1981) administered BAP ("highly pure") orally to Sprague-Dawley rats by caffeine gavage. The rats were treated until moribund or dead; all rats were subjected to terminal histopathologic examination. Gavaged rats were divided into 3 dose groups of 32 rats/sex/group; the groups received 0.15 mg/kg per gavage either every 9th day (Group A), every 3rd day (Group B) or 5 times per week (Group C); these treatments resulted in annual average doses of 6, 18 or 39 mg/kg, respectively. Untreated and gavage (5 times/week) controls (32 rats/sex/group) were included. The median survival times for the untreated control group; the gavage control group; and groups A, B and C were 129, 102, 112, 113 and 87 weeks, respectively. The survival time of Group C was short compared with controls and may have precluded tumor formation (Knauf and Rice, 1992). The combined tumor incidence in the forestomach, esophagus and larynx was 3/64, 6/64, 13/64, 26/64 and 14/64 for the untreated control group, gavage control group, group A, group B and group C, respectively. There was a statistically significant association between the dose and the proportions of rats with tumors of the forestomach, esophagus or larynx. This association is not characterized by a linear trend. The linearity was affected by the apparently reduced tumor incidence that is seen in the high-dose group (Knauf and Rice, 1992).

Intratracheal instillation and inhalation studies in guinea pigs, hamsters and rats have resulted in elevated incidences of respiratory tract and upper digestive tract tumors (U.S. EPA, 1991a). Male Syrian golden hamsters (24/group) were exposed by inhalation to 0, 2.2, 9.5 or 46.5 mg BAP/cu.m in a

sodium chloride aerosol (Thyssen et al., 1981). (Greater than 99% of the particles had diameters between 0.2 and 0.5 um.) For the first 10 weeks of the study, the hamsters were exposed to BAP daily for 4.5 hours/day; thereafter, daily for 3 hours/day. Animals dying within the first year of the study were replaced; the effective number of hamsters in the control, low-, mid- and high-dose groups was 27, 27, 26 and 25, respectively. (The total time of treatment, although over 60 weeks, was not stated.) During the first 10 weeks, animals in the 3 dose groups reportedly lost weight. After week 10, however, the body weights in all groups were similar until week 60 when the body weights of hamsters in the high-dose group decreased and the mortality increased significantly. The incidence of respiratory tract tumors (including tumors of the nasal cavity, larynx and trachea) in the control, low-, mid- and high-dose groups was 0/27, 0/27, 9/26 and 13/25, respectively; the incidences of upper digestive tract tumors (including tumors of the pharynx, esophagus and forestomach) were 0/27, 0/27, 7/26 and 14/25, respectively. Trend analysis for incidences of both respiratory tract tumors and upper gastrointestinal tract tumors showed a statistically significant tendancy for the proportion of animals with either tumor type to increase steadily with increased dose (Knauf and Rice, 1992).

Intraperitoneal BAP injections have caused increases in the number of injection site tumors in mice and rats (reviewed in U.S. EPA, 1991a). Subcutaneous BAP injections have caused increases in the number of injection site tumors in mice, rats, guinea pigs, hamsters and some primates (IARC, 1983; U.S. EPA, 1991a). BAP is commonly used as a positive control in many dermal application bioassays and has been shown to cause skin tumors in mice, rats, rabbits and guinea pigs. BAP is both an initiator and a complete carcinogen in mouse skin (IARC, 1983). Increased incidences of distant site tumors have also been reported in animals as a consequence of dermal BAP exposure (reviewed in U.S. EPA, 1991a).

BAP has also been reported to be carcinogenic in animals when administered by the following routes: i.v.; transplacentally; implantation in the stomach wall, lung, renal parenchyma and brain; injection into the renal pelvis; and vaginal painting (U.S. EPA, 1991a).

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o SUPPORTING DATA :

Benzo[a]pyrene has been shown to cause genotoxic effects in a broad range of prokaryotic and mammalian cell assay systems (U.S. EPA, 1991a). In prokaryotes, BAP tested positive in DNA damage assays and in both reverse and forward mutation assays. In mammalian cell culture assays, BAP tested positive in DNA damage assays, forward mutation assays, chromosomal effects assays and cell transformation assays.

CARO -

o CLASSIFICATION : B2; probable human carcinogen

o BASIS FOR CLASSIFICATION : Human data specifically linking benzo[a]pyrene (BAP) to a carcinogenic effect are lacking. There are, however, multiple

animal studies in many species demonstrating BAP to be carcinogenic following administration by numerous routes. BAP has produced positive results in numerous genotoxicity assays. NOTE: At the June 1992 CRAVE Work Group meeting, a revised risk estimate for benzo[a]pyrene was verified (see Additional Comments for Oral Exposure). This section provides information on three aspects of the carcinogenic risk assessment for the agent in question; the U.S. EPA classification, and quantitative estimates of risk from oral exposure and from inhalation exposure. The classification reflects a weight-of-evidence judgment of the likelihood that the agent is a human carcinogen. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000 or 1 in 1,000,000. The Carcinogenicity Background Document provides details on the rationale and methods used to derive the carcinogenicity values found in IRIS. Users are referred to the Oral Reference Dose (RfD) and Reference Concentration (RfC) sections for information on long-term toxic effects other than carcinogenicity.

o ORAL SLOPE FACTOR : 7.3E+0 per (mg/kg)/day
o DRINKING WATER UNIT RISK : 2.1E-4 per (ug/L)
o DOSE EXTRAPOLATION METHOD : Risk estimate based on a geometric mean of four slope
o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	5E-1 ug/L
E-5 (1 in 100,000)	5E-2 ug/L
E-6 (1 in 1,000,000)	5E-3 ug/L

o ORAL DOSE-RESPONSE DATA :

Tumor Type -- forestomach, squamous cell papillomas and carcinomas
Test Animals -- CFW mice, sex unknown
Route -- oral, diet
Reference -- Neal and Rigdon, 1967

a) Conditional upper bound two-stage model with terms for promotion (modification of Moolgavkar-Venson-Knudson, generalized forms of two-stage model)

Administered Dose (ppm)	Tumor Incidence
0	0/289
1	0/25
10	0/24
20	1/23
30	0/37
40	1/40
45	4/40
50	24/34
100	19/23
250	66/73

Tumor Type -- squamous cell carcinoma of the forestomach
Test Animals -- SWR/J Swill mice
Route -- oral, diet
Reference -- Rabstein et al., 1973

Administered Dose (ppm)	Tumor Incidence
0	2/268* male
0	1/402* female

*See additional comments concerning the use of control data from other studies that utilized similar mouse strains.

b) Same data as above. Upper bound estimate by extrapolation from 10% response point to background of empirically fitted dose-response curve. (Procedure using two-stage model described in (a)).

c) Same data as above except the additional 2 control groups (Rabstein et al., 1973) were excluded. Generalized Weibull-type dose-response model.

d) Tumor Type -- forestomach, larynx and esophagus, papillomas and carcinomas (combined). Linearized Multistage Model, Extra Risk.

Test Animals -- Sprague-Dawley rats, males and females
Route -- oral, diet
Reference -- Brune et al., 1981

Dose (mg/kg diet/year)	Tumor Incidence
0	3/64
6	3/64
39	10/64

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o ADDITIONAL COMMENTS :

NOTE: The range of oral slope factors calculated was: 4.5E+0 to 11.7E+0 per (mg/kg)/day.

At the June 1992 CRAVE Work Group meeting, it was noted that an error had been made in the 1991 document "Dose-Response Analysis of Ingested Benzo[a]pyrene" which is quoted in the Drinking Water Criteria Document for PAH. In the calculation of the doses in the Brune et al. (1981) study it was erroneously concluded that doses were given in units of mg/year, whereas it was in fact mg/kg/year. When the doses are corrected the slope factor is correctly calculated as 11.7 per (mg/kg)/day, as opposed to 4.7 per (mg/kg)/day as reported in the Drinking Water Criteria Document. The correct range of slope factors is 4.5 to 11.7 per (mg/kg)/day, with a geometric mean of 7.3 per (mg/kg)/day. A drinking water unit risk based on the revised slope factor is 2.1E-4 per (ug/L). Therefore, these values have been changed on IRIS and an Erratum to the Drinking Water Criteria Document is being prepared.

Risk estimates were calculated from two different studies in two species of outbred rodents (Neal and Rigdon, 1967; Brune et al., 1981). These studies have several commonalities including mode of administration, tumor sites, tumor types and the presumed mechanisms of action. The data sets were not combined prior to modeling (the preferred approach) because they employed significantly dissimilar protocols.

The geometric mean from several slope factors, each considered to be of equal merit, was used to calculate a single unit risk. These four slope factor estimates span less than a factor of three and each is based on an acceptable, but less-than-optimal, data set. Each estimate is based on a low-dose extrapolation procedure which entails the use of multiple assumptions and default procedures.

Clement Associates (1990) fit the Neal and Rigdon (1967) data to a two-stage dose response model. In this model the transition rates and the growth rate of preneoplastic cells were both considered to be exposure-dependent. (The functional form for the dose-dependence of preneoplastic cell growth rate was simple saturation.) A term to permit the modeling of BAP as its own promoter was also included. Historical control stomach tumor data from a related, but not identical, mouse strain, SWR/J Swill (Rabstein et al., 1973) and the CFW Texas colony (Neal and Rigdon, 1967) were used in the modeling. In calculating the lifetime unit risk for humans several standard assumptions were made: mouse food consumption was 13% of its body weight/day; human body weight was assumed to be 70 kg and the assumed body weight of the mouse 0.034 kg. The standard assumption of surface area equivalence between mice and humans was the cube root of 70/0.034. A conditional upper bound estimate was calculated to be 5.9 per (mg/kg)/day (U.S. EPA, 1991a).

A U.S. EPA report (1991b) argued that the upper-bound estimate calculated in Clement Associates (1990) involved the use of unrealistic conditions placed on certain parameters of the equation. Other objections to this slope factor were also raised. The authors of this report used the Neal and Rigdon (1967) data to generate an upper-bound estimate extrapolated linearly from the 10% response point to the background of an empirically fitted dose-response curve (Clement Associates, 1990). Other results, from similar concepts and approaches used for other compounds, suggest that the potency slopes calculated in this manner are comparable to those obtained from a linearized

multistage procedure for the majority of the other compounds. The upper bound estimate calculated in U.S. EPA (1991b) is 9.0 per (mg/kg)/day.

The authors of U.S. EPA (1991b) selected a model to reflect the partial lifetime exposure pattern over different parts of the animals' lifetimes. The authors thought that this approach more closely reflected the Neal and Rigdon (1967) regimen. A Weibull-type dose-response model was selected to accommodate the partial lifetime exposure; the upper-bound slope factor calculated from this method was 4.5 per (mg/kg)/day.

Using the dietary portion of the Brune et al. (1981) rat data, a linearized multistage procedure was used to calculate an upper bound slope factor for humans. In the interspecies conversion the assumed human body weight was 70 kg and the rat 0.4 kg. The slope factor calculated by this method was 11.7 per (mg/kg)/day.

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o DISCUSSION OF CONFIDENCE :

The data are considered to be less than optimal, but acceptable. There are precedents for using multiple data sets from different studies using more than one sex, strain and species; the use of the geometric mean of four slope factors is preferred because it makes use of more of the available data. The use of the geometric means was based on arguments presented in a personal communication (Stiteler, 1991).

CARI - NO DATA
CARDR-
o CARCINOGENICITY SOURCE :

Source Document -- U.S. EPA, 1991a,b

The 1991 Drinking Water Criteria Document for the polycyclic aromatic hydrocarbons has received agency review.
DOCUMENT

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o REVIEW DATES : 01/07/87, 12/04/91, 06/03/92, 08/05/93, 02/02/94,
o VERIFICATION DATE : 12/04/91
o EPA CONTACTS :

Robert E. McGaughy / OHEA -- (202)260-5889

Rita Schoeny / OHEA -- (513)569-7544

HAONE- NO DATA

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA - NO DATA

WQCHU-

Water and Fish Consumption: 2.8E-3 ug/L

Fish Consumption Only: 3.11E-2 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. However, zero may not be obtainable at this time, so the recommended criteria represents a E-6 estimated incremental increase of cancer over a lifetime. The values given represent polynuclear aromatic hydrocarbons as a class.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute LEC -- none
Chronic LEC -- none

Marine:

Acute LEC -- 3.0E+2 ug/L
Chronic LEC -- none

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LEC's are given when the minimum data required to derive water quality criteria are not available. The values given represent polynuclear aromatic hydrocarbons as a class.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value -- 0 mg/L (Proposed,1990)

Considers technological or economic feasibility? -- NO

Discussion -- The proposed MCLG for benzo(a)pyrene is zero based on the evidence of carcinogenic potential (B2).

Reference -- 55 FR 30370 (07/25/90)

EPA Contact -- Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value -- 0.0002 mg/L (Proposed, 1990)

Considers technological or economic feasibility? -- YES

Discussion -- The proposed MCL is equal to the PQL and is associated with a maximum lifetime individual risk of 1 E-4.

Monitoring requirements -- Community and non-transient water system monitoring based on state vulnerability assessment; vulnerable systems to be monitored quarterly for one year; repeat monitoring dependent upon detection and size of system.

Analytical methodology -- High pressure liquid chromatography (EPA 550, 550.1); gas chromatographic/mass spectrometry (EPA 525): PQL= 0.0002 mg/L.

Best available technology -- Granular activated carbon

Reference -- 55 FR 30370 (07/25/90)

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

Status -- Listed (Proposed, 1991)

Discussion -- "Unregulated" contaminants are those contaminants for which EPA establishes a monitoring requirement but which do not have an associated final MCLG, MCL, or treatment technique. EPA may regulate these contaminants in the future.

Monitoring requirement -- All systems to be monitored unless a vulnerability assessment determines the system is not vulnerable.

Analytical methodology -- Gas chromatography/mass spectrometry (EPA 525); high pressure liquid chromatography (EPA 550, 550.1).

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

SMCL - NO DATA

FISTD- NO DATA

FIREV- NO DATA

CERC -

Value -- 1 pound (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The RQ for benzo(a)pyrene is based on potential carcinogenicity (group B2). This chemical is currently under assessment for carcinogenicity and chronic toxicity and the RQ is subject to change in future rulemaking.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline (800) 424-9346 / (202) 260-3000 / FTS 260-3000

SARA - NO DATA

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - None
IREF - None
CREF - Brune, H., R.P. Deutsch-Wenzel, M. Habs, S. Ivankovic and D. Schmahl. 1981. Investigation of the tumorigenic response to benzo[a]pyrene in aqueous caffeine solution applied orally to Sprague-Dawley rats. J. Cancer Res. Clin. Oncol. 102(2): 153-157.
CREF - Clement Associates. 1990. Ingestion dose-response model to benzo(a)pyrene. EPA Control No. 68-02-4601.
CREF - IARC (International Agency for Research on Cancer). 1983. Certain Polycyclic Aromatic Hydrocarbons and Heterocyclic Compounds. Monographs on the Evaluation of Carcinogenic Risk of the Chemical to Man, Vol. 3. Lyon, France.
CREF - Knauf, L. and G. Rice. 1992. Statistical Evaluation of Several Benzo[a]pyrene Bioassays. Memorandum to R. Schoeny, U.S. EPA, Cincinnati, OH. January 2.
CREF - Neal, J. and R.H. Rigdon. 1967. Gastric tumors in mice fed benzo[a]pyrene -- A quantitative study. Tex. Rep. Biol. Med. 25(4): 553-557.
CREF - Rabstein, L.S., R.L. Peters and G.J. Spahn. 1973. Spontaneous tumors and pathologic lesions in SWR/J mice. J. Natl. Cancer Inst. 50: 751-758.
CREF - Stiteler, W. 1991. Syracuse Research Corporation, Syracuse, NY. Personal communication with R. Schoeny, U.S. EPA, Cincinnati, OH.
CREF - Thyssen, J., J. Althoff, G. Kimmerle and U. Mohr. 1981. Inhalation studies with benzo[a]pyrene in Syrian golden hamsters. J. Natl. Cancer Inst. 66: 575-577.
CREF - U.S. EPA. 1991a. Drinking Water Criteria Document for PAH. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards, Washington, DC.
CREF - U.S. EPA. 1991b. Dose-Response Analysis of Ingested Benzo[a]pyrene (CAS No. 50-32-8). Human Health Assessment Group, Office of Health and Environmental Assessment, Washington, DC. EPA/600/R-92/045.
HAREF- None

Beryllium

1 - IRIS
NAME - Beryllium
RN - 7440-41-7
IRSN - 11
DATE - 930201
UPDT - 02/01/93, 1 field
STAT - Oral RfD Assessment (RDO) on-line 02/01/93
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) on-line 09/01/92
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 03/01/88 RDO Reference dose table clarified
IRH - 03/01/88 RDO Text added
IRH - 09/07/88 CAR Carcinogen summary on-line
IRH - 01/01/90 CAREV References clarified
IRH - 01/01/90 CAREV Text revised
IRH - 01/01/90 CARO Quantitative estimate for oral exposure section added
IRH - 01/01/90 CARI Text revised
IRH - 01/01/90 CARDR Work group review dates and verification date added
IRH - 01/01/90 REFS Bibliography on-line
IRH - 02/01/90 OREF Puzanova et al. 1978 citation corrected
IRH - 02/01/90 CREF Wagner et al. 1969 citation corrected
IRH - 09/01/90 RDO Morgareidge ref. now Cox (same study-authors reversed)
IRH - 09/01/90 RCRA EPA contact changed
IRH - 09/01/90 OREF Morgareidge ref. now Cox (same study-authors reversed)
IRH - 01/01/91 CAR Text edited
IRH - 01/01/91 CARI Inhalation slope factor removed (global change)
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 09/01/92 CAREV U.S. EPA citation year corrected, paragraph 3
IRH - 09/01/92 CARDR Source document year corrected
IRH - 09/01/92 CARDR Review statement revised
IRH - 09/01/92 CREF U.S. EPA reference year corrected
IRH - 02/01/93 RDO Primary contact changed
RLEN - 27537
SY - Beryllium
SY - Beryllium-9
SY - Glucinum
SY - RCRA waste number P015
SY - UN 1567

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
No adverse effects Rat, Chronic Oral Bioassay	NOAEL: 5 ppm in drinking water (0.54 mg/kg bw/day)	100 mg/kg/day	1	5E-3
Schroeder and Mitchner, 1975	LOAEL: none			

*Conversion Factors: 5 ppm (5 mg/L) x 0.035 L/day / 0.325 kg bw = 0.54 mg/kg

bw/day

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o ORAL RFD STUDIES :

Schroeder, H.A. and M. Mitchner. 1975. Life-term studies in rats: Effects of aluminum, barium, beryllium and tungsten. J. Nutr. 105: 421-427.

Fifty-two weanling Long-Evans rats of each sex received 0 or 5 ppm beryllium (as BeSO4, beryllium sulfate) in drinking water. Exposure was for the lifetime of the animals. At natural death the rats were dissected and gross and microscopic changes were noted in heart, kidney, liver, and spleen. There were no effects of treatment on these organs or on lifespan, urinalysis, serum glucose, cholesterol, and uric acid, or on numbers of tumors. Male rats experienced decreased growth rates from 2 to 6 months of age.

Similar studies were carried out on Swiss (CD strain) mice in groups of 54/sex at doses of approximately 0.95 mg/kg/day (Schroeder and Mitchner, 1975). Female animals showed decreased body weight compared with untreated mice at 6 of 8 intervals. Male mice exhibited slight increases in body weight. These effects were not considered adverse, therefore, 0.95 mg/kg/day is considered a NOAEL.

An unpublished investigation by Cox et al. (1975) indicates a much higher dose level (approximately 25 mg/kg/day) in the diet may be a NOEL.

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o ORAL RFD UNCERTAINTY :

UF -- The uncertainty factor of 100 reflects a factor of 10 each for interspecies conversion and for the protection of sensitive human subpopulations.

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o ORAL RFD MODIFYING FACTOR :

MF -- None

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o ORAL RFD COMMENTS :

This RfD is limited to soluble beryllium salts. Data on the terato- genicity or reproductive effects of beryllium are limited. It has been reported to produce embryolethality and terata in chick embryos (Puzanova et al., 1978).

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o ORAL RFD CONFIDENCE :

Study -- Low
Data Base -- Low
RfD -- Low

Confidence in the study is rated as low because only one dose level was administered. Although numerous inhalation investigations and a supporting chronic oral bioassay in mice exist, along with the work by Cox et al. (1975) which indicates that a higher dose level might be a NOEL, these studies are considered as low to medium quality; thus, the data base is given a low confidence rating. The overall confidence in the RfD is low, reflecting the need for more toxicity data by the oral route.

o ORAL RFD SOURCE DOCUMENT :

Source Document – U.S. EPA, 1985

The 1985 Drinking Water Criteria Document for Beryllium is currently undergoing Agency review.

o REVIEW DATES : 12/02/85
o VERIFICATION DATE : 12/02/85
o EPA CONTACTS :

Linda R. Papa / OHEA – (513)569-7587

Krishan Khanna / OST – (202)260-7588

RDI - NO DATA
CAREV-
o CLASSIFICATION : B2; probable human carcinogen.
o BASIS FOR CLASSIFICATION : Beryllium has been shown to induce lung cancer via inhalation in rats and monkeys and to induce osteosarcomas in rabbits via intravenous or intramedullary injection. Human epidemiology studies are considered to be inadequate.
o HUMAN CARCINOGENICITY DATA :

Inadequate. Reported increases, while apparently associated with exposure, did not take a variety of possible confounding factors into account.

Wagoner et al. (1980) observed 47 deaths from cancer among 3055 white males employed in beryllium-processing with a median duration of employment of 7.2 months. Among the 2068 followed for 25 years or more, 20 lung cancer deaths were observed. These increased incidences were statistically significant. When lung cancer mortality data became available for 1968-1975, the number of expected deaths was recalculated and the increased incidence was statistically significant only among workers followed 25 years or more (Bayliss, 1980; MacMahon, 1977, 1978). When the number of expected deaths was adjusted for smoking, the increased incidence was no longer significant (U.S. EPA, 1986).

An earlier study of workers from this same beryllium processing plant, and several studies of workers from this plant combined with workers from other beryllium plants, have reported a statistically significant increased incidence of lung cancer (Bayliss and Wagoner, 1977; Mancuso, 1970, 1979, 1980). No adjustment was made for smoking in these studies, and all were limited in their ability to detect a possible increased incidence of lung cancer because of methodological constraints and deficiencies.

o ANIMAL CARCINOGENICITY DATA :

Sufficient. Based on the evidence for induction of tumors by a variety of beryllium compounds in male and female monkeys and in several strains of rats of both sexes, via inhalation and intratracheal instillation, and the induction of osteosarcomas in rabbits by intravenous or intramedullary injection in multiple studies.

Slight increases in cancer incidence (not statistically significant in comparison with controls) were reported in Long-Evans rats (52/sex/group) administered 5 ppm beryllium sulfate in the drinking water for a lifetime. The authors reported a slight excess of grossly observed tumors in the 5 ppm group (9/33) over controls (4/26) in the male rats. The power of this test to detect a carcinogenic effect was reduced by high mortality (approximately 60% survived a pneumonia epidemic at 20 months) (Schroeder and Mitchener, 1975a). Schroeder and Mitchener (1975b) administered 5 ppm beryllium sulfate in drinking water to Swiss mice (54/sex/group) over a lifetime. A non-statistically significant increase in incidence of lymphoma leukemias were reported in the females (9/52) relative to controls (3/47).

An increase in reticulum cell sarcomas of the lungs was seen in male, but not female Wistar-derived rats administered beryllium sulfate in the diet at 5 and 50 ppm, but not at 500 ppm (Morgareidge et al., 1977). The incidence in males equaled 10/49, 17/35, 16/40 and 12/39 for the control, low, intermediate and high dose groups, respectively. Since the results were published only as an abstract, and since no response was seen at the highest dose, these results are considered to be only suggestive for the induction of cancer via this route.

Osteogenic sarcomas were induced in rabbits by intravenous injection of beryllium compounds in at least 12 different studies and by intramedullary injection in at least four studies (U.S. EPA, 1991). Bone tumors were induced by beryllium oxide, zinc beryllium silicate, beryllium phosphate, beryllium silicate and beryllium metal. No bone tumors were reported to be induced by intravenous injection of beryllium oxide or zinc beryllium silicate in rats or guinea pigs (Gardner and Heslington, 1946). Positive results, however, were reported in mice injected with zinc beryllium silicate, although the numbers were not listed (Cloudman et al., 1949). The sarcomas were generally reported to be quite malignant and metastasized to other organs.

Lung tumors, primarily adenomas and adenocarcinomas, have been induced via the inhalation route in both male and female Sprague-Dawley rats during exposure periods of up to 72 weeks by beryllium sulfate (Reeves et al., 1967), in both male and female Sherman and Wistar rats by beryllium phosphate, beryllium fluoride and zinc beryllium silicate (Schepers, 1961), in male Charles River CR-CD rats by beryl ore (Wagner et al., 1969) and in both male and female rhesus monkeys by beryllium sulfate (Vorwald, 1968). Positive results were seen in rats exposed to beryllium sulfate at concentrations as low as 2 ug/cu.m (Vorwald, 1968).

Tumors were also induced by intratracheal instillation of metallic beryllium, beryllium-aluminum alloys and beryllium oxide in both Wistar rats and rhesus monkeys. Adenomas, adenocarcinomas and malignant lymphomas were seen in the lungs, with lymphosarcomas and fibrosarcomas present at extrapulmonary sites (Groth et al., 1980; Ishinishi et al., 1980).

o SUPPORTING DATA :

Beryllium sufate and beryllium chloride have been shown to be nonmutagenic in bacterial and yeast gene mutation assays (Simmon et al., 1979). In contrast, gene mutation studies in Chinese hamster V79 and CHO cells were positive (Miyaki et al., 1979; Hsie et al., 1979). Chromosomal aberrations and sister chromatid exchange were also induced by beryllium in cultured human lymphocytes and Syrain hamster embryo cells (Larramendy et al., 1981).

CARO -

o CLASSIFICATION : B2; probable human carcinogen.

o BASIS FOR CLASSIFICATION : *Beryllium has been shown to induce lung* cancer via inhalation in rats and monkeys and to induce osteosarcomas in rabbits via intravenous or intramedullary injection. Human epidemiology studies are considered to be inadequate.

o ORAL SLOPE FACTOR : 4.3 per(mg/kg)/day

o DRINKING WATER UNIT RISK : 1.2E-4 per(ug/L)

o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk

o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	8.3E-1 ug/L
E-5 (1 in 100,000)	8.3E-2 ug/L
E-6 (1 in 1,000,000)	8.3E-3 ug/L

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o ORAL DOSE-RESPONSE DATA :

Tumor Type – gross tumors, all sites combined
Test Animals – rat/Long-Evans, male
Route – drinking water
Reference – Schroeder and Mitchener, 1975a

Administered ppm	Dose (mg/kg)/day	Human Equivalent Dose (mg/kg/day)	Tumor Incidence
0	0	0	4/26
5	0.54	0.09	9/33

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o ADDITIONAL COMMENTS :

The solubility and speciation of beryllium in air and water media vary, with ambient air characterized by relatively insoluble beryllium compounds such as beryllium oxide and metallic beryllium, and water characterized by

more soluble forms. Carcinogenic potency varies according to the form of beryllium present.

Human equivalent doses were calculated using a human body weight of 70 kg, an animal weight of 0.325 kg and length of exposure, experiment and lifespan of 1126 days for treated and control animals.

The unit risk should not be used if the water concentration exceeds 8.3E+1 ug/L, since above this concentration the unit risk may not be appropriate.

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o DISCUSSION OF CONFIDENCE :

The estimate is derived from a study which did not show a significant increase in tumorigenic response. While this study is limited by use of only one non-zero dose group, the occurrence of high mortality and unspecified type and site of the tumors, it was used as the basis of the quantitative estimate because exposure occurred via the most relevant route. Oral risk estimates derived by extrapolation from studies in other species/strains for the intravenous and inhalation routes (also highly uncertain) are within an order of magnitude.

CARI -
o CLASSIFICATION : B2; probable human carcinogen.
o BASIS FOR CLASSIFICATION : Beryllium has been shown to induce lung cancer via inhalation in rats and monkeys and to induce osteosarcomas in rabbits via intravenous or intramedullary injection. Human epidemiology studies are considered to be inadequate.
o INHALATION UNIT RISK : 2.4E-3 per (ug/cu.m)
o DOSE EXTRAPOLATION METHOD : Relative risk
o RISK/AIR CONCENTRATIONS :

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	4E-2 ug/cu.m
E-5 (1 in 100,000)	4E-3 ug/cu.m
E-6 (1 in 1,000,000)	4E-4 ug/cu.m

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o INHALATION DOSE-RESPONSE DATA :

Tumor Type –
Test Animals – humans
Route – inhalation, occupational exposure
Reference –

Beryllium Concentration	Fraction	95 percent Effective	Upper-bound	Unit

in Workplace (ug/cu.m)	of Lifetime	dose (ug/cu.m)	Estimate of Relative Risk	Risk /ug/cu.m
100	1.00	21.92	1.98	1.61E-3
			2.09	1.79E-3
	0.25	5.48	1.98	6.44E-3
			2.09	7.16E-3
1000	1.00	219.18	1.98	1.61E-4
			2.09	1.79E-4
	0.25	54.79	1.98	6.44E-4
			2.09	7.16E-4

o ADDITIONAL COMMENTS :

Human data were used for the inhalation exposure quantitation despite limitations in the study. Humans are most likely to be exposed by inhalation to beryllium oxide, rather than other beryllium salts. Animal studies by inhalation of beryllium oxide have utilized intratracheal instillation, rather than general inhalation exposure.

Effective dose was determined by adjusting for duration of daily (8/24 hours) and annual (240/365 days) exposure, and the fraction of the lifetime at risk (i.e., time from onset of employment to termination of follow-up). The risk estimates were based on the data of Wagoner et al. (1980) in which the smoking adjusted, expected lung cancer deaths were found to range from 13.91 to 14.67, in comparison to 20 observed. Relative risk estimates of 1.36 and 1.44 were derived and the 95% confidence limits of these estimates, 1.98 and 2.09, respectively, were used to estimate the lifetime cancer risk. Note that all of the above estimates are based on one data set using a range of estimated exposure and exposure times. Because of uncertainties regarding workplace beryllium concentration and exposure duration, unit risks were derived using two estimates each of concentration, fraction of lifetime exposed and relative risk. The recommended value is the arithmetic mean of the 8 derived unit risks.

The unit risk should not be used if the air concentration exceeds 4 ug/cu.m, since above this concentration the unit risk may not be appropriate.

o DISCUSSION OF CONFIDENCE :

The estimate of risk for inhalation exposure was based upon an epidemiologic study having several confounding variables. The estimates of exposure levels and duration were also somewhat uncertain. While a quantitative assessment based on several animal studies resulted in a similar estimate of risk (which increases the confidence somewhat), the quality of the available studies was poor (that is, they were conducted at single dose levels or lacked control groups)

CARDR-
o CARCINOGENICITY SOURCE :

Source Document – U.S. EPA, 1986, 1991

Source Document Review – The values in 1986 Health Assessment Document for Beryllium and the 1991 Drinking Water Cntena Document for Beryllium received Agency and external review.

Other EPA Documentation – None
DOCUMENT

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o REVIEW DATES : 05/04/88, 02/01/89, 12/07/89
o VERIFICATION DATE : 05/04/88 (inhalation); 02/01/89 (oral)
o EPA CONTACTS :

William Pepelko / OHEA – (202)260-5904

David Bayliss / OHEA – (202)260-5726

HAONE- NO DATA

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA -

Considers technological or economic feasibility? – YES

Discussion – Beryllium was listed as a hazardous air pollutant under section 112 of the CAA in 1971 on the basis that it can cause the chronic lung disease berylliosis. Emission standards promulgated for extraction, ceramic, and propellant plants, foundries, incinerators, and machine shops are 10 g/24 hr or attainment of an ambient concentration near the source of 0.01 ug/cu.m, 30 day average. This ambient concentration was judged adequate to protect the public health with an ample margin of safety. More complex standards were also promulgated for beryllium rocket motor firing. The NESHAPs are now under review, and will consider new health evidence that beryllium may be a carcinogen. Reporting of releases of massive forms of this hazardous substance is not required if the diameter of the pieces released exceeds 100 micrometers (0.004 inches).

Reference – 40 CFR Part 61, Subparts C & D

EPA Contact – Emissions Standards Division, OAQPS
(917)541-5571 / FTS 629-5571

WQCHU-

Water and Fish Consumption: 6.8E-3 ug/L

Fish Consumption Only: 1.17E-1 ug/L

Considers technological or economic feasibility? – NO

Discussion – For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. However, zero may not be attainable at this time, so the recommended criterion represent a E-6 estimated incremental increase of cancer risk over a lifetime.

Reference – 45 FR 79318 (11/28/80); Quality Criteria for Water, EPA 440/5-86-001 (5/87).

EPA Contact – Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute LEC – 1.3E+2 ug/L
Chronic LEC – 5.3E+0 ug/L

Marine: None

Considers technological or economic feasibility? – NO

Discussion – The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LECs are given when the minimum data required to derive water quality criteria are not available. Hardness has a substantial effect on acute toxicity.

Reference – 45 FR 79318 (11/28/80)

EPA Contact – Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value – 0 mg/L (Proposed, 1990)

Considers technological or economic feasibility? – NO

Discussion – The proposed MCLG for beryllium is zero based on the evidence of carcinogenic potential (B2).

Reference – 55 FR 30370 (07/25/90)

EPA Contact – Health and Ecological Criteria Division / OST / (202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value – 0.001 mg/L (Proposed, 1990)

Considers technological or economic feasibility? – YES

Discussion – The MCL is based on 5x the MDL, which is associated with a maximum lifetime individual risk of 1 E-4.

Monitoring requirements – Ground water systems every 3 years; surface water systems annually; will allow monitoring at up to 10-year intervals after the system completes 3 rounds of sampling at <50% of the MCL.

Analytical methodology – Atomic absorption/furnace technique (EPA 210.2; ASTM D-3645; SM 304); inductively-coupled plasma (EPA 200.7; SM 305); ICP mass spectrometry (EPA 200.8): PQL= 0.001 mg/L.

Best available technology – Activated alumina;ion exchange; reverse osmosis; lime softening; coagulation/filtration.

Reference – 55 FR 30370 (07/25/90)

EPA Contact – Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

Status – Listed (Final, 1991)

Discussion – "Unregulated" contaminants are those contaminants for which EPA establishes a monitoring requirement but which do not have an associated final MCLG, MCL, or treatment technique. EPA may regulate these contaminants in the future.

Monitoring requirement – All systems to be monitored unless a vulnerability assessment determines the system is not vulnerable.

Analytical methodology – Atomic absorption/furnace technique (EPA 210.2; SM 304; ASTM D-3645); inductively coupled plasma (EPA 200.7; SM 305); spectrophotometric
(EPA 200.8).

Reference – 56 FR 3526 (01/30/91)

EPA Contact – Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

SMCL - NO DATA

FISTD- NO DATA

FIREV- NO DATA

CERC -

Value (status) – 10 pounds (Final, 1989)

Considers technological or economic feasibility? – NO

Discussion – The RQ for beryllium is based on potential carcinogenicity. Available data indicate a hazard ranking of medium based on a potency factor of 79.70/mg/kg/day and a weight-of-evidence group B2, which correspond to an RQ of 10 pounds. Reporting of releases of massive forms of this hazardous substance is not required if the diameter of the pieces released exceeds 100 micrometers (0.004 inches).

Reference – 54 FR 33418 (08/14/89)

EPA Contact – RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

SARA - NO DATA

RCRA -

Status – Listed

Reference – 52 FR 25942 (07/09/87)

EPA Contact – RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - Cox, G.E., D.E. Bailey and K. Morgareidge. 1975. Chronic feeding studies with beryllium sulfate in rats. Unpublished report submitted by the Food and Drug Research Laboratories, Inc., to the Aluminum Company of America, Pittsburgh, PA.

OREF - Puzanova, L., M. Doskocil and A. Doubkova. 1978. Disturbances of the development of chick embryos after the administration of beryllium chloride at early stages of embryogenesis. Folia. Morphologica. 26(3): 228-231.

OREF - Schroeder, H.A. and M. Mitchener. 1975. Life-term studies in rats: Effects of aluminum, barium, beryllium and tungsten. J. Nutr. 105: 421-427.

OREF - U.S. EPA. 1985. Drinking Water Criteria Document for Beryllium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.

IREF - None

CREF - Bayliss, D.L. 1980. U.S. EPA, Washington, DC. Letter to William H. Foege, M.D., Center for Disease Control, Atlanta, GA. November 12.

CREF - Bayliss, D.L. and J.K. Wagoner. 1977. Bronchogenic cancer and cardio-respiratory disease mortality among white males employed in a beryllium production facility. OSHA Beryllium Hearing, 1977, Exhibit 13.F.

CREF - Cloudman, A.M., D. Vining, S. Barkulis and J.J. Nickson. 1949. Bone changes following intravenous injections of beryllium. Am. J. Pathol. 25: 810-811.

CREF - Gardner, L.U. and H.F. Heslington. 1946. Osteo-sarcoma from intravenous beryllium compounds in rabbits. Fed. Proc. 5: 221. (Cited in U.S. EPA, 1987)

CREF - Groth, D.H., C. Kommineni and G.R. Mackay. 1980. Carcinogenicity of beryllium hydroxide and alloys. Environ. Res. 21(1): 63-84.

CREF - Hsie, A.W., J.P. O'Neill, J.R. San Sebastian, et al. 1979. Quantitative mammalian cell genetic toxicology: Study of the cytotoxicity and mutagenicity of seventy individual environmental agents related to energy technologies and three subfractions of crude synthetic oil in the CHO/HGPRT system. Environ. Sci. Res. 15: 219-315.

CREF - Ishinishi, N., M. Mizunoe, T. Inamasu and A. Hisanga. 1980. Experimental study on carcinogenicity of beryllium oxide and arsenic trioxide to the lung of rats by an intratracheal instillation. Fukuoka Igaku Zasshi. 71(1): 19-26. (Jap. with Eng. abstract)

CREF - Larramendy, M.L., N.C. Popescu and J.A. DiPaola. 1981. Induction by inorganic metal salts of sister chromatid exchanges and chromosome aberrations in human and Syrian hamster cell strains. Environ. Mutagen. 3: 597-606.

CREF - MacMahon, B. 1977. Evaluation of epidemiological materials. January 10, 1978. Brush Wellman, Cleveland, OH. OSHA Beryllium Hearings: 5.

CREF - MacMahon, B. 1978. OSHA Beryllium Hearings, comment on recent post-hearing submissions. Docket No. H005. February 9. 1979.
CREF - Mancuso, T.F. 1970. Relation of duration of employment and prior respiratory illness to respiratory cancer among beryllium workers. Environ. Res. 3: 251-275.
CREF - Mancuso, T.F. 1979. Occupational lung cancer among beryllium workers in dusts and disease. In: Proc. Conference on Occupational Exposure to Fibrous and Particulate Dust and Their Extension into the Environment, R. Lemen and J. Dement, Ed. Pathrotox Publishers, Inc.
CREF - Mancuso, T.F. 1980. Mortality study of beryllium industry workers' occupational lung cancer. Environ. Res. 21: 48-55.
CREF - Miyaki, M., N. Akamatsu, T. Ono, H. Koyama. 1979. Mutagenicity of metal cations in cultured cells from chinese hamster. Mutat. Res. 68: 259-263.
CREF - Morgareidge, K., G.E. Cox, D.E. Bailey and M.A. Gallo. 1977. Chronic oral toxicity of beryllium in the rat. Toxicol. Appl. Pharmocol. 41(1): 204-205.
CREF - Reeves, A.L., D. Deitch, and A.J. Vorwald. 1967. Beryllium carcinogenesis: I. Inhalation exposure of rats to beryllium sulfate aerosol. Cancer Res. 27(1): 439-445.
CREF - Schepers, G.W.H. 1961. Neoplasia experimentally induced by beryllium compounds. Prog. Exp. Tumor Res. 2: 203-244.
CREF - Schroeder, H.A. and M. Mitchener. 1975a. Life-term studies in rats: Effects of aluminum, barium, beryllium and tungsten. J. Nutr. 105: 421-427.
CREF - Schroeder, H.A. and M. Mitchener. 1975b. Life-term effects of mercury, methyl mercury and nine other trace metals on mice. J. Nutr. 105: 452-458.
CREF - Simmon, V.F., H.S. Rosenkranz, E. Zeiger and L.A. Poirier. 1979. Mutagenic activity of chemical carcinogens and related compounds in the intraperitoneal host-mediated assay. J. Natl. Cancer Inst. 62(4): 911-918.
CREF - U.S. EPA. 1986. Health Assessment Document for Beryllium. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA 600/8-84-026F.
CREF - U.S. EPA. 1991. Drinking Water Criteria Document for Beryllium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.
CREF - Vorwald, A.J. 1968. Biologic manifestations of toxic inhalants in monkeys. In: Use of Nonhuman Primates in Drug Evaluation, H. Vagtborg, Ed. University of Texas Press, Austin, TX. p. 222-228.
CREF - Wagner, W.D., D.H. Groth, J.L. Holtz, G.E. Madden and H.E. Stokinger. 1969. Comparative chronic inhalation toxicity of beryllium ores, bertrandite and beryl, with production of pulmonary tumors by beryl. Toxicol. Appl. Pharmacol. 15: 10-29.
CREF - Wagoner, J.K., P.F. Infante and D.L. Bayliss. 1980. Beryllium: An etiologic agent in the induction of lung cancer, nonneoplastic respiratory disease, and heart disease among industrially exposed workers. Environ. Res. 21: 15-34.
HAREF- None

[IRIS] SS 4 /cf?
USER:

gamma-Chlordane

1 - IRIS
NAME - Chlordane
RN - 57-74-9
IRSN - 139
DATE - 930701
UPDT - 07/01/93, 6 fields
STAT - Oral RfD Assessment (RDO) on-line 07/01/89
STAT - Inhalation RfC Assessment (RDI) pending
STAT - Carcinogenicity Assessment (CAR) on-line 07/01/93
STAT - Drinking Water Health Advisones (DWHA) on-line 08/01/90
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 09/30/87 CAR Carcinogenicity section added
IRH - 03/01/88 RDO Dose conversion clarified
IRH - 03/01/88 RDO Text clarified in paragraph 3
IRH - 03/01/88 CAREV Basis for classification clarified
IRH - 03/01/88 HADV Health Advisory added
IRH - 04/01/89 RDO Withdrawn; new RfD verified (in preparation)
IRH - 06/01/89 RDO Revised oral RfD summary added
IRH - 06/01/89 REFS Bibliography on-line
IRH - 07/01/89 RDO Reference clarified in paragraph 2
IRH - 07/01/89 CAR Velsicol (1983) references clarified
IRH - 07/01/89 CREF Carcinogen references added
IRH - 03/01/90 RDI Inhalation RfD now under review
IRH - 08/01/90 HALIF DWEL changed reflecting change in RfD
IRH - 08/01/90 HADR Primary contact changed
IRH - 08/01/90 RCRA EPA contact changed
IRH - 01/01/91 CAR Text edited
IRH - 01/01/91 CARI Inhalation slope factor removed (global change)
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 07/01/93 CARDR Secondary contact's phone number changed
RLEN - 28857
SY - Belt
SY - CD 68
SY - Chlordane
SY - Chlorindan
SY - Chlor Kil
SY - Corodan
SY - Dowchlor
SY - ENT 9.932
SY - HCS 3260
SY - Kypchlor
SY - M 140
SY - M 410
SY - 4,7-Methanoindan, 1,2,4,5,6,7,8,8-Octachloro-3a,4,7,7a-Tetrahydro-
SY - 4,7-Methano-1H-Indene,
1,2,4,5,6,7,8,8-Octachloro-2,3,3a,4,7,7a-Hexahydro-
SY - NCI-C00099
SY - Niran
SY - Octachlorodihydrodicyclopentadiene
SY - 1,2,4,5,6,7,8,8-Octachloro-2,3,3a,4,7,7a-Hexahydro-4,7-Methano-indene
SY - 1,2,4,5,6,7,8,8-Octachloro-3a,4,7,7a-Hexahydro-4,7-Methylene Indane
SY - Octachloro-4,7-Methanohydroindane
SY - Octachloro-4,7-Methanotetrahydroindane
SY - Octa-Klor
SY - Oktaterr

SY - Ortho-Klor
SY - Synklor
SY - TAT Chlor 4
SY - Topiclor
SY - Toxichlor
SY - Velsicol 1068

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Regional liver hypertrophy in females	NOEL: 1 ppm (0.055 mg/kg/day)	1000	1	6E-5 mg/kg/day
30-Month Rat Feeding Study	LEL: 5 ppm (0.273 mg/kg/day)			
Velsicol Chemical Co., 1983a				

*Conversion Factors: Actual dose tested

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o ORAL RFD STUDIES :

Velsicol Chemical Company. 1983a. MRID No. 00138591, 00144313. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Charles River Fischer 344 rats (80/sex/dose) were fed technical chlordane at dietary levels of 0, 1, 5, and 25 ppm for 130 weeks. Body weight, food consumption, and water uptake were monitored at regular intervals. Clinical laboratory studies were performed and organ weights measured on eight animals/sex/group at weeks 26 and 52, and on all survivors at week 130. Gross and microscopic pathology were performed on all tissues. Daily dose level of 0.045, 0.229, and 1.175 mg/kg/day for males and 0.055, 0.273, and 1.409 mg/kg/day for females for the 1, 5, and 25 ppm treatment groups, respectively, were calculated from food consumption and body weight data.

Following the submission of a 30-month chronic feeding/oncogenicity study in Fischer 344 rats, the Agency reviews by the Office of Pesticides Programs and the Cancer Assessment Group of these data indicated that male rats at the highest dosage exhibited an increase in liver tumors (ICF Clement, 1987). The registrant, Velsicol Chemical Company, subsequently convened the Pathology Working Group to reevaluate the slides of livers of the chlordane-treated rats reported in MRID No. 00138591. It was concluded that liver lesions had not occurred in male rats and that 25 ppm (0.1175 mg/kg/day) was the NOEL for males. Liver lesions (hypertrophy), however, had occurred in female rats at 5 ppm (0.273 mg/kg/day), which was considered an LEL. Therefore an NOEL of 1 ppm (0.055 mg/kg/day) (LDT) was established for female rats.

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o ORAL RFD UNCERTAINTY :

UF – An uncertainty factor of 100 was used to account for the inter- and intraspecies differences. An additional UF of 10 was used to account for the lack of an adequate reproduction study and adequate chronic study in a second

mammalian species, and the generally inadequate sensitive endpoints studied in existing studies, particularly since chlordane is known to bioaccumulate over a chronic duration.

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o ORAL RFD MODIFYING FACTOR :

MF – None

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o ORAL RFD COMMENTS :

Data Considered for Establishing the RfD

1) 30-Month Feeding (oncogenic) - rat: Principal study - see previous description; core grade minimum

2) 24-Month Chronic Toxicity - mouse: NOEL=1 ppm (0.15 mg/kg/day); LEL=5 ppm (0.75 mg/kg/day) (hepatocellular swelling and necrosis in males; hepatocyte swelling in males, and increased live weight in males and females); At 12.5 ppm (1.875 mg/kg/day) (HDT); core grade minimum (Velsicol Chemical Co., 1983b)

Data Gap(s): Chronic Dog Feeding Study, Rat Reproduction Study, Rat Teratology Study, Rabbit Teratology Study

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o ORAL RFD CONFIDENCE :

Study – Medium
Data Base – Low
RfD – Low

The critical study is of adequate quality and is given a medium rating. The data base is given a low confidence rating because of 1) the lack of an adequate reproduction study and adequate chronic study in a second mammalian species and 2) inadequate sensitive endpoints studied in existing studies, particularly since chlordane is known to bioaccumulate over a chronic duration. Low confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

Source Document – This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation – Pesticide Registration Standard, November 1986; Pesticide Registration Files

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o REVIEW DATES : 12/18/85, 03/22/89
o VERIFICATION DATE : 03/22/89
o EPA CONTACTS :

George Ghali / OPP – (703)557-7490

William Burnam / OPP – (703)557-7491

RDI -
o INHALATION RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

o REVIEW DATES : 02/22/90
CAREV-
o CLASSIFICATION : B2; probable human carcinogen
o BASIS FOR CLASSIFICATION : Sufficient evidence in studies in which benign and malignant liver tumors were induced in four strains of mice of both sexes and in F344 male rats; structurally related to other liver carcinogens
o HUMAN CARCINOGENICITY DATA :

Inadequate. There were 11 case reports involving central nervous system effects, blood dyscrasias and neuroblastomas in children with pre-/postnatal exposure to chlordane and heptachlor (Infante et al., 1978). As no other information was available, no conclusions can be drawn.

There were three epidemiologic studies of workers exposed to chlordane and/or heptachlor. One study of pesticide applicators was considered inadequate in sample size and duration of follow-up. This study showed marginal statistically significant increased mortality from bladder cancer (3 observed) (Wang and McMahon, 1979a). The other two studies were of pesticide manufacturing workers. Neither of them showed any statistically significantly increased cancer mortality (Wang and McMahon, 1979b; Ditraglia et al., 1981). Both these populations also had confounding exposures from other chemicals.

o ANIMAL CARCINOGENICITY DATA :

Sufficient. Chlordane has been studied in four mouse and four rat long-term carcinogenesis bioassays. Dose-related incidences of liver carcinoma constitute the major finding in mice. Becker and Sell (1979) tested chlordane (90:10 mixture of chlordane to heptachlor) in C57B1/6N mice, a strain historically known not to develop spontaneous liver tumors. An unspecified number of mice were fed chlordane at 0, 25 and 50 ppm (0, 3.57, 7.14 mg/kg bw) for 18 months. None of the controls developed tumors or nodular lesions of the liver. Twenty-seven percent (16 mice) of the surviving treated mice developed primary hepatocellular carcinomas. Velsicol (1973) fed groups of 100 male and 100 female CD-1 mice diets with 0, 5, 25 or 50 ppm analytical grade chlordane for 18 months. A significant ($p<0.01$) dose-related increase in nodular hyperplasias in the liver of male and female mice was reported at the the two highest dose levels. A histological review by Reuber (U.S. EPA, 1985) reported a high incidence ($p<0.01$) of hepatic carcinomas instead of hyperplastic nodules at 25 and 50 ppm.

A dose-related increase ($p<0.001$ after lifetable adjustment) of hepatocellular carcinomas was also observed in both sexes of B6C3F1 mice (NCI, 1977). Male and female mice were fed technical-grade chlordane (purity\= 94.8%) at TWA concentrations (TWAC) of 29.9 and 56.2 ppm and 30.1 and 63.8 ppm, respectively, for 80 weeks. In this study there were individual matched controls for the low and high dose groups. ICR male mice developed

hepatocellular adenomas and hemangiomas when fed 12.5 ppm chlordane for 24 months. No tumors were observed in the female mice when tested at the same concentrations: 0, 1, 5, and 12.5 ppm (Velsicol, 1983a).

Velsicol (1983b) reported a long-term (130 weeks) carcinogenesis bioassay on 80 male and 80 female F344 rats fed concentrations of 0, 1, 5, and 25 ppm chlordane. A significant increase in adenomas of the liver was observed in male rats receiving 25 ppm. Although no tumors were observed in female rats, hepatocellular swelling was significantly increased at 25 ppm. The NCI (1977) reported a significant increase ($p<0.05$) of neoplastic nodules of the liver in low-dose Osborne-Mendel female rats (TWAC of 120.8 ppm) but not in the high-dose group (TWAC of 241.5 ppm). No tumor incidence was reported for the males fed TWAC of 203.5 and 407 ppm. Loss of body weight and a dose-related increase in mortality was observed in all treated groups. High mortality and reduced growth rates in Osborne-Mendel rats was also observed by Ingle (1952) when the rats were exposed to 150 and 300 ppm chlordane but not at 5, 10, and 30 ppm. No treatment-related incidence of tumors was reported. Significantly enlarged livers and liver lesions were found in male and female albino rats fed chlordane at greater than or equal to 80 ppm (Ambrose et al., 1953a,b). No treatment-related increase in tumors was found, but the study duration (400 days) was short.

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o SUPPORTING DATA :

Gene mutation assays indicate that chlordane is not mutagenic in bacteria (Wildeman and Nazar, 1982; Probst et al., 1981; Gentile et al., 1982). Positive results have been reported in Chinese hamster lung V79 cells and mouse lymphoma L5178Y cells with and without exogenous metabolism, as well as in plant assays. Chlordane did not induce DNA repair in bacteria, rodent hepatocytes (Maslansky and Williams, 1981), or human lymphoid cells (Sobti et al., 1983). It is a genotoxicant in yeast (Gentile et al., 1982; Chambers and Dutta, 1976), human fibroblasts (Ahmed et al., 1977), and fish (Vigfusson et al., 1983).

Five compounds structurally related to chlordane (aldrin, dieldrin, heptachlor, heptachlor epoxide, and chlorendic acid) have produced liver tumors in mice. Chlorendic acid has also produced liver tumors in rats.

CARO -
o CLASSIFICATION : B2; probable human carcinogen
o BASIS FOR CLASSIFICATION : Sufficient evidence in studies in which benign and malignant liver tumors were induced in four strains of mice of both sexes and in F344 male rats; structurally related to other liver carcinogens
o ORAL SLOPE FACTOR : 1.3E+0 per (mg/kg)/day
o DRINKING WATER UNIT RISK : 3.7E-5 per (ug/L)
o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk
o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	3E+0 ug/L
E-5 (1 in 100,000)	3E-1 ug/L
E-6 (1 in 1,000,000)	3E-2 ug/L

o ORAL DOSE-RESPONSE DATA :

Tumor Type – hepatocellular carcinoma
Test Animals – mouse/CD-1 (Velsicol); mouse/B6C3F1 (NCI)
Route – diet
Reference – Velsicol, 1973; NCI, 1977

Administered Dose (ppm)	Human Equivalent Dose (mg/kg-day)	Tumor Incidence	Reference
female			
0	0.000	0/45	Velsicol,
5	0.052	0/61	1973
25	0.260	32/50	
50	0.520	26/37	
male			
0	0.000	3/33	Velsicol,
5	0.052	5/55	1973
25	0.260	41/52	
50	0.520	32/39	
male			
0	0.00	2/18	NCI, 1977
29.9	0.31	16/48	
56.2	0.58	43/49	
female			
0	0.00	0/19	NCI, 1977
30.1	0.31	3/47	
63.8	0.66	34/49	

o ADDITIONAL COMMENTS :

Four data sets for mice and one data set for rats showed a significant increase in liver tumors; namely hepatocellular carcinomas in mice (NCI, 1977; Velsicol, 1973) and hepatocellular adenomas in rats (Velsicol, 1983a). The quantitative estimate is based on the geometric mean from the four mouse data sets as mice were the more sensitive species tested and as risk estimates for a similar compound (heptachlor) were similarly derived from mouse tumor data. The slope factors for the data sets are these: 2.98 per (mg/kg)/day for CD-1 female mice, 4.74 per (mg/kg)/day for CD-1 male mice, 0.76 per (mg/kg)/day for B6C3F1 male mice, and 0.25 per (mg/kg)/day for B6C3F1 female mice. Low and high dose groups in the NCI (1977) study had individual matched controls.

The unit risk should not be used if the water concentration exceeds 300 ug/L, since above this concentration the unit risk may not be appropriate.

o DISCUSSION OF CONFIDENCE :

Liver carcinomas were induced in mice of both sexes in two studies. An

adequate number of animals was observed. and dose-response effects were reported in all studies. The geometric mean of slope factors (0.25 to 4 74 per (mg/kg)/day for the most sensitive species is consistent with that derived from rat data (1.11/mg/kg/day).

CARI -
o CLASSIFICATION : B2; probable human carcinogen
o BASIS FOR CLASSIFICATION : Sufficient evidence in studies in which benign and malignant liver tumors were induced in four strains of mice of both sexes and in F344 male rats; structurally related to other liver carcinogens
o INHALATION UNIT RISK : 3.7E-4 per (ug/cu.m)
o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk
o RISK/AIR CONCENTRATIONS :

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	3E-1 ug/cu.m
E-5 (1 in 100,000)	3E-2 ug/cu.m
E-6 (1 in 1,000,000)	3E-3 ug/cu.m

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o INHALATION DOSE-RESPONSE DATA :

The inhalation risk estimates were calculated from the oral data presented in CARO.

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o ADDITIONAL COMMENTS :

The unit risk should not be used if the air concentration exceeds 30 ug/cu.m, above this concentration the unit risk may not be appropriate.

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o DISCUSSION OF CONFIDENCE :

See CARO.

CARDR-
o CARCINOGENICITY SOURCE :

Source Document – U.S. EPA, 1986, 1985

The values in the 1986 Carcinogenicity Assessment for Chlordane and Heptachlor/Heptachlor Epoxide have been reviewed by the Carcinogen Assessment Group.

DOCUMENT

o REVIEW DATES 04/01/87
o VERIFICATION DATE 04/01/87
o EPA CONTACTS :

Dharm V. Singh / OHEA – (202)260-5958

Jim Cogliano / OHEA – (202)260-3814

HAONE-

Appropriate data for calculating a One-day HA are not available. It is recommended that the Ten-day HA of 0.06 mg/L be used as the One-day HA.

HATEN-

Ten-day HA – 6E-2 mg/L

LOAEL – 6.25 mg/kg/day
UF – 1000 (allows for interspecies and intrahuman variability with the use of a LOAEL from an animal study)
Assumptions – 1 L/day water consumption for a 10-kg child

Principal Study – Ambrose et al., 1953

The toxic effects in rats resulting from daily gastric intubation of chlordane at doses of 6.25, 12.5, 25.0, 50.0, 100.0, or 200 mg/kg for 15 days were histologic changes in the liver of the treated animals at all dose levels and central nervous system effects at higher dose levels. Only minimal histopathologic changes characterized by the presence of abnormal intra-cytoplasmic bodies of various diameters were evident at the lowest dose level (6.25 mg/kg). That dose level was identified as the LOAEL in this study.

HALTC-

Appropriate data for calculating a Longer-term HA are not available. It is recommended that the modified DWEL (adjusted for a 10-kg child) of 0.5 ug/L be used as the Longer-term HA.

HALTA-

Appropriate data for calculating a Longer-term HA are not available. It is recommended that the DWEL of 2 ug/L be used as the Longer-term HA for the 70-kg adult.

HALIF-

Drinking Water Equivalent Level (DWEL) – 2E-3 mg/L

Assumptions – 2 L/day water consumption for a 70-kg adult

RfD Verification Date = 03/22/89 (see RDO)

Lifetime HA – None

Chlordane is considered to be a probable human carcinogen. Refer to CAR for information on the carcinogenicity of this substance.

Principal Study (DWEL) – Velsicol Chemical Corporation, 1983 (This study was used in the derivation of the chronic oral RfD; see RDO)

OLEP -

No data available

ALAB -

Determination of chlordane is by a liquid-liquid extraction gas chromatographic procedure.

TREAT-

Treatment technologies which are capable of removing chlordane from drinking water include adsorption by granular or powdered activated carbon and air stripping.

HADR -
o HEALTH ADVISORY SOURCE :

U.S. EPA. 1985. Final Draft of the Drinking Water Criteria Document on Chlordane. Office of Drinking Water, Washington, DC.
DOCUMENT

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o HEALTH ADVISORY REVIEW :

EPA review of HAs in 1985.

Public review of HAs following notification of availability in October, 1985.

Scientific Advisory Panel review of HAs in January, 1986.

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o EPA DRINKING WATER CONTACT :

Jennifer Orme Zavaleta / OST – (202)260-7586

Edward V. Ohanian / OST – (202)260-7571

CAA - NO DATA

WQCHU-

Water and Fish Consumption: 4.6E-4 ug/L

Fish Consumption Only: 4.8E-4 ug/L

Considers technological or economic feasibility? – NO

Discussion – For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. However, zero may not be obtainable at this time, so the recommended criteria represents an E-6 estimated incremental increase of cancer risk over a lifetime.

Reference – 45 FR 79318 (11/28/80)

EPA Contact – Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute – 2.4 E+ 0 ug/L (at any time)
Chronic – 4.3 E- 3 ug/L (24-hour average)

Marine:

Acute – 9.0 E-2 ug/L (at any time)
Chronic – 4.0 E-3 ug/L (24-hour average)

Considers technological or economic feasibility? – NO

Discussion – Criteria were derived from a minimum data base consisting of acute and chronic tests on a variety of species. Requirements and methods are covered in the reference to the Federal Register.

Reference – 45 FR 79318 (11/28/80)

EPA Contact – Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value (status) – 0 mg/L (Final, 1991)

Considers technological or economic feasibility? – NO

Discussion – An MCLG of 0 mg/L for chlordane is promulgated based upon carcinogenic effects (B2).

Reference – 56 FR 3526 (01/30/91)

EPA Contact – Health and Ecological Criteria Division / OST / (202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value – 0.002 mg/L (Final, 1991)

Considers technological or economic feasibility? – YES

Discussion – EPA has set a MCL equal to the PQL of 0.002, which is associated with a lifetime individual risk of 1.5 E-4.

Monitoring requirements – All systems monitored for four consecutive quarters every three years; repeat monitoring dependent upon detection, vulnerability status and system size.

Analytical methodology – Microextraction/gas chromatography (EPA 505); electron-capture/gas chromatography (EPA 508); gas chromatography/mass spectrometry (EPA 525); PQL= 0.002 mg/L.

Best available technology – Granular activated carbon

Reference – 56 FR 3526 (01/30/91)

EPA Contact – Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

SMCL - NO DATA

FISTD-

Status – Issued (1986)

Reference – Chlordane Pesticide Registration Standard. December, 1986 (NTIS No. PB87-175816).

EPA Contact – Registration Branch, OPP / (703)557-7760 / FTS 557-7760

FIREV-

Action – Cancellation of many termiticide products (1988)

Considers technological or economic feasibility? – YES

Summary of regulatory action – 43 FR 12372 (03/24/78) - Cancellation of all but termiticide use; under the provisions of the Administrator's acceptance of the settlement plan to phase out certain uses of chlordane, most registered products containing chlordane were effectively canceled or the applications for registration were denied by 12/31/80. A summary of those uses not affected by this settlement, or a previous suspension, follows: 1) subsurface ground insertion for termite control (clarified by 40 FR 30522, July 21, 1975, to apply to the use of emulsifiable or oil concentrate formulations for controlling subterranean termites on structural sites such as buildings, houses, barns, and sheds, using current control practices), 2) dipping of nonfood roots and tops./52 FR 42145 (11/03/87) - Negotiated agreement on termiticide use. The agreement (order) accepted voluntary cancellations of the registration of certain pesticide products and imposed limitations on the continued sale, distribution, and use of existing stocks of such products/ criterion of concern: oncogenicity,

Reference – 43 FR 12372 (03/24/78); 52 FR 42145 (11/03/87); 53 FR 11798 (04/08/88)

EPA Contact – Special Review Branch / OPP
(703)557-7400 / FTS 557-7400

CERC -

Value (status) – 1 pound (Final, 1989)

Considers technological or economic feasibility? – NO

Discussion – The RQ for chlordane is 1 pound, based on aquatic toxicity, as established under CWA Section 311 (40 CFR 117.3). Available data indicate the aquatic 96-hour Median Threshold Limit for chlordane is less than 0.1 ppm. This corresponds to an RQ of 1 pound. Chlordane has also been found to bioaccumulate in the tissues of aquatic and marine organisms, and has the potential to concentrate in the food chain.

Reference – 54 FR 33418 (08/14/89)

EPA Contact – RCRA/Superfund Hotline

(800)424-9346 / (202)260-3000 / FTS 260-3000

SARA - NO DATA

RCRA -

Status – Listed

Reference – 52 FR 25942 (07/09/87)

EPA Contact – RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - ICF-Clement. 1987. MRID No. 40433701. Available from EPA. Write to FOI, EPA, Washington DC 20460.

OREF - Velsicol Chemical Co. 1983a. MRID No. 00138591, 00144313. Available from EPA. Write to FOI, EPA, Washington DC 20460.

OREF - Velsicol Chemical Co. 1983b. MRID No. 00144312. Available from EPA. Write to FOI, EPA, Washington DC 20460.

IREF - None

CREF - Ahmed, F.E., R.W. Hart and N.J. Lewis. 1977. Pesticide induced DNA damage and its repair in cultured human cells. Mutat. Res. 42: 161-174.

CREF - Ambrose, A.M., H.E. Christensen, D.J. Robbins and L.J. Rather. 1953a. Toxi- cological and pharmacological studies on chlordane. Arch. Ind. Hyg. Occup. Med. 7: 197-210.

CREF - Ambrose, A.M., H.E. Christensen and D.J. Robbins. 1953b. Pharmacological observations on chlordane. Fed. Proceed. 12: 298. (Abstract #982)

CREF - Becker, F.F. and S. Sell. 1979. Fetoprotein levels and hepatic alterations during chemical carcinogenesis in C57BL/6N mice. Cancer Res. 39: 3491-3494.

CREF - Chambers, D. and S.K. Dutta. 1976. Mutagenic tests of chlordane on different microbial tester strains. Genetics. 83: s13. (Abstract)

CREF - Ditraglia, D., D.P. Brown, T. Namekata and N. Iverson. 1981. Mortality study of workers employed at organochlorine pesticide manufacturing plants. Scand. J. Work Environ. Health. 7(4): 140-146.

CREF - Gentile, J.M., G.J. Gentile, J. Bultman, R. Sechriest, E.D. Wagner and M.J. Plewa. 1982. An evaluation of the genotoxic properties of insecticides following plant and animal activation. Mutat. Res. 101: 19-29.

CREF - Infante, P.F., S.S. Epstein and W.A. Newton. 1978. Blooddyscrasis and childhood tumors and exposure to chlordane and heptachlor. Scand. J. Work Environ. Health. 4: 137-150.

CREF - Ingle. L. 1952. Chronic oral toxicity of chlordan to rats. Arch. Ind. Hyg. Occup. Med. 6. 357-367.

CREF - Maslansky, C.J. and G.M. Williams. 1981. Evidence for an epigenetic mode of action in organochlorine pesticide hepatocarcinogenicity: A lack of genotoxicity in rat, mouse, and hamster hepatocytes. J. Toxicol. Environ. Health. 8: 121-130.

CREF - NCI (National Cancer Institute). 1977. Bioassay of Chlordane for possible Carcinogenicity. NCI Carcinogenesis Tech. Rep. Ser. No. 8. U.S. DHEW Publ. No. (NIH) 77-808. Bethesda, MD.

CREF - Probst, G.S., R.E. McMahon, L.E. Hill, C.Z. Thompson, J.K. Epp and S.B. Neal. 1981. Chemically-induced unscheduled DNA synthesis in primary rat hepatocyte cultures: A comparison with bacterial mutagenicity using 218 compounds. Environ. Mutagen. 3: 11-31.

CREF - Sobti, R.C., A. Krishan and J. Davies. 1983. Cytokinetic and cytogenetic effect of agricultural chemicals on human lymphoid cells in vitro. Arch. Toxicol. 52: 221-231.

CREF - U.S. EPA. 1985. Hearing Files on Chlordane, Heptachlor Suspension (unpub- lished draft). Available for inspection at U.S. EPA, Washington, DC.

CREF - U.S. EPA. 1986. Carcinogenicity Assessment of Chlordane and Heptachlor/ Heptachlor Epoxide. Prepared by the Office of Health and Environmental Assessment, Carcinogen Assessment Group, Washington, DC.

CREF - Velsicol Chemical Corporation. 1973. MRID No. 00067568. Available from EPA. Write to FOI, EPA, Washington, DC. 20460.

CREF - Velsicol Chemical Corporation. 1983a. MRID No. 00144312, 00132566. Available from EPA. Write to FOI, EPA, Washington, DC. 20460.

CREF - Velsicol Chemical Corporation. 1983b. MRID No. 00138591. Available from EPA. Write to FOI, EPA, Washington, DC. 20460.

CREF - Vigfusson, N.V., E.R. Vyse, C.A. Pernsteiner and R.J. Dawson. 1983. In vivo induction of sister-chromatid exchange in Umbra limi by the insecticides endrin, chlordane, diazinon and guthion. Mutat. Res. 118: 61-68.

CREF - Wang, H.H. and B. MacMahon. 1979a. Mortality of workers employed in the manufacture of chlordane and heptachlor. J. Occup. Med. 21(11): 745-748.

CREF - Wang, H.H. and B. MacMahon. 1979b. Mortality of pesticide applicators. J. Occup. Med. 21(11): 741-744.

CREF - Wildeman, A.G. and R.N. Nazar. 1982. Significance of plant metabolism in the mutagenicity and toxicity of pesticides. Can. J. Genet. Cytol. 24: 437-449.

HAREF- Ambrose, A.M., H.E. Christensen, D.J. Robbins and L.J. Rather. 1953. Toxi- cological and pharmacological studies on chlordane. Arch. Ind. Hyg. Occup. Med. 7: 197-210.

HAREF- U.S. EPA. 1985. Final Draft of the Drinking Water Criteria Document on Chlordane. Office of Drinking Water, Washington, DC.

HAREF- Velsicol Chemical Corp. 1983. MRID No. 00138591. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Chlorobenzene

1 - IRIS
NAME - Chlorobenzene
RN - 108-90-7
IRSN - 386
DATE - 930701
UPDT - 07/01/93, 3 fields
STAT - Oral RfD Assessment (RDO) on-line 07/01/93
STAT - Inhalation RfC Assessment (RDI) pending
STAT - Carcinogenicity Assessment (CAR) on-line 03/01/91
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 08/01/89 RDO Oral RfD summary on-line
IRH - 08/01/89 REFS Bibliography on-line
IRH - 06/01/90 RDI Inhalation RfD now under review
IRH - 11/01/90 RDO Text edited
IRH - 11/01/90 CAR Carcinogen assessment on-line
IRH - 11/01/90 CREF Carcinogen references added
IRH - 03/01/91 RDO EPA contacts changed
CONTINUE PRINTING? (YES/NO/CONT)
USER:
c

IRH - 03/01/91 CARDR EPA contacts changed
IRH - 01/01/92 EXSR Regulatory actions on-line
IRH - 07/01/93 RDO Source document review statement clarified
RLEN - 20409
SY - BENZENE CHLORIDE
SY - BENZENE, CHLORO-
SY - CHLOORBENZEEN (Dutch)
SY - CHLORBENZENE
SY - CHLORBENZOL
SY - CHLOROBENZEN (Polish)
SY - CHLOROBENZENE
SY - CHLOROBENZENU (Czech)
SY - CHLOROBENZOL
SY - CLOROBENZENE (Italian)
SY - MCB
SY - MONOCHLOORBENZEEN (Dutch)
SY - MONOCHLORBENZENE
SY - MONOCHLORBENZOL (German)
SY - MONOCHLOROBENZENE
SY - MONOCLOROBENZENE (Italian)
SY - NCI-C54886
SY - PHENYL CHLORIDE
SY - UN 1134

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Histopathologic changes in liver	NOAEL: 27.25 mg/kg/day (adjusted dose:	1000	1	2E-2 mg/kg/day

19 mg/kg/day)
13-Week Dog Study.
Oral Exposure (capsule) LOAEL: 54 5 mg/kg/day

Monsanto Co., 1967a.
Knapp et al., 1971

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*Conversion Factors: Doses adjusted for dosing schedule of 5 days/7 days.

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o ORAL RFD STUDIES :

Monsanto Company. 1967a. 13-week oral administration - dogs. Monchlorobenzene. Final report. Prepared by Hazelton Laboratories, Project No. 241-105, February 24.

Knapp, W.K., W.M. Busey and W. Kundzins. 1971. Subacute oral toxicity of monochlorobenzene in dogs and rats. Toxicol. Appl. Pharmacol. 19: 393 (Abstract).

Male and female beagle dogs given chlorobenzene orally by capsule at doses of 27.25, 54.5, or 272.5 mg/kg/day, 5 days/week, for 13 weeks. NOAEL = 27.25 mg/kg/day; LOAEL = 54.5 mg/kg/day (slight bile duct proliferation, cytologic alternations, and leukocytic infiltration of the stroma, all in liver).
Death; body weight loss; changes in hematology, clinical chemistry, and urine analysis; and pathologic changes in liver (bile duct hyperplasia, cytologic changes, leukocytic infiltration, centrolobular degeneration), kidney, gastrointestinal mucosa, and hematopoietic tissue were observed at 272.5 mg/kg/day.

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o ORAL RFD UNCERTAINTY :

UF -- The UF of 1000 allows for uncertainty in the extrapolation of dose levels from laboratory animals to humans (10A), uncertainty in the threshold for sensitive humans (10H), and uncertainty in the effect of duration when extrapolating from subchronic to chronic exposure (10S).

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o ORAL RFD MODIFYING FACTOR :

MF -- None

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o ORAL RFD COMMENTS :

Other Data Reviewed:

1) 90-Day Feeding - rat: NOAEL=50 mg/kg/day; LOAEL=100 mg/kg/day; (increased liver and kidney weights) (Monsanto Co., 1967b; Knapp et al., 1971)

2) 6-Month Gavage - rat: NOAEL=14.4 mg/kg/day; LOAEL=144 mg/kg/day; (liver histopathology) (Irish, 1963)

3) 90-Day Gavage - rat: NOAEL=60 mg/kg/day; LOAEL=125 mg/kg/day; (increased liver weights) (NTP, 1985)

4) 90-Day Gavage - mouse: NOAEL=60 mg/kg/day; LOAEL=125 mg/kg/day; (increased

liver weights) (NTP, 1985)

5) 2-Year Gavage - rat: NOAEL=60 mg/kg/day; LOAEL=120 mg/kg/day; (liver histopathology in rats) (NTP, 1985)

6) 2-Year Gavage - mouse: NOAEL=60 mg/kg/day; LOAEL=120 mg/kg/day; (liver histopathology in rats) (NTP, 1985)

7) Developmental - rat: Developmental NOAEL=590 ppm (6 hour/day, equivalent to 216 mg/kg/day) (exposed by inhalation during periods of major organogenesis); Maternal NOAEL=210 ppm (77 mg/kg/day) (John et al., 1984)

8) Developmental - rabbit: Developmental NOAEL=590 ppm (6 hour/day, equivalent to 125 mg/kg/day; (exposed by inhalation during periods of major organogenesis); Maternal NOAEL=75 ppm (16 mg/kg/day) (John et al., 1984)

9) 2-Generation Reproduction (inhalation) - rat: Reproductive NOAEL=450 ppm (165 mg/kg/day); Systemic NOAEL=50 ppm (18 mg/kg/day) (Nair et al., 1987)

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o ORAL RFD CONFIDENCE :

Study -- Medium
Data Base -- Medium
RfD -- Medium

The referenced subchronic study was given medium confidence since it provided both a NOAEL and a LOAEL and incorporated several biochemical and biological endpoints. Several subchronic, chronic, developmental, and reproductive toxicity studies provide supportive data, but they did not give a complete assessment of toxicity. Thus, the data base was given medium confidence. A medium level of confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

Source Document -- U.S. EPA, 1986

U.S. EPA, 1986 received EPA Science Advisory Board Reveiw 7/86.

Other EPA Documentation -- None

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o REVIEW DATES : 06/24/85, 07/08/85, 07/22/85, 04/16/87, 01/19/89.
o VERIFICATION DATE : 01/19/89
o EPA CONTACTS :

Charles Abernathy / OST -- (202)260-5374

Edward V. Ohanian / OST -- (202)260-7571

RDI -

o INHALATION RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

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o REVIEW DATES : 06/23/88, 05/16/90, 06/21/90
CAREV-
o CLASSIFICATION : D; not classifiable as to human carcinogenicity
o BASIS FOR CLASSIFICATION : No human data, inadequate animal data and predominantly negative genetic toxicity data in bacterial, yeast, and mouse lymphoma cells.
o HUMAN CARCINOGENICITY DATA :

None.

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o ANIMAL CARCINOGENICITY DATA :

Inadequate. Only one study, an NTP (1985a) gavage study in rats and mice has been performed. Groups of F344/N rats and B6C3F1 mice (50/sex/dose) were administered chlorobenzene (>99% pure) by gavage in corn oil 5 days/week for 103 weeks. Fifty animals/sex/species served as untreated and vehicle controls. The rats received 60 or 120 mg/kg/day; the female mice groups received 60 or 120 mg/kg/day and the male mice groups 30 or 60 mg/kg/day. Body weights in all groups of both species were comparable.

At the end of the study survival in the male rats was 68, 78, 64, and 52% for the untreated-control, vehicle-control, low-, and high-dose groups, respectively; survival in the female rat groups was 74, 58, 60, and 62% for the untreated-control, vehicle-control, low-, and high-dose groups, respectively. Using the pairwise comparison test, only the high-dose male rats had a statistically significant elevation in mortality relative to their vehicle controls. No chlorobenzene-related signs of clinical toxicity were observed in the rats during the experiment; histological examination of the liver showed hepatocellular necrosis (graded as minimal to mild in all groups) caused by chlorobenzene. A statistically significant positive trend in the incidence of hepatocellular neoplastic nodules was observed in male rats; the incidence was 4/50, 2/50, 4/49 and 8/49 for the untreated-control, vehicle-control, low-, and high-dose groups, respectively. No hepatocellular tumors were observed in dosed male rats and none of the high-dose males with neoplastic nodules showed signs of hepatocellular necrosis. There was no increase in neoplastic nodules, hepatocellular carcinomas or combined neoplastic nodules and hepatocellular carcinomas in females. In the high-dose females, there was a single incidence of hepatocellular carcinoma. The incidence of neoplastic liver nodules in female rats was 1/49, 0/50, 1/50, and 1/50 in the untreated-control, vehicle-control, low-, and high-dose groups, respectively. No other significant increase in site-specific tumors or neoplastic pathology was observed in rats; however, a transitional cell bladder papilloma was observed in one low-dose and in one high-dose male rat and a kidney tubular cell carcinoma was observed in a high-dose female. These rare tumor types were not observed in the untreated or vehicle control groups.

In the female mice, survival was approximately 78% in all groups. In male

mice, however, survival was reduced in both dosed groups: it was 70, 78, 56, and 58 in the untreated-control, vehicle-control, low- and high-dose groups, respectively. The mortality in the dosed males was statistically significantly increased. No chlorobenzene-related clinical toxicity and no significant increase in site-specific tumors or neoplastic pathology were observed in any mouse group.

In 1986, the Environmental Health Committee of the Science Advisory Board reviewed this study; overall, the Committee considered the study to be of good quality, but the Comittee questioned the biological significance of the increased incidence of neoplastic liver nodules (Doull and Abrahamson, 1986). The NTP report does not specify the number of liver sections pathologically examined or which sections of the liver were examined; the location of the liver sections could cause the difference in the number and the diagnosis of lesions formed. Liver nodules are currently not considered necessarily to be progressive, and consequently lethal to the host. In this study, there was no incidence of hepatocellular carcinoma in male rats after 2 years of the study.

The incidence of liver nodules in the untreated control male rats, however, was significantly higher than the recent historical NTP controls (67/3618, 1.9%).

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o SUPPORTING DATA :

Chlorobenzene was not mutagenic for Salmonella typhimurium strains TA98, TA100, TA1535, TA1537 or TA1538, with or without addition of rat liver or hamster liver homogenate (duPont, 1977; Lawlor et al., 1979; Merck, 1978; Monsanto, 1976a; NTP, 1982; Simmon et al., 1979). Chlorobenzene did not induce DNA damage in Escherichia coli strains WP2 uvr A+ rec A+ or WP100 uvr A- rec A- or S. typhimurium strains TA1978 uvr B+ or TA1538 uvr B- (Lawlor et al., 1979; Simmon et al., 1979). Chlorobenzene caused increases in the number of revertants in Actinomycetes antibioticus-400 (Keskinova, 1968) and Aspergillus nidulans (Prasad, 1970; Prasad and Pramer, 1968) and mitotic disturbances in Allium cepa (Ostergen and Levan, 1943). Chlorobenzene did not induce specific locus forward mutations in mouse lymphoma L5178Y cells, either with or without metabolic activation (Monsanto, 1976b). Chlorobenzene induced reciprocal recombination in Saccharomyces cerevasiae strain D3 with metabolic activation (Simmon et al., 1979), but was ineffective in S. cerevesiae strain D4 with or without metabolic activation (Monsanto, 1976a).

In carcinogenesis studies with 1,2-dichlorobenzene and 1,3-dichlorobenzene in rats and mice (NTP, 1985b, 1987), 1,3-dichlorobenzene induced kidney tumors in male rats and liver tumors in both sexes of mice. Neither compound induced liver tumors in rats.

The NTP (1985a) study speculated that chlorobenzene was related to the carcinogen, benzene, because of similarities in structure, metabolism and hematological effects in rodents; and therefore, a toxic human response could be predicted based upon a rodent model.

CARO - NO DATA
CARI - NO DATA
CARDR-
o CARCINOGENICITY SOURCE :

Source Document -- U.S. EPA, 1986, 1989

The Health Effects Criteria Document on Chlorobenzene by the Office of Drinking Water has undergone public review and Science Advisory Board review (1986).

The proposed drinking water standard for chlorobenzene has been reviewed by EPA and the public (1989 Federal Register notice).
DOCUMENT

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o REVIEW DATES : 04/04/90
o VERIFICATION DATE : 04/04/90
o EPA CONTACTS :

Charles Abernathy / OST -- (202)260-5374

Julie Du / OST -- (202)260-7583

HAONE- NO DATA

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA - NO DATA

WQCHU-

Water and Fish Consumption: 4.88E+0 ug/L

Fish Consumption Only: None

Considers technological or economic feasibility? -- NO

Discussion -- To control undesirable taste and odor qualities of ambient water, the estimated concentration is 2.0E+1 ug/L. There is no demonstrated

relationship between organoleptic endpoints and adverse health effects.

Reference --45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute LEC -- 2.5E+2 ug/L
Chronic LEC -- 5.0E+1 ug/L

Marine:

Acute LEC -- 1.6E+2 ug/L
Chronic LEC -- 1.29E+2 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LECs are given when the minimum data required to derive water quality criteria are not available. The criteria are based on chlorinated benzenes as a class.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value -- 0.1 mg/L (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion -- A MCLG of 0.1 mg/L is promulgated based on potential adverse effects (hepatic toxicity) reported in a subchronic study of dogs. The MCLG is based upon a DWEL of 0.7 mg/L and an assumed drinking water contribution of 20 percent.

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value – 0 1 mg/L (Final, 1991)

Considers technological or economic feasibility? – YES

Discussion – EPA has promulgated a MCL equal to the MCLG of 0.1 mg/L.

Monitoring requirements – All systems initially monitored for four consecutive quarters; repeat monitoring dependent upon detection, vulnerability status and system size.

Analytical methodology – Capillary column gas chromatography/mass spectrometry (EPA 524.2). PQL= 0.005 mg/L.

Best available technology – Granular activated carbon; packed tower aeration.

Reference – 56 FR 3526 (01/30/91); 56 FR 30266 (07/01/91)

EPA Contact – Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED"CONTAMINANTS

Status – Listed (Final, 1991)

Discussion – "Unregulated" contaminants are those contaminants for which EPA establishes a monitoring requirement but which do not have an associated final MCLG, MCL, or treatment technique. EPA may regulate these contaminants in the future.

Monitoring requirement – Monitoring required for all water systems at a minimum frequency of once every 5 years.

Analytical methodology – Gas chromatography (EPA 502.1, 502.2, 503.1); gas chromatographic/mass spectrometry (EPA 524.1, 524.2).

Reference – 56 FR 25690 (07/08/87)

EPA Contact – Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

SMCL - NO DATA

FISTD- NO DATA

FIREV- NO DATA

CERC -

Value (status) -- 100 pounds (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ for chlorobenzene is based on aquatic toxicity as established under CWA Section 311 (40 CFR 117.3). The available data indicate that the aquatic 96-Hour Median Threshold Limit is 1-10 ppm, which corresponds to an RQ of 100 pounds.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

SARA - NO DATA

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - Irish, D.D. 1963. Halogenated hydrocarbons. II. Cyclic. In: Patty's Industrial Hygiene and Toxicology, Vol. II., D.W. Fassett and D.D. Irish, Ed. Interscience Publishers, New York, NY. p. 1333-1362.

OREF - John, J.A., W.C. Hayes, T.R. Hanley, Jr., K.A. Johnson, T.S. Gushow and K.S. Rao. 1984. Inhalation teratology study on monochlorobenzene in rats and rabbits. Toxicol. Appl. Pharmacol. 76: 365-373.

OREF - Knapp, W.K., W.M. Busey and W. Kundzins. 1971. Subacute oral toxicity of monochlorobenzene in dogs and rats. Toxicol. Appl. Pharmacol. 19: 393. (Abstract).

OREF - Monsanto Chemical Company. 1967a. 13-week oral administration -- dogs. Monochlorobenzene. Final report. Prepared by Hazelton Laboratories, Project No. 241-105, February 24.

OREF - Monsanto Chemical Company. 1967b. Three-month subacute oral study – rats. Monchlorobenzene. Final report. Prepared by Hazelton Laboraties. Project No. 241-104. March 9.
OREF - Nair, R.S., J.A. Barter, R.E. Schroeder, A. Knezevich and C.R. Stack. 1987. A two-generation reproduction study with monochlorobenzene vapor in rats. Fund. Appl. Toxicol. 9: 678-686.
OREF - NTP (National Toxicology Program). 1985. Toxicology and carcinogenesis studies of chlorobenzene (CAS No. 108-90-7) in F344/N rats and B6C3Fl mice (gavage studies). U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health. NTP No. 261. NIH Publ. No. 83-2517.
OREF - U.S. EPA. 1986. Drinking Water Criteria Document for Chlorobenzene. Office of Drinking Water, Washington, DC.
IREF - None
CREF - E.I. duPont deNemours and Company. 1977. Mutagenic activity of monochlorobenzene in the Salmonella-microsome assay. Haskell Laboratory for Toxicology and Industrial Medicine. Unpublished report.
CREF - Doull, J. and S. Abrahamson. 1986. Chair, Halogenated Organics Subcommittee of Science Advisory Board U.S. EPA and Vice-Chair Halogenated Organics Subcommittee of Science Advisory Board U.S. EPA. Memorandum to R.A. Griesemer, Chair Environmental Health Committee of Science Advisory Board, U.S. EPA. Technical comments of the Halogenated Organics Subcommittee on EPA's Draft Drinking Water Criteria Document for Monochlorobenzene. December 16.
CREF - Keskinova, D.V. 1968. The effect of dimethylcyclodiazomethane in chlorobenzene solution on the process of mutagenesis in Actinomyces antibioticus-400. Genetika. 4(8): 121-125.
CREF - Lawlor, T., S.R. Haworth and P. Voytek. 1979. Evaluation of the genetic activity of nine chlorinated phenols, seven chlorinated benzenes, and three chlorinated hexanes. Environ. Mutagen. 1: 143. (Abstract)
CREF - Merck and Company. 1978. Summary of monochlorobenzene bacterial mutagen test (Ames Test). Office of Pesticide and Toxic Substances, U.S. EPA, Washington, DC. TSCA Sec 8(d) submission 8DHQ-1078-0302.
CREF - Monsanto Company. 1976a. Mutagenicity evaluation of B10-76-86-CP-5535 (WGK). Final report. LBI Project No. 2457. Litton Bionetics, Kensington, MD. Unpublished.
CREF - Monsanto Company. 1976b. Mutagenicity evaluation of B10-76-86-CP-5535 (LOX). Final report. LBI Project No. 2547. Litton Bionetics, Kensington, MD. Unpublished.
CREF - NTP (National Toxicology Program). 1982. Environmental Mutagen Test Development Program, Research Triangle Park, NC. (Cited in NTP, 1985a)
CREF - NTP (National Toxicology Program). 1985a. Toxicology and carcinogenesis studies of chlorobenzene (CAS No. 108-90-7) in F344/N rats and B6C3F1 mice (gavage studies). U.S. Department of Health and Human Services. Public Health Service. National Institutes of Health. NTP No. 261. NIH Publ. No. 83-2517.
CREF - NTP (National Toxicology Program). 1985b. Toxicology and carcinogenesis studies of 1,2-dichlorobenzene (CAS No. 95-50-1) in F344/N rats and B6C3F1 mice (gavage studies). NTP TR 235. NIH Publ. No. 86-2511.
CREF - NTP (National Toxicology Program). 1987. Toxicology and carcinogenesis studies of 1,4-dichlorobenzene (CAS No. 106-46-7) in F344/N rats and B6C3F1 mice (gavage studies). NTP TR 319. NIH Publ. No. 86-2575.
CREF - Ostergren, G. and A. Levin. 1943. The connection between c-mitotic activity and water solubility in some monocyclic compounds. Hereditas.

29: 496-498.
CREF - Prasad, I. 1970. Mutagenic effects of the herbicide 3.4-dichloro-ropionanilide and its degradation products. Can. J. Biochem. 16: 369-372.
CREF - Prasad, I. and D. Pramer. 1968. Mutagenic activity of some chloroanilines and chlorobenzenes. Genetics. 60: 212-213.
CREF - Simmon, V.F., E.C. Ricco and M.V. Pierce. 1979. In vitro microbiological genotoxicity tests of chlorobenzene, m-dichlorobenzene, o-dichlorobenzene and p-dichlorobenzene. Final report. SRI International, Menlo Park, CA. Unpublished.
CREF - U.S. EPA. 1986. Draft Health Effects Criteria Document for Chlorobenzene. Criteria and Standards Division, Office of Drinking Water, Washington, DC.
CREF - U.S. EPA. 1989. National Primary and Secondary Drinking Water Regulations; Proposed Rule. Monochlorobenzene. Federal Register. 54(97): 22087-22088.
HAREF- None

[IRIS] SS 2 /cf?
USER:

bis(Chloroethyl)ether

1 - IRIS
NAME - Bis(chloroethyl)ether (BCEE)
RN - 111-44-4
IRSN - 134
DATE - 940207
UPDT - 02/07/94, 3 fields
STAT - Oral RfD Assessment (RDO) no data
STAT - Inhalation RfC Assessment (RDI) message 10/01/91
STAT - Carcinogenicity Assessment (CAR) on-line 02/01/94
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 03/01/88 CARO Text revised
IRH - 03/01/88 CARI Text revised
IRH - 02/01/89 WQCHU Reference added
IRH - 11/01/89 REFS Bibliography on-line
IRH - 01/01/91 CAR Text edited
IRH - 01/01/91 CARI Inhalation slope factor removed (global change)
IRH - 08/01/91 RDI Inhalation RfC now under review
CONTINUE PRINTING? (YES/NO/CONT)
USER:
c

IRH - 08/01/91 RCRA EPA contact changed
IRH - 10/01/91 RDI Inhalation RfC message on-line
IRH - 10/01/91 IREF Inhalation RfC references added
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 02/01/94 CARDR Primary contact's phone number changed
RLEN - 10625
SY - BCEE
SY - beta,beta'-DICHLOROETHYL ETHER
SY - Bis(chloroethyl)ether
SY - BIS(2-CHLOROETHYL) ETHER
SY - BIS(beta-CHLOROETHYL) ETHER
SY - CHLOREX
SY - 1-CHLORO-2-(beta-CHLOROETHOXY)ETHANE
SY - CHLOROETHYL ETHER
SY - CLOREX
SY - DCEE
SY - 2,2'-DICHLOORETHYLETHER
SY - 2,2'-DICHLOR-DIAETHYLAETHER
SY - 2,2'-DICHLORETHYL ETHER
SY - beta,beta-DICHLORODIETHYL ETHER
SY - DICHLOROETHER
SY - DICHLOROETHYL ETHER
SY - DI(2-CHLOROETHYL) ETHER
SY - 2,2'-DICHLOROETHYL ETHER
SY - DI(beta-CHLOROETHYL)ETHER
SY - sym-DICHLOROETHYL ETHER
SY - DICHLOROETHYL OXIDE
SY - 2,2'-DICLOROETILETERE
SY - DWUCHLORODWUETYLOWY ETER
SY - ENT 4,504

SY - ETHANE, 1,1'-OXYBIS(2-CHLORO-
SY - ETHER, BIS(2-CHLOROETHYL)
SY - ETHER DICHLORE
SY - 1,1'-OXYBIS(2-CHLORO)ETHANE
SY - OXYDE DE CHLORETHYLE
SY - RCRA WASTE NUMBER U025
SY - UN 1916

RDO - NO DATA

RDI -
o INHALATION RFD SUMMARY :

The health effects data for bis(chloroethyl)ether have been reviewed by the RfD/RfC Work Group and determined to be inadequate for derivation of an inhalation RfC. The verification status of this chemical is currently not verifiable. For additional information on health effects of this chemical interested parties are referred to the EPA documentation listed below.

U.S. EPA. 1980. Ambient Water Quality Criteria Document for Chloroalkyl Ethers. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards Criteria and Standards Division, Washington, DC. EPA 440/5-80-030. NTIS PB81-117418/AS.

U.S. EPA. 1987. Health Effects Assessment for Bis(2-chloroethyl)ether. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH. EPA/600/8-88/023.

---

o REVIEW DATES : 07/17/91
CAREV-
o CLASSIFICATION : B2; probable human carcinogen
o BASIS FOR CLASSIFICATION : Positive carcinogenicity results in two strains of mice and evidence of mutagenicity
o HUMAN CARCINOGENICITY DATA :

None.

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o ANIMAL CARCINOGENICITY DATA :

BCEE was marginally sarcomatogenic at s.c. injection sites in female ICR/Ha mice (Van Duurren et al., 1968). Innes et al. (1969) administered BCEE by gavage to two hybrid mouse strains, (C57Bl/6 x C3H/Anf)F1 and (C57Bl/6 x AKR)F1. Groups of 18 mice/sex/strain began treatment with 100 mg/kg at 7 days of age. At 4 weeks, after weaning, exposure was continued through the diet. Concentrations in diet were calculated so as to deliver the maximally tolerated dose (300 ppm), and treatment was continued for 18 months. Increased evidence of hepatomas were noted in male and female (C57Bl/6 x C3H/Anf)F1 mice and in male (C57Bl/6 x AKR)F1 mice.

By contrast to the above, Theiss et al. (1977) saw no increases in pulmonary tumors in strain A mice given repeated i.p. injections of BCEE. Futhermore, an NCI study (for which statistical analyses were not available) indicated that BCEE was not carcinogenic for Charles River rats by the oral

route (Ulland et al., 1973; U.S. EPA, 1980).

o SUPPORTING DATA :

BCEE is a direct-acting mutagen producing base pair exchange mutations in E. coli, S. typhimurium, and B. subtilis (Shirasu et al., 1975). Vapor-phase exposure of Salmonella frameshift mutant strains TA1538 and TA98 also resulted in a weak positive response (Fishbein, 1977). BCEE is mutagenic in S. cerevisiae (Fishbein, 1977) but did not induce heritable translocations in mice (Jorgenson et al., 1977). BCEE is structurally related to bis(chloromethyl)ether (BCME), a human carcinogen.

CARO -

o CLASSIFICATION : B2; probable human carcinogen
o BASIS FOR CLASSIFICATION : Positive carcinogenicity results in two strains of mice and evidence of mutagenicity
o ORAL SLOPE FACTOR : 1.1E+0 per (mg/kg)/day
o DRINKING WATER UNIT RISK : 3.3E-5 per (ug/L)
o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk
o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	3E+0 ug/L
E-5 (1 in 100,000)	3E-1 ug/L
E-6 (1 in 1,000,000)	3E-2 ug/L

o ORAL DOSE-RESPONSE DATA :

Tumor Type -- hepatomas
Test Animals -- mouse/(C57B1/6 x C3H/AnF)F1, male
Route -- gavage followed by diet
Reference -- Innes et al., 1969

Administered Dose (mg/kg/day)	Human Equivalent Dose (mg/kg/day)	Tumor Incidence
0	0	8/79
39	2.94	14/16

o ADDITIONAL COMMENTS :

The tumor incidence in control mice is derived from pooling the tumor incidence of four untreated control groups (8/63) and one treated (gavaged with gelatin suspension) control group (0/16).

The unit risk should not be used if the water concentration exceeds 3E+2 ug/L, since above this concentration the unit risk may not be appropriate.

o DISCUSSION OF CONFIDENCE :

A small number of animals was treated at only one dose.

CARI -
o CLASSIFICATION : B2; probable human carcinogen
o BASIS FOR CLASSIFICATION : Positive carcinogenicity results in two strains of mice and evidence of mutagenicity
o INHALATION UNIT RISK : 3.3E-4 per (ug/cu.m)
o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk
o RISK/AIR CONCENTRATIONS :

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	3E-1 ug/cu.m
E-5 (1 in 100,000)	3E-2 ug/cu.m
E-6 (1 in 1,000,000)	3E-3 ug/cu.m

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o INHALATION DOSE-RESPONSE DATA :

The inhalation risk estimates were calculated from the oral exposure data in CARO.

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o ADDITIONAL COMMENTS :

The unit risk should not be used if the air concentration exceeds 3E+1 ug/cu.m, since above this concentration the unit risk may not be appropriate.

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o DISCUSSION OF CONFIDENCE :

See CARO.

CARDR-
o CARCINOGENICITY SOURCE :

Source Document -- U.S. EPA, 1980

The 1980 Ambient Water Quality Criteria Document for Chloroalkyl Ethers received extensive peer and public review.
DOCUMENT

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o REVIEW DATES : 07/23/86
o VERIFICATION DATE : 07/23/86
o EPA CONTACTS :

Robert E. McGaughy / OHEA -- (202)260-5889

Herman J. Gibb / OHEA – (202)260-5898

HAONE- NO DATA

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA - NO DATA

WQCHU-

Water and Fish Consumption – 3.0E-2 ug/L

Fish Consumption Only – 1.36E+0 ug/L

Considers technological or economic feasibility? – NO

Discussion – For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. However, zero may not be attainable at this time, so the recommended criteria represents a E-6 estimated incremental increase of cancer risk over a lifetime. The values given represent chloroalkyl ethers as a class.

Reference – 45 FR 79318 (11/28/80); NTIS No. PB81-117418

EPA Contact – Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute LEC – 2.38E+5 ug/L
Chronic – None

Marine: None

Considers technological or economic feasibility? – NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LECs are given when the minimum data required to derive water quality criteria are not available. The values given represent chloroalkyl ethers as a class. The values given represent chloroalkyl ethers as a class.

Reference -- 45 FR 79318 (11/28/80); NTIS No. PB81-117418

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG - NO DATA

MCL - NO DATA

SMCL - NO DATA

FISTD- NO DATA

FIREV- NO DATA

CERC -

Value (status) -- 10 pounds (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The RQ for BCEE is 10 pounds, based on its potential carcinogenicity. The available data indicate a hazard ranking of medium, based on a potency factor of 13.29/mg/kg/day and weight-of-evidence group B2, which corresponds to an RQ of 10 pounds.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

SARA - NO DATA

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - None
IREF - U.S. EPA. 1980. Ambient Water Quality Criteria Document for Chloroalkyl Ethers. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards Criteria and Standards Division, Washington, DC. EPA 440/5-80-030. NTIS PB81-117418/AS.
IREF - U.S. EPA. 1987. Health Effects Assessment for Bis(2-chloroethyl)ether. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH. EPA/600/8-88/023.
CREF - Fishbein, L. 1977. Potential industrial carcinogens and mutagens. U.S. EPA, Office of Toxic Substances, EPA 560/5-77-005. Washington, DC.
CREF - Innes, J.R.M., B.M. Ulland, M.G. Valerio, et al. 1969. Bioassay of pesticides and industrial chemicals for tumorigenicity in mice: A preliminary note. J. Natl. Cancer Inst. 42: 1101-1114.
CREF - Jorgenson, T.A., C.J. Rushbrook, G.W. Newell and R.G. Tardiff. 1977. Study of mutagenic potential of bis(2-chloroethyl) and bis(2-Chloroisopropyl)ethers in mice by the heritable translocation test. Toxicol. Appl. Pharmacol. 41: 196.
CREF - Shirasu, Y., M. Moriya, K. Kato and T. Kada. 1975. Mutagenicity screening of pesticides in microbial systems. Mutat. Res. 31: 268.
CREF - Theiss, J.C., G.D. Stoner, M.B. Shimkin and E.K. Weisburger. 1977. Test for carcinogenicity of organic contaminants of United States drinking waters by pulmonary tumor response in strain A mice. Cancer Res. 37: 2717.
CREF - Ulland, B., E.K. Weisburger and J.H. Weisburger. 1973. Chronic toxicity and carcinogenicity of industrial chemicals and pesticides. Toxicol. Appl. Pharmacol. 25: 446.
CREF - U.S. EPA. 1980. Ambient Water Quality Criteria Document for Chloroalkyl Ethers. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-030. NTIS PB 81-117418.
CREF - Van Duuren, B.L., B.M. Goldschmidt, C. Katz, L. Langseth, G. Mercado and A. Sivak. 1968. Alpha-haloethers: A new type of alkylating carcinogen. Arch. Environ. Health. 16: 472.
HAREF- None

[IRIS] SS 3 /cf?
USER:

Chromium(VI)

1 - IRIS
NAME - Chromium(VI)
RN - 18540-29-9
IRSN - 141
DATE - 950202
UPDT - 02/02/95, 1 field
STAT - Oral RfD Assessment (RDO) on-line 02/01/95
STAT - Inhalation RfC Assessment (RDI) pending
STAT - Carcinogenicity Assessment (CAR) on-line 03/01/91
STAT - Drinking Water Health Advisories (DWHA) on-line 03/01/88
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 04/01/92
IRH - 09/30/87 CAREV Citation corrected
IRH - 03/01/88 CARI Confidence statement revised
IRH - 03/01/88 CARDR Contacts switched
IRH - 03/01/88 HADV Health Advisory added
IRH - 12/01/89 RDI Inhalation RfD now under review
IRH - 06/01/90 CAREV Basis - Text revised
IRH - 06/01/90 CARO Text revised
IRH - 06/01/90 CAA Area code for EPA contact corrected
IRH - 06/01/90 RCRA EPA contact changed
IRH - 06/01/90 REFS Bibliography on-line
IRH - 01/01/91 CAR Text edited
IRH - 01/01/91 CARI Inhalation slope factor removed (global change)
IRH - 03/01/91 CAREV Text revised
IRH - 03/01/91 CAREV Text revised
IRH - 03/01/91 CARO Text revised
IRH - 01/01/92 RDO Secondary contact changed
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 04/01/92 CAA CAA regulatory action withdrawn
IRH - 09/01/94 RDO Work group review date added
IRH - 02/01/95 RDO RfD noted as pending change: Work Grp Mtg on 08/03/94
RLEN - 31280
SY - 7440-47-3
SY - CHROMIC ION
SY - CHROMIUM
SY - CHROMIUM, ION
SY - Chromium(VI)
SY - CHROMIUM (VI) ION

RDO -
o ORAL RFD SUMMARY :

NOTE: The Oral RfD for chromium(VI), soluble salts may change in the near future pending the outcome of a further review now being conducted by the RfD/RfC Work Group.

Critical Effect	Experimental Doses*	UF	MF	RfD
No effects reported	NOAEL: 25 mg/L of chromium as K2CrO4	500	1	5E-3 mg/kg/day
Rat, 1-Year Drinking Study	(converted to 2.4 mg of chromium(VI)/kg/day)			
MacKenzie et al.,	LOAEL: none			

1958

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*Conversion Factors: Drinking water consumption = 0.097 L/kg/day (reported)

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o ORAL RFD STUDIES :

MacKenzie, R.D., R.U. Byerrum, C.F. Decker, C.A. Hoppert and R.F. Langham. 1958. Chronic toxicity studies. II. Hexavalent and trivalent chromium administered in drinking water to rats. Am. Med. Assoc. Arch. Ind. Health. 18: 232-234.

Groups of eight male and eight female Sprague-Dawley rats were supplied with drinking water containing 0-11 ppm (0-11 mg/L) hexavalent chromium (as K_2CrO_4) for 1 year. The control group (10/sex) received distilled water. A second experiment involved three groups of 12 males and 9 female rats. One group was given 25 ppm (25 mg/L) chromium (as K_2CrO_4); a second received 25 ppm chromium in the form of chromic chloride; and the controls again received distilled water. No significant adverse effects were seen on appearance, weight gain, or food consumption, and there were no pathologic changes in the blood or other tissues in any treatment group. The rats receiving 25 ppm of chromium (as K_2CrO_4) showed an approximate 20% reduction in water consumption. This dose corresponds to 2.4 mg chromium(VI)/kg/day based on actual body weight and water consumption data.

For rats treated with 0-11 ppm (in the diet), blood was examined monthly, and tissues (livers, kidneys and femurs) were examined at 6 months and 1 year. Spleens were also examined at 1 year. The 25 ppm groups (and corresponding controls) were examined similarly, except that no animals were killed at 6 months. An abrupt rise in tissue chromium concentrations was noted in rats treated with greater than 5 ppm. The authors stated that "apparently, tissues can accumulate considerable quantities of chromium before pathological changes result." In the 25 ppm treatment groups, tissue concentrations of chromium were approximately 9 times higher for those treated with hexavalent chromium than for the trivalent group.

Similar no-effect levels have been observed in dogs and humans. Anwar et al. (1961) observed no significant effects in female dogs (2/dose group) given up to 11.2 ppm chromium(VI) (as K_2CrO_4) in drinking water for 4 years. The calculated doses were 0.012-0.30 mg/kg of chromium(VI). In humans, no adverse health effects were detected (by physical examination) in a family of four persons who drank for 3 years from a private well containing chromium(VI) at approximately 1 mg/L (0.03 mg/kg/day for a 70-kg human).

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o ORAL RFD UNCERTAINTY :

UF -- The uncertainty factor of 500 represents two 10-fold decreases in dose to account for both the expected interhuman and interspecies variability in the toxicity of the chemical in lieu of specific data, and an additional factor of 5 to compensate for the less-than-lifetime exposure duration of the principal study.

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o ORAL RFD MODIFYING FACTOR :

MF -- None

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o ORAL RFD COMMENTS :

This RfD is limited to metallic chromium(VI) of soluble salts. Examples of soluble salts include potassium dichromate (K2CR2O7), sodium dichromate (Na2Cr2O7), potassium chromate (K2CrO4) and sodium chromate (Na2CrO4).

Trivalent chromium is an essential nutrient. There is some evidence to indicate that hexavalent chromium is reduced in part to trivalent chromium in vivo (Petrilli and DeFlora, 1977, 1978; Gruber and Jennette, 1978).

The literature available on possible fetal damage caused by chromium compounds is limited. No studies were located on teratogenic effects resulting from ingestion of chromium.

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o ORAL RFD CONFIDENCE :

Study -- Low
Data Base -- Low
RfD -- Low

Confidence in the chosen study is low because of the small number of animals tested, the small number of parameters measured and the lack of toxic effect at the highest dose tested. Confidence in the data base is low because the supporting studies are of equally low quality, and teratogenic and reproductive endpoints are not well studied. Low confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

U.S. EPA. 1984. Health Effects Assessment for Hexavalent Chromium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.

U.S. EPA. 1985. Drinking Water Health Advisory for Chromium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. (Draft)

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o REVIEW DATES : 11/21/85, 02/05/86, 08/03/94
o VERIFICATION DATE : 02/05/86
o EPA CONTACTS :

Kenneth L. Bailey / OST -- (202)260-5535

Sue Velazquez / OHEA -- (513)569-7571

RDI -
o INHALATION RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

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o REVIEW DATES : 11/16/89, 09/20/90
CAREV-
o CLASSIFICATION : A; human carcinogen
o BASIS FOR CLASSIFICATION : Results of occupational epidemiologic studies of chromium-exposed workers are consistent across investigators and study populations. Dose- response relationships have been established for chromium exposure and lung cancer. Chromium-exposed workers are exposed to both chromium III and chromium VI compounds. Because only chromium VI has been found to be carcinogenic in animal studies, however, it was concluded that only chromium VI should be classified as a human carcinogen.
o HUMAN CARCINOGENICITY DATA :

Sufficient. Epidemiologic studies of chromate production facilities in the United States (Machle and Gregorius, 1948; Brinton et al., 1952; Mancuso and Hueper, 1951, Mancuso, 1975; Baetjer, 1950; Taylor, 1966; Enterline, 1974; Hayes et al., 1979; Hill and Ferguson, 1979), Great Britain (Bidstrup, 1951; Bidstrup and Case, 1956; Alderson et al., 1981), Japan (Watanabe and Fukuchi, 1975; Ohsaki et al., 1978; Sano and Mitohara, 1978; Satoh et al., 1981) and West Germany (Korallus et al., 1982; Bittersohl, 1971) have established an association between chromium (Cr) exposure and lung cancer. Most of these studies did not attempt to determine whether Cr III or Cr VI compounds were the etiologic agents.

Three studies of the chrome pigment industry, one in Norway (Langard and Norseth, 1975), one in England (Davies, 1978, 1979), and the third in the Netherlands and Germany (Frentzel-Beyme, 1983) also found an association between occupational chromium exposure (predominantly to Cr VI) and lung cancer.

Results of two studies of the chromium plating industry (Royle, 1975; Silverstein et al., 1981) were inconclusive, while the findings of a Japanese study of chrome platers were negative (Okubo and Tsuchiya, 1979). The results of studies of ferrochromium workers (Pokrovskaya and Shabynina, 1973; Langard et al., 1980; Axelsson et al., 1980) were inconclusive as to lung cancer risk.

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o ANIMAL CARCINOGENICITY DATA :

Sufficient. Hexavalent chromium compounds were carcinogenic in animal assays producing the following tumor types: intramuscular injection site tumors in Fischer 344 and Bethesda Black rats and in C57BL mice (Furst et al., 1976; Maltoni, 1974, 1976; Payne, 1960; Heuper and Payne, 1959); intra-plural implant site tumors for various chromium VI compounds in Sprague-Dawley and Bethesda Black rats (Payne, 1960; Heuper 1961; Heuper and Payne, 1962); intrabronchial implantation site tumors for various Cr VI compounds in Wistar rats (Levy and Martin, 1983; Laskin et al., 1970; Levy as quoted in NIOSH, 1975); and subcutaneous injection site sarcomas in Sprague-Dawley

rats (Maltoni, 1974, 1976).

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o SUPPORTING DATA :

A large number of chromium compounds have been assayed in in vitro genetic toxicology assays. In general, hexavalent chromium is mutagenic in bacterial assays whereas trivalent chromium is not (Lofroth, 1978; Petrellie and Flora, 1977, 1978). Likewise Cr VI but not Cr III was mutagenic in yeasts (Bonatti et al., 1976) and in V79 cells (Newbold et al., 1979). Chromium III and VI compounds decrease the fidelity of DNA synthesis in vitro (Loeb et al., 1977), while Cr VI compounds inhibit replicative DNA synthesis in mammalian cells (Levis et al., 1978) and produce unscheduled DNA synthesis, presumably repair synthesis, as a consequence of DNA damage (Raffetto, 1977). Chromate has been shown to transform both primary cells and cell lines (Fradkin et al., 1975; Tsuda and Kato, 1977; Casto et al., 1979). Chromosomal effects produced by treatment with chromium compounds have been reported by a number of authors; for example, both Cr VI and Cr III salts were clastogenic for cultured human leukocytes (Nakamuro et al., 1978).

There are no long-term studies of ingested Cr VI. There appears to be significant in vivo conversion of Cr VI to Cr III and III to VI; Cr III is an essential trace element.

CARO - NO DATA
CARI -
o CLASSIFICATION : A; human carcinogen
o BASIS FOR CLASSIFICATION : Results of occupational epidemiologic studies of chromium-exposed workers are consistent across investigators and study populations. Dose- response relationships have been established for chromium exposure and lung cancer. Chromium-exposed workers are exposed to both chromium III and chromium VI compounds. Because only chromium VI has been found to be carcinogenic in animal studies, however, it was concluded that only chromium
o INHALATION UNIT RISK : 1.2E-2 per (ug/cu.m)
o DOSE EXTRAPOLATION METHOD : Multistage, extra risk
o RISK/AIR CONCENTRATIONS :

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	8E-3 ug/cu.m
E-5 (1 in 100,000)	8E-4 ug/cu.m
E-6 (1 in 1,000,000)	8E-5 ug/cu.m

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o INHALATION DOSE-RESPONSE DATA :

Species/Strain Tumor Type	Dose	Tumor Incidence		Reference
human	Route: Occupational exposure (inhalation)			
Age (years)	Midrange (ug/cu.m)	Deaths from Lung Cancer	Person Years	
50	5.66	3	1345	Mancuso, 1975
	25.27	6	931	
	46.83	6	299	
60	4.68	4	1063	
	20.79	5	712	
	39.08	5	211	
70	4.41	2	401	
	21.29	4	345	

o ADDITIONAL COMMENTS :

The cancer mortality in Mancuso (1975) was assumed to be due to Cr VI, which was further assumed to be no less than one-seventh of total chromium. It was also assumed that the smoking habits of chromate workers were similar to those of the U.S. white male population. The unit risks of Langard et al. (1980), Axelsson et al. (1980), and Pokrovskaya and Shabynina (1973) are 1.3E-1, 3.5E-2 and 9.2E-2 per (ug/cu.m), respectively.

Hexavalent chromium compounds have not produced lung tumors in animals by inhalation. Trivalent chromium compounds have not been reported as carcinogenic by any route of administration.

The unit risk should not be used if the air concentration exceeds 8E-1 ug/cu.m, since above this concentration the unit risk may not be appropriate.

o DISCUSSION OF CONFIDENCE :

Results of studies of chromium exposure are consistent across investigators and countries. A dose-relationship for lung tumors has been established. The assumption that the ratio of Cr III to Cr VI is 6:1 may lead to a 7-fold underestimation of risk. The use of 1949 hygiene data, which may underestimate worker exposure, may result in an overestimation of risk. Further overestimation of risk may be due to the implicit assumption that the smoking habits of chromate workers were similar to those of the general white male population, since it is generally accepted that the proportion of smokers is higher for industrial workers than for the general population.

CARDR-
o CARCINOGENICITY SOURCE :

U.S. EPA. 1984. Health Assessment Document for Chromium. Prepared by

the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. EPA 600/8-83-014F.

The quantification of cancer risk in the 1984 Health Assessment Document has received peer review in public sessions of the Environmental Health Committee of the U.S. EPA's Science Advisory Board.

DOCUMENT

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o REVIEW DATES : 06/26/86
o VERIFICATION DATE : 06/26/86
o EPA CONTACTS :

Herman J. Gibb / OHEA -- (202)260-5898

Chao W. Chen / OHEA -- (202)260-5719

HAONE-

NOTE: All chromium HAs are based on total chromium (III and VI).

Appropriate data for calculating a One-day HA are not available. It is recommended that the Ten-day HA of 1.4 mg/L be used as the One-day HA.

HATEN-

NOTE: All chromium HAs are based on total chromium (III and VI).

Ten-day HA -- 1.4E+0 mg/L

NOAEL -- 14.4 mg/kg/day
UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)
Assumptions -- 1 L/day water consumption for a 10-kg child

Principal Study -- Gross and Heller, 1946

Rats were exposed to drinking water containing Cr(VI) (K_2CrO_4) at levels of 80 or 134 mg Cr(VI)/L for 60 days (8.3 or 14.4 mg Cr(VI)/kg/day, respectively) without adverse effects. Therefore, a NOAEL of 14.4 mg/kg/day is identified.

HALTC-

NOTE: All chromium HAs are based on total chromium (III and VI).

Longer-term (Child) HA -- 2.4E-1 mg/L

NOAEL -- 2.4 mg/kg/day
UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)
Assumptions -- 1 L/day water consumption for a 10-kg child

Principal study -- MacKenzie et al., 1958

In a 1-year drinking water study, consumption of water containing either Cr(III) ($CrCl_3$) or Cr(VI) (K_2CrO_4) (0 to 1.87 mg/kg/day for male rats and 0 to 2.41 mg/kg/day for female rats) produced no significant differences in weight gain, appearance, or pathological changes in the blood or other tissue. Therefore, a NOAEL of 2.41 mg/kg/day is identified.

HALTA-

NOTE: All chromium HAs are based on total chromium (III and VI).

Longer-term (Adult) HA -- 8.4E-1 mg/L

NOAEL -- 2.4 mg/kg/day
UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)
Assumptions -- 2 L/day water consumption for a 70-kg adult

Principal study -- MacKenzie et al., 1958 (study described in HALTC)

HALIF-

NOTE: All chromium HAs are based on total chromium (III and VI).

Drinking Water Equivalent Level (DWEL) -- 1.7E-1 mg/L

Assumptions -- 2 L/day water consumption for a 70-kg adult

RfD Verification Date = 02/05/86 (see RDO)

Lifetime HA -- 1.2E-1 mg/L

Assumptions -- 71% exposure by drinking water

Principal study -- MacKenzie et al., 1958 (This study was used in the derivation of the chronic oral RfD; see RDO)

OLEP -

No data available

ALAB -

Determination of chromium is by an atomic absorption technique using either direct aspiration into a flame or a furnace.

TREAT-

The treatment technologies that are available to remove chromium from water include coagulation/filtration, lime softening, ion exchange, and reverse osmosis.

HADR -
o HEALTH ADVISORY SOURCE :

U.S. EPA. 1985. Draft of the Drinking Water Criteria Document on Chromium. Office of Drinking Water, Washington, DC.
DOCUMENT

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o HEALTH ADVISORY REVIEW :

EPA review of HAs in 1985.

Public review of HAs following notification of availability in October, 1985.

Scientific Advisory Panel review of HAs in January, 1986.

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o EPA DRINKING WATER CONTACT :

Kenneth Bailey / OST -- (202)260-5535

Edward V. Ohanian / OST -- (202)260-7571

CAA - NO DATA

WQCHU-

Water and Fish Consumption -- 5.0E+1 ug/L

Fish Consumption Only -- None

Considers technological or economic feasibility? -- NO

Discussion -- The WQC of 5.0E+1 ug/L is based on consumption of contaminated aquatic organisms and water.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 1.6E+1 ug/L (1-hour average)

Chronic -- 1.1E+1 ug/L (4-day average)

Marine:

Acute -- 1.1E+3 ug/L (1-hour average)
Chronic -- 5.0E+1 ug/L (4-day average)

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base on all forms of chromium consisting of acute and chronic tests on a variety of species. Requirements and methods are covered in the reference to the Federal Register.

Reference -- 50 FR 30784 (07/28/85)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value (status) -- 0.1 mg/L [total chromium] (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion -- An MCLG of 0.1 mg/L for total chromium (Cr III and Cr VI) is based on the EPA's RfD methodology for Cr VI, the more toxic chromium species. The MCLG is based upon a DWEL of 0.17 mg/L calculated from available human and animal data and an assumed drinking water contribution of 20 percent. An uncertainty factor of 500 was applied. The MCLG also falls into the safe and adequate daily dietary intake range of 50 to 200 mg/day for Cr III established by the National Research Council in the National Academy of Sciences (NAS, 1989).

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value (status) -- 0.1 mg/L [total chromium] (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion -- The EPA has established an MCL equal to the MCLG of 0.1 mg/L.

Monitoring requirements -- Ground water systems monitored every three years; surface water systems monitored annually; systems out of compliance must begin monitoring quarterly until system is reliably and consistently below MCL.

Analytical methodology -- Atomic absorption/furnace technique (EPA 218.2; SM 304); inductively coupled plasma (EPA 200.7): PQL= 0.01 mg/L.

Best available technology -- Coagulation/filtration; ion exchange; reverse osmosis.

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

SMCL - NO DATA

FISTD- NO DATA

FIREV- NO DATA

CERC -

Value (status) -- 10 pound (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The RQ for chromium (VI) is based on potential carcinogenicity. Available epidemiological data on inhalation of hexavalent chromium indicate a hazard ranking of high based on a potency factor of 388.99/mg/kg/day and assignment to weight-of-evidence group A. This corresponds to an RQ of 1 pound. In addition, a 10 pound adjustment has been applied based on BHP.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline (800)424-9346 / (202)260-3000 / FTS 260-3000

SARA - NO DATA

RCRA -

Status -- Listed (total chromium)

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

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OREF - U.S. EPA. 1985. Drinking Water Health Advisory for Chromium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.

IREF - None

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CREF - Petrilli, F.L. and S. DeFlora. 1977. Toxicity and mutagencity of hexavalent chromium on Salmonella typhimurium. Appl. Environ. Microbiol. 33(4): 805-809.
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HAREF- U.S. EPA. 1985. Drinking Water Criteria Document on Chromium. Office of Drinking Water, Washington, DC. (Draft)

Dibenz(a,h)anthracene

1 - IRIS
NAME - Dibenz[a,h]anthracene
RN - 53-70-3
IRSN - 450
DATE - 940302
UPDT - 03/02/94, 2 fields
STAT - Oral RfD Assessment (RDO) no data
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) on-line 03/01/94
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 12/01/90 CAR Carcinogen assessment on-line
IRH - 12/01/90 REFS Bibliography on-line
IRH - 01/01/92 EXSR Regulatory Action section on-line
IRH - 09/01/93 CAR Carcinogenicity assessment noted as pending change
IRH - 09/01/93 CARDR Work group review date added
IRH - 11/01/93 CARDR Work group review date added
IRH - 03/01/94 CAR Pending change note removed; no change
IRH - 03/01/94 CARDR Work group review date added
RLEN - 21677
SY - Dibenz(a,h)anthracene
SY - DB(a,h)A
SY - DBA
SY - dibenz(a,h)anthracene
SY - DIBENZO(a,h)ANTHRACENE
SY - HSDB 5097
SY - NSC 22433
SY - RCRA WASTE NUMBER U063
SY - 1,2,5,6-DIBENZANTHRACEEN [Dutch]
SY - 1,2,5,6-dibenzanthracene
SY - 1,2:5,6-BENZANTHRACENE
SY - 1,2:5,6-DIBENZ(a)ANTHRACENE
SY - 1,2:5,6-Dibenzanthracene
SY - 1,2:5,6-DIBENZOANTHRACENE

RDO - NO DATA

RDI - NO DATA
CAREV-
o CLASSIFICATION : B2; probable human carcinogen
o BASIS FOR CLASSIFICATION : Based on no human data and sufficient data from animal bioassays. Dibenz[a,h]anthracene produced carcinomas in mice following oral or dermal exposure and injection site tumors in several species following subcutaneous or intramuscular administration.
Dibenz[a,h]anthracene has induced DNA damage and gene mutations in bacteria as well as gene mutations and transformation in several types of mammalian cell cultures.
o HUMAN CARCINOGENICITY DATA :

None. Although there are no human data that specifically link exposure to dibenz[a,h]anthracene with human cancers, dibenz[a]anthracene is a component

of mixtures that have been associated with human cancer. These include coal tar, soots, coke oven emissions and cigarette smoke (U.S. EPA, 1984, 1990; IARC, 1984).

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o ANIMAL CARCINOGENICITY DATA :

Sufficient. Dibenz[a,h]anthracene has been shown to be carcinogenic when administered to mice by the oral route (Snell and Stewart, 1962, 1963). Instead of drinking water DBA/2 mice (21/sex) were given a water-olive oil emulsion containing 0.2 mg/mL dibenz[a,h]anthracene ad libitum. Average exposure was estimated to be 0.85 mg/day for males and 0.76 mg/day for females. The control groups (25 male and 10 female) received the water-olive oil emulsion in place of water. The mice did not tolerate the olive oil vehicle well and all 4 groups lost weight after a few weeks exposure and eventually became emaciated and dehydrated. Animals that died spontaneously or that became moribund were examined for tumors. The duration of the experiment was 279 and 237 days for males and females, respectively, in the dosed groups and 351 and 226 days for male and female controls. Mice developed pulmonary adenomas (treated males, 14/14; control males 1/23; treated females, 13/13; control females, 0/6), pulmonary carcinomas (treated males, 14/14; control males, 0/23; treated females, 10/13; control females, 0/6), mammary carcinoma (treated females, 12/13; control females, 0/6) and hemangioendothelioma (treated males, 10/14; control males, 0/23; treated females, 6/13; control females, 0/6). No statistical analyses appear to have been performed.

Mammary carcinomas were observed in two strains of female mice following gavage with dibenz[a,h]anthracene (Biancifiori and Caschera, 1962; Berenblum and Haran, 1955). Biancifiori and Caschera (1962) observed mammary carcinomas when female Balb/c (1/20) and pseudo-pregnant female (obtained by mating virgin females with vasectomized males) Balb/c (13/24) mice were treated for 15 weeks with a twice-weekly gavage containing 0.5% dibenz[a,h]anthracene (total dose was 15 mg/animal). Mammary carcinomas occurred in 2/30 pseudo-pregnant females not dosed with dibenz[a,h]anthracene. Previous studies indicated that mammary carcinomas did not occur in virgin Balb/c females (Biancifiori et al., 1959). A single 1.5-mg dose of dibenz[a,h]anthracene in polyethylene glycol [average molecular weight (a.m.u.) 400] (PEG-400) produced forestomach papillomas in 2/42 male Swiss mice after 30 weeks. In this short-term study no mice developed tumors when treated with PEG alone (1 time/week) for 30 weeks (0/20) (Berenblum and Haran, 1955).

Dibenz[a,h]anthracene has produced positive results in mouse skin painting assays for complete carcinogenicity. Swiss mice developed carcinomas following dermal exposure to dibenz[a,h]anthracene at concentrations of 0.001% or greater (Wynder and Hoffman, 1959; Van Duuren et al., 1967). Numerous studies that demonstrate complete carcinogenic activity and initiating activity are summarized in IARC (1973) and U.S. EPA (1990).

Subcutaneous injection of dibenz[a,h]anthracene induced sarcomas at the site of injection in several animal species. Groups (>19) of C3H mice received single subcutaneous injections of dibenz[a,h]anthracene in tricaprylin at doses ranging from 0.0019-8 mg (approximately 0.09-360 mg/kg). No controls appear to have been used in this experiment (Bryan and Shimkin, 1943). Tumor latency appeared to decrease and the incidence of iniection site

sarcomas appeared to increase with dose (>76% at doses >0.06 mg or 2.8 mg/kg).

A single subcutaneous injection of 2.4, 4.7, 9.3, 18.7, 37.5, or 75 ug dibenz[a,h]anthracene into groups of 100 NMRI mice was reported to produce a dose-related increase in tumor incidence (37/100, 39/100, 44/100, 56/100, 65/100, and 69/100, respectively) by the 114th week after injection (Pfeiffer, 1977). No concurrent controls were reported; however, a spontaneous tumor rate for NMRI mice was previously reported to be 0-2% (Pfeiffer, 1973). The development of fibrosarcomas from a single subcutaneous injection of 150 ug dibenz[a,h]anthracene was shown to be higher in AHH+ strains of mice than in AHH- strains.

Lubet et al. (1983) found that subcutaneous injections of dibenz[a,h]anthracene were associated with fibrosarcoma development in mice, but only for some strains. Four strains of mice used included two, C3H/HeJ and C57B1/6J, that respond to 3-methylcholanthrene treatment with increased levels and types of hepatic enzymes, including AHH. Two strains, AKR/J and DBA/2J were nonresponders. Groups of 30 animals were injected with a single dose of 150 mg dibenz[a,h]anthracene in 0.05 mL trioctanoin and observed for 9 months. A control group for each strain, consisting of 10 animals each, received a subcutaneous injection of 0.05 mL trioctanoin alone. The tumor incidence in the treated animals varied between 0 and 80%, depending on the strain. Tumor incidences were higher in the C3H and C57B1 mice but not in AKR or DBA mice. Likewise, the average latency period (in days) for fibrosarcoma development varied with the strain and tended to be inversely correlated with the tumor incidence rate. Numerous earlier studies that demonstrate the carcinogenicity of parenterally injected dibenz[a,h]anthracene in a variety of species are summarized in IARC (1973) and U.S. EPA (1990).

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o SUPPORTING DATA :

Dibenz[a,h]anthracene has produced positive results in bacterial DNA damage and mutagenicity assays and in mammalian cell DNA damage, mutagenicity and cell transformation assays. In bacterial DNA damage assays, positive results were obtained in Escherichia coli and Bacillus subtilis at exposure levels of 12-50 ug/well. Dibenz[a,h]anthracene tested positive for reverse mutation in Salmonella typhimurium strains TA100 and TA98 (3-5 ug/plate) and positive for forward mutation in strain TM677 (21 ug/mL) (McCann et al., 1975; Andrews et al., 1978; Baker et al., 1980; Hermann, 1981; Kaden et al., 1979).

In mammalian cell DNA damage assays, positve results were obtained in human foreskin epithelial cells not activated with mixed-function oxidase (MFO) inducers (1-100 ug/mL) and in HeLa cells (28 ng/mL) activated with 3-methylcholanthrene (Lake et al., 1978; Martin et al., 1978). When Syrian hamster embryo cells and rat hepatocytes not activated with MFO inducers were exposed to 20-30 ug/mL the results were not positive (Casto, 1979; Probst et al., 1981). Dibenz[a,h]anthracene induced forward mutations in Chinese hamster embryo cells exposed to concentrations of 1 ug/mL or greater (Huberman and Sachs, 1976; Krahn and Heidelberger, 1977; Huberman, 1978). It transformed several types of mammalian cells exposed to concentrations of 10 ug/mL or greater; these cell types included: Syrian hamster embryo cells, mouse C3H10T 1/2 cells and mouse prostate C3H cells (DiPaolo et al., 1969; Chen and Heidelberger, 1969; Pienta et al., 1977; Casto et al., 1977; Casto, 1979; Reznikoff et al., 1973; Lubet et al., 1983).

Current theories on mechanisms of metabolic activation of polycyclic aromatic hydrocarbons are consistent with a carcinogenic potential for dibenz[a,h]anthracene. Dibenz[a,h]anthracene has a "bay-region" structure (Jerina et al., 1978). It is metabolized by mixed-function oxidases to dihydrodiols that are mutagenic in bacteria and tumorigenic in mouse skin painting assays and when injected into newborn mice (Wood et al., 1978; Nordqvist et al., 1979; Slaga et al., 1980; Buening et al., 1979).

CARO - NO DATA
CARI - NO DATA
CARDR-
o CARCINOGENICITY SOURCE :

Source Document -- U.S. EPA, 1984, 1990

The 1990 Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons has received Agency and external review.
DOCUMENT

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o REVIEW DATES : 02/07/90, 08/05/93, 09/21/93, 02/02/94
o VERIFICATION DATE : 02/07/90
o EPA CONTACTS :

Rita S. Schoeny / OHEA -- (513)569-7813

Robert E. McGaughy / OHEA -- (202)260-5889

HAONE- NO DATA

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA - NO DATA

WQCHU-

Water and Fish Consumption: 2.8E-3 ug/L

Fish Consumption Only: 3.11E-2 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. However, zero may not be obtainable at this time, so the recommended criteria represents a E-6 estimated incremental increase of cancer over a lifetime. The values given represent polynuclear aromatic hydrocarbons as a class.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute LEC -- none
Chronic LEC -- none

Marine:

Acute LEC -- 3.0E+2 ug/L
Chronic LEC -- none

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LEC's are given when the minimum data required to derive water quality criteria are not available. The values given represent polynuclear aromatic hydrocarbons as a class.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value -- 0 mg/L (Proposed, 1990)

Considers technological or economic feasibility? -- NO

Discussion -- The proposed MCLG is zero. This value is based on carcinogenic PAH's as a class.

Reference -- 55 FR 30370 (07/25/90)

EPA Contact -- Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value -- 0.0002 mg/L (Proposed, 1990)

Considers technological or economic feasibility? -- YES

Discussion -- The proposed MCL is equal to the PQL and is associated with a maximum lifetime individual risk of 1 E-4.

Monitoring requirements -- Community and non-transient water system monitoring based on state vulnerability assessment; vulnerable systems to be monitored quarterly for one year; repeat monitoring dependent upon detection and size of system.

Analytical methodology -- High pressure liquid chromatography (EPA 550, 550.1); gas chromatographic/mass spectrometry (EPA 525): PQL= 0.0002 mg/L.

Best available technology -- Granular activated carbon.

Reference -- 55 FR 30370 (07/25/90)

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

Status -- Listed (Proposed, 1991)

Discussion -- "Unregulated" contaminants are those contaminants for which EPA establishes a monitoring requirement but which do not have an associated final MCLG, MCL, or treatment technique. EPA may regulate these contaminants in the future.

Monitoring requirement -- All systems to be monitored unless a vulnerability assessment determines the system is not vulnerable.

Analytical methodology -- Gas chromatography/mass spectrometry (EPA 525); high pressure liquid chromatography (EPA 550, 550.1).

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

SMCL - NO DATA

FISTD- NO DATA

FIREV- NO DATA

CERC -

Value (status) -- 1 pound (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ for benz[a,h]anthracene is based on potential carcinogenicity. Available data indicate a hazard ranking of high and a weight of evidence classification of Group B2, which corresponds to an RQ of 1 pound.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

SARA - NO DATA

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - None
IREF - None

CREF - Andrews, A.W., L.H. Thibault and W. Lijinsky. 1978. The relationship between carcinogenicity and mutagenicity of some polynuclear hydrocarbons. Mutat. Res. 51: 311-318.

CREF - Baker, R.S.U., A.M. Bonin, I. Stupans and G.M. Holder. 1980. Comparison of rat and guinea pig as sources of the S9 fraction in the Salmonella/mammalian microsome mutagenicity test. Mutat. Res. 71: 43-52.

CREF - Berenblum, I. and N. Haran. 1955. The influence of croton oil and of polyethylene glycol-400 on carcinogenesis in the forestomach of the mouse. Cancer Res. 15: 510-516.

CREF - Biancifiori, C. and F. Caschera. 1962. The relation between pseudo-pregnancy and the chemical induction by four carcinogens of mammary and ovarian tumours in BALB/c mice. Br. J. Cancer. 16: 722-730.

CREF - Biancifiori, C., G.M. Bonser and F. Caschera. 1959. The influence of pseudo- pregnancy on the induction of mammary tumors by methylcholanthrene in mice of Balb/c strain. Br. J. Cancer. 13: 662-668.

CREF - Bryan, W.R. and M.B. Shimkin. 1943. Quantitative analysis of dose-response data obtained with three carcinogenic hydrocarbons in strain C3H male mice. J. Natl. Cancer Inst. 3: 503-531.

CREF - Buening, M.K., W. Levin, A.W. Wood, et al. 1979. Tumorigenicity of the dihydrodiols of dibenzo[a,h]anthracene on mouse skin and in newborn mice. Cancer Res. 39: 1310-1314.

CREF - Casto, B.C. 1979. Polycyclic hydrocarbons and Syrian hamster embryo cells: Cell transformation, enhancement of viral transformation and analysis of DNA damage. In: Polynuclear Aromatic Hydrocarbons, P.W. Jones and P. Leber, Ed. Ann Arbor Science Publ., Ann Arbor, MI. p. 51-66.

CREF - Casto, B.C., N. Janosko and J.A. DiPaolo. 1977. Development of a focus assay model for transformation of hamster cells in vitro by chemical carcinogens. Cancer Res. 37: 3508-3515.

CREF - Chen, T.T. and C. Heidelberger. 1969. Quantitative studies on the malignant transformation of mouse prostate cells by carcinogenic hydrocarbons in vitro. Int. J. Cancer. 4: 166-178.

CREF - DiPaolo, J.A., P. Donovan and R.L. Nelson. 1969. Quantitative studies of in vitro transformation by chemical carcinogens. J. Natl. Cancer Inst. 42(5): 867-874.

CREF - Hermann, M. 1981. Synergistic effects of individual polycyclic aromatic hydrocarbons on the mutagenicity of their mixtures. Mutat. Res. 90(4): 399-409.

CREF - Huberman, E. 1978. Cell transformation and mutability of different genetic loci in mammalian cells by metabolically activated carcinogenic polycyclic hydrocarbons. In: Polycyclic Hydrocarbons and Cancer, Vol. 2. Molecular and Cell Biology, H.V. Gelboin and P.O.P. Tso, Ed. Academic Press, NY. p. 161-174.

CREF - Huberman, E. and L. Sachs. 1976. Mutability of different genetic loci in mammalian cells by metabolically activated carcinogenic polycyclic hydrocarbons. Proc. Natl. Acad. Sci. USA. 73(1): 188-192.

CREF - IARC (International Agency for Research on Cancer). 1973. Certain Polycyclic Aromatic Hydrocarbons and Heterocyclic Compounds. Monographs on the Evaluation of the Carcinogenic Risk of the Chemical to Man. Polynuclear Aromatic Compounds. Vol. 3. World Health Organization, Lyon, France.

CREF - IARC (International Agency for Research on Cancer). 1984. Monographs on

the Evaluation of the Carcinogenic Risk of the Chemical to Man. Polynuclear Aromatic Hydrocarbons. Part 3. Industrial Exposures in Aluminum Production, Coal Gasification, Coke Production, and Iron and Steel Founding. Vol. 34. World Health Organization, Lyon, France.

CREF - Jerina, D.M., H. Yagi, R.E. Lehr, et al. 1978. The bay-region theory of carcinogenesis by polycyclic aromatic hydrocarbons. In: Polycyclic Hydrocarbons and Cancer, Vol. 1, Environment, Chemistry and Metabolism, H.V. Gelboin and P.O.P. Ts'o, Ed. Academic Press, NY.

CREF - Kaden, D.A., R.A. Hites and W.G. Thilly. 1979. Mutagenicity of soot and associated polycyclic aromatic hydrocarbons to Salmonella typhimurium. Cancer Res. 39: 4152-4159.

CREF - Krahn, D.F. and C. Heidelberger. 1977. Liver homogenate-mediated mutagenesis in Chinese hamster V79 cells by polycyclic aromatic hydrocarbons and aflatoxins. Mutat. Res. 46: 27-44.

CREF - Lake, R.S., M.L. Kropko, M.R. Pezzutti, R.H. Shoemaker and H.J. Igel. 1978. Chemical induction of unscheduled DNA synthesis in human skin epithelial cell cultures. Cancer Res. 38: 2091-2098.

CREF - Lubet, R.A., E. Kiss, M.M. Gallagher, C. Dively, R.E. Kouri and L.M. Schectman. 1983. Induction of neoplastic transformation and DNA single-strand breaks in C3H/10T1/2 clone 8 cells by polycyclic hydrocarbons and alkylating agents. J. Natl. Cancer Inst. 71(5): 991-997.

CREF - Martin, C.N., A.C. McDermid and R.C. Garner. 1978. Testing of known carcinogens and noncarcinogens for their ability to induce unscheduled DNA synthesis in HeLa cells. Cancer Res. 38: 2621-2627.

CREF - McCann, J.E., E. Choi, E. Yamasaki and B.N. Ames. 1975. Detection of carcinogens as mutagens in the Salmonella/microsome test: Assay of 300 chemicals. Proc. Natl. Acad. Sci. USA. 72(12): 5135-5139.

CREF - Nordqvist, M., D.R. Thakker, W. Levin, et al. 1979. The highly tumorigenic 3,4-dihydrodiol is a principal metabolite formed from dibenzo[a,h]anthracene by liver enzymes. Mol. Pharmacol. 16: 643-655.

CREF - Pfeiffer, E.H. 1973. Investigations on the carcinogenic burden by air polution in man. VII. Studies on the oncogenetic interaction of polycyclic aromatic hydrocarbons. Zbl. Bakt. Hyg., I. Abt. Orig. B. 158: 69-83.

CREF - Pfeiffer, E.H. 1977. Oncogenic interaction of carcinogenic and non-carcinogenic polycyclic aromatic hydrocarbons in mice. In: Air Pollutants and Cancer in Man, U. Mohr, D. Schmaehl and L. Tomatis, Ed. IARC, Lyon, France. p. 69-77.

CREF - Pienta, R.J., J.A. Poiley and W.B. Lebherz, III. 1977. Morphological transformation of early passage golden Syrian hamster embryo cells derived from cryopreserved primary cultures as a reliable in vitro bioassay for identifying diverse carcinogens. Int. J. Cancer. 19: 642-655.

CREF - Probst, G.S., R.E. McMahon, L.E. Hill, C.Z. Thompson, J.K. Epp and S.B. Neal. 1981. Chemically-induced unscheduled DNA synthesis in primary rat hepatocyte cultures: A comparison with bacterial mutagenicity using 218 compounds. Environ. Mutagen. 3: 11-32.

CREF - Reznikoff, C.A., J.S. Bertram, D.W. Brankow and C. Heidelberger. 1973. Quatitiative and qualitiative studies of chemcial transformation of cloned C3H mouse embryo cells sensitive to postconfluence inhibition of cell division. Cancer Res. 33: 3239-3249.

CREF - Slaga, T.J., G.L. Gleason, G. Mills, et al. 1980. Comparison of the skin tumor-initiating activities of dihydrodiols and diol-epoxides of various polycyclic aromatic hydrocarbons. Cancer Res. 40: 1981-1984.

CREF - Snell, K.C. and H.L. Stewart. 1962. Pulmonary adenomatosis induced in DBA/2 mice by oral administration of dibenz[a,h]anthracene. J. Natl. Cancer Inst. 28(5): 1043-1049.
CREF - Snell, K.C. and H.L. Stewart. 1963. Induction of pulmonary adenomatose in DBA/2 mice by the oral administration of dibenz[a,h]anthracene. Acta. Un. Int. Cancer. 19: 692-694.
CREF - U.S. EPA. 1984. Carcinogen Assessment of Coke Oven Emissions. Office of Health and Environmental Assessment, Washington, DC. EPA 600/6-82-003F. NTIS PB 84-170181.
CREF - U.S. EPA. 1990. Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons (PAHs). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. Final Draft. ECAO-CIN-D010, September, 1990.
CREF - Van Duuren, B.L., L. Langseth, B.M. Goldschmidt, et al., 1967. Carcinogenicity of epoxides, lactones and peroxy compounds. VI. Structure and carcinogenic activity. J. Natl. Cancer Inst. 21: 623.
CREF - Wood, A.W., W. Levin, P.E. Thomas, et al. 1978. Metabolic activation of dibenzo[a,h]anthracene and its dihydrodiols to bacterial mutagens. Cancer Res. 38(7): 1967-1973.
CREF - Wynder, E.L. and D. Hoffmann. 1959. A study of tobacco carcinogenesis. VII. The role of higher polycyclic hydrocarbons. Cancer. 12: 1079-1086.
HAREF- None

Dieldrin

1 - IRIS
NAME - Dieldrin
RN - 60-57-1
IRSN - 221
DATE - 930701
UPDT - 07/01/93, 4 fields
STAT - Oral RfD Assessment (RDO) on-line 09/01/90
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) on-line 07/01/93
STAT - Drinking Water Health Advisories (DWHA) on-line 09/01/90
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 09/07/88 RDO Oral RfD summary on-line
IRH - 09/07/88 CAR Carcinogen summary on-line
IRH - 03/01/90 CAREV Ditraglia citation clarified
IRH - 03/01/90 CAREV Reuber citation year and Deichman spelling corrected
IRH - 03/01/90 CAREV Shirasu citation year corrected
IRH - 03/01/90 CARO Reuber citation year corrected
IRH - 03/01/90 REFS Bibliography on-line
IRH - 04/01/90 CREF Treon and Cleveland, 1955 citation corrected
IRH - 09/01/90 RDO Text edited
IRH - 09/01/90 CAR Text edited
IRH - 09/01/90 HADV Health Advisory on-line
IRH - 09/01/90 REFS Health Advisory references added
IRH - 01/01/91 CAR Text edited
IRH - 01/01/91 CARI Inhalation slope factor removed (global change)
IRH - 01/01/92 EXSR Regulatory Action section on-line
IRH - 07/01/93 CARDR Secondary contact's phone number changed
RLEN - 26908
SY - ALVIT
SY - COMPOUND 497
SY - DIELDREX
SY - Dieldrin
SY - DIELDRINE
SY - DIELDRITE
SY - 1,4:5,8-DIMETHANONAPHTHALENE,
1,2,3,4,10,10-HEXACHLORO-6,7-EPOXY-1,4,4a,5,6,7,
SY - 8,8a-OCTAHYDRO, endo,exo-
SY - ENT 16,225
SY - HEOD
SY - HEXACHLOROEPOXYOCTAHYDRO-endo,exo-DIMETHANONAPHTHALENE
SY - 3,4,5,6,9,9-HEXACHLORO-1a,2,2a,3,6,6a,7,7a-OCTAHYDRO-2,7:3,6-DIMETHANON
APHTH
SY - (2,3-b)OXIRENE
SY - ILLOXOL
SY - NA 2761
SY - NCI-C00124
SY - OCTALOX
SY - PANORAM D-31
SY - QUINTOX
SY - RCRA WASTE NUMBER P037

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Liver lesions 2-Year Rat Feeding Study Walker et al., 1969	NOAEL: 0.1 ppm (0.005 mg/kg/day) LOAEL: 1.0 ppm (0.05 mg/kg/day)	100	1	5E-5 mg/kg/day

*Conversion Factors: 1 ppm = 0.05 mg/kg/day (assumed rat food consumption)

o ORAL RFD STUDIES :

Walker, A.I.T., D.E. Stevenson, J. Robinson, R. Thorpe and M. Roberts. 1969. The toxicology and pharmacodynamics of dieldrin (HEOD): Two-year oral exposures of rats and dogs. Toxicol. Appl. Pharmacol. 15: 345-373.

Walker et al. (1969) administered dieldrin (recrystallized, 99% active ingredient) to Carworth Farm "E" rats (25/sex/dose; controls 45/sex) for 2 years at dietary concentrations of 0, 0.1, 1.0, or 10.0 ppm. Based on intake assumptions presented by the authors, these dietary levels are approximately equal to 0, 0.005, 0.05 and 0.5 mg/kg/day. Body weight, food intake, and general health remained unaffected throughout the 2-year period, although at 10.0 ppm (0.5 mg/kg/day) all animals became irritable and exhibited tremors and occasional convulsions. No effects were seen in various hematological and clinical chemistry parameters. At the end of 2 years, females fed 1.0 and 10.0 ppm (0.05 and 0.5 mg/kg/day) had increased liver weights and liver-to-body weight ratios ($p<0.05$). Histopathological examinations revealed liver parenchymal cell changes including focal proliferation and focal hyperplasia. These hepatic lesions were considered to be characteristic of exposure to an organochlorine insecticide. The LOAEL was identified as 1.0 ppm (0.005 mg/kg/day) and the NOAEL as 0.1 ppm (0.005 mg/kg/day).

o ORAL RFD UNCERTAINTY :

UF – The UF of 100 allows for uncertainty in the extrapolation of dose levels from laboratory animals to humans (10A) and uncertainty in the threshold for sensitive humans (10H).

o ORAL RFD MODIFYING FACTOR :

MF = None

o ORAL RFD COMMENTS :

Data considered for establishing the RfD:

1) 2-Year Feeding - rat: Principal study - see previous description

2) 2-Year Feeding (oncogenic) - dog: Systemic NOEL=0.005 mg/kg/day; LEL= 0.05 mg/kg/day (increased liver weight and liver/body weight ratios, increased plasma alkaline phosphatase, and decreased serum protein concentration) (Walker et al., 1969)

3) 2-Year Feeding - rat: Systemic LEL=0.5 ppm (approximately 0.025 mg/kg/day).

(liver enlargement with histopathology); (Fitzhugh et al., 1964)

4) 2-Year Feeding (oncogenic) - mouse: Systemic LEL=0.1 ppm (0.015 mg/kg/day), (liver enlargement with histopathology); (Walker et al., 1972)

5) 25-Month Feeding - dog: Systemic NOEL=0.2 mg/kg/day; LEL=0.5 mg/kg/day, (weight loss and convulsions); (Fitzhugh et al., 1964)

6) Teratology - mouse: Teratogenic NOEL=6.0 mg/kg/day (HDT, gestational days 7-16); Maternal LEL=6.0 mg/kg/day (HDT, decrease in maternal weight gain); Fetotoxic LEL=6.0 mg/kg/day (HDT, decreased numbers of caudal ossification centers and increases in supernumerary ribs); (Chernoff et al., 1975). This study was not considered since 41% of the test dams died at the highest dose tested.

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o ORAL RFD CONFIDENCE :

Study – Low
Data Base – Medium
RfD – Medium

The principal study is an older study for which detailed data are not available and in which a wide range of doses was tested. The chronic toxicity evaluation is relatively complete and supports the critical effect, if not the magnitude of effects. Reproductive studies are lacking. The RfD is given a medium confidence rating because of the support for the critical effect from other dieldrin studies, and from studies on organochlorine insecticides in general.

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o ORAL RFD SOURCE DOCUMENT :

Source Document – U.S. EPA, 1987

Other EPA Documentation – None

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o REVIEW DATES : 04/16/87
o VERIFICATION DATE : 04/16/87
o EPA CONTACTS :

Krishan Khanna / OST – (202)260-7588

Henry Spencer / OST – (202)557-4383

RDI - NO DATA
CAREV-
o CLASSIFICATION : B2; probable human carcinogen
o BASIS FOR CLASSIFICATION : Dieldrin is carcinogenic in seven strains of mice when administered orally. Dieldrin is

structurally related to compounds (aldrin, chlordane, heptachlor, heptachlor epoxide, and chlorendic acid) which produce tumors in rodents.

o HUMAN CARCINOGENICITY DATA :

Inadequate. Two studies of workers exposed to aldrin and to dieldrin reported no increased incidence of cancer. Both studies were limited in their ability to detect an excess of cancer deaths. Van Raalte (1977) observed two cases of cancer (gastric and lymphosarcoma) among 166 pesticide manufacturing workers exposed 4-19 years and followed from 15-20 years. Exposure was not quantified, and workers were also exposed to other organochlorine pesticides (endrin and telodrin). The number of workers studied was small, the mean age of the cohort (47.7 years) was young, the number of expected deaths was not calculated, and the duration of exposure and of latency was relatively short.

In a retrospective mortality study, Ditraglia et al. (1981) reported no statistically significant excess in deaths from cancer among 1155 organochlorine pesticide manufacturing workers [31 observed vs. 37.8 expected, Standardized Mortality Ratio (SMR) = 82]. Workers were employed for 6 months or more and followed 13 years or more (24,939 person-years). Workers with no exposure (for example, office workers) were included in the cohort. Vital status was not known for 112 or 10% of the workers, and these workers were assumed to be alive; therefore additional deaths may have occurred but were not observed. Exposure was not quantified and workers were also exposed to other chemicals and pesticides (including endrin). Increased incidences of deaths from cancer were seen at several specific sites: esophagus (2 deaths observed, SMR = 235); rectum (3, SMR = 242); liver (2, SMR = 225); and lymphatic and hematopoietic system (6, SMR = 147), but these site-specific incidences were not statistically significantly increased.

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o ANIMAL CARCINOGENICITY DATA :

Sufficient. Dieldrin has been shown to be carcinogenic in various strains of mice of both sexes. At different dose levels the effects range from benign liver tumors, to hepatocarcinomas with transplantation confirmation, to pulmonary metastases.

The Food and Drug Administration (FDA) conducted a long-term carcinogenesis bioassay for dieldrin (Davis and Fitzhugh, 1962). Ten ppm dieldrin was administered orally to 218 male and female C3HeB/Fe mice for 2 years. The study was compromised by the poor survival rate, lack of detailed pathology, loss of a large percentage of the animals to the study, and failure to treat the data for males and females separately. A statistically significant increase in incidence of hepatomas was observed in the treated groups versus the control groups in both males and females. In FDA follow-up study, Davis (1965) examined 100 male and 100 female C3H mice which had been orally administered 10 ppm dieldrin. The same limitations as the previous study were reported. The incidence of benign hepatomas and hepatic carcinomas was significantly increased in the dieldrin group. A reevaluation of the histological material of both studies was done by Reuber in 1974 (Epstein, 1975a,b; 1976). He concluded that the hepatomas were malignant and that dieldrin was hepatocarcinogenic for male and female C3HeB/Fe and C3H mice.

Walker et al. (1972) conducted several studies of dieldrin in CF1 mice of both sexes. Dieldrin was administered orally at concentrations of 0, 0.1, 1.0, and 10 ppm. Treatment groups varied from 87 to 288 animals of each sex. Surviving animals were sacrificed during weeks 132-140. Incidence of tumors was related to the number of dose levels and the dose administered. Effects were detected at the lowest dieldrin level tested (0.1 ppm) in both male and female mice. Dieldrin also produced significant increases (<0.05) in the incidence of pulmonary adenomas, pulmonary carcinomas, lymphoid tumors, and "other" tumors in female mice.

Diets containing 10 ppm dieldrin were fed to groups of 30 CF1 mice of both sexes for 110 weeks (Thorpe and Walker, 1973). The control group consisted of 45 mice of both sexes. A statistically significant increase ($p<0.01$) in incidence of liver tumors was found in both sexes of treated animals relative to controls. The liver tumors appeared much earlier in treated animals than controls.

Technical-grade dieldrin (>96%) was fed to B6C3F1 mice (50/sex/dose) at TWA doses of 0, 2.5, or 5 ppm for 80 weeks followed by an observation period of 10 to 13 weeks (NCI, 1978a). Matched control groups consisted of 20 untreated males and 10 untreated females. No significant difference in survival was noted. A significant dose-related increase in hepatocellular carcinoma was found in male mice when compared with pooled controls.

Tennekes et al. (1981) fed groups of 19 to 82 male CF1 mice control or dieldrin-supplemented (10 ppm) diets or control diets for 110 weeks. Dieldrin produced a statistically significant increased incidence of hepatocellular carcinomas in the treated group.

Dieldrin (>99%) was continuously fed in the diet for 85 weeks to 50 C3H/He, 62 B6C3FI, and 71 C57Bl/6J male mice (Meierhenry et al., 1983). Controls were 50 to 76 males of each strain. Dieldrin produced a significant increase in the incidence of hepatocellular carcinomas compared with controls in all three strains.

Seven studies with four strains of rats fed 0.1 to 285 ppm dieldrin varying in duration of exposure from 80 weeks to 31 months did not produce positive results for carcinogenicity (Treon and Cleveland, 1955; Fitzhugh et al., 1964; Song and Harville, 1964; Walker et al., 1969; Deichmann et al., 1970; NCI, 1978a,b). Three of these studies used Osborne-Mendel rats, two studies used Carworth rats, and one each used Fischer 344 and Holtzman strains. Only three of the seven studies are considered adequate in design and conduct. The others used too few animals, had unacceptably high levels of mortality, were too short in duration, and/or had inadequate pathology examination or reporting.

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o SUPPORTING DATA:

Dieldrin causes chromosomal aberrations in mouse cells (Markaryan, 1966; Majumdar et al., 1976) and in human lymphoblastoid cells (Trepanier et al., 1977), forward mutation in Chinese hamster V79 cells (Ahmed et al., 1977), and unscheduled DNA synthesis in rat (Probst et al., 1981) and human cells (Rocchi et al., 1980). Dieldrin did not produce responses in 13 other mutagenicity tests. Negative responses were given in assays for gene conversion in S.

cerevisiae, back-mutation in S. marcesans, forward mutation (Gal Rz2 in E. coli), and forward mutation to streptomycin resistance in E. coli (Fahrig, 1974). Negative responses were produced in reverse mutation assays with six strains of S. typhimurium with or without metabolic activation (Bidwell et al., 1975; Marshall et al., 1976; Shirasu et al., 1976; Wade et al., 1979; Haworth et al., 1983). Majumdar et al. (1977), however, reported that dieldrin was mutagenic for S. typhimurium with and without metabolic activation.

Five compounds structurally related to dieldrin - aldrin, chlordane, heptachlor, heptachlor epoxide, and chlorondic acid - have induced malignant liver tumors in mice. Chlorendic acid has also induced liver tumors in rats.

CARO -
o CLASSIFICATION : B2; probable human carcinogen
o BASIS FOR CLASSIFICATION : Dieldrin is carcinogenic in seven strains of mice when administered orally. Dieldrin is structurally related to compounds (aldrin, chlordane, heptachlor, heptachlor epoxide, and chlorendic acid) which produce tumors in rodents.
o ORAL SLOPE FACTOR : 1.6E+1 per (mg/kg)/day
o DRINKING WATER UNIT RISK : 4.6E-4 per (ug/L)
o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk
o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	2E-1 ug/L
E-5 (1 in 100,000)	2E-2 ug/L
E-6 (1 in 1,000,000)	2E-3 ug/L

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o ORAL DOSE-RESPONSE DATA :

Tumor Type -- liver carcinoma
Test Animals -- mouse
Route -- diet
Reference -- see table

Sex/Strain	Slope Factor	Reference
Male, C3H	22	Davis (1965), reevaluated by Reuber, 1974 (cited in Epstein, 1975a)
Female, C3H	25	Davis (1965), reevaluated by Reuber, 1974 (cited

in Epstein, 1975a)

Male, CF1	25	Walker et al. (1972)
Female, CF1	28	Walker et al. (1972)
Male, CF1	15	Walker et al. (1972)
Female, CF1	7.1	Walker et al. (1972)
Male, CF1	55	Thorpe and Walker (1973)
Female, CF1	26	Thorpe and Walker (1973)
Male, B6C3F1	9.8	NCI (1978a,b)
Male, CF1	18	Tennekes et al. (1981)
Male, C57B1/6J	7.4	Meierhenry et al. (1983)
Male, C3H/He	8.5	Meierhenry et al. (1983)
Male, B6C3F1	11	Meierhenry et al. (1983)

o ADDITIONAL COMMENTS :

The slope factor is the geometric mean of 13 slope factors calculated from liver carcinoma data in both sexes of several strains of mice. Inspection of the data indicated no strain or sex specificity of carcinogenic response.

The unit risk should not be used if the water concentration exceeds 20 ug/L, since above this concentration the unit risk may not be appropriate.

o DISCUSSION OF CONFIDENCE :

The individual slope factors calculated from 13 independent data sets range within a factor of 8.

CARI -

o CLASSIFICATION : B2; *probable human carcinogen*

o BASIS FOR CLASSIFICATION : Dieldrin is carcinogenic in seven strains of mice when administered orally. Dieldrin is structurally related to compounds (aldrin, chlordane, heptachlor, heptachlor epoxide, and chlorendic acid) which produce tumors in rodents.

o INHALATION UNIT RISK : 4.6E-3 per (ug/cu.m)

o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk

o RISK/AIR CONCENTRATIONS :

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	2E-2 ug/cu.m
E-5 (1 in 100,000)	2E-3 ug/cu.m
E-6 (1 in 1,000,000)	2E-4 ug/cu.m

o INHALATION DOSE-RESPONSE DATA :

Calculated from oral data in CARO.

o ADDITIONAL COMMENTS :

The unit risk should not be used if air concentrations exceed 2 ug/cu.m, since above this concentration the unit risk may not be appropriate.

o DISCUSSION OF CONFIDENCE :

This inhalation risk estimate was based on oral data.

CARDR-
o CARCINOGENICITY SOURCE :

Source Document – U.S. EPA, 1986
DOCUMENT

o REVIEW DATES : 03/05/87
o VERIFICATION DATE : 03/05/87
o EPA CONTACTS :

Dharm Singh /OHEA – (202)260-5958

Jim Cogliano / OHEA – (202)260-3814

HAONE-

Appropriate data for calculating a One-day HA are not available. It is recommended that the modified DWEL of 0.0005 mg/L be used as the One-day HA.

HATEN-

Appropriate data for calculating a Ten-day HA are not available. It is recommended that the modified DWEL of 0.0005 mg/L be used as the Ten-day HA.

HALTC-

Appropriate data for calculating Longer-term HAs for dieldrin are not available. It is recommended that the modified DWEL of 0.0005 mg/L be used as the Longer-term HA for the 10-kg child.

HALTA-

Appropriate data for calculating Longer-term HAs for dieldrin are not available. It is recommended that the modified DWEL of 0.002 mg/L be used as the Longer-term HA for the 70-kg adult.

HALIF-

Drinking Water Equivalent Level (DWEL) -- 2E-3 mg/L

Assumptions -- 2 L/day water consumption for a 70-kg adult

RfD Verification Date -- 04/16/87 (see Section I.A. in this file)

Lifetime HA -- None

Dieldrin is considered to be a probable human carcinogen. Lifetime HAs are not recommended for known or probable human carcinogens. The estimated excess cancer risk associated with lifetime exposure to drinking water containing dieldrin at the DWEL of 2 ug/L is approximately 8.05 x 10-4. Refer to Section II for the carcinogenicity assessment for dieldrin.

Principal Study -- Walker et al., 1969 (This study was used in the derivation of the chronic oral RfD; see RDO)

OLEP -

The odor threshold for dieldrin in water is reported as 0.04 mg/L.

ALAB -

Determination of dieldrin is by a liquid-liquid extraction gas chromatographic procedure.

TREAT-

Available data indicate that reverse osmosis, granular activated carbon

adsorption, ozonation, and conventional treatment will remove dieldrin from water.

HADR -
o HEALTH ADVISORY SOURCE :

U.S. EPA. 1989. Drinking Water Health Advisories: Pesticides. Lewis Publishers, Chelsea, MI. p. 299-312.
DOCUMENT

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o HEALTH ADVISORY REVIEW :

EPA review of HAs in 1987.

Public review of HAs in January-March 1988.

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o EPA DRINKING WATER CONTACT :

Krishan Khanna / OST -- (202)260-7588

Edward V. Ohanian / OST -- (202)260-7571

CAA - NO DATA

WQCHU-

Water and Fish Consumption: 7.1E-5 ug/L

Fish Consumption Only: 7.6E-5 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient concentration should be zero. However, zero may not be attainable at this time, so the recommended criterion represents a E-6 estimated incremental increase of cancer risk over a lifetime.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute – 1.0E+0 ug/L
Chronic – 1.9E-3 ug/L

Marine:

Acute – 7.1E-1 ug/L
Chronic – 1.9E-3 ug/L

Considers technological or economic feasibility? – NO

Discussion – Criteria were derived from a minimum data base consisting of acute tests on a variety of species. Requirements and methods are covered in the reference to the Federal Register.

Reference – 45 FR 79318 (11/28/80)

EPA Contact – Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

No data available

MCL -

No data available

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

Status – Listed (Proposed, 1991)

Discussion – "Unregulated" contaminants are those contaminants for which EPA establishes a monitoring requirement but which do not have an associated final MCLG, MCL, or treatment technique. EPA may regulate these contaminants in the future.

Monitoring requirement – All systems to be monitored unless a vulnerability assessment determines the system is not vulnerable.

Analytical methodology – Microextraction/gas chromatography (EPA 505); electron-capture/gas chromatography (EPA 508); gas chromatographic/mass

spectrometry (EPA 525).

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

SMCL - NO DATA

FISTD-

No data available

FIREV-

Action -- Registration canceled (1974)

Considers technological or economic feasibility? -- NO

Summary of regulatory action -- Cancellation of all but termiticide and use. Criteria of concern: carcinogenicity, bio-accumulation, hazard to wildlife, and other chronic effects.

Reference -- 39 FR 37246 (10/18/74)

EPA Contact -- Special Review Branch / OPP (703)557-7400 / FTS 557-7400

CERC -

Value (status) -- 1 pound (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The RQ for dieldrin is based on aquatic toxicity as established under CWA Section 311 (40 CFR 117.3) and potential carcinogenicity. The available data indicate that the aquatic 96-Hour Median threshold Limit is less than 0.1 ppm, which corresponds to an RQ of 1 pound. Available data also indicate a hazard ranking of high and a weight of evidence classification of Group B2, which corresponds to an RQ of 1 pound.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline

(800)424-9346 / (202)260-3000 / FTS 260-3000

SARA - NO DATA

RCRA -

Status – Listed

Reference – 52 FR 25942 (07/09/87)

EPA Contact – RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - Chernoff, N., R.J. Kavlock, J.R. Kathrein, J.M. Dunn and J.K. Haseman. 1975. Prenatal effects of dieldrin and photodieldrin in mice and rats. Toxicol. Appl. Pharmacol. 31: 302-308.

OREF - Fitzhugh, O.G., A.A. Nelson and M.L. Quaife. 1964. Chronic oral toxicity of aldrin and dieldrin in rats and dogs. Food Cosmet. Toxicol. 2: 551-562.

OREF - U.S. EPA. 1987. Dieldrin: Health Advisory. Office of Drinking Water, Washington, DC. NTIS PB 88-113543/AS.

OREF - Walker, A.I.T., D.E. Stevenson, J. Robinson, E. Thorpe and M. Roberts. 1969. The toxicology and pharmacodynamics of dieldrin (HEOD): Two-year oral exposures of rats and dogs. Toxicol. Appl. Pharmacol. 15: 345-373.

OREF - Walker, A.I.T., E. Thorpe and D.E. Stevenson. 1972. The toxicology of dieldrin (HEOD). I. Long-term oral toxicity studies in mice. Food Cosmet. Toxicol. 11: 415-432.

IREF - None

CREF - Ahmed, F.E., R.W. Hart and N.J. Lewis. 1977. Pesticide induced DNA damage and its repair in cultured human cells. Mutat. Res. 42: 161-174.

CREF - Bidwell, K., E. Weber, I. Neinhold, T. Connor and M.S. Legator. 1975. Comprehensive evaluation for mutagenic activity of dieldrin. Mutat. Res. 31: 314. (Abstract)

CREF - Davis, K.J. 1965. Pathology report on mice fed aldrin, dieldrin, heptachlor or heptachlor epoxide for two years. Internal FDA memorandum to Dr. A.J. Lehman. July 19. (Cited in: U.S. EPA, 1986)

CREF - Davis, K.J. and O.G. Fitzhugh. 1962. Tumorigenic potential of aldrin and dieldrin for mice. Toxicol. Appl. Pharmacol. 4: 187-189.

CREF - Deichmann, W.B., W.E. MacDonald, E. Blum, et al. 1970. Tumorigenicity of aldrin, dieldrin and endrin in the albino rat. Ind. Med. Surg. 39: 426-434.

CREF - Ditraglia, D., D.P. Brown, T. Namekata and M. Iverson. 1981. Mortality study of workers employed at organochlorine pesticide manufacturing plants. Scand. J. Work. Env. Health. 7 (Suppl. 4): 140-146.
CREF - Epstein, S.S. 1975a. The carcinogenicity of dieldrin. Part 1. Sci. Total Environ. 4: 1-52.
CREF - Epstein, S.S. 1975b. The carcinogenicity of dieldrin. Part 2. Sci. Total Environ. 4: 205-217.
CREF - Epstein, S.S. 1976. Case study 5: Aldrin and dieldrin suspension based on experimental evidence and evaluation and societal needs. Ann. NY. Acad. Sci. 271: 187-195.
CREF - Fahrig, R. 1974. Comparative mutagenicity studies with pesticides. IARC Scientific Press No. 10.
CREF - Fitzhugh, O.G., A.A. Nelson and M.L. Quaife. 1964. Chronic oral toxicity of aldrin and dieldrin in rats and dogs. Food Cosmet. Toxicol. 2: 551-562.
CREF - Haworth, S., T. Lawlor, K. Mortelmans, W. Speck and E. Zeigler. 1983. Salmonella mutagenicity test results for 250 chemicals. Environ. Mutag. 5(Suppl. 1): 1-142.
CREF - Majumdar, S.K., H.A. Kopelman and M.J. Schnitman. 1976. Dieldrin-induced chromosome damage in mouse bone-marrow and WI-38 human lung cells. J. Hered. 67: 303-307.
CREF - Majumdar, S.K., L.G. Maharam and G.A. Viglianti. 1977. Mutagenicity of dieldrin in the Salmonella-microsome test. J. Hered. 68: 184-185.
CREF - Markaryan, D.S. 1966. Cytogenic effect of some chlorinated insecticides on mouse bone-marrow cell nuclei. Soviet Genetics. 2(1): 80-82.
CREF - Marshall, T.C., H.W. Dorough and H.E. Swim. 1976. Screening of pesticides for mutagenic potential using Salmonella typhimurium mutants. J. Agric. Chem. 24: 560-563.
CREF - Meierhenry, E.F., B.H. Reuber, M.E. Gershwin, L.S. Hsieh and S.W. French. 1983. Deildrin-induced mallory bodies in hepatic tumors of mice of different strains. Hepatology. 3: 90-95.
CREF - NCI (National Cancer Institute). 1978a. Bioassays of aldrin and dieldrin for possible carcinogenicity. DHEW Publication No. (NIH) 78-821. National Cancer Institute Carcinogenesis Technical Report Series, No. 21. NCI-CG-TR-21.
CREF - NCI (National Cancer Institute). 1978b. Bioassays of aldrin and dieldrin for possible carcinogenicity. DHEW Publication No. (NIH) 78-822. National Cancer Institute Carcinogenesis Technical Report Series, No. 22. NCI-CG-TR-22.
CREF - Probst, G.S., R.E. McMahon, L.W. Hill, D.Z. Thompson, J.K. Epp and S.B. Neal. 1981. Chemically induced unscheduled DNA synthesis in primary rat hepatocyte cultures: A comparison with bacterial mutagenicity using 218 chemicals. Environ. Mutagen. 3: 11-32.
CREF - Reuber, M.D. 1974. Exhibit 42. Testimony at hearings on aldrin/dieldrin. (Cited in: Epstein, 1975a)
CREF - Rocchi, P., P. Perocco, W. Alberghini, A. Fini and G. Prodi. 1980. Effect of pesticides on scheduled and unscheduled DNA synthesis of rat thymocytes and human lymphocytes. Arch. Toxicol. 45: 101-108.
CREF - Shirasu, Y., M. Moriya, K. Kato, A. Furuhashi and T. Kada. 1976. Mutagenicity screening of pesticides in the microbial system. Mutat. Res. 40(1): 19-30.
CREF - Song, J. and W.E. Harville. 1964. Carcinogenicity of aldrin and dieldrin in mouse and rat liver. Fed. Proc. Fed. Am. Soc. Exp. Biol. 23: 336

CREF - Tennekes. H.A., A.S. Wright. K.M. Dix and J.H. Koeman. 1981. Effects of dieldrin, diet. and bedding on enzyme function and tumor incidence in livers of male CF-1 mice. Cancer Res. 41: 3615-3620.
CREF - Thorpe. E. and A.I.T. Walker. 1973. The toxicology of dieldrin (HEOD). Part
HAREF- NO DATA

1 - IRIS
NAME - Di(2-ethylhexyl)phthalate (DEHP)
RN - 117-81-7
IRSN - 13
DATE - 930201
UPDT - 02/01/93, 3 fields
STAT - Oral RfD Assessment (RDO) on-line 05/01/91
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) on-line 02/01/93
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 03/01/88 RDO Text added to paragraph 1
IRH - 09/07/88 CAR Carcinogen summary on-line
IRH - 02/01/89 CAREV Study description revised
IRH - 02/01/89 CARDR Primary contact's phone number corrected
IRH - 07/01/89 REFS Bibliography on-line
IRH - 08/01/89 RDO Text revised
IRH - 05/01/90 CAREV Text revised
CONTINUE PRINTING? (YES/NO/CONT)
USER:
c

IRH - 05/01/90 CREF Kozumbo et al., 1982 citation added
IRH - 05/01/91 RDO Corrected principal study title
IRH - 05/01/91 RDO 2nd para, line 3 units corrected from g/kg to mg/kg
IRH - 08/01/91 CARDR Primary and secondary contacts changed
IRH - 08/01/91 RCRA EPA contact changed
IRH - 01/01/92 RDO Secondary contact changed
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 02/01/93 CARDR Primary contact changed
RLEN - 20331
SY - BEHP
SY - Bis(2-ethylhexyl)-1,2-benzene-dicarboxylate
SY - Bis(2-ethylhexyl)phthalate
SY - Bisoflex 81
SY - Bisoflex DOP
SY - Compound 889
SY - DAF 68
SY - DEHP
SY - Di(2-ethylhexyl)orthophthalate
SY - Di(2-ethylhexyl)phthalate
SY - Dioctyl phthalate
SY - Di-sec-octyl phthalate
SY - DOP
SY - Ergoplast FDO
SY - Ethylhexyl phthalate
SY - 2-Ethylhexyl phthalate
SY - Eviplast 80
SY - Eviplast 81
SY - Fleximel
SY - Flexol DOP
SY - Flexol plasticizer DOP

SY - Good-Rite GP 264
SY - Hatcol DOP
SY - Hercoflex 260
SY - Kodaflex DOP
SY - Mollan O
SY - NCI- C52733
SY - Nuoplaz DOP
SY - Octoil
SY - Octyl phthalate
SY - Palatinol AH
SY - Phthalic acid, Bis(2-ethylhexyl) ester
SY - Phthalic acid, dioctyl ester
SY - Pittsburgh PX-138
SY - Platinol DOP
SY - RC Plasticizer DOP
SY - RCRA waste number U028
SY - Reomol D 79P
SY - Reomol DOP
SY - Sicol 150
SY - Staflex DOP
SY - Truflex DOP
SY - Vestinol AH
SY - Vinicizer 80
SY - Witcizer 312

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Increased relative liver weight	NOAEL: none LOAEL: 0.04% of diet (19 mg/kg bw/day)	1000	1	2E-2 mg/kg/day
Guinea Pig Sub-chronic-to-Chronic Oral Bioassay				

Carpenter et al., 1953

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*Conversion Factors: none

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o ORAL RFD STUDIES :

Carpenter, C.P., C.S. Weil and H.F. Smyth. 1953. Chronic oral toxicity of di(2-ethylhexyl)phthalate for rats and guinea pigs. Arch. Indust. Hyg. Occup. Med. 8: 219-226.

The following numbers of guinea pigs were fed diets containing DEHP for a period of 1 year: 24 males and 23 females consumed feed containing 0.13% DEHP; 23 males and 23 females consumed feed containing 0.04% DEHP; and 24 males and 22 females were fed the control diet. These dietary levels corresponded to 64 or 19 mg/kg bw/day based on measured food consumption. No treatment-related effects were observed on mortality, body weight, kidney weight, or gross pathology and histopathology of kidney, liver, lung, spleen,

or testes. Statistically significant increases in relative liver weights were observed in both groups of treated females (64 and 19 mg/kg bw/day).

Groups of 32 male and 32 female Sherman rats were maintained for 2 years on diets containing either 0.04, 0.13 or 0.4% DEHP (equivalent to 20, 60, and about 195 mg/kg bw/day based on measured food consumption). An F1 group of 80 animals was fed the 0.04% diet for 1 year. Mortality in the F1 treated and control groups was high; 46.2 and 42.7%, respectively, survived to 1 year. There was, however, no effect of treatment on either parental or F1 group mortality, life expectancy, hematology, or histopathology of organs. Both parental and F1 rats receiving the 0.4% DEHP diet were retarded in growth and had increased kidney and liver weights.

It appears that guinea pigs offer the more sensitive animal model for DEHP toxicity. A LOAEL in this species is determined to be 19 mg/kg/day.

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o ORAL RFD UNCERTAINTY :

UF -- Factors of 10 each were used for interspecies variation and for protection of sensitive human subpopulations. An additional factor of 10 was used since the guinea pig exposure was longer than subchronic but less than lifetime, and because, while the RfD is set on a LOAEL, the effect observed was *considered to be minimally adverse*.

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o ORAL RFD MODIFYING FACTOR :

MF -- None

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o ORAL RFD COMMENTS :

Dietary levels of 0, 0.01, 0.1, and 0.3% DEHP (greater than 99% pure) were administered to male and female CD-1 mice that were examined for adverse fertility and reproductive effects using a continuous breeding protocol. DEHP was a reproductive toxicant in both sexes significantly decreasing fertility and the proportion of pups born alive per litter at the 0.3% level, and inducing damage to the seminiferous tubules. DEHP has been observed to be both fetotoxic and teratogenic (Singhe, 1972; Shiot and Nishimura, 1982).

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o ORAL RFD CONFIDENCE :

Study -- Medium
Data Base -- Medium
RfD -- Medium

The study by Carpenter et al. (1953) utilized sufficient numbers of guinea pigs and measured multiple endpoints. The fact that there were only two concentrations of DEHP tested precludes a rating higher than medium. Since there are corroborating chronic animal bioassays, the data base is likewise rated medium. Medium confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

The RfD has been reviewed by the RfD Work Group. Documentation may be found in the meeting notes of 01/22/86.

o REVIEW DATES : 01/22/86
o VERIFICATION DATE : 01/22/86
o EPA CONTACTS :

Michael L. Dourson / OHEA -- (513)569-7533

W. Bruce Peirano / OHEA -- (513)569-7553

RDI - NO DATA
CAREV-
o CLASSIFICATION : B2; probable human carcinogen.
o BASIS FOR CLASSIFICATION : Orally administered DEHP produced significant dose-related increases in liver tumor responses in rats and mice of both sexes.
o HUMAN CARCINOGENICITY DATA :

Inadequate. Thiess et al. (1978) conducted a mortality study of 221 DEHP production workers exposed to unknown concentrations of DEHP for 3 months to 24 years. Workers were followed for a minimum of 5 to 10 years (mean follow-up time was 11.5 years). Eight deaths were reported in the exposed population. Deaths attributable to pancreatic carcinoma (1 case) and uremia (1 case in which the workers also had urethral and bladder papillomas) were significantly elevated in workers exposed for >15 years when compared to the corresponding age groups in the general population. The study is limited by a short follow-up period and unquantified worker exposure. Results are considered inadequate for evidence of a causal association.

o ANIMAL CARCINOGENICITY DATA :

Sufficient. In an NTP (1982) study, 50 male and 50 female fisher 344 rats per group were fed diets containing 0, 6000 or 12,000 ppm DEHP for 103 weeks. Similarly, groups of 50 male and 50 female B6C3F1 mice were given 0, 3000 or 6000 ppm DEHP in the diet for 103 weeks. Animals were killed and examined histologically when morbund or after 105 weeks. No clinical signs of toxicity were observed in either rats or mice. A statistically significant increase in the incidence of hepatocellular carcinomas and combined incidence of carcinomas and adenoma were observed in female rats and both sexes of mice. The combined incidence of neoplastic nodules and hepatocellular carcinomas was statistically significantly increased in the high-dose male rats. A positive dose response trend was also noted.

Carpenter et al. (1953) found no malignant tumors in treated groups of 32 male and 32 female Sherman rats. Animals were given 400, 1300 or 4000 ppm DEHP in the diet for 1 year and reduced to a maximum of 8 males and 8 females and treated for another year. Controls, F1 and 4000 ppm groups were sacrificed after being maintained on control or 4000 ppm diets for 1 year. Only 40 to 47% of the animals in each group, including F1 animals, survived 1 year. Thus, an insufficient number of animals were available for a lifetime evaluation.

Carpenter et al. (1953) did not find a carcinogenic effect in guinea pigs and dogs exposed to 1300 or 4000 ppm DEHP Both guinea pigs and dogs were terminated after 1 year of exposure. The treatment and survival periods for these animals were considerably below their lifetimes.

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o SUPPORTING DATA :

Studies indicate that DEHP is not a direct acting mutagen in either a forward mutation assay in Salmonella typhimurium (Seed, 1982) or the rec assay in Bacillus subtilis (Tomita et al., 1982). DEHP did not induce mutations in a modified reverse mutation plate incorporation assay in Salmonella strains TA100 and TA98 at concentrations up to 1000 ug/plate in the presence or absence of S9 hepatic homogenate (Kozumbo et al., 1982). MEHP, the monoester form of DEHP and a metabolite is positive in the rec assay and in the reverse mutation assay in Salmonella. In the absence of exogenous metabolism MEHP produced chromosomal aberrations and sister chromatid exchanges in V79 cells. Both DEHP and MEHP induced chromosomal aberrations and morphological transformation in cultured fetal Syrian hamster cells exposed in utero (Tomita et al., 1982). Chromosomal effects were not found in CHO mammalian cells (Phillips et al., 1982) exposed to DEHP. DEHP was weakly positive with metabolic activation in only one of several studies testing for mutagenic activity at the thymidine kinase locus in L5178Y mouse lymphoma cells (Ashby et al., 1985). DEHP is a potent inducer of hepatic peroxisomal enzyme activity (Ganning et al., 1984).

CARO -
o CLASSIFICATION : B2; probable human carcinogen.
o BASIS FOR CLASSIFICATION : Orally administered DEHP produced significant dose-related increases in liver tumor responses in rats and mice of both sexes.
o ORAL SLOPE FACTOR : 1.4E-2/mg/kg/day
o DRINKING WATER UNIT RISK : 4.0E-7 per (ug/L)
o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk
o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	3E+2 ug/L
E-5 (1 in 100,000)	3E+1 ug/L
E-6 (1 in 1,000,000)	3E+0 ug/L

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o ORAL DOSE-RESPONSE DATA :

Tumor Type -- Mouse/B6C3FI, male
Test Animals -- hepatocellular carcinoma and adenoma
Route -- diet
Reference -- NTP, 1982

—— Dose ——

The values in the 1988 Drinking Water Criteria Document for Phthalic Acid Esters (External Review Draft) have received Agency review.
DOCUMENT

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o REVIEW DATES : 08/26/87; 10/07/87
o VERIFICATION DATE : 10/07/87
o EPA CONTACTS :

Brian J. Commons / OST -- (202)260-7589

Linda R. Papa / OHEA -- (513)569-7587

HAONE- NO DATA

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA - NO DATA

WQCHU-

Water and Fish Consumption: 1.5E+4 ug/L

Fish Consumption Only: 5E+4 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The WQC of 1.5E+4 ug/L is based on consumption of contaminated aquatic organisms and water. A WQC of 5E+4 ug/L has also been established based on consumption of contaminated aquatic organisms alone.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 4.0E+2 ug/L
Chronic -- 3.6E+2 ug/L

Marine:

Acute -- 4.0E+2 ug/L
Chronic -- 3.6E+2 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute and chronic tests on a variety of species. EPA is currently considering withdrawing some or all the values.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value -- 0 mg/L (Proposed, 1990)

Considers technological or economic feasibility? -- NO

Discussion -- The proposed MCLG for di(2-ethylhexyl)phthalate is zero based on the evidence of carcinogenic potential (B2)

Reference -- 55 FR 30370 (07/25/90)

EPA Contact -- Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value -- 0.004 mg/L (Proposed, 1990)

Considers technological or economic feasibility? -- YES

Discussion -- The MCL is based on 10x the MDL and is associated with a maximum lifetime individual risk of 1 E-6.

Monitoring requirements -- Community and non-transient water system monitoring based on state vulnerability assessment; vulnerable systems to be monitored quarterly for one year; repeat monitoring dependent upon detection and size of system.

Analytical methodology -- Photoionization/gas chromatography (EPA 502.2); gas chromatographic/mass spectrometry (EPA 524.1, 524.2): PQL= 0.004 mg/L.

Best available technology -- Granular activated carbon

Reference -- 55 FR 30370 (07/25/90)

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

Status -- Listed (Final, 1991)

Discussion -- "Unregulated" contaminants are those contaminants for which EPA establishes a monitoring requirement but which do not have an associated final MCLG, MCL, or treatment technique. EPA may regulate these contaminants in the future.

Monitoring requirement -- All systems to be monitored unless a vulnerability assessment determines the system is not vulnerable.

Analytical methodology -- Gas chromatography (EPA 506); gas chromatography/ mass spectrometry (EPA 525).

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

SMCL - NO DATA

FISTD- NO DATA

FIREV- NO DATA

CERC -

Value (status) -- 100 pounds (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The 100-pound RQ is based on assessment for potential carcinogenicity. Available data indicate a hazard ranking of low based on a potency factor of 0.015/mg/kg/day and weight-of-evidence group B2, which

corresponds to an RQ of 100 pounds.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

SARA - NO DATA

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - Carpenter, C.P., C.S. Weil and H.F. Smyth. 1953. Chronic oral toxicity of di(2-ethylhexyl)phthalate for rats and guinea pigs. Arch. Indust. Hyg. Occup. Med. 8: 219-226.

OREF - NTP (National Toxicology Program). 1984. Di(2-ethylhexyl)phthalate: Reproduction and fertility assessment in CD-1 mice when administered by gavage. Final Report. NTP-84-079. NTP, Research Triangle Park, NC.

OREF - Shiota, K. and H. Nishimura. 1982. Teratogenicity of di-2-ethylhexyl phthalate and di-n-butyl phthalate in mice. Environ. Health Perspect. 45(0): 65-70.

OREF - Singhe, A.R., W.H. Lawrence and J. Autian. 1972. Teratogenicity of phthalate esters in rats. J. Pharmacol. Sci. 61: 51.

IREF - None

CREF - Albro, P.W., J.T. Corbett, J.L. Schroeder, et al. 1982. Pharmacokinetics, interactions with macromolecules and species differences in metabolism of DEHP. Environ. Health Perspect. 45: 19-25.

CREF - Ashby, J., F.J. de Serres, M. Draper, et al. 1985. Evaluation of short-term tests for carcinogens. Report of the International Programme on Chemical Safety's Collaborative Study on In Vitro Assays. Elsevier Science Publishers, Amsterdam.

CREF - Carpenter, C.P., C.S. Weil and H.F. Smith, Jr. 1953. Chronic oral toxicity of di-(2-ethylhexyl)phthalate for rats, guinea pigs and dogs. AMA Arch. Ind. Hyg. Occup. Med. 8: 219-226.

CREF - Ganning, A.E., V. Brunk and G. Dallner. 1984. Phthalate esters and their effect on the liver. Hepatology. 4(3): 541-547.

CREF - Kluwe, W.M. 1982. Overview of phthalate ester pharmacokinetics in mammalian species. Environ. Health Perspect. 45: 3-10.
CREF - Kozumbo, W.J., R. Kroll and R.J. Rubin. 1982. Assessment of the mutagenicity of phthalate esters. Environ. Health Perspect. 45: 103-109.
CREF - Lhuguenot, J.C., A.M. Mitchell, G. Milner, E.A. Lock and C.R. Elcombe. 1985. The metabolism of di-(2-ethylhexyl)phthalate (DEHP) and mono-(2-ethylhexyl)phthalate (MEHP) in rats: In vivo and in vitro dose and time dependency of metabolism. Toxicol. Appl. Pharmacol. 80: 11-22.
CREF - NTP (National Toxicology Program). 1982. Carcinogenesis bioassay of di-(2-ethylhexyl)phthalate (CAS No. 117-81-7) in F344 rats and B6C3F, mice (feed study). NTP Tech. Rep. Ser. TR No. 217, NTP, Research Triangle Park, NC.
CREF - Phillips, B.J., T.E.B. James and S.D. Gangolli. 1982. Genotoxicity studies of di-(2-ethylhexyl)phthalate and its metabolites in CHO cells. Mutat. Res. 102: 297-304.
CREF - Pollack, G.M., R.C. Li, J.C. Ermer and D.D. Shen. 1985. Effects of route of administration and repetitive dosing on the disposition kinetics of di-(2-ethylhexyl)phthalate and its mono-de-esterified metabolite in rats. Toxicol. Appl. Pharmacol. 79: 246-256.
CREF - Seed, J.L. 1982. Mutagenic activity of phthalate esters in bacterial liquid suspension assays. Environ. Health Perspect. 45: 111-114.
CREF - Tanaka, A., T. Adachi, T. Takahashi and T. Yamaha. 1975. Biochemical studies on phthalic esters. I. Elimination, distribution and metabolism of di-(2-ethylhexyl)phthalate in rats. Toxicology. 4: 253-264.
CREF - Thiess, A.M., R. Frentzel-Beyme and R. Wieland. 1978. Mortality study in workers exposed to di-(2-ethylhexyl)phthalate (DOP). In: Moglichkerten und Grenzen des Biological Monitoring. Arbeitsmedizinische Probleme des Dienstleistungsqewerbes. Arbeitsmedizinische kolloquium [Possibilities and Limits of Biological Monitoring. Problems of Occupational Medicine in Small Industries. Colloquim in Occupational Medicine], Frankfurt/M., May 1978. Stuttgart, A.W. Gentner, p. 155-164. (Ger.)
CREF - Tomita, I., Y. Nakamura, N. Aoki and N. Inui. 1982. Mutagenic/carcinogenic potential of DEHP and MEHP. Environ. Health Perspect. 45: 119-125.
CREF - Williams, D.T. and B.J. Blanchfield. 1975. The retention, distribution, excretion and metabolism of dibutylphthalate-7-14C in the rat. J. Agric. Food Chem. 23: 854-857.
CREF - U.S. EPA. 1988. Drinking Water Criteria Document for Phthalic Acid Esters. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. (External Review Draft).
HAREF- None

[IRIS] SS 4 /cf?
USER:

1 - IRIS
NAME - Manganese
RN - 7439-96-5
IRSN - 372
DATE - 950607
UPDT - 06/07/95, 3 fields
STAT - Oral RfD Assessment (RDO) on-line 06/01/95
STAT - Inhalation RfC Assessment (RDI) on-line 12/01/93
STAT - Carcinogenicity Assessment (CAR) on-line 03/01/94
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 09/26/88 CAR Carcinogen summary on-line
IRH - 09/01/89 REFS Bibliography on-line
IRH - 06/01/90 RDO Oral RfD now under review
IRH - 08/01/90 RDO Oral RfD summary on-line
IRH - 08/01/90 CAR Text edited
IRH - 08/01/90 REFS Oral RfD references added
IRH - 09/01/90 RDI Inhalation RfC now under review
IRH - 12/06/90 RDI Inhalation RfC on-line
IRH - 12/06/90 IREF Inhalation RfC references added
IRH - 01/01/92 EXSR Regulatory Action section on-line
IRH - 06/01/92 IREF Iregren, 1990 and Nishiyama et al., 1975 pages corrected
IRH - 08/01/92 RDO Oral RfD noted as pending change
IRH - 08/01/92 RDO Work group review date added
IRH - 10/01/92 RDO Oral RfD withdrawn; new summary in preparation
IRH - 10/01/92 RDO Work group review date added
IRH - 10/01/92 OREF Oral RfD references withdrawn
IRH - 01/01/93 RDO Oral RfD replaced (RfD changed)
IRH - 01/01/93 OREF Oral RfD references replaced
IRH - 05/01/93 RDO Work group review date added
IRH - 07/01/93 CAREV 'Inadequate' added to 1st paragraph
IRH - 07/01/93 CARDR EPA Documentation clarified
IRH - 11/01/93 RDI Inhalation RfC noted as pending changed
IRH - 11/01/93 RDI Work group review date added
IRH - 12/01/93 RDI Inhalation RfC replaced; RfC changed
IRH - 12/01/93 IREF Inhalation RfC references replaced
IRH - 01/01/94 RDO Oral RfD noted as pending change
IRH - 01/01/94 RDO Work group review date added
IRH - 03/01/94 CARDR Primary contact changed
IRH - 04/01/94 RDO Text revised
IRH - 04/01/94 RDO Text revised
IRH - 04/01/94 RDO Text revised
IRH - 04/01/94 RDO Text revised
IRH - 04/01/94 OREF Oral RfD references revised
IRH - 06/01/95 RDO Oral RfD noted as pending change
IRH - 06/01/95 RDO Work group review date added
RLEN - 83986
SY - COLLOIDAL MANGANESE
SY - MAGNACAT
SY - MANGAN
SY - Manganese
SY - MANGAN NITRIDOVANY
SY - TRONAMANG

Manganese

RDO -
o ORAL RFD SUMMARY :

NOTE: The Oral RfD for manganese may change in the near future pending the outcome of a further review now being conducted by the RfD/RfC Work Group.

Critical Effect	Experimental Doses*	UF	MF	RfD
CNS effects Human Chronic Ingestion Data Kondakis et al., 1989	NOAEL (water): 0.005 mg/kg-day LOAEL (water): 0.06 mg/kg-day	1 mg/kg-day (water)	1	5E-3
CNS effects Human Chronic Ingestion Data WHO, 1973; Schroeder et al., 1966; NRC, 1989	NOAEL (food): 0.14 mg/kg-day LOAEL (food): None	1 mg/kg-day (food)	1	1.4E-1

*Conversion Factors and Assumptions: The arithmetic mean of the range of manganese concentrations for the NOAEL and LOAEL are 167 ug/L and 1950 ug/L, respectively. Assuming a water consumption of 2 L/day and a body weight of 70 kg, these are equivalent to 0.005 mg/kg-day and 0.06 mg/kg-day, respectively. The water RfD assumes a separate dietary intake of manganese, as this essential element is found in varying amounts in all diets. The NOAEL of 10 mg/day (0.14 mg/kg-day for 70 kg adult) for chronic human consumption of manganese in the diet is based on a composite of data from all three references. WHO (1973) reported no adverse effects in humans consuming supplements of 8-9 mg Mn/day (0.11-0.13 mg/kg-day). Schroeder et al. (1966) reported a chronic human NOAEL of 11.5 mg Mn/day (0.16 mg/kg-day).

o ORAL RFD STUDIES :

WHO (World Health Organization). 1973. Trace Elements in Human Nutrition: Manganese. Report of a WHO Expert Committee. Technical Report Service, 532, WHO, Geneva, Switzerland. p. 34-36.

Schroeder, H.A., J.J. Balassa and I.H. Tipton. 1966. Essential trace metals in man: Manganese, a study in homeostasis. J. Chron. Dis. 19: 545-571.

NRC (National Research Council). 1989. Recommended Dietary Allowances, 10th ed. Food and Nutrition Board, National Research Council, National Academy Press, Washington, DC. p. 230-235.

Kondakis, X.G., N. Makris, M. Leotsinidis, M. Prinou and T. Papapetropoulos. 1989. Possible health effects of high manganese concentration in drinking water. Arch. Environ. Health. 44: 175-178.

The World Health Organization (WHO) reviewed several investigations of adult diets and reported the average daily consumption of manganese to range from 2.0-8.8 mg Mn/day. Higher manganese intakes are associated with diets high in whole-grain cereals, nuts, green leafy vegetables, and tea. From manganese balance studies, the WHO concluded that 2-3 mg/day is adequate for adults and 8-9 mg/day is "perfectly safe."

Evaluations of standard diets from the United States, England, and Holland reveal average daily intakes of 2.3-8.8 mg Mn/day. Depending on individual diets, however, a normal intake may be well over 10 mg Mn/day, especially from a vegetarian diet. While the actual intake is higher, the bioavailability of manganese from a vegetarian diet is lower, thereby decreasing the actual absorbed dose. This is discussed in more detail in the Additional Studies / Comments Section.

No signs of toxicity were reported in patients (number not specified) given 30 mg manganese citrate (9 mg Mn/day) for many months. Assuming the patients also consumed 2.5 mg Mn/day in food, the total manganese intake would be approximately 11.5 mg Mn/day.

The Food and Nutrition Board of the National Research Council (NRC, 1989) determined an "adequate and safe" intake of manganese to be 2-5 mg/day for adults. This level was chosen because it includes an "extra margin of safety" from the level of 10 mg/day, which the NRC considered to be safe for an occasional intake.

There is one epidemiologic study of manganese in drinking water performed by Kondakis et al. (1989). Three areas in northwest Greece were chosen for this study, with manganese concentrations in natural well water of 3.6-14.6 ug/L in area A, 81.6-252.6 ug/L in area B, and 1600-2300 ug/L in area C. The total population of the three areas studied ranged from 3200 to 4350 people. The study included only individuals over the age of 50 drawn from a random sample of 10% of all households (n=62, 49, and 77 for areas A, B, and C, respectively). The authors reported that "all areas were similar with respect to social and dietary characteristics," but few details were reported. The three areas are located within a 200-square km region. Although the amount of manganese in the diet was not reported, the authors indicated that most of the food was purchased from markets and is expected to be comparable for all three areas. Chemicals other than manganese in the well water were reported to be within Economic Community (EC) standards, except for hardness (120-130 mg calcium carbonate per liter). The individuals chosen were submitted to a neurologic examination, the score of which represents a composite of the presence and severity of 33 symptoms (e.g., weakness/fatigue, gait disturbances, tremors, dystonia). Whole blood and hair manganese concentrations also were determined. The mean concentration of manganese in hair was 3.51, 4.49, and 10.99 ug/g dry weight for areas A, B, and C, respectively ($p<0.0001$ for area C versus A). The concentration of manganese in whole blood did not differ between the three areas, but this is not considered to be a reliable indicator of manganese exposure. The mean (x) and range (r) of neurologic scores were as follows: Area A (males: x=2.4, r=0-21; females: x=3.0, r=0-18; both x=2.7, r=0-21); Area B (males x=1.6, r=0-6; females: x=5.7 r=0-43; both: x=3.9, r=0-43); and Area C (males: x=4.9, r=0-29; females: x=5.5, r=0-21; both x=5.2, r=0-29). The authors indicate that the difference in mean scores for area C versus A was significantly increased

(Mann-Whitney z=3.16, p=0.002 for both sexes combined). In a subsequent analysis, logistic regression indicated that there is a significant difference between areas A and C even when both age and sex are taken into account (Kondakis, 1990). Therefore, the LOAEL for this study is defined by Area C (1600-2300 ug/L) and NOAEL by Area B (81.6-252.6 ug/L). Using the arithmetic means of the ranges of manganese provided (1950 ug/L for Area C and 167 ug/L for Area B), and assuming a water consumption of 2 L/day and a body weight of 70 kg, the LOAEL is equivalent to 0.06 mg Mn/kg-day and the NOAEL is equivalent to 0.005 mg Mn/kg-day.

The individuals examined in the Greek epidemiologic study also had exposure to manganese in their diet. This was roughly estimated to be 10-15 mg/day because of the high intake of vegetables (Kondakis, 1990). In a subsequent correspondence from the investigator, a lower dietary estimate of 5-6 mg Mn/day was stated (Kondakis, 1993), but data have not been supplied to substantiate either estimate. Because of the uncertainty in the amount of manganese in the diet, it is difficult to estimate a total oral intake. The lack of dietary data is recognized as a source of significant uncertainty in this assessment.

The use of the Kondakis et al. (1989) study in supporting the derivation of a separate "water RfD" assumes that the manganese exposure from drinking water is in addition to that found in the diet. This assumption is made primarily because the differences in the bioavailability of manganese in food as compared with that of manganese in water may be such that it is inappropriate to add these intakes together. Unfortunately, while it is agreed that the bioavailability of manganese may vary substantially, relatively few data are available to quantitate these differences, and the number of variables that may affect the uptake of manganese are such that to determine a single value for the absorption of manganese from any medium is not appropriate. These issues are discussed further in the Additional Studies / Comments Section. If the water RfD is used to support a drinking water standard for manganese, no relative source contribution needs to be applied because dietary intake of manganese is already assumed. An RfD of 0.005 mg/kg-day for manganese in water is equivalent to a drinking water standard of 0.2 mg/L.

A report by Kawamura et al. (1941) is the only epidemiologic study describing toxicologic responses in humans consuming large amounts of manganese dissolved in drinking water. The manganese came from about 400 dry-cell batteries buried near a drinking water well resulting in high levels of both manganese and zinc in the water. Twenty-five cases of manganese poisoning were reported, with symptoms including lethargy, increased muscle tonus, tremor and mental disturbances. The most severe symptoms were seen in elderly people, while children appeared to be unaffected. Three individuals died, one from suicide. The cause of death for the other two was not reported, but the autopsy of one individual revealed manganese concentration in the liver to be 2-3 times higher than in control autopsies. Zinc levels also were increased in the liver. The well water was not analyzed until 1 month after the outbreak, at which time it was found to contain approximately 14 mg Mn/L. When re-analyzed 1 month later, however, the levels were decreased by about half. Therefore, by retrospective extrapolation, the concentration of manganese at the time of exposure may have been as high as 28 mg Mn/L. Consumption of 2 liters of water per day containing 28 mg/L of

manganese approximates an intake of 0.8 mg/kg-day for a 70-kg adult. The severe effects seen in this study support the LOAEL estimated from the 1989 Kondakis et al. study of 0.06 mg/kg-day, which was associated with less severe effects.

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o ORAL RFD UNCERTAINTY :

UF -- The information used to determine the RfD for manganese in food was taken from many large populations consuming normal diets over an extended period of time with no adverse health effects. As long as physiologic systems are not overwhelmed, humans exert an efficient homeostatic control over manganese such that body burdens are kept constant with variations in diet. It is recognized that manganese is an essential element, being required for normal human growth and maintenance of health. It has also been suggested that children are less susceptible to manganese intoxication and may require slightly higher levels of manganese than adults. This is not true for infants, who may represent a more sensitive subpopulation (see discussion in the Additional Studies / Comments Section). The available information providing a chronic NOAEL in many cross-sections of human populations, taken in conjunction with the essentiality of manganese, warrants an uncertainty factor of 1. For the drinking water RfD, the study by Kondakis et al. (1989) describes a population of older adults (over 50 years) who had consumed well water containing various concentrations of manganese for a lifetime. The study group is considered to represent a sensitive subpopulation, hence an uncertainty factor of 1 is applied.

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o ORAL RFD MODIFYING FACTOR :

MF -- The modifying factors are 1 for both the dietary RfD and the drinking water RfD.

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o ORAL RFD COMMENTS :

While the Greek epidemiologic study (Kondakis et al., 1989) is considered to be acceptable for deriving a separate water RfD for manganese, it is also recognized as being limited in scope. Arguments for using the Greek study are strengthened when it is reviewed in the context of additional information. In addition to the Greek and Japanese studies of humans ingesting manganese in drinking water, one limited oral study has been performed in a group of four Rhesus monkeys (Gupta et al., 1980). Muscular weakness and rigidity of the lower limbs developed after 18 months of exposure to 6.9 mg Mn/kg-day (as MnC12.4H2O). Histologic analysis showed degenerated neurons in the substantia nigra and scanty neuromelanin granules in some other pigmented cells.

A pattern is seen when the two human studies are viewed in conjunction with the study in Rhesus monkeys. Kondakis et al. (1989) identified a NOAEL for humans ingesting manganese in drinking water of 0.005 mg/kg-day and a LOAEL associated with minor neurologic effects of 0.06 mg/kg-day. Kawamura et al. (1941) reported severe effects in a small group of people ingesting about 0.8 mg/kg-day of manganese in water. Some subpopulations (e.g., children) appeared to be unaffected. Finally, Gupta et al. (1980) reported severe effects confirmed by histopathologic analysis in monkeys receiving 6.9 mg/kg manganese.

A couple of case studies have also pointed to the potential for manganese poisoning by routes other than inhalation. One involved a 59-year-old male who was admitted to the hospital with symptoms of classical manganese poisoning, including dementia and a generalized extrapyramidal syndrome (Banta and Markesbery, 1977). The patient's serum, hair, urine, feces, and brain were found to have manganese "elevated beyond toxic levels," perhaps a result of his consumption of "large doses of vitamins and minerals for 4 to 5 years." Unfortunately, no quantitative data were reported.

Another case study of manganese intoxication involved a 62-year-old male who had been receiving total parenteral nutrition that provided 2.2 mg of manganese (form not stated) daily for 23 months (Ejima et al., 1992). The patient's whole blood manganese was found to be elevated, and he was diagnosed as having parkinsonism, with dysarthria, mild rigidity, hypokinesia with masked face, a halting gait, and severely impaired postural reflexes. To be able to compare the manganese load in this individual with that corresponding to an oral intake, the difference between the direct intravenous exposure and the relatively low level of absorption of manganese from the gastrointestinal tract must be taken into account. Assuming an average absorption of roughly 5% of an oral dose, the intravenous dose of 2.2 mg Mn/day would be approximately equivalent to an oral intake of 40 mg Mn/day. This is consistent with the severe neurological effects reported in the Japanese drinking water poisoning (Kawamura et al., 1941) in which it is estimated that the individuals were exposed to about 58 mg Mn/day.

Several oral studies have been performed in rodents, also demonstrating biochemical changes in the brain following administration of 1 mg MnC12.4H2O/mL in drinking water (approximately 38.9 mg Mn/kg-day) (Lai et al., 1981, 1982; Leung et al., 1981; Chandra and Shukla, 1981). However, rodents do not exhibit the same neurologic deficits that humans do following exposure to manganese; thus the relevance of these biochemical changes has been challenged. The problem with using rodents is exemplified by the disease of parkinsonism, which is characterized by effects very similar to those seen in manganese poisoning. Marsden and Jenner (1987) hypothesize that the ability of certain drugs to induce parkinsonism in primates but not in rodents is due to the relative lack of neuromelanin in rodents. Because manganese selectively accumulates in pigmented regions of the brain (e.g., the substantia nigra), this species difference is fundamentally important.

It is also important to recognize the substantial difference in species' requirements for manganese. For rats, the estimated requirement is 50 mg Mn/kg diet (Rogers, 1979). Assuming a food consumption equivalent to 5% of body weight, this corresponds to a requirement of about 2.5 mg Mn/kg-day. Although the dietary requirement for manganese has not been determined quantitatively for humans, the "Estimated Safe and Adequate Daily Dietary Intake" is 2-5 mg/day, or about 0.03-0.07 mg Mn/kg-day, assuming a reference body weight of 70 kg. The dietary requirement for manganese in rats then, is about 50-fold higher than the estimated safe and adequate intake for humans, suggesting that data derived from rodent studies may not be appropriate for use in deriving quantitative risk estimates for manganese in humans.

Another issue of great importance to consider in the risk assessment for manganese concerns the bioavailability of different forms of manganese consumed under different exposure conditions. The separate RfDs for food and

water indicate a potentially higher bioavailability of manganese from drinking water. In considering dietary sources, it is also important to recognize that various dietary factors as well as the form of manganese can have a significant bearing on the dose absorbed from the gastrointestinal tract. Many constituents of a vegetarian diet (e.g., tannins, oxalates, phytates, fiber, calcium, and phosphorus) have been found to inhibit manganese absorption presumably by forming insoluble complexes in the gut. Individuals who are deficient in iron demonstrate an increase in manganese absorption. It is also recognized that manganese uptake and elimination are under homeostatic control, generally allowing for a wide range of dietary intakes considered to be safe. These factors and others are described in a review by Kies (1987). In addition to the influence of extrinsic variables, significant interindividual differences in manganese absorption and retention have been reported. In humans administered a dose of radiolabeled manganese in an infant formula, the mean absorption was 5.9 +/- 4.8%, but the range was 0.8-16%, a 20-fold difference (Davidsson et al., 1989). Retention at day 10 was 2.9 +/- 1.8%, but the range was 0.6-9.2%, again indicating substantial differences between individuals.

In a 100-day dietary study in 6-week-old male mice, Komura and Sakamoto (1991) demonstrated significant differences in tissue levels of manganese in mice fed equivalent amounts of manganese as $MnCl_2.4H_2O$, $Mn(Ac)_2.4H_2O$, $MnCO_3$ and MnO_2. Mice receiving the two soluble forms of manganese (the chloride and acetate salts) were found to gain significantly less weight than controls, while mice consuming the insoluble forms of manganese (the carbonate and dioxide salts) appeared to actually gain slightly more weight than controls. However, the acetate and carbonate groups had significantly higher manganese levels in the liver and kidney compared with the chloride and dioxide groups, both of which were elevated above control levels. Reduced locomotor activity in the carbonate and acetate groups was also reported, perhaps related to the higher tissue levels of manganese. This study points out a need for understanding the effects of the various forms of manganese, of which relatively little is known. More information on manganese speciation can be found in the RfC file on IRIS.

It is also recognized that neonates may be at increased risk of toxicity resulting from exposure to manganese because of a higher level of uptake from the gastrointestinal tract. The uptake and retention of manganese have been reviewed by Lonnerdal et al. (1987). In rats, manganese absorption decreased dramatically as the animals matured. While 24-hour retention values are as high as 80% in 14-day-old pups, this value drops to about 30% by day 18. Low levels of manganese absorption (about 3-4%) have also been reported for mature humans, but few data are available for infants.

Collipp et al. (1983) found that hair manganese levels in newborn infants was found to increase significantly from birth (0.19 ug/g) to 6 weeks of age (0.865 ug/g) and 4 months of age (0.685 ug/g) when the infants were given formula, but that the increase was not significant in babies who were breast-fed (0.330 ug/g at 4 months). While human breast milk is relatively low in manganese (7-15 ug/L), levels in infant formulas are 3-100 times higher. It was further reported in this study that the level of manganese in the hair of learning disabled children (0.434 ug/g) was significantly increased in comparison with that of normal children (0.268 ug/g). Other investigators also have reported an association between elevated levels of manganese in hair

and learning disabilities in children (Barlow and Kapel, 1979; Pihl and Parkes, 1977). Although no causal relationship has been determined for learning disabilities and manganese intake, further research in this area is warranted. High levels of manganese in infant formulas may be of concern because of the increased absorption and retention of manganese that has been reported in neonatal animals (Lonnerdal et al., 1987). Also, manganese has been shown to cross the blood-brain barrier, with the rate of penetration in animal experiments being 4 times higher in neonates than in adults (Mena, 1974). It was suggested by Dieter et al. (1992) that "if there were a toxicological limit to manganese according to the principles of preventive health care, then it would have to be set at 0.2 mg/L of manganese for infants as a group at risk."

Although conclusive evidence is lacking, some investigators have also linked increased intakes of manganese with violent behavior. Gottschalk et al. (1991) found statistically significantly elevated levels of manganese in the hair of convicted felons (1.62 +/- 0.173 ppm in prisoners compared with 0.35 +/- 0.020 ppm in controls). The authors suggest that "a combination of cofactors, such as the abuse of alcohol or other chemical substances, as well as psychosocial factors, acting in concert with mild manganese toxicity may promote violent behavior." Caution should be exercised to prevent reading too much into these data, but support for this hypothesis is provided by studies of a population of Aborigines in Groote Eylandt. Several clinical symptoms consistent with manganese intoxication are present in about 1% of the inhabitants of this Australian island, and it may not be coincidental that the proportion of arrests in this native population is the highest in Australia (Cawte and Florence, 1989; Kilburn, 1987). The soil in this region is very high in manganese (40,000-50,000 ppm), and the fruits and vegetables grown in the region also are reported to be high in manganese. Quantitative data on oral intakes have not been reported, but elevated concentrations of manganese have been determined in the blood and hair of the Aborigines (Stauber et al., 1987). In addition to the high levels of environmental manganese, other factors common to this population may further increase the propensity for manganism: high alcohol intake, anemia, and a diet deficient in zinc and several vitamins (Florence and Stauber, 1989).

In addition to the toxicity information available by the oral route, several inhalation toxicity studies on manganese also have been performed in laboratory animals, demonstrating an effect on both the brain and lungs. An in-depth discussion of the psychologic and neurologic effects associated with inhalation of manganese can be found in the RfC file on IRIS.

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o ORAL RFD CONFIDENCE :

Study -- Not applicable
Data Base -- Not applicable
RfD -- Not applicable

Many studies have reported similar findings with regard to the normal dietary intake of manganese by humans. These data are considered to be superior to any data obtained from animal toxicity studies, especially as the physiologic requirements for manganese vary quite a bit among different species, with man requiring less than rodents (Schroeder et al., 1966). There is no one study used to derive the dietary RfD for manganese. While several

studies have determined average levels of manganese in various diets, no quantitative information is available to indicate toxic levels of manganese in the diet of humans. Because of the homeostatic control humans maintain over manganese, it is generally not considered to be very toxic when ingested with the diet. It is important to recognize that while the RfD process involves the determination of a point estimate of an oral intake, it is also stated that this estimate is associated "with uncertainty spanning perhaps an order of magnitude." Numerous factors, both environmental factors (e.g., the presence or absence of many dietary constituents) and biological or host factors (e.g., age, alcohol consumption, anemia, and general nutritional status) can significantly influence an individual's uptake of manganese from the diet. As discussed in the Additional Studies / Comments Section, there is significant variability in the absorption of manganese by humans. The determination of a single intake of manganese in the diet must be recognized as a process that is limited in its ability to reflect the variable nature of manganese toxicity. Such a determination may both over- and underestimate the risk, depending on the specific combination of environmental and individual circumstances. Confidence in the data base is medium and confidence in the dietary RfD for manganese is also medium.

It is again emphasized that this oral RfD is based on a total dietary intake of manganese and is not intended to be applied directly to drinking water situation. Because of the greater bioavailability of manganese from water, a separate RfD for water is proposed. This is based on the Greek epidemiologic study by Kondakis et al. (1989). The major advantage of this study is that it examined a sensitive subpopulation of humans exposed to varying concentrations of manganese in the drinking water for a lifetime. A significant weakness of this study is that the actual manganese content in the diet was not measured. The author did indicate, however, that the people in the three areas were age-matched and had similar social, economic and educational backgrounds, and that the food consumed by these subjects was purchased at the marketplace and was not expected to vary much in manganese content. The confidence in the critical study can be considered low-to-medium. It is not higher primarily because of the lack of data on dietary manganese in the three populations under study. Also, many of the endpoints scored in the neurological exam are not specific for manganese poisoning and are, in fact, associated with the normal process of aging. Confidence in the data base also can be considered medium-to-low. The Greek study is supported by the more severe effects occurring at higher levels in the Japanese study (Kawamura et al., 1941) and the study in rhesus monkeys (Gupta et al., 1980). Overall confidence in the drinking water RfD can be considered medium-to-low. While the RfD process involves the determination of a single point estimate of an oral intake, it must be recognized that this estimate is associated with several sources of uncertainty. Available data indicate that numerous factors, both environmental factors (e.g., the presence of high or low levels of other inorganics in drinking water) and biological or host factors (e.g., age, alcohol consumption, and nutritional status, particularly anemia) can significantly influence the uptake of manganese by an individual. The determination of a single concentration of manganese in drinking water, then, must be recognized as a process that is limited in its ability to reflect the variable nature of manganese toxicity.

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o ORAL RFD SOURCE DOCUMENT :

Source Document -- U.S. EPA, 1993

The Drinking Water Criteria Document for Manganese has received Agency review.

Other EPA Documentation -- U.S. EPA, 1984

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o REVIEW DATES : 05/17/90, 06/21/90, 06/24/92, 09/22/92, 03/31/93, 12/14/93, 05/10/95
o VERIFICATION DATE : 09/22/92
o EPA CONTACTS :

Susan Velazquez / NCEA -- (513)569-7571

Julie Du / OST -- (202)260-7583

RDI -
o INHALATION RFD SUMMARY :

Critical Effect	Exposures*	UF	MF	RfC
Impairment of neuro-behavioral function	NOAEL: None LOAEL: 0.15 mg/cu.m	1000	1	5E-5
Occupational exposure to manganese dioxide	LOAEL(ADJ): 0.05 mg/cu.m LOAEL(HEC): 0.05 mg/cu.m			

Roels et al., 1992

Impairment of neuro-behavioral function	NOAEL: None LOAEL: 0.97 mg/cu.m			
Occupational exposure to manganese oxides and salts	LOAEL(ADJ): 0.34 mg/cu.m LOAEL(HEC): 0.34 mg/cu.m			

Roels et al., 1987

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*Conversion Factors and Assumptions: Roels et al., 1992: The LOAEL is derived from an occupational-lifetime integrated respirable dust (IRD) concentration of manganese dioxide (MnO_2) (based on 8-hour TWA occupational exposure multiplied by individual work histories in years) expressed as mg manganese (Mn)/cu.m x years. The IRD concentrations ranged from 0.040 to 4.433 mg Mn/cu.m x years, with a geometric mean of 0.793 mg Mn/cu.m x years and a geometric standard deviation of 2.907. The geometric mean concentration (0.793 mg/cu.m x years) was divided by the average duration of MnO_2 exposure (5.3 years) to obtain a LOAEL TWA of 0.15 mg/cu.m. The LOAEL refers to an extrarespiratory effect of particulate exposure and is based on an 8-hour TWA occupational exposure. MVho = 10 cu.m/day, MVh = 20 cu.m/day. LOAEL(HEC) = 0.15 mg/cu.m x (MVho/MVh) x 5 days/7 days = 0.05 mg/cu.m.

Roels et al., 1987: The LOAEL is based on an 8-hour TWA occupational exposure. The TWA of total airborne manganese dust ranged from 0.07 to 8.61 mg/cu.m, and the median was 0.97 mg/cu.m. This is an extrarespiratory effect of a particulate exposure. MVho = 10 cu.m/day, MVh = 20 cu.m/day. LOAEL(HEC) = 0.97 mg/cu.m x (MVho/MVh) x 5 days/7 days = 0.34 mg/cu.m.

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o INHALATION RFD STUDIES :

Roels, H., R. Lauwerys, J.-P. Buchet et al. 1987. Epidemiological survey among workers exposed to manganese: Effects on lung, central nervous system, and some biological indices. Am. J. Ind. Med. 11: 307-327.

Roels, H.A., P. Ghyselen, J.P. Buchet, E. Ceulemans, and R.R. Lauwerys. 1992. Assessment of the permissible exposure level to manganese in workers exposed to manganese dioxide dust. Br. J. Ind. Med. 49: 25-34.

Roels et al. (1992) conducted a cross-sectional study of 92 male workers exposed to manganese dioxide (MnO_2) dust in a Belgian alkaline battery plant. A control group of 101 male workers was matched for age, height, weight, work schedule, coffee and alcohol consumption, and smoking; educational level was slightly higher in the control group ($p = 0.046$ by chi square test).

The manganese (Mn)-exposed group had been exposed to MnO_2 for an average of 5.3 years (range: 0.2-17.7 years). The geometric means of the workers' TWA airborne Mn concentrations, as determined by personal sampler monitoring at the breathing zone, were 0.215 mg Mn/cu.m for respirable dust and 0.948 mg Mn/cu.m for total dust. No data on particle size or purity were presented, but the median cut point for the respirable dust fraction was 5 um according to information provided by Roels et al. (1992) and Roels (1993). Total and respirable dust concentrations were highly correlated ($r = 0.90$, $p < 0.001$), with the Mn content of the respirable fraction representing on average 25% of the Mn content in the total dust. The authors noted that the personal monitoring data were representative of the usual exposure of the workers because work practices had not changed during the last 15 years of the operation of the plant.

Occupational-lifetime integrated exposure to Mn was estimated for each worker by multiplying the current airborne Mn concentration for the worker's job classification by the number of years for which that classification was held and adding the resulting (arithmetic) products for each job position a worker had held. The geometric mean occupational-lifetime integrated respirable dust (IRD) concentration was 0.793 mg Mn/cu.m x years (range: 0.040-4.433 mg Mn/cu.m x years), with a geometric standard deviation of 2.907 mg Mn/cu.m x years, based on information provided by Roels (1993). The geometric mean occupational-lifetime integrated total dust (ITD) concentration was 3.505 mg Mn/cu.m x years (range: 0.191-27.465 mg Mn/cu.m x years). Geometric mean concentrations of blood Mn (MnB) (0.81 ug/dL) and urinary Mn (MnU) (0.84 ug/g creatinine) were significantly higher in the Mn-exposed group than in the control group, but on an individual basis no significant correlation was found between either MnB or MnU and various external exposure parameters. Current respirable and total Mn dust concentrations correlated significantly with geometric mean MnU on a group basis (Spearman $r = 0.83$, $p < 0.05$).

A self-administered questionnaire focused on occupational and medical history, neurological complaints, and respiratory symptoms. Lung function was evaluated by standard spirographic measures. Neurobehavioral function was evaluated by tests of audio-verbal short-term memory, visual simple reaction time, hand steadiness, and eye-hand coordination. Blood samples were assayed for several hematological parameters (erythrocyte count, leukocyte count, hemoglobin, hematocrit, mean corpuscular volume, mean corpuscular hemoglobin, platelets, and differential leukocyte count); Mn; lead; zinc protoporphyrin; and serum levels of calcium, iron, follicle stimulating hormone (FSH), luteinizing hormone (LH), and prolactin. Urinary Mn, cadmium, and mercury concentrations were also determined.

Responses to the questionnaire indicated no significant differences between groups in either respiratory or neurological symptoms, nor were spirometric, hormonal, or calcium metabolism measurements significantly different for the two groups. In addition, a separate report (Gennart et al., 1992) indicated no significant difference in the fertility of 70 of these workers, in contrast to earlier findings in 85 workers exposed not only to MnO2 but also to other Mn oxides and salts at higher concentrations (Lauwerys et al., 1985). Erythropoietic parameters and serum iron concentrations were consistently and significantly lower in the Mn-exposed workers, albeit within the normal range of values.

Of particular note, Mn workers performed worse than controls on several measures of neurobehavioral function. Visual reaction time was consistently and significantly slower in the Mn-exposed workers measured in four 2-minute periods, with more pronounced slowing over the total 8-minute period and significantly greater variability in reaction times for the Mn-exposed group. Abnormal values for mean reaction times (defined as greater than or equal to the 95th percentile of the control group) also were significantly more prevalent in the Mn-exposed group during three of four 2-minute intervals of the 8-minute testing period.

Five measures of eye-hand coordination (precision, percent precision, imprecision, percent imprecision, and uncertainty) reflected more erratic control of fine hand-forearm movement in the Mn-exposed group than in the controls, with mean scores on all five measures being highly significantly different for the two groups. There was also a significantly greater prevalence of abnormal values for these five measures in the Mn-exposed group. The hole tremormeter test of hand steadiness indicated a consistently greater amount of tremor in the Mn-exposed workers, with performance for two of the five hole sizes showing statistically significant impairment.

Roels et al. (1992) performed an exposure-response analysis by classifying IRD values into three groups (<0.6, 0.6-1.2, and >1.2 mg Mn/cu.m x years) and comparing the prevalence of abnormal scores for visual reaction time, hand steadiness, and eye-hand coordination with controls. This analysis indicated that the prevalence of abnormal eye-hand coordination values was significantly greater in workers whose IRD levels were less than 0.6 mg Mn/cu.m x years. However, the relationship between exposure and response was not linear across groups. Visual reaction time and hand steadiness showed linear exposure-related trends but did not achieve statistical significance except at levels of >1.2 mg Mn/cu.m x years. As noted by the authors, "analysis of the data on a group basis ... does not permit us to identify a threshold effect level for

airborne Mn." Although suggestive of a LOAEL of <0.6 mg Mn/cu.m x years, the exposure-response analysis by Roels et al. (1992) possibly could reflect the small disparity in educational level between exposed and control workers that was noted above with regard to the matching criteria for this study. If educational level were in fact a covariate of exposure as well as neurobehavioral performance, it could confound the exposure-response analysis. Although it is not clear that such was the case, the possibility of confounding suggests that the LOAEL should not be based on the results of the exposure-response analysis until these results can be confirmed by other studies. Also, statistical correction for multiple comparisons should be included in the exposure response analysis.

A LOAEL may be derived from the Roels et al. (1992) study by using the IRD concentration of MnO2, expressed as mg Mn/cu.m x years (based on 8-hour TWA occupational exposures for various job classifications, multiplied by individual work histories in years). Dividing the geometric mean IRD concentration (0.793 mg/cu.m x years) by the average duration of the workers' exposure to MnO2 (5.3 years) yields a LOAEL of 0.15 mg/cu.m. The LOAEL(HEC) is 0.05 mg/cu.m.

Roels et al. (1987) conducted a cross-sectional study in 141 male workers exposed to MnO2, manganese tetroxide (Mn3O4), and various Mn salts (sulfate, carbonate, and nitrate). A matched group of 104 male workers was selected as a control group. The two groups were matched for socioeconomic status and background environmental factors; in addition, both groups had comparable work-load and work-shift characteristics.

The TWA of total airborne Mn dust ranged from 0.07 to 8.61 mg/cu.m, with an overall arithmetic mean of 1.33 mg/cu.m, a median of 0.97 mg/cu.m, and a geometric mean of 0.94 mg/cu.m. The duration of employment ranged from 1 to 19 years, with a mean of 7.1 years. The particle size and purity of the dust were not reported. Neurological examination, neurobehavioral function tests (simple reaction time, short-term memory, eye-hand coordination, and hand tremor), spirographic measurements, blood and urine tests, and a self-administered questionnaire were used to assess possible toxic effects of Mn exposure. The questionnaire was designed to detect CNS and respiratory symptoms.

Significant differences in mean scores between Mn-exposed and reference subjects were found for objective measures of visual reaction time, eye-hand coordination, hand steadiness, and audio-verbal short-term memory. The prevalence of abnormal scores on eye-hand coordination and hand steadiness tests showed a dose-response relationship with blood Mn levels; short-term memory scores were related to years of Mn exposure but not to blood Mn levels. The prevalence of subjective symptoms was greater in the exposed group than in controls for 20 of 25 items on the questionnaire, with four items being statistically significant: fatigue, tinnitus, trembling of fingers, and irritability.

A significantly greater prevalence of coughs during the cold season, dyspnea during exercise, and recent episodes of acute bronchitis was self-reported in the exposed group. Lung function parameters were only slightly (<10%) lower in the Mn-exposed workers, with the only significant alterations evident in Mn-exposed smokers. These mild changes in Mn-exposed workers

(apart from the effects of smoking) and the absence of respiratory effects in the more recent study by Roels et al. (1992) suggest that the nervous system is a more sensitive target for Mn toxicity.

Based upon the findings of impaired neurobehavioral function in workers whose average Mn exposure was estimated by the geometric mean TWA of total airborne Mn dust at the time of the study, a LOAEL of 0.97 mg/cu.m was identified, with a LOAEL(HEC) of 0.34 mg/cu.m. Note that this LOAEL(HEC) is based on total Mn dust of mixed forms, whereas the LOAEL(HEC) from the more recent Roels et al. (1992) study is based on the measured respirable dust fraction of MnO2 only. However, the geometric mean total dust concentrations in the 1987 and 1992 studies by Roels et al. were approximately the same (0.94 and 0.95 mg/cu.m, respectively).

The findings of Roels et al. (1987, 1992) are supported by other recent reports that provide comparable and consistent indications of neurobehavioral dysfunction in Mn-exposed workers (Mergler et al., 1993; Iregren, 1990; Wennberg et al., 1991, 1992).

Mergler et al. (1993) conducted a cross-sectional study of 115 male ferromanganese and silicomanganese alloy workers in southwest Quebec. A matched-pair design was employed because of presumptively high environmental pollutant levels; 74 pairs of workers and referents were matched on age, educational level, smoking status, number of children, and length of residency in the region.

Air concentrations of respirable and total dust were sampled by stationary monitors during silicomanganese production. The geometric mean of a series of 8-hour TWAs was 0.035 mg Mn/cu.m (range: 0.001-1.273 mg Mn/cu.m) for respirable dust and 0.225 mg Mn/cu.m (range: 0.014-11.480 mg Mn/cu.m) for total dust. The authors noted that past dust levels at certain job sites had been considerably higher. The mean duration of the workers' Mn exposure was 16.7 years and included Mn fumes as well as mixed oxides and salts of Mn. Geometric mean MnB was significantly higher in the Mn alloy workers, but MnU did not differ significantly between exposed workers and controls.

The number of discordant pairs, in which workers reported undesirable symptoms on a self-administered questionnaire but their matched pairs did not, was statistically significant for 33 of 46 items, including the following: fatigue; emotional state; memory, attention, and concentration difficulties; nightmares; sweating in the absence of physical exertion; sexual dysfunction; lower back pain; joint pain; and tinnitus. Workers did not report symptoms typical of advanced Mn poisoning (e.g., hand tremor, changes in handwriting, loss of balance when turning, difficulty in reaching a fixed point) significantly more than referents, which suggests that the other reported symptoms were probably not due to bias on the part of the workers.

The greatest differences in neurobehavioral function were evident in tests of motor function, especially tests requiring coordinated alternating and/or rapid movements. Workers performed significantly worse on the motor scale of a neuropsychological test battery both in overall score and in eight subscales of rapid sequential or alternating movements. Worker performance also was significantly worse on tests of hand steadiness, parallel-line drawing performance, and ability to rapidly identify and mark specified alphabetic

characters within strings of letters. Performance on a variety of other tests of psychomotor function, including simple reaction time, was worse in Mn-exposed workers but marginally significant ($0.05 < p < 0.10$). In addition, Mn alloy workers differed significantly from referents on measures of cognitive flexibility and emotional state. Olfactory perception also was significantly enhanced in the Mn-alloy workers.

The matched-pair design of Mergler et al. (1993) helped reduce differences between exposed and referent subjects that might otherwise have confounded the study. However, to the extent that the referents also may have had significant exposure to Mn in the ambient atmosphere, such exposure may have reduced the differences in neurobehavioral performance between workers and referents. This possibility is supported by the fact that the finger-tapping speed of both workers and referents on a computerized test was slower than that of Mn-exposed workers assessed on the same test by Iregren (1990) in Sweden. In the absence of a NOAEL, the LOAEL from the study of Mergler et al. (1993) is based on the geometric mean respirable dust level (0.035 mg Mn/cu.m), with a LOAEL(HEC) of approximately 0.01 mg/cu.m, which is about five-fold lower than the LOAEL(HEC) identified in the study by Roels et al. (1992).

Workers exposed to Mn in two Swedish foundries (15 from each plant) were evaluated in a study first reported by Iregren (1990). The exposure to Mn varied from 0.02 to 1.40 mg/cu.m (mean = 0.25 mg/cu.m; median = 0.14 mg/cu.m) for 1-35 years (mean = 9.9 years). Earlier monitoring measurements made in both factories suggested that essentially no changes in exposure had occurred in either factory for the preceding 18 years. Each exposed worker was matched for age, geographical area, and type of work to two workers not exposed to Mn in other industries. Neurobehavioral function was assessed by eight computerized tests and two manual dexterity tests. There were significant differences between exposed and control groups for simple reaction time, the standard deviation of reaction time, and finger-tapping speed of the dominant hand. In addition, digit-span short-term memory, speed of mental addition, and verbal (vocabulary) understanding differed significantly between exposed and control groups. The difference in verbal understanding suggested that the two groups were not well matched for general cognitive abilities. With verbal performance used as an additional matching criterion, differences between the groups in simple reaction time, the standard deviation of reaction time, and finger-tapping speed remained statistically significant, despite a decrease in statistical power due to reducing the size of the reference group to 30 workers. Further analyses using verbal test scores as a covariate also indicated that these same three measures of neurobehavioral function were statistically different in exposed and control workers. No significant correlation was found within the exposed group to establish a concentration-response relationship.

Additional reports of neurobehavioral and electrophysiological evaluations of these same workers have been published by Wennberg et al. (1991, 1992). Although none of the latter results achieved statistical significance at $p = 0.05$, increased frequency of self-reported health symptoms, increased prevalence of abnormal electroencephalograms, slower brain-stem auditory-evoked potential latencies, and slower diadochokinesometric performance were found in the exposed workers. Diadochokinesis refers to the ability to perform rapidly alternating movements such as supination and pronation of the

forearm, and is an indicator of extrapyramidal function (see Additonal Comments/Studies). Also, an increase in P-300 latency, as suggested by these results, has been observed in patients with parkinsonism according to Wennberg et al. (1991), who viewed these results in Mn-exposed workers as early (preclinical) signs of disturbances similar to parkinsonism. Based on the impairments in reaction time and finger-tapping speed reported in the Swedish studies (Iregren, 1990; Wennberg et al., 1991, 1992), the LOAEL(HEC) is calculated to be 0.05 mg/cu.m. Although numerically the same value as that derived from Roels et al. (1992), the Swedish study measured total dust. However, Wennberg et al. (1991) stated that the respirable dust level was 20-80% of total dust, which implies that the LOAEL(HEC) from the Swedish studies could be somewhat lower than that from Roels et al. (1992).

All of the above studies taken together provide a consistent pattern of evidence indicating that neurotoxicity is associated with low-level occupational Mn exposure. The fact that the speed and coordination of motor function are especially impaired is consistent with other epidemiological, clinical, and experimental animal evidence of Mn intoxication (see Additional Comments/Studies). Moreover, the LOAEL(HEC)s obtained from these studies are not appreciably different. Nevertheless, some differences between the studies are evident in the durations of exposure and forms of Mn to which workers were exposed. In the Roels et al. (1992) study, the mean period of exposure was 5.3 years (range: 0.2-17.7 years), and exposure was limited to MnO2. In the other studies, mixed forms of Mn were present, and the mean durations of exposure were longer: 7.1 years in Roels et al. (1987), 9.9 years in Iregren (1990), and 16.7 years in Mergler et al. (1993). The findings of Mergler et al. (1993) suggest that the LOAEL(HEC) could be at least as low as approximately 0.01 mg/cu.m. However, the variable concentrations and mixed compounds of Mn to which workers were exposed make it difficult to rely primarily upon the findings of Mergler et al. (1993) in deriving the RfC. Nevertheless, their results provide support for the findings of Roels et al. (1992) and suggest that the longer period of exposure (16.7 years in Mergler et al. (1993) vs. 5.3 years in Roels et al., 1992) may have contributed to the apparent differences in sensitivity. Although analyses by Roels et al. (1987, 1992) and Iregren (1990) generally did not indicate that duration of exposure correlated significantly with neurobehavioral outcomes, none of these studies involved average exposures as long as those in the Mergler et al. (1993) study. Also, the oldest worker in the Roels et al. (1992) study was less than 50 years old, and the average age in that study was only 31.3 years vs. 34.3 years in Roels et al. (1987), 43.4 years in Mergler et al. (1993), and 46.4 in Iregren (1990). These points suggest that chronic exposure to Mn and/or interactions with aging could result in effects at lower concentrations than would be detected after shorter periods of exposure and/or in younger workers.

Based on the findings of neurobehavioral impairment by Roels et al. (1987, 1992), with supporting evidence from Mergler et al. (1993) and the Swedish reports (Iregren, 1990; Wennberg et al., 1991, 1992), the LOAEL for derivation of the RfC is 0.15 mg/cu.m, and the LOAEL(HEC) is 0.05 mg/cu.m.

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o INHALATION RFD UNCERTAINTY :

UF -- An uncertainty factor of 1000 reflects 10 to protect sensitive individuals, 10 for use of a LOAEL, and 10 for database limitations reflecting both the less-than-chronic periods of exposure and the lack of developmental

data, as well as potential but unquantified differences in the toxicity of different forms of Mn.

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o INHALATION RFD MODIFYING :

MF -- None
FACTOR

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o INHALATION RFD COMMENTS :

Manganese toxicity varies depending upon the route of exposure. When ingested, Mn is considered to be among the least toxic of the trace elements. In the normal adult, between 3 and 10% of dietary Mn is absorbed. Total body stores normally are controlled by a complex homeostatic mechanism regulating absorption and excretion. As detailed in the Uncertainty Factor Text and the Additonal Comments/Studies for the oral RfD, toxicity from ingested Mn is rarely observed. However, deficiencies of calcium and iron have been noted to increase Mn absorption (Mena et al., 1969; Murphy et al., 1991). Also, Mena et al. (1969) found that anemic subjects absorbed 7.5% of ingested Mn, whereas normal subjects absorbed 3%. Interestingly, manganism patients absorbed 4%, and apparently healthy Mn miners absorbed only 3%. These differences suggest that certain subpopulations, such as children, pregnant women, elderly persons, iron- or calcium-deficient individuals, and individuals with liver impairment, may have an increased potential for excessive Mn body burdens due to increased absorption or altered clearance mechanisms, which may be of particular importance for those exposed to Mn by multiple routes.

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o INHALATION RFD CONFIDENCE : Study -- Medium Data Base -- Medium RfC -- Medium Confidence in the principal studies (Roels et al., 1987, 1992) is medium. Neither of the principal studies identified a NOAEL for neurobehavioral effects, nor did either study directly measure particle size or provide information on the particle size distribution. The 1992 study by Roels et al. did provide respirable and total dust measurements, but the 1987 study measured only total dust. These limitations of the studies are mitigated by the fact that the principal studies found similar indications of neurobehavioral dysfunction, and these findings were consistent with the results of other human studies (Mergler et al., 1993; Iregren, 1990; Wennberg et al., 1991, 1992; as well as various clinical studies). In addition, the exposure history of the workers in the 1992 study by Roels et al. was well characterized and essentially had not changed over the preceding 15 years, thereby allowing calculation of integrated exposure levels for individual workers. However, individual integrated exposures were not established in the 1987 study of Roels et al. data base is medium. The duration of exposure was

relatively limited in all of the principal and supporting studies, ranging from means of 5.3 and 7.1 years in the co-principal studies by Roels et al. (1992 and 1987, respectively) to a maximum of 16.7 years in the study by Mergler et al. (1993). Moreover, the workers were relatively young, ranging from means of 31.3 and 34.3 years in the co-principal studies (Roels et al., 1992 and 1987, respectively) to a maximum of 46.4 years (Iregren, 1990). These temporal limitations raise concerns that longer durations of exposure and/or interactions with aging might result in the detection of effects at lower concentrations, as suggested by results from studies involving longer exposure durations and lower concentrations (Mergler et al., 1993; Iregren, 1990). In addition, except for the 1992 study by Roels et al., in which Mn exposure was limited to MnO2, the other principal and supporting studies did not specify the species of Mn and the proportions of the different compounds of Mn to which workers were exposed. It is not clear whether certain compounds or oxidation states of Mn are more toxic than others. Thus, it is not possible to distinguish the relative toxicity of different Mn compounds in these studies, despite some indications in the literature regarding the differential toxicity of various oxidation states of Mn. Although the primary neurotoxicological effects of exposure to airborne Mn have been qualitatively well characterized by the general consistency of effects across studies, the exposure-effect relationship remains to be well quantified, and a no-effect level for neurotoxicity has not been identified in any of these studies thus far. Finally, the effects of Mn on development and reproduction have not been studied adequately. Insufficient information on the developmental toxicity of Mn by inhalation exposure exists, and the same is true for information on female reproductive function. The study of the reproductive toxicity of inhaled Mn in males also needs to be characterized more fully. Reflecting medium confidence in the principal studies and medium confidence in the data base, confidence in the inhalation RfC is medium.

o INHALATION RFD SOURCE :

Source Document -- This assessment is not presented in any existing U.S. EPA

document.

Other EPA Documentation -- U.S. EPA, 1984
DOCUMENT

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o REVIEW DATES : 08/23/90, 09/19/90, 09/23/93
o VERIFICATION DATE : 09/23/93
o EPA CONTACTS :

J. Michael Davis / NCEA -- (919)541-4162

Annie M. Jarabek / NCEA -- (919)541-4847

CAREV-
o CLASSIFICATION : D; not classifiable as to human carcinogenicity
o BASIS FOR CLASSIFICATION : Existing studies are inadequate to assess the carcinogenicity of manganese. NOTE: Manganese is an element considered essential to human health.
o HUMAN CARCINOGENICITY DATA :

None.

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o ANIMAL CARCINOGENICITY DATA :

Inadequate. DiPaolo (1964) subcutaneously or intraperitoneally injected DBA/1 mice with 0.1 mL of an aqueous of solution 1% manganese chloride twice weekly for 6 months. A larger percentage of the mice exposed subcutaneously (24/36; 67%) and intraperitoneally (16/39; 41%) to manganese developed lymphosarcomas compared with controls injected with water (16/66; 24%). In addition, tumors appeared earlier in the exposed groups than in the control groups. The incidence of tumors other than lymphosarcomas (i.e., mammary adenocarcinomas, leukemias, injection site tumors) did not differ significantly between the exposed groups and controls. A thorough evaluation of the results of this study was not possible because the results were published in abstract form.

Stoner et al. (1976) tested manganous sulfate in a mouse lung adenoma screening bioassay. Groups of strain A/Strong mice (10/sex), 6-8 weeks old, were exposed by intraperitoneal injection to 0, 6, 15 or 30 mg/kg manganous sulfate 3 times/week for 7 weeks (a total of 21 injections). The animals were observed for an additional 22 weeks after the dosing period, before sacrifice at 30 weeks. Lung tumors were observed in 12/20, 7/20, and 7/20 animals in the high, medium, and low dosage groups, respectively. The percentage of mice with tumors was elevated, but not significantly, at the highest dose level (Fisher Exact test) compared with that observed in the vehicle controls. In addition, there was an apparent increase in the average number of pulmonary adenomas per mouse both at the mid and high doses, as compared with the vehicle controls (10 mice/sex), but the increase was significant only at the high dose (Student's t-test, $p<0.05$).

In the mouse lung adenoma bioassay, certain specific criteria should be met in order for a response to be considered positive (Shimkin and Stoner,

1975). Among these criteria are an increase in the mean number of tumors per mouse and an evident dose-response relationship. While the results of this study are suggestive of carcinogenicity, the data cannot be considered conclusive since the mean number of tumors per mouse was significantly increased at only one dose, and the evidence for a dose-response relationship was marginal.

Furst (1978) exposed groups of F344 rats (25/sex) intramuscularly or by gavage to manganese powder, manganese dioxide, and manganese (II) acetylacetonate (MAA). Treatment consisted of either 9 i.m. doses of 10 mg each of manganese powder or manganese dioxide, 24 doses of 10 mg manganese powder by gavage, or 6 i.m. doses of 50 mg of MAA. In addition, female swiss mice (25/group) were exposed intramuscularly to manganese powder (single 10 mg dose) and manganese dioxide (6 doses of 3 or 5 mg each). There was an increased incidence of fibrosarcomas at the injection site in male (40%) and female (24%) rats exposed intramuscularly to MAA compared with vehicle controls (4% male, 4% female). EPA (1984) determined that these increases were statistically significant and noted that the study results regarding MAA, an organic manganese compound, cannot necessarily be extrapolated to pure manganese or other inorganic manganese compounds. No difference in tumor incidence was found between rats and mice exposed to manganese powder and manganese dioxide and controls.

Sunderman et al. (1974, 1976) exposed male 344 rats to 0.5 to 4.4 mg manganese dust intramuscularly and found that no tumors were induced at the injection site. It was further observed that co-administration of manganese with nickel subsulfide resulted in decreased sarcoma production by comparison to nickel subsulfide alone. Subsequent studies by Sunderman et al. (1980) suggest that manganese dust may inhibit local sarcoma induction by benzo(a)pyrene.

Witschi et al. (1981) exposed female A/J mice intraperitoneally to 80 mg/kg methylcyclopentadienyl manganese tricarbonyl (MMT) and found that although cell proliferation was produced in the lungs, lung tumor incidence did not increase.

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o SUPPORTING DATA :

None.

CARO - NO DATA
CARI - NO DATA
CARDR-
o CARCINOGENICITY SOURCE :

Source Document -- U.S. EPA, 1984, 1988

The Drinking Water Criteria Document for Manganese has received OHEA review.
DOCUMENT

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o REVIEW DATES : 05/25/88

o VERIFICATION DATE : 05/25/88
o EPA CONTACTS :

Susan Velazquez / NCEA -- (513)569-7571

Julie Du / OST -- (202)260-7583

HAONE- NO DATA

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA - NO DATA

WQCHU-

Water and Fish Consumption: 1.0E+2 ug/L

Fish Consumption Only: None

Considers technological or economic feasibility? -- NO

Discussion -- A criterion for domestic water supplies of 5.0E+1 ug/L should minimize the objectionable qualities, such as staining of laundry and undesirable taste. There is no demonstrated relationship between organoleptic endpoints and adverse health effects.

Reference -- Quality Criteria for Water, July 1976 (PB-263943).

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

No data available

MCLG - NO DATA

MCL - NO DATA

SMCL - NO DATA

FISTD- NO DATA

FIREV- NO DATA

CERC - NO DATA

SARA - NO DATA

RCRA - NO DATA

TSCA -

No data available

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HAREF- None

[IRIS] SS 2 /cf?
USER:
vanadium (name)

Search in progress

2-Nitroaniline

1 - IRIS
NAME - 2-Nitroaniline
RN - 88-74-4
IRSN - 627
DATE - 920807
UPDT - 08/07/92. 52 fields
STAT - Oral RfD Assessment (RDO) pending 08/01/92
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) no data
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) no data
IRH - 08/01/92 RDO Oral RfD now under review
RLEN - 1240
SY - Benzenamine, 2-nitro-
SY - O-NITROANILINE
SY - 2-NITROANILINE
SY - AI3-02916
SY - Aniline, o-nitro-
SY - AZOENE FAST ORANGE GR BASE
SY - AZOENE FAST ORANGE GR SALT
SY - AZOFIX ORANGE GR SALT
SY - AZOGENE FAST ORANGE GR
SY - AZOIC DIAZO COMPONENT 6
SY - BRENTAMINE FAST ORANGE GR BASE
SY - BRENTAMINE FAST ORANGE GR SALT
SY - C.I. AZOIC DIAZO COMPONENT 6
SY - C.I. 37025
SY - CCRIS 2317
SY - DEVOL ORANGE B
SY - DEVOL ORANGE SALT B
SY - DIAZO FAST ORANGE GR
SY - FAST ORANGE BASE GR
SY - FAST ORANGE BASE JR
SY - FAST ORANGE GR BASE
SY - FAST ORANGE O BASE
SY - FAST ORANGE O SALT
SY - FAST ORANGE SALT GR
SY - FAST ORANGE SALT JR
SY - HILTONIL FAST ORANGE GR BASE
SY - HILTOSAL FAST ORANGE GR SALT
SY - HINDASOL ORANGE GR SALT
SY - HSDB 1132
SY - NATASOL FAST ORANGE GR SALT
SY - O-AMINONITROBENZENE
SY - o-NITRANILINE
SY - o-NITROANILINE
SY - ONA
SY - ORANGE BASE CIBA II
SY - ORANGE GRS SALT
SY - ORANGE SALT CIBA II
SY - ORANGE SALT IRGA II
SY - Orthonitroaniline
SY - 1-AMINO-2-NITROBENZENE

SY - 2-AMINONITROBENZENE
SY - 2-NITROANILINE
SY - 2-NITROBENZENAMINE

RDO -
o ORAL RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

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o REVIEW DATES : 06/23/92

RDI - NO DATA
CAREV- NO DATA
CARO - NO DATA
CARI - NO DATA
CARDR- NO DATA

HAONE- NO DATA

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA - NO DATA

WQCHU- NO DATA

WQCAQ- NO DATA

MCLG - NO DATA

MCL - NO DATA

SMCL - NO DATA

FISTD- NO DATA

FIREV- NO DATA

CERC - NO DATA

SARA - NO DATA

RCRA - NO DATA

TSCA - NO DATA
OREF - NO DATA
IREF - NO DATA
CREF - NO DATA
HAREF- NO DATA

PCB 1248

1 - IRIS
NAME - Aroclor 1248
RN - 12672-29-6
IRSN - 631
DATE - 940406
UPDT - 04/06/94, 5 fields
STAT - Oral RfD Assessment (RDO) message 04/01/94
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) no data
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) no data
IRH - 09/01/92 RDO Oral RfD now under review
IRH - 12/01/92 RDO Work group review date added
IRH - 07/01/93 RDO Work group review date added
IRH - 08/01/93 RDO Work group review date added
IRH - 04/01/94 RDO Oral RfD message on-line
IRH - 04/01/94 OREF Oral RfD references on-line
RLEN - 10476SY - Aroclor 1248
SY - HSDB 6356

RDO -
o ORAL RFD SUMMARY :

The health effects data for Aroclor 1248 were reviewed by the U.S. EPA RfD/RfC Work Group and determined to be inadequate for the derivation of an oral RfD. The verification status for this chemical currently is NOT VERIFIABLE. For additional information on the health effects of this chemical, interested parties are referred to the U.S. EPA documentation listed below.

NOT VERIFIABLE status indicates that the U.S. EPA RfD/RfC Work Group deemed the data base at the time of review to be insufficient to derive an oral RfD according to the current Agency guidelines. This status does not preclude the use of information in cited references for assessment by others.

Derivation of an oral RfD for Aroclor 1248 is not recommended because a Frank Effect (death of an infant) was noted at the lowest dose tested in a sensitive animal species, rhesus monkeys (Macaca mulatta). In general, Rhesus monkeys have shown adverse effects to PCB mixtures at doses 10-fold lower than in other species. The data indicated a dose-response relationship for this effect.

Schantz et al. (1989) evaluated neurobehavioral performance in offspring of rhesus monkeys that had been exposed to 0.03, 0.1 and 0.2 mg/kg-day of dietary Aroclor 1248 for different durations. Group I consisted of infants whose dams had received 0.03 mg/kg-day. Of the seven dams for this group, six delivered viable offspring. Necropsy of the infant who died at the time of weaning showed signs of PCB intoxication that included thymic atrophy and skin hyperpigmentation. Group II consisted of offspring of 4/8 females fed 0.1 mg/kg-day of Aroclor 1248. Of the eight dams of this group, one delivered a dead infant and one delivered an infant that died shortly after weaning with signs of PCB intoxication. Group III consisted of offspring of 3/7 females fed 0.2 mg/kg-day of Aroclor 1248. Of the seven females that were dams in this group, only three delivered live infants. Information on maternal

toxicity was not provided in the report. Mild dermatological lesions and hyperpigmentation about the hairline developed in offspring in all treated groups during nursing, but no signs of toxicity were evident at the time of neurological testing (age 14 months). Offspring weights at birth and weaning were significantly reduced in Group III. Offspring in Groups I and II did not differ from controls on spatial, color or shape in two-choice discrimination reversal learning tests, but decreased performance on a shape discrimination problem was observed in Group III when irrelevant cues were inserted. On the basis of thymic atrophy and chloracne and death of 1 of 7 infants, it is concluded that 0.03 mg/kg-day represents a FEL for developmental effects.

Adult female Rhesus monkeys were fed 0, 2.5 or 5 ppm (0, 0.1 and 0.2 mg/kg-day) of Aroclor 1248 incorporated in food pellets for up to 14 months (Barsotti et al., 1976; Barsotti, 1980). The exposure period ran from 7 months prior to breeding through gestation, and then for an additional 4 months until the infants were weaned. Some treated females began showing skin changes, such as hyperpigmentation and alopecia, characteristic signs of PCB intoxication, during the first 2 months of dosing. Monkeys with less body fat were the first to show clinical signs, regardless of the dose group to which they were assigned. All treated females showed signs of PCB intoxication to some degree by 6 months. A progressive increase in SGPT values was observed for all treated monkeys and this increase was found to be statistically significant ($p<0.05$) by the 22nd month of the study, even though dosing stopped at the end of the 14th month. One female in each dose group developed severe shigellosis and died, and other dosed females developed clinical signs of shigellosis but did not die. Necropsies of deceased monkeys showed focal necrosis and lipid deposition of the liver, as well as marked subcutaneous edema. Increased menstrual duration was noted as well as occasional amenorrhea.

For the experimental breeding trial, conducted during the dosing period, all low-dose monkeys (8/8) conceived; 3/8 aborted and 5/8 delivered live infants. However, 3 of these 5 livebom infants showed clinical signs of PCB toxicity and, being unable to withstand the stress of weaning, died when separated from their dams. Among the high-dose monkeys, 6/8 conceived. Among these six conceptions, four ended in abortion, one infant went to term, but was stillborn. Only one normal birth occurred among this group; however, at the time of weaning, this infant showed clinical signs of PCB toxicity and died.

The investigators realized that PCB mixtures might have latent effects that could appear long after dosing had ceased. Thus, they included three additional recovery breeding periods after dosing had been completed.

The first recovery breeding trial occurred approximately 22 months after the initiation of Aroclor 1248 dosing and 8 months after dosing had stopped. For the low-dose dams, 8/8 conceived. One of these eight conceptions resulted in abortion. Of the seven livebirths, two infants died at or before weaning. Among the high-dose mothers, 7/7 conceived. There was one abortion and one stillbirth among this group of seven mothers, and five livebirths. Among the group of five livebirths, three infants died at or before weaning.

A second recovery breeding trial was conducted approximately 36 months after the completion of Aroclor 1248 dosing. Among the low-dose mothers, 5/7

conceived. There was one stillbirth and four live births. All four of the liveborn infants survived past weaning and were available for behavioral testing at 14 months and 4 years of age. Among the high-dose mothers, 4/6 conceived for this breeding trial. There were no abortions among the four conceptions, but one stillbirth did occur; there were three livebirths.

The third recovery breeding trial was conducted 55 months after the completion of Aroclor 1248 dosing. Among the low-dose dams, 7/7 conceived. There were no abortions among this group but two stillbirths did occur. All five liveborn infants survived past weaning. For the high-dose mothers, only five had normal reproductive cycles and 4/5 conceived. Among the four conceptions, one ended in abortion, another infant was stillborn and two were born live.

In the first recovery breeding trial the average birth weights for the dosed groups were found to be reduced when compared with controls. For the second recovery breeding trial, the mean weight of the test group infants was 15 and 22% below the control group.

Results of this prolonged recovery period revealed impairment of reproductive function in female Rhesus monkeys lasting for more than 4 years after dosing ceased. In the groups of infants for which birth-weight data are available, a significant reduction in mean birth weight for PCB-exposed infants is evident.

Thomas and Hinsdill (1978) performed immunologic tests after Rhesus monkeys had been fed 0, 2.5 and 5 ppm dietary Aroclor 1248 for 11 months. All treated monkeys developed facial acne and edema and swollen eyelids to varying degrees after 6 months, with pronounced alopecia occurring in the 0.2 mg/kg-day group. Following the treatment period, the monkeys were inoculated with sheep red blood cells (SRBC) and tetanus toxoid. Anti-SRBC antibody titers were significantly reduced in the 0.2 mg/kg-day group at weeks 1 and 12 after inoculation, but antibody response to tetanus toxoid was not affected by treatment at either dosage level.

Groups of three female New Zealand white rabbits were fed 0, 10, 100 or 250 ppm of Aroclor 1248 for 4 weeks and bred with untreated males (Thomas and Hinsdill, 1980). No maternal toxicity was evident. Body-weight gain was significantly reduced in the offspring in the high-dose group.

Barsotti, D.A., R.J. Marlar and J.R. Allen. 1976. Reproductive dysfunction in rhesus monkeys exposed to low levels of polychlorinated biphenyls (Aroclor 1248). Food Cosmet. Toxicol. 14: 99-103.

Barsotti, D.A. 1980. Gross, Clinical, and Reproductive Effects of Polychlorinated Biphenyls in the Rhesus Monkey. August. Ph.D. Thesis, available through the University Library, University of Wisconsin, Madison, WI.

Schantz, S.L., E.D. Levin, R.W. Bowman et al. 1989. Effects of perinatal PCB exposure on discrimination-reversal learning in monkeys. Neurotoxicol. Teratol. 11: 243-250.

Thomas, P.T. and R.D. Hinsdill. 1978. Effect of polychlorinated biphenyls on

the immune responses of mesus monkeys and mice. Toxicol. Appl. Pharmacol. 44: 41-51.

Thomas. P.T. and R.D Hinsdill. 1980. Perinatal PCB exposure and its effect on the immune system of young rabbits. Drug Chem. Toxicol. 3: 173-184.

o REVIEW DATES : 08/12/92, 11/04/92, 06/16/93, 07/20/93

RDI - NO DATA
CAREV- NO DATA
CARO - NO DATA
CARI - NO DATA
CARDR- NO DATA

HAONE- NO DATA

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA - NO DATA

WQCHU- NO DATA

WQCAQ- NO DATA

MCLG - NO DATA

MCL - NO DATA

SMCL - NO DATA

FISTD- NO DATA

FIREV- NO DATA

CERC - NO DATA

SARA - NO DATA

RCRA - NO DATA

TSCA - NO DATA

OREF - Barsotti, D.A., R.J. Marlar and J.R. Allen. 1976. Reproductive dysfunction in rhesus monkeys exposed to low levels of polychlorinated biphenyls (Aroclor 1248). Food Cosmet. Toxicol. 14: 99-103.

OREF - Barsotti, D.A. 1980. Gross, Clinical, and Reproductive Effects of Polychlorinated Biphenyls in the Rhesus Monkey. August. Ph.D. Thesis, available through the University Library, University of Wisconsin, Madison, WI.

OREF - Schantz, S.L., E.D. Levin, R.W. Bowman et al. 1989. Effects of perinatal PCB exposure on discrimination-reversal learning in monkeys. Neurotoxicol. Teratol. 11: 243-250.

OREF - Thomas, P.T. and R.D. Hinsdill. 1978. Effect of polychlorinated biphenyls on the immune responses of rhesus monkeys and mice. Toxicol. Appl. Pharmacol. 44: 41-51.

OREF - Thomas, P.T. and R.D. Hinsdill. 1980. Perinatal PCB exposure and its effect on the immune system of young rabbits. Drug Chem. Toxicol. 3: 173-184.

IREF - None

CREF - None

HAREF- None

PCB 1254

1 - IRIS
NAME - Aroclor 1254
RN - 11097-69-1
IRSN - 662
DATE - 941003
UPDT - 10/03/94, 5 fields
STAT - Oral RfD Assessment (RDO) on-line 10/01/94
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) no data
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) no data
STAT - Supplementary Data no data
IRH - 07/01/93 RDO Oral RfD now under review
IRH - 03/01/94 RDO Work group review date added
IRH - 10/01/94 RDO Oral RfD summary on-line
IRH - 10/01/94 OREF Oral RfD references on-line
RLEN - 63965
SY - Aroclor 1254
SY - Arochlor 1254
SY - CHLORIERTE BIPHENYLE, CHLORGEHALT 54% [German]
SY - CLORODIFENILI, CLORO 54% [Italian]
SY - DIPHENYLE CHLORE, 54% DE CHLORE [French]
SY - HSDB 6357
SY - NCI-C02664

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Ocular exudate, inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes Monkey Clinical and Immunologic Studies Arnold et al., 1994a,b; Tryphonas et al., 1989, 1991a,b	NOAEL: None LOAEL: 0.005 mg/kg-day	300	1	2E-5 mg/kg-day

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*Conversion Factors and Assumptions -- None

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o ORAL RFD STUDIES :

Arnold, D.L., F. Bryce, R. Stapley et al. 1993a. Toxicological consequences of Aroclor 1254 ingestion by female Rhesus (Macaca mulatta) monkeys, Part 1A: Prebreeding phase - clinical health findings. Food Chem. Toxicol. 31: 799-810.

Arnold, D.L., F. Bryce, K. Karpinski et al. 1993b. Toxicological consequences of Aroclor 1254 ingestion by female Rhesus (Macaca mulatta) monkeys. Part 1B: Prebreeding phase -clinical and analytical laboratory findings. Food Chem. Toxicol. 31: 811-824.

Tryphonas, H., S. Hayward, L. O'Grady et al. 1989. Immunotoxicity studies of PCB (Aroclor 1254) in the adult rhesus (Macaca mulatta) monkey – preliminary report. Int. J. Immunopharmacol. 11: 199-206.

Tryphonas, H., M.I. Luster, G. Schiffman et al. 1991a. Effect of chronic exposure of PCB (Aroclor 1254) on specific and nonspecific immune parameters in the rhesus (Macaca mulatta) monkey. Fund. Appl. Toxicol. 16(4): 773-786.

Tryphonas, H., M.I. Luster, K.L. White et al. 1991b. Effects of PCB (Aroclor 1254) on non-specific immune parameters in Rhesus (Macaca mulatta) monkeys. Int. J. Immunopharmacol. 13: 639-648.

Groups of 16 adult female rhesus monkeys ingested gelatin capsules containing Aroclor 1254 (Monsanto Lot No. KA634) in 1:1 glycerol: corn oil vehicle daily at dosages of 0, 5, 20, 40 or 80 ug/kg-day for more than 5 years. The Aroclor mixture contained 5.19 ppm of polychlorinated dibenzofurans and undetectable levels of polychlorinated dibenzo-p-dioxins (Truelove et al., 1990). At study initiation the monkeys were 11.1 +/- 4.1 years old (Tryphonas et al., 1991a,b; Arnold et al., 1993a,b). After 25 months of exposure the monkeys had achieved a pharmacokinetic steady-state based on PCB concentrations in adipose tissue and/or blood (Tryphonas et al., 1989). Results of general health and clinical pathology evaluations conducted during the first 37 months of exposure were reported by Arnold et al. (1993a,b). Results of immunologic assessments after 23 and 55 months of exposure were reported by Tryphonas et al. (1989, 1991a,b). Results of reproductive endocrinology evaluations after 24 or 29 months of exposure were reported by Truelove et al. (1990) and Arnold et al. (1993a). Effects on hydrocortisone levels during the first 22 months of exposure were reported by Loo et al. (1989) and Arnold et al. (1993b). All of the aforementioned evaluations were performed during the prebreeding phase of the study. Results of reproduction and histopathology evaluations in these monkeys are not fully available (Arnold, 1992).

General health status was evaluated daily, and body weight measurements, feed conversion ratio calculations, and detailed clinical evaluations were performed weekly throughout the study. Analyses of clinical signs of toxicity were limited to the occurrence of eye exudate, inflammation and/or prominence of the eyelid Meibomian (tarsal) glands, and particular changes in finger and toe nails (prominent nail beds, separation from nail beds, elevated nail beds, and nails folding on themselves). Each endpoint was analyzed for individual treatment-control group differences and dose-related trends with respect to incidence rate, total frequency of observed occurrences, and the onset time of the condition. With respect to effects on the eyes, the treatment-control group comparisons showed statistically significant (p less than or equal to 0.05) increases in the total frequency of inflamed and/or prominent Meibomian glands at 0.005, 0.02 and 0.08 mg/kg-day, and decreased onset time for these effects at 0.08 mg/kg-day. Significant dose-related trends (p less than or equal to 0.05) were observed for increased total frequencies of inflamed and/or prominent Meibomian glands, decreased onset time of inflamed and/or

prominent Meibomian glands and increased incidences of eye exudate. With respect to effects on finger and/or toe nails, the treatment-control group comparisons showed significantly (p less than or equal to 0.05) increased incidence of certain nail changes at 0.005 mg/kg-day (nail folding) and 0.08 mg/kg-day (elevated nails), increased total frequency of certain nail changes at 0.005 mg/kg-day (nail separation), 0.04 mg/kg-day (nail folding and separation) and 0.08 mg/kg-day (nail folding and separation, prominent beds, elevated nails), and decreased onset time of certain nail changes at 0.005 mg/kg-day (elevated nails) and 0.08 mg/kg-day (nail folding, prominent beds, elevated nails). Significant dose-related trends (p less than or equal to 0.05) were observed for certain nail changes (prominent beds, elevated nails) when adjusted for onset time, total frequencies of certain nail changes (nail folding and separation, prominent beds, elevated nails), and decreases in onset time of certain nail changes (nail folding, prominent beds, elevated nails).

Immunologic assessment showed significant ($p<0.01$ or <0.05) reductions in IgG (at all doses of Aroclor 1254) and IgM (all doses but 0.02 mg/kg-day) antibody levels in response to injected sheep red blood cells (SRBC) after 23 months of exposure (Tryphonas et al., 1989). A significant ($p<0.05$) decrease in the percent of helper T-lymphocytes, a significant ($p<0.05$) increase in the percent and absolute level of suppressor T-lymphocytes (TS) and a significant ($p<0.01$) reduction in TH/TS ratio was observed at 0.08 mg/kg-day. The antibody response to SRBC is an antigen-driven response that requires the interaction of several distinct cell types (i.e., antigen processing and presentation by macrophages, participation by T-helper cells and finally proliferation and differentiation of B cells into plasma cells that secrete the antibody), which result in the production and secretion of antibodies specific for SRBC from plasma cells. Perturbation in any of the cells or cell-to-cell interactions by physical, chemical or biological agents can result in aberrant antibody responses. The necessity for the interaction of the three principal cells of the immune system (i.e., macrophage, B lymphocyte and T lymphocyte), in response to SRBC, is the main reason why this response has been so widely used in immunotoxicity testing as a surrogate for infection with a pathogenic organism.

In a recent evaluation of the sensitivity and predictability of various immune function assays used for immunotoxicity testing in the mouse (Luster et al., 1992), the antibody plaque-forming cell (PFC) response to SRBC was found to show the highest association with immunotoxic compounds. Essentially this means that the antibody PFC response to SRBC is a very good predictor of immunotoxicants. Also, it has recently been demonstrated that measurement of serum antibody titer to SRBC using the ELISA assay is as sensitive as the PFC assay for determining the response to SRBC (Butterworth et al., 1993).

There were no exposure-related effects on total B-lymphocytes, total T-lymphocytes, total serum immunoglobulin levels, total serum protein, serum protein fractions after 23 months. No exposure-related effects on serum hydrocortisone levels were observed although the SRBC assay is considered a good surrogate (Tryphonas et al., 1989; Loo et al., 1989; Arnold et al., 1993b).

After 55 months of exposure, there was a significant dose-related decrease ($p<0.0005$ for pairwise comparisons and trend test) in the IgM antibody

response to injected SRBC at greater than or equal to 0.005 mg/kg-day at all times of evaluation (1-4 weeks postimmunization) (Tryphonas et al., 1991a). IgG antibody response to injected SRBC was significantly ($p<0.01$) decreased only at 0.04 mg/kg-day, although the overall trend for dose-response was significant ($p=0.033$). The antibody response to pneumococcus antigen did not differ significantly among all test groups (including controls) at any time tested and showed no dose-related trend. However, the antibody response to pneumococcus antigen is a T cell-independent response and the fact that there is no change with this antigen is not inconsistent with the depressed response to the T cell-dependent SRBC antigen. Other data corroborate the significance of Aroclor 1254 suppression of the antibody response to SRBC and point to effects on T lymphocytes including the dose-related suppression of the Con A and PHA lymphoproliferative responses. The monkeys treated with greater than or equal to 0.005 mg/kg-day had significantly ($p<0.0001$) lower mean percentage levels of total T-lymphocytes and significant trend for dose-response, but absolute numbers of T-lymphocytes were similar among test groups. Flow cytometric analysis showed no treatment-related effects on peripheral blood T-helper, T-suppressor or B-lymphocytes or TH/TS lymphocyte ratio. A statistically significant, dose-related increase was noted for thymosin alpha-1-levels but not for thymosin beta-2-levels. Serum complement activity was significantly ($p<0.025$) increased at greater than or equal to 0.005 mg/kg-day but showed no significant ($p=0.1$) dose-related trend. Natural killer cell activity at effect or target ratios of 25:1, 50:1 or 75:1 was not significantly ($p>0.05$) increased at any dosage, although there was a significant ($p=0.03$) dose-related trend. No signs of microbial infection were noted in any of the preceding reports.

Clinical pathology was evaluated during the first 37 months of the study (Arnold et al., 1993b). These evaluations included monthly measurements of hematology and serum biochemistry (including serum protein, RBC indices, semi-monthly measurements of thyroid function, and daily measurements of urinary porphyrins during the 33rd month of dosing). Significant (p0.05) decreases in average dose-group values compared with controls were found for serum cholesterol at 0.04 mg/kg-day, and reticulocyte count, serum cholesterol, total bilirubin, and alpha-1 + alpha-2-globulins at 0.08 mg/kg-day. Significant dose-related decreasing linear trends were also observed for reticulocyte count ($p=0.002$), cholesterol (p less than or equal to 0.001), and total bilirubin ($p=0.005$). Dose-related decreasing linear trends were also observed for red blood cell count ($p=0.019$), mean platelet volume ($p=0.034$), hematocrit ($p=0.064$), hemoglobin concentration ($p=0.041$). With regard to thyroid endpoints [serum thyroxine (T4), serum triiodothyronine (T3) uptake ratio, percent T3 uptake, and free thyroxine index], dose-response analysis consisted of group mean comparisons and an assessment of parallelism in the response profiles (an absence of parallelism would indicate time-dose interactive effects). No statistically significant changes were observed for any of the thyroid endpoints.

After approximately 2 years of dosing, each dose group was randomly divided into two test groups for daily analyses of serum progesterone and estrogen concentrations during one menstrual cycle (Truelove et al., 1990; Arnold et al., 1993b). There were no statistically significant differences between treated and control monkeys in menstrual cycle length or menses duration, and no apparent treatment-related effects on incidence of anovulatory cycles or temporal relationship between estrogen peak and menses

onset, menses end or progesterone peak (Truelove et al., 1990; Arnold et al., 1993a,b).

To summarize the above, monkeys that ingested 0.005-0.08 mg/kg-day doses of Aroclor 1254 showed ocular exudate, prominence and inflammation of the Meibomian glands and distortion in nail bed formation. These changes were seen at the lowest dose tested, 0.005 mg/kg-day, and a dose-dependent response was demonstrated. Similar changes have been documented in humans for accidental oral ingestion of PCBs. Among the various immunologic function tests that were performed, the increases in IgM and IgG antibodies to sheep erythrocytes are most significant. IgG and IgM antibodies in response to SRBC were reduced after 23 months of exposure but only the IgM antibodies were clearly decreased after 55 months. Particular importance is attributed to the immune response to sheep erythrocytes since it involves participation by the three principal cells of the immune system: the macrophage, B lymphocytes and T lymphocytes and has been shown to be the most predictive immunotoxicity test of those currently in use (Luster et al., 1992). On the basis the studies described, a LOAEL of 0.005 mg/kg-day was established for Aroclor 1254.

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o ORAL RFD UNCERTAINTY :

UF -- A 10-fold factor is applied to account for sensitive individuals. A factor of 3 is applied to extrapolation from rhesus monkeys to humans. A full 10-fold factor for interspecies extrapolation is not considered necessary because of similarities in toxic responses and metabolism of PCBs between monkeys and humans and the general physiologic similarity between these species. A partial factor is applied for the use of a minimal LOAEL since the changes in the periocular tissues and nail bed see at the 0.05 mg/kg-day are not considered to be of marked severity. The duration of the critical study continued for approximately 25% of the lifespan of rhesus monkeys so that a reduced factor was used for extrapolation from subchronic exposure to a chronic RfD. The immunologic and clinical changes that were observed did not appear to be dependent upon duration which further justifies using a factor of 3 rather than 10 for extrapolation from subchronic to chronic, lifetime exposure. The total UF is 300.

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o ORAL RFD MODIFYING FACTOR :

MF -- None

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o ORAL RFD COMMENTS :

Human data available for risk assessment of Aroclor 1254 are useful only in a qualitative manner. Studies of the general population who were exposed to PCBs by consumption of contaminated food, particularly neurobehavioral evaluations of infants exposed in utero and/or through lactation, have been reported, but the original PCB mixtures, exposure levels and other details of exposure are not known (Kreiss et al., 1981; Humphrey, 1983; Fein et al., 1984a,b; Jacobson et al., 1984a, 1985, 1990a,b; Rogan et al., 1986; Gladen et al., 1988). Most of the information on health effects of PCB mixtures in humans is available from studies of occupational exposure. Some of these studies examined workers who had some occupational exposure to Aroclor 1254, but sequential or concurrent exposure to other Aroclor mixtures nearly always occurred, exposure involved dermal as well as inhalation routes (relative

contribution by each route not known), and monitoring data are lacking or inadequate (Alvares et al., 1977; Brown and Jones, 1981; Colombi et al., 1982; Fischbein et al., 1979, 1982, 1985; Fischbein, 1985; Warshaw et al., 1979; Smith et al., 1982; Taylor et al., 1984; Lawton et al., 1985). Insufficient data are available in these studies to determine possible contributions of Aroclor 1254 alone, extent of direct skin exposure and possible contaminants. However, it is relevant to note that dermal and ocular effects, including skin irritation, chloracne, hyperpigmentation and eyelid and conjunctival irritation, have been observed in humans occupationally exposed to Aroclor 1254 and other Aroclor formulations.

Aroclor 1254 was fed to groups of eight female and four male adult rhesus monkeys once daily in dosages of 0, 5, 25 or 100 ug/kg for 14 months, followed by an observation period of 7 months (Levinskas et al., 1984). The Aroclor 1254 was dissolved in corn oil and offered to the animals in apple sauce prior to each day's feeding, and the control mixture (corn oil in applesauce) was used during the observation period. Dosages were adjusted biweekly for changing body weight as necessary. The monkeys were selected on the basis of a successful reproductive history, estimated to be at least 6 years old, and had been in captivity for 2-9 years. After 6 months of treatment the monkeys were bred to untreated males or females from the same colony over an 8-month period and offspring were observed for 2 months. Breeding was continued until conception was diagnosed by digital examination of the uterus and alterations in the menstrual cycle. Evaluations of adult animals included hematology and clinical chemistry. Urinalysis was also performed every 3 months during the study. Semen analyses were performed monthly from just prior to the start of treatment until the end of the treatment period. After 2 months of observation; sperm concentration, total sperm, sperm motility, percent abnormal cells and live/dead ratios were evaluated. Based upon these parameters, no effect was observed upon male reproductive capacity. Necropsies including histological examinations were performed on all adult animals that died during the study or were euthanized at the end of the observation period. Birth weight and somatic measurements were taken for all offspring of exposed females or males. The infants of the exposed females were subsequently evaluated monthly for body weight and complete blood cell counts were performed. Infants that did not show signs of intoxication were euthanized after 2 months and those showing signs were weaned, observed for reversal of signs, and euthanized at the end of the study along with the adults. Necropsies including histological examinations were performed on all infants that died or were euthanized.

Death or euthanasia in extremis occurred in 1/12, 0/12, 1/12 and 5/12 of the adult monkeys in the control, low-, mid- and high-dose groups, respectively. All of the deaths occurred in females except for one male in the high-dose group, and the only deaths considered to be related to treatment were in four of the high-dose animals (3 females, 1 male). Characteristic signs of PCB intoxication developed in the high-dose group after 9 months of exposure, including effects on the eyelids (redness and/or edema, wrinkling) in approximately half the animals and swelling of the lips in all animals. Other characteristic signs included bleeding gums, abnormal fingernail/toenail growth pattern and increased alopecia (including eyelashes) in several of the high-dose monkeys. In general, the signs of intoxication appeared to subside during the post-treatment period. Some of the monkeys in the mid-dose group showed signs of intoxication (swelling of the lips in one male and one female)

after 15 and 18 months, respectively, and alopecia and abnormal nail growth, but no signs attributable to exposure occurred in the low-dose group. Hematologic effects at the high dose were observed including reduced packed cell volume, erythrocyte count, hemoglobin and platelet counts. In addition, increased serum iron and reduced serum cholesterol were observed, particularly in the monkeys that died. Some of the high-dose monkeys also had prolonged bleeding and improper healing at biopsy sites. Dermal histological changes characteristic of PCB poisoning were prominent in the high-dose group, occurring in 11/12 monkeys (8 females, 3 males), and included loss of secretory epithelium in the Meibomian (eyelid) glands and sebaceous glands, partial or total atrophy of sebaceous glands, follicular keratosis and/or squamous cysts. Dermal changes also occurred in four of the mid-dose monkeys, but not in the low-dose or control groups. Other histological alterations included squamous metaplasia in glandular ducts of the tongue or lip (3 high-dose females, 1 high-dose male), subgingival epithelial cysts of the mandible (1 high-dose male, 1 high-dose female, 1 mid-dose male) and hyperplasia in the bile and pancreatic ducts and gall bladder (1 high-dose female). Nonspecific bone marrow alterations (decreased cellularity and/or granulocyte maturation) occurred in 6/12 high-dose monkeys (5 females, 1 male) and may have been compound-related because they correlated with the hematologic changes.

There was no apparent effect on male fertility based on conception rate following matings with the untreated females or the semen analyses (Levinskas et al., 1984). In the female control, low-, mid- and high-dose groups, the numbers of known pregnancies were 7, 7, 7 and 5, respectively; the numbers of live births were 6, 5, 7 and 1, respectively. Analysis of the preceding data showed that there was a statistically significant reduction in fertility in the high-dose group; this analysis refers only to the decreased number of live births. There was a clear exposure-related effect on birth weight and infant body weight gain. When compared with control group infants (mean birth weight 495.2 g) the 0.025 mg/kg-day infants (mean birth weight 392.2 g) showed a statistically significant reduction in birth weight ($p<0.005$). Most of the infants in the mid-dose group and all of the infants in the high-dose had abnormal clinical signs. These changes included being born with or developed dermal signs that were consistent with those in the adults (e.g., swollen lips, swollen eyelids and/or scanty eyelashes) and more severe at the high dose, and also developed pulmonary signs (e.g., respiratory wheezing). Histological changes in the infants were generally similar to those observed in the adults. These effects included changes in the Meibomian and sebaceous glands, pancreatic ducts and bone marrow. Other histological changes included thymic atrophy in one mid-dose and the high-dose infant, and other effects in the high-dose infant (e.g., retarded kidney cortical maturation, bile duct hyperplasia and gastric mucosal gland cysts).

To summarize the above, no treatment-related effects were observed in the low-dose adults or their infants, indicating that 0.005 mg/kg-day is a NOAEL. For the mid-dose infants there was a 15% reduction in birth weight of infants that was statistically significant ($p<0.005$). When these infants reached 2 months of age the reduced body weight was 22% below controls and this difference was also found to be statistically significant ($p=0.05$). Ocular and dermal signs and/or histological changes characteristic of PCB intoxication developed in a some adults receiving 25 and 100 ug/kg-day, as well as in most of the infants in these groups. Based on these effects the 0.025 mg/kg-day dosage is a LOAEL. Other effects at the high dose included

decreased adult survival, female fertility and numbers of live births, indicating that 0.1 mg/kg-day is a FEL. This FEL is supported by results of the Truelove study (Truelove et al., 1982).

Aroclor 1254 was fed to 1, 2 or 1 pregnant rhesus monkeys in reported average daily doses of 0, 0.1 or 0.2 mg/kg-day, respectively, 3 days/week for up to 267 days starting on gestation day 60 (Truelove et al., 1982). The exposure period included gestation and lactation. One of the adult monkeys in the low-dose group and the one adult in the high-dose group lost their fingernails after 233 and 242 days of PCB treatment, but other overt signs of intoxication were not observed. There was a significant reduction in antibody production in response to injected SRBC in the exposed monkeys, but levels of antibody production to tetanus toxoid were not appreciably different from control. The two low-dosage monkeys delivered dead infants. The infant of the high-dosage monkey died at age 139 days; this infant showed impaired immune function as assessed by antibody production following SRBC injections. Hematological evaluation performed bimonthly following parturition in adults and the surviving infant were inconclusive. Although evaluation of the dead infants and other results of this study is complicated by the small number of animals, the characteristic dermal sign of PCB toxicity in the exposed monkeys and lack of effects in controls strongly indicate that the developmental toxicity is exposure-related. Therefore, based on the stillbirths, 0.1 mg/kg-day is a FEL in monkeys.

Groups of four young adult female rhesus monkeys were fed 0 or 0.28 mg/kg doses of Aroclor 1254 for 5 days/week for 114-121 weeks (Tryphonas et al., 1986a,b; Arnold et al., 1990). Groups of four mature adult female cynomolgus monkeys that had a poor breeding history were similarly exposed for 55-58 weeks (Tryphonas et al., 1986a; Arnold et al., 1990). The Aroclor mixture contained no detectable polychlorinated dibenzo-p-dioxin contaminants. Adjusting for the partial weekly exposure gives an average daily dosage of 0.2 mg/kg-day. Prominent clinical signs appeared in all exposed rhesus monkeys during the first 2-12 months of exposure, including facial and periorbital edema, loss of eyelashes, Meibomian gland enlargement and impaction, conjunctivitis, nail lesions progressing from dryness to detachment and gingival hyperplasia and necrosis of varying severity. Two of the exposed rhesus monkeys developed overwhelming infections of the eye or periodontal tissue after 27 months of exposure prompting sacrifice within 48 hours. The hematology and serum biochemistry evaluations showed various changes in the exposed rhesus monkeys, particularly slight or moderate normocytic anemia, depressed erythropoiesis in bone marrow and increased triglycerides and SGOT. The immunologic testing was inconclusive due to large interspecies variability. Pathology findings in the exposed rhesus monkeys included effects in the liver of three monkeys (30-55% increased relative liver weight, hepatocellular hypertrophy and necrosis, bile duct epithelial hypertrophy and hyperplasia, gall bladder epithelial hypertrophy), thyroid of two monkeys (enlargement, occasional follicular cell desquamation) and stomach of two monkeys (hypertrophic gastropathy). The cynomolgus monkeys had effects that were generally consistent with but less extensive and severe than those observed in the rhesus monkeys. After 38 weeks of exposure the rhesus monkeys were mated with untreated males; cynomolgus monkeys were not mated. The control and exposed rhesus monkeys became pregnant within 7 and 8 matings, respectively. Following extended post-implant bleeding all of the treated rhesus monkeys aborted within 30-60 days of gestation. Following recovery

from the abortions the monkeys were bred again up to a maximum of seven times but none appeared to conceive. The menstrual cycle lengths and durations became erratic and longer during and subsequent to the breeding. Based on the abortions, reproductive impairment and pronounced overt signs of toxicity, the 0.2 mg/kg-day dosage is an FEL in monkeys.

Aulerich and Ringer (1977) performed a breeding study in which groups of eight female and two male adult mink were fed diets containing 0 or 2 ppm Aroclor 1254 for 39 weeks or until the kits were 4 weeks of age. The Aroclor was dissolved in acetone which was evaporated from the diet prior to feeding. Using assumed values of 150 g/day for food consumption and 0.8 kg for body weight for female mink (Bleavins et al., 1980), the estimated dosage of Aroclor 1254 is 0.4 mg/kg-day. Approximately monthly determinations reportedly showed no statistically significant ($p<0.05$) differences between the control and treated mink in body weight gain, hemoglobin, and hematocrit. Only two of seven mated females gave birth, producing one infant each. Of the two infants, one was born dead and the other had low body weight and was dead by age 4 weeks. Based on the reproductive and/or fetal toxicity resulting in nearly complete lack of births, 0.4 mg/kg-day is a FEL for Aroclor 1254 in mink.

Twelve female and four male adult ranch-bred mink (age 8 months, body weight not reported) were fed a diet containing 1 ppm Aroclor 1254 for 6 months (Wren et al., 1987a,b). Groups of 15 females and five males were used for unexposed controls. The mink were bred after approximately 12-14 weeks of exposure and exposure was continued until weaning at age 5 weeks. Using assumed values for food consumption and for body weight for female mink (Bleavins et al., 1980), the estimated dosage of Aroclor 1254 is 0.15 mg/kg-day. Offspring mortality during the first week of life was 75.8% higher in the exposed group than in the controls. Average body weight was significantly lower in the exposed offspring at age 3 and 5 weeks, but not at age 1 week, suggesting that transfer of PCBs by lactation may have contributed to the effect. There were no exposure-related effects on adult survival or mating performance, number of offspring per female mated or female that delivered, adult thyroid plasma T3 or T4 levels during the exposure period, adult scrotal diameter, offspring survival or relative liver weight at weaning or organ weights or histology (brain, kidney, adrenal, pituitary, thyroid). Teratogenicity was not evaluated. The neonatal mortality indicates that 0.15 mg/kg-day is an FEL in mink.

Groups of 10 female Sprague-Dawley rats were fed 0, 1, 5, 10 or 50 ppm Aroclor 1254 in the diet for approximately 5-6 months (Byrne et al., 1987). The Aroclor was dissolved in acetone which was evaporated from the diet prior to feeding. Based on reported body weight and food consumption data the dosages are estimated to be 0.09, 0.43, 0.61 and 4.3 mg/kg-day. Serum thyroxine (T4) and triiodothyronine (T3) were evaluated at five different times during 140 and 175 days of treatment, respectively. Serum T4 levels were significantly reduced at 0.09 and 0.43 mg/kg-day by day 35 and at greater than or equal to 0.61 mg/kg-day by day 14. T3 levels were significantly reduced at 0.09 mg/kg-day by day 40 and at greater than or equal to 0.4 mg/kg-day by day 20. The suppressions were generally dose-related for T4 throughout the treatment period and T3 after 75 days. Disappearance rate of injected L-[125I] T4 was significantly decreased at greater than or equal to 0.09 mg/kg-day. Rats treated with only 0.43 or 0.61 mg/kg-day for approximately 5 months

and challenged with i.p. injected TSH had diminished response of serum T4 and T3. Thyroid histology was not evaluated. There were no treatment-related effects on relative thyroid weight, body weight or food consumption. The findings of this study indicate that the decreased serum T3 and T4 resulted primarily from direct damage to the thyroid rather than suppression of the hypothalamo-pituitary axis or any enhanced peripheral catabolism (e.g., liver). Insufficient data are available to determine if the decreases in circulating thyroid hormones were physiologically significant. However, because the effects are indicative of impaired organ function, they are at least potentially adverse and 0.09 mg/kg-day is considered to represent a LOAEL in rats.

Groups of 10 female Sprague-Dawley rats were fed 0, 1, 5, 10 or 50 ppm Aroclor 1254 in the diet for 5 months (Byrne et al., 1988). The Aroclor was dissolved in acetone which was evaporated from the diet prior to feeding. Using a rat food consumption factor of 0.05 kg food/kg body weight, the dosages are estimated to be 0.05, 0.25, 0.5 and 2.5 mg/kg-day. Serum levels of adrenal cortex hormones were evaluated in 8-10 rats 3-5 times during the treatment period. Serum corticosterone was significantly ($p<0.05$) decreased at greater than or equal to 0.25 mg/kg-day after approximately 60 days of exposure. Serum dehydroepiandrosterone and dehydroepiandrosterone sulfate were significantly ($p<0.05$) decreased at 0.25 and 0.5 mg/kg-day (not evaluated at other dosages) after approximately 100 days and 25 days of exposure, respectively. Serum corticosterone is the principal glucocorticoid in rats. Adrenal weight, adrenal histology and non-adrenal endpoints other than food consumption were not evaluated. Food consumption did not significantly differ between and among control and treatment groups. The results of this study are suggestive of toxicity to the adrenal rather than response to stress which would be expected to increase the release of glucocorticoids. Insufficient data are available to determine if the decreases in circulating adrenal cortex hormones were physiologically significant. However, because the effects are indicative of impaired organ function, they are at least potentially adverse. The dosages of 0.05 and 0.25 mg/kg-day therefore are considered to represent a NOEL and LOAEL, respectively, in rats.

Hepatotoxicity is a prominent effect of Aroclor 1254 that is well characterized in rats (U.S. EPA, 1990). The spectrum of effects includes hepatic microsomal enzyme induction, increased serum levels of liver-associated enzymes indicative of possible hepatocellular damage, liver enlargement, lipid deposition, fibrosis and necrosis. Estimated subchronic dosages as low as 1.25-2.5 mg/kg-day have been reported to produce increased liver weight and hepatic biochemical alterations in rats, but the lowest dosages producing signs of hepatic effects are generally higher than the lowest dosages that caused thyroid, adrenal and bone changes (Litterset et al., 1972; Bruckner et al., 1974; Kling and Gamble, 1982; Andrews et al., 1989). Rats fed 6.8 mg/kg-day for 8 months (Kimbrough et al., 1972) or an estimated dosage of 50 mg/kg-day for 30 days (Kling et al., 1978) developed fatty and necrotic degenerative hepatic histologic changes. Chronic dietary exposure to 1.25-5 mg/kg-day for approximately 2 years produced only preneoplastic and neoplastic liver lesions in rats (NCI, 1978; Ward, 1985).

A two-generation reproduction study was performed in which groups of 20 female and 10 male Sherman rats (age 3-4 weeks, body weight not reported) were fed 0, 1, 5, 20 or 100 ppm dietary Aroclor 1254 (Monsanto Lot No. AK-38) in

peanut oil vehicle (Linder et al., 1974). Reported dosages were 0.06, 0.32, 1.5 and 7.6 mg/kg-day, and different controls were used for the less than or equal to 0.32 and greater than or equal to 1.5 mg/kg-day groups. Exposure times (before mating or conception-to-mating) ranged from 62-274 days. Exposure-related effects included increased relative liver weight in F1a weanlings at greater than or equal to 0.06 mg/kg-day, enlarged and vacuolated hepatocytes in F2a weanlings at greater than or equal to 1.5 mg/kg-day, and 15-72% reduced litter size at greater than or equal to 1.5 mg/kg-day in the F1b, F2a and F2b generations and at 7.6 mg/kg-day in the F1a generation. Relative testes weights were increased in adult F1b males at 7.6 mg/kg-day (other groups not evaluated). The highest NOAEL is 0.32 mg/kg-day based on the increased liver weight without altered histology. The decreased litter size indicates that 1.5 mg/kg-day is a FEL.

A one-generation reproduction study was performed in which groups of 10 male and 10 female Sherman rats were fed 0, 100 or 500 ppm dietary Aroclor 1254 for 67 or 186 days prior to pair-mating for the F1a and F1b generations, respectively (Linder et al., 1974). The F0 rats received reported dosages of 0, 7.2 and 37.0 mg/kg-day and were sacrificed after a total exposure duration of 8 months for hematology, organ weight and liver histology evaluation. The study was terminated after the F1b pups were weaned. Effects in the P1 rats included increased liver weight in both sexes greater than or equal to 7.2 mg/kg-day, increased relative testis weight (absolute weight unchanged) at 37.0 mg/kg-day, decreased body weight gain in both sexes at 37.0 mg/kg-day, and changes in hematological values (reduced hematocrit and hemoglobin in both sexes, increased total leukocytes with normal differential count in females) at 37.0 mg/kg-day. Specific information on liver pathology was not reported but degenerative changes similar to those found in the Kimbrough et al. (1972) subchronic study were indicated for both dosages. Effects on the offspring included reduced survival to weaning at 7.2 mg/kg-day (85.9 and 68.1% survival in F1a and F1b pups, respectively, compared with 95.5% in controls), and reduced litter size and number and 100% pup mortality by day 3 in F1a rats at 37.0 mg/kg-day. The decreases in postnatal survival indicate that both dosages are FELs.

Groups of six to eleven female Wistar rats were fed 2.5, 26 or 269 ppm Aroclor 1254 in the diet during gestation and lactation (Overman et al., 1987). A control group was fed untreated diet that contained 0.02 ppm PCBs (i.e., no added PCBs). Using a rat food consumption factor of 0.05 kg food/kg body weight, the dosages are estimated to be 0.001, 0.13, 1.3 and 13.5 mg/kg-day. The following neurobehavioral endpoints were significantly delayed or reduced in the pups: appearance of the auditory startle response at 0.13 and 1.3 mg/kg-day at age 6 days (slightly delayed), development of righting ability at 1.3 mg/kg-day at days of age (slightly delayed) and performance on a motor coordination test at 1.3 mg/kg-day at age 7 and 8 days (slower performance). Grip strength and appearance of eye opening were not affected by exposure. Other effects attributable to exposure included increased relative liver weight in pups at weaning at greater than or equal to 1.3 mg/kg-day and reduced birth weight, 50% mortality by 2 days of age and retarded growth in pups at 13.5 mg/kg-day. There were no exposure-related effects on maternal weight gain, gestation length, litter size, pup sex ratios, number of live and dead pups or physical appearance, relative spleen and thymus weight or relative and absolute brain weight of pups. Brain PCB levels increased from birth to weaning in all groups. Based on the evidence

for impaired motor coordination in developing infants the 0.13 and 1.3 mg/kg-day dosages are a NOAEL and LOAEL, respectively.

Dietary Aroclor 1254 was administered to groups of 4-10 female ICR mice in concentrations of 0, 1, 10 or 100 ppm from 90 days before mating through gestation day 18 (Welsch, 1985). The investigators estimated the dosages to be 0.125, 1.25 and 12.5 mg/kg-day. No developmental toxicity was observed as judged by number of litters, number of dead and reabsorbed fetuses, fetal weight, incidence of gross malformations or skeletal development. Fetuses were not examined for internal malformations. Maternal effects other than significantly increased relative liver weight at greater than or equal to 0.125 mg/kg-day were not observed. No developmental effects were observed in mice treated with the same doses of PCB only on gestation days 6-18. Based on the increased maternal liver weight the highest NOAEL is 12.5 mg/kg-day.

Groups of seven adult male New Zealand white rabbits were fed dietary Aroclor 1254 in reported estimated dosages of 0, 0.18, 0.92, 2.10 or 6.54 mg/kg-day for 8 weeks (Street and Sharma, 1975). Immunological testing was started after 4 weeks of treatment at which time the rabbits were immunized with injected SRBCs. No treatment-related changes in serum antibody titers to SRBC (hemolysin and hemagglutination) were observed. SRBC-induced increases in serum gamma-globulin were consistently but not statistically significantly decreased by exposure, and the number of globulin-producing cells in popliteal lymph nodes was significantly decreased at 0.92 and 6.54 mg/kg-day. Skin sensitivity to tuberculin was generally lower in the treated groups but none of the decreases were statistically significant. Marked histologic atrophy of the thymus cortex was observed at 0.18 mg/kg-day and higher dosages except 0.92 mg/kg-day. There were no treatment-related effects on leukocyte count, histology of the spleen, thymus, liver, kidneys or spleen, relative kidney or adrenal weight, terminal body weight or food consumption. Relative liver and spleen weights were significantly increased at greater than or equal to 2.10 mg/kg-day; the increase in liver weight was 74% at the highest dosage. The 0.18 mg/kg-day dosage is a LOAEL based on the thymic cortical atrophy.

Limited specific information is available on the oral absorption of Aroclor 1254. Pregnant ferrets that ingested a single oral dose of Aroclor 1254 (approximately 0.06 mg/kg) absorbed approximately 85% of the initial amount (Bleavins et al., 1984). Studies predominately of individual chlorobiphenyl congeners indicate, in general, that PCBs are readily and extensively absorbed by animals. These studies have found oral absorption efficiency on the order of 75 to >90% in rats, mice and monkeys (Albro and Fishbein, 1972; Allen et al., 1974; Tanabe et al., 1981; Clevenger et al., 1989). A study of a non-Aroclor 54% chlorine PCB mixture prepared by the investigators provides direct evidence of absorption of PCBs in humans after oral exposure (Buhler et al., 1988), and indirect evidence of oral absorption of PCBs by humans is available from studies of ingestion of contaminated fish by the general population (Schwartz et al., 1983; Kuwabara et al., 1979). There are no quantitative data regarding inhalation absorption of PCBs in humans but studies of workers exposed suggest that PCBs are well absorbed by the inhalation and dermal routes (Maroni et al., 1981a,b; Smith et al., 1982; Wolff, 1985). PCBs distribute preferentially to adipose tissue and concentrate in human breast milk due to its high fat content (Jacobson et al., 1984b; Ando et al., 1985).

The metabolism of PCBs following oral and parenteral administration in animals has been extensively studied and reviewed, but studies in animals following inhalation or dermal exposure are lacking (Sundstrom and Hutzinger, 1976; Safe, 1980; Sipes and Schnellmann, 1987). Information on metabolism of PCBs in humans is limited to occupationally exposed individuals whose intake is derived mainly from inhalation and dermal exposure (Jensen and Sundstrom, 1974; Wolff et al., 1982; Schnellmann et al., 1983; Safe et al., 1985; Fait et al., 1989). In general, metabolism of PCBs depends on the number and position of the chlorine atoms on the phenyl ring of the constituent congeners (i.e., congener profile of the PCB mixture) and animal species. Although only limited data are available on metabolism of PCBs following inhalation exposure, there is no reason to suspect that PCBs are metabolized differently by this route.

Data exist on the in vitro hepatic metabolism and in vivo metabolic clearance of 2,2',3,3',6,6'-hexachlorobiphenyl and 4,4'-dichlorobiphenyl congeners in humans, monkeys, dogs and rats (Schnellmann et al., 1985). The hexachlorobiphenyl congener is a constituent of Aroclor 1254. For each congener, the Vmax values for metabolism in the monkey, dog and rat are consistent with the respective metabolic clearance values found in vivo. Thus, the kinetic constants for PCB metabolism obtained from the dog, monkey and rat hepatic microsomal preparations were good predictors of in vivo metabolism and clearance for these congeners. In investigations directed at determining which species most accurately predicts the metabolism and disposition of PCBs in humans, the in vitro metabolism of these congeners was also studied using human liver microsomes (Schnellmann et al., 1983, 1984). Available data suggest that metabolism of PCBs in humans would most closely resemble that of the monkey and rat. For example, the in vitro apparent Km and Vmax are comparable between humans and monkeys. These studies show consistency between the in vitro and in vivo findings and collectively indicate that metabolism of the two congeners is similar in monkeys and humans.

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o ORAL RFD CONFIDENCE :

Study – Medium
Data Base – Medium
RfD – Medium

Confidence in the principal study is medium. Groups of 16 rhesus monkeys were tested at four dose levels and LOAEL was established on the basis of clinical signs and immunologic alterations. Data for female and male reproductive function and developmental data in a nonhuman primate species is taken from an unpublished study (Levinskas et al., 1984) which established a NOAEL for reproductive effects at 0.005 mg/kg-day. The Arnold study also included evaluation of reproductive function but the data have not been completely analyzed. Preliminary examination of the Arnold et al. data indicate that the LOAEL for female reproductive function may be 0.005 mg/kg-day. This inconsistency in effect levels for reproductive toxicity was viewed as a limitation to the data base. Furthermore, there is a limitation in the characterization of reproductive toxicology because results of an unpublished study have been considered. An extensive number of laboratory animal and human studies were available for review, including two-generation reproductive studies. The chronic, 2-year bioassays performed in F344 rats showed evidence

of degenerative hepatocellular changes in addition to the neoplastic changs that were observed. Only limited assessment of nonhepatic changes were made. Human occupational and environmental data is available for commercial PCB mixtures in general but not specifically for Aroclor 1254. The data base is rated medium on the basis of these considerations. Overall confidence in the RfD is medium.

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o ORAL RFD SOURCE DOCUMENT :

Source Document -- This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation -- U.S. EPA, 1984, 1989, 1990

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o REVIEW DATES : 06/16/93, 02/16/94
o VERIFICATION DATE : 02/16/94
o EPA CONTACTS :

John L. Cicmanec / OHEA -- (513)569-7481

Michael L. Dourson / OHEA -- (513)569-7533

RDI - NO DATA
CAREV- NO DATA
CARO - NO DATA
CARI - NO DATA
CARDR- NO DATA

HAONE- NO DATA

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA - NO DATA

WQCHU- NO DATA

WQCAQ- NO DATA

MCLG - NO DATA

MCL - NO DATA

SMCL - NO DATA

FISTD- NO DATA

FIREV- NO DATA

CERC - NO DATA

SARA - NO DATA

RCRA - NO DATA

TSCA - NO DATA

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IREF - None

CREF - None

HAREF- None

PCB 1260

1 - IRIS
NAME - Polychlorinated biphenyls (PCBs)
RN - 1336-36-3
IRSN - 288
DATE - 940601
UPDT - 06/01/94, 3 fields
STAT - Oral RfD Assessment (RDO) message 06/01/94
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) on-line 01/01/90
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 05/01/89 CAR Carcinogen summary on-line
IRH - 01/01/90 CAR Text edited
IRH - 01/01/90 REFS Bibliography on-line
IRH - 01/01/92 EXSR Regulatory Action section on-line
IRH - 06/01/94 RDO Message only
RLEN - 24527
SY - AROCLOR
SY - AROCLOR 1221
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SY - AROCLOR 1248
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SY - AROCLOR 1260
SY - AROCLOR 1262
SY - AROCLOR 1268
SY - AROCLOR 2565
SY - AROCLOR 4465
SY - AROCLOR 5442
SY - BIPHENYL, POLYCHLORO-
SY - CHLOPHEN
SY - CHLOREXTOL
SY - CHLORINATED BIPHENYL
SY - CHLORINATED DIPHENYL
SY - CHLORINATED DIPHENYLENE
SY - CHLORO BIPHENYL
SY - CHLORO 1,1-BIPHENYL
SY - CLOPHEN
SY - DYKANOL
SY - FENCLOR
SY - INERTEEN
SY - KANECHLOR
SY - KANECHLOR 300
SY - KANECHLOR 400
SY - MONTAR
SY - NOFLAMOL
SY - PCB
SY - PCBs
SY - PHENOCHLOR
SY - PHENOCLOR
SY - POLYCHLORINATED BIPHENYL
SY - Polychlorinated Biphenyls
SY - POLYCHLOROBIPHENYL
SY - PYRALENE

SY - PYRANOL
SY - SANTOTHERM
SY - SANTOTHERM FR
SY - SOVOL
SY - THERMINOL FR-1
SY - UN 2315

RDO -
o ORAL RFD SUMMARY :

Please check the following individual aroclor files for RfD assessments: Aroclor 1016, Aroclor 1248, Aroclor 1254, Aroclor 1260.

RDI - NO DATA
CAREV-
o CLASSIFICATION : B2; probable human carcinogen
o BASIS FOR CLASSIFICATION : hepatocellular carcinomas in three strains of rats and two strains of mice and inadequate yet suggestive evidence of excess risk of liver cancer in humans by ingestion and inhalation or dermal contact.
o HUMAN CARCINOGENICITY DATA :

Inadequate. Although there are many studies, the data are inadequate due to confounding exposures or lack of exposure quantification. The first documentation of carcinogenicity associated with PCB exposure was reported at a New Jersey petrochemical plant involving 31 research and development employees and 41 refinery workers (Bahn et al., 1976, 1977). Although a statistically significant increase in malignant melanomas was reported, the two studies failed to report a quantified exposure level and to account for the presence of other potential or known carcinogens. In an expanded report of these studies, NIOSH (1977) concurred with the Bahn et al. (1976) findings.

Brown and Jones (1981) reported a retrospective cohort mortality study on 2567 workers who had completed at least 3 months of employment at one or two capacitor manufacturing plants. Exposure levels were 24-393 mg/cu.m at plant A and 318-1260 mg/cu.m at plant B. No excess risk of cancer was observed. In a 7-year follow-up study, Brown (1987) reported a statistically significant excess risk of liver and biliary cancer, with four of the five liver cancers in female workers at plant B. A review of the pathology reports indicated that two of the liver tumors counted in the follow-up study were not primary liver tumors. When these tumors are excluded the elevation in incidence is not statistically significant. The results also may be confounded by population differences in alcohol consumption, dietary habits, and ethnic composition.

Bertazzi et al. (1987) conducted a mortality study of 544 male and 1556 female employees of a capacitor-making facility in Northern Italy. Aroclor 1254 and Pyralene 1476 were used in this plant until 1964. These were progressively replaced by Pyralenes 3010 and 3011 until 1970, after which

lower chlorinated Pyralenes were used exclusively. In 1980 the use of PCBs was abandoned. Some employees also used trichloroethylene but, according to the authors, were presumed to be protected by efficient ventilation. Air samples were collected and analyzed for PCBs in 1954 and 1977 because of reports of chloracne in workers. Quantities of PCBs on workers' hands and workplace surfaces also were measured in 1977. In 18 samples, levels ranged from 0.2-159.0 ug/sq.m on workplace surfaces and 0.3-9.2 ug/sq.m on workers' hands.

The authors compared observed mortality with that expected between 1946 and 1982 based on national and local Italian mortality rates. With vital status ascertainment 99.5% complete, relatively few deaths were reported by 1982 [30 males (5.5%) and 34 females (2.2%)]. In cohort males, the number of deaths from malignant tumors was significantly higher than expected compared with local or national rates, as was the number of deaths from cancer of the GI tract (6 observed vs. 1.7 national expected and 2.2 local expected). Of the six GI cancer deaths, one was due to liver cancer and one to biliary tract cancer. Deaths from hematologic neoplasms in males were also higher than expected, but the excess was not statistically significant. Total cancer deaths in females were significantly elevated in comparison to local rates (12 observed vs. 5.3 expected). None of these were liver or biliary cancers. The number of deaths from hematologic neoplasms in females was higher than expected when compared with local rates (4 observed vs. 1.1 expected). This study is limited by several factors, particularly the small number of deaths that occurred by the cut-off period. The power of the study is insufficient to detect an elevated risk of site-specific cancer. In addition, the authors stated, after an examination of the individual cases, that interpretation of the increase in GI tract cancer in males was limited, as it appeared likely that some of these individuals had only limited PCB exposure. Confounding factors may have included possible contamination of the PCBs by dibenzofurans and exposure of some of the workers to trichloroethylene, alkylbenzene, and epoxy resins.

Two occurrences of ingestion of PCB-contaminated rice oil have been reported: the Yusho incident of 1968 in Japan and the Yu-Cheng incident of 1979 in Taiwan. Amano et al. (1984) completed a 16-year retrospective cohort mortality study of 581 male and 505 female victims of the Yusho incident. A consistently high risk of liver cancer in females over the entire 16 years was observed; liver cancer in males was also significantly increased. Several serious limitations are evident in this study. There was a lack of information regarding job histories or the influence of alcoholism or smoking.

The information concerning the diagnosis of liver cancer was obtained from the victims' families, and it is not clear whether this information was independently verified by health professionals. For some of the cancers described, the latency period is shorter than would be expected. Furthermore, the contaminated oils contained polychlorinated dibenzofurans and polychlorinated quinones as well as PCBs, and the study lacks data regarding exposure to the first two classes of compounds. There is strong evidence indicating that the health effects seen in Yusho victims were due to ingestion of polychlorinated dibenzofurans, rather than to PCBs themselves (reviewed in EPA, 1988). The results of the Amano et al. study can, therefore, be considered as no more than suggestive of carcinogenicity of PCBs.

o ANIMAL CARCINOGENICITY DATA

Sufficient. PCB mixtures assayed in the following studies were commercial preparations and may not be the same as mixtures of isomers found in the environment. Although animal feeding studies demonstrate the carcinogenicity of commercial PCB preparations, it is not known which of the PCB congeners in such preparations are responsible for these effects, or if decomposition products, contaminants or metabolites are involved in the toxic response. Early bioassays with rats (Kimura and Baba, 1973; Ito et al., 1974) were inadequate to assess carcinogenicity due to the small number of animals and short duration of exposure to PCB. A long-term bioassay of Aroclor 1260 reported by Kimbrough et al. (1975) produced hepatocellular carcinomas in female Sherman rats when 100 ppm was administered for 630 days to 200 animals.

Hepatocellular carcinomas and neoplastic nodules were observed in 14 and 78%, respectively, of the dosed animals, compared with 0.58 and 0%, respectively, of the controls.

The NCI (1978) reported results for 24 male and 24 female Fischer 344 rats treated with Aroclor 1254 at 25, 50, or 100 ppm for 104 to 105 weeks. Although carcinomas of the gastrointestinal tract were observed among the treated animals only, the incidence was not statistically significantly elevated. An apparent dose-related incidence of hepatic nodular hyperplasia in both sexes as well as hepatocellular carcinomas among mid- to high-dose treated males was reported (4-12%, compared to 0% in controls).

Norback and Weltman (1985) fed 70 male and 70 female Sprague-Dawley rats a diet containing Aroclor 1260 in corn oil at 100 ppm for 16 months, followed by a 50 ppm diet for an additional 8 months, then a basal diet for 5 months. Control animals (63 rats/sex) received a diet containing corn oil for 18 months, then a basal diet alone for 5 months. Among animals that survived for at least 18 months, females exhibited a 91% incidence (43/47) of hepatocellular carcinoma. An additional 4% (2/47) had neoplastic nodules. In males corresponding incidences were 4% (2/46) for carcinoma and 11% (5/46) for neoplastic nodules. Concurrent liver morphology studies were carried out on tissue samples obtained by partial hepatectomies of three animals/group at eight time points. These studies showed the sequential progression of liver lesions to hepatocellular carcinomas.

Orally administered PCB resulted in increased incidences of hepatocellular carcinomas in two mouse strains. Ito et al. (1973) treated male dd mice (12/group) with Kanechlors 500, 400 and 300 each at dietary levels of 100, 250 or 500 ppm for 32 weeks. The group fed 500 ppm of Kanechlor 500 had a 41.7% incidence of hepatocellular carcinomas and a 58.3% incidence of nodular hyperplasia. Hepatocelluar carcinomas and nodular hyperplasia were not observed in mice fed 100 or 250 ppm of Kanechlor 500, nor among those fed Kanechlors 400 or 300 at any concentrations.

Schaeffer et al. (1984) fed male Wistar rats diets containing 100 ppm of the PCB mixtures Clophen A 30 (30% chlorine by weight) or Clophen A 60 (60% chlorine by weight) for 800 days. The PCB mixtures were reported to be free of furans. Clophen A 30 was administered to 152 rats, Clophen A 60 to 141 rats, and 139 rats received a standard diet. Mortality and histologic lesions were reported for animals necropsied during each 100-day interval for all

three groups. Of the animals that survived the 800-day treatment period, 1/53 rats (2%) in the control group, 3/87 (3%) in the Clophen A 30 group and 52/85 (61%) in the Clophen A 60 group had developed hepatocellular carcinoma. The incidence in the Clophen A 60 group was significantly elevated in comparison to the control group. Neoplastic nodules were reported in 2/53 control, 35/87 Clophen A 30, and 34/85 Clophen A 60-treated animals. The incidence of nodules was significantly increased in both treatment groups in comparison to the control group. Neoplastic liver nodules and hepatocellular carcinomas appeared earlier and at higher incidence in the Clophen A 60 group relative to the Clophen A 30 group. The authors interpreted the results as indicative of a carcinogenic effect related to the degree of chlorination of the PCB mixture. The authors also suggested that these findings support those of others, including Ito et al. (1973) and Kimbrough et al. (1975), in which hepatocellular carcinomas were produced by more highly chlorinated mixtures.

Kimbrough and Linder (1974) dosed groups of 50 male BALB/cJ mice (a strain with a low spontaneous incidence of hepatoma) with Aroclor 1254 at 300 ppm in the diet for 11 months or 6 months, followed by a 5-month recovery period. Two groups of 50 mice were fed a control diet for 11 months. The incidence of hepatomas in survivors fed Aroclor 1254 for 11 months was 10/22. One hepatoma was observed in the 24 survivors fed Aroclor 1254 for 6 months.

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o SUPPORTING DATA :

Most genotoxicity assays of PCBs have been negative. The majority of microbial assays of PCB mixtures and various congeners showed no evidence of mutagenic effects (Schoeny et al., 1979; Schoeny, 1982; Wyndham et al., 1976).

Of various tests on the clastogenic effect of PCBs (Heddle and Bruce, 1977; Green et al., 1975), only Peakall et al. (1972) reported results indicative of a possible clastogenic action by PCBs in dove embryos.

Chlorinated dibenzofurans (CDFs), known contaminants of PCBs, and chlorinated dibenzodioxins (CDDs) are structurally related to and produce certain biologic effects similar to those of PCB congeners. While the CDDs are known to be carcinogenic, the carcinogenicity of CDFs is still under evaluation.

CARO -
o CLASSIFICATION : B2; probable human carcinogen
o BASIS FOR CLASSIFICATION : hepatocellular carcinomas in three strains of rats and two strains of mice and inadequate yet suggestive evidence of excess risk of liver cancer in humans by ingestion and inhalation or dermal contact.
o ORAL SLOPE FACTOR : 7.7/mg/kg/day
o DRINKING WATER UNIT RISK : 2.2E-4/ug/L
o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk
o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	5E-1 ug/L
E-5 (1 in 100,000)	5E-2 ug/L
E-6 (1 in 1,000,000)	5E-3 ug/L

o ORAL DOSE-RESPONSE DATA :

Tumor Type – trabecular carcinoma/adenocarcinoma, neoplastic nodule
Test Animals – rat/Sprague-Dawley, female
Route – diet
Reference – Norback and Weltman, 1985

Administered Dose (mg/kg)/day (TWA)	Human Equivalent Dose (mg/kg)/day	Tumor Incidence
0	0	1/49
3.45	0.59	45/47

o ADDITIONAL COMMENTS :

Human equivalent dosage assumes a TWA daily dose of 3.45 mg/kg/day. This reflects the dosing schedule of 5 mg/kg/day (assuming the rat consumes an amount equal to 5% of its bw/day) for the first 16 months, 2.5 mg/kg/day for the next 8 months, and no dose for the last 5 months.

A slope factor of 3.9/mg/kg/day was based on data from the Kimbrough et al. (1975) study of female Sherman rats fed Aroclor 1260. The estimate based on the data of Norback and Weltman (1985) is preferred because Sprague-Dawley rats are known to have low incidence of spontaneous hepatocellular neoplasms. Moreover, the latter study spanned the natural life of the animal, and concurrent morphologic liver studies showed the sequential progression of liver lesions to hepatocellular carcinomas.

Although it is known that PCB congeners vary greatly as to their potency in producing biological effects, for purposes of this carcinogenicity assessment Aroclor 1260 is intended to be representative of all PCB mixtures. There is some evidence that mixtures containing more highly chlorinated biphenyls are more potent inducers of hepatocellular carcinoma in rats than mixtures containing less chlorine by weight (reviewed in Kimbrough, 1987 and Schaeffer et al., 1984).

The unit risk should not be used if the water concentration exceeds 50 ug/L, since above this concentration the slope factor may differ from that stated.

o DISCUSSION OF CONFIDENCE :

The Norback and Weltman study used an adequate number of animals, observed for their normal lifespan. Only one non-zero test dose was used. A second risk estimate was also calculated based on the numbers of malignant tumors alone, as called for in the EPA's guidelines for carcinogen risk assessment. The slope factor thus derived is 5.7/mg/kg/day, which is 26% less than that derived using combined malignant tumors and neoplastic nodules. This risk

estimate is supported by one based on data of Kimbrough et al. (1975).

PCB mixtures in drinking water may not be the same as the mixtures introduced or used for testing carcinogenicity in animals.

CARI - NO DATA
CARDR-
o CARCINOGENICITY SOURCE :

Source Document – U.S. EPA, 1988

The 1988 Drinking Water Criteria Document for PCBs has received OHEA review.
DOCUMENT

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o REVIEW DATES : 04/22/87
o VERIFICATION DATE : 04/22/87
o EPA CONTACTS :

Charli Hiremath / OHEA – (202)260-5725

Debdas Mukerjee / OHEA – (513)569-7572

HAONE- NO DATA

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA - NO DATA

WQCHU-

Water and Fish Consumption: 7.9E-5 ug/L

Fish Consumption Only: 7.9E-6 ug/L

Considers technological or economic feasibility? – NO

Discussion – For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. Since zero, however, may not be attainable at this time, the recommended criteria represents an E-6 estimated incremental increase of cancer risk over a lifetime.

Reference – 45 FR 79318 (11/28/80)
EPA Contact – Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute – 2.0E+0 ug/L
Chronic – 1.4E-2 ug/L

Marine:

Acute – 1.0E+1 ug/L
Chronic – 3.0E-2 ug/L

Considers technological or economic feasibility? – NO

Discussion – Criteria were derived from a minimum data base consisting of acute tests on a variety of species. Requirements and methods are covered in the reference to the Federal Register.

Reference – 45 FR 79318 (11/28/80)

EPA Contact – Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value – 0.0 mg/L (Final, 1991)

Considers technological or economic feasibility? – NO

Discussion – The MCLG for polychlorinated biphenyls is zero based on the evidence of carcinogenic potential (classification B2).

Reference – 56 FR 3526 (01/30/91)

EPA Contact – Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value -- 0.0005 mg/L (Final, 1991)

Considers technological or economic feasibility? -- YES

Discussion -- The MCL is based on a PQL of 0.0005 mg/L and is associated with a maximum lifetime individual risk of E-4.

Monitoring requirements -- All systems monitored initially for four consecutive quarters every three years; repeat monitoring dependent upon detection, vulnerability status and system size.

Analytical methodology -- Microextraction/gas chromatography (EPA 505); electron capture detector (EPA 508); perchlorination/gas chromatography (EPA 508A). PQL= 0.0005 mg/L.

Best available technology -- Granular activated carbon

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

SMCL - NO DATA

FISTD- NO DATA

FIREV- NO DATA

CERC -

Value (status) -- 1 pound (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ for polychlorinated biphenyls is based on aquatic toxicity. The available data indicate that the 96-Hour Median Threshold Limit is less than 0.1 ppm, which corresponds to an RQ of 1 pound.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

SARA - NO DATA

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

___IV.E.1. TSCA, SECTION 6

Status -- Final Rule (1988)

Discussion -- Prohibits the manufacture, processing, distribution in commerce, or the use of PCBs other than in a "totally enclosed manner" unless specifically exempted by the EPA. Reporting, disposal and record-keeping requirements. Advance notice of proposed rulemaking [56 FR 26738, (06/10/91)] to amend TSCA PCB disposal regulations.

Reference -- 52 FR 27322 (07/19/88); 55 FR 21033 (05/22/90)

EPA Contact -- Chemical Control Division / OTS
(202) 260-3749 / FTS 260-3749

OREF - None
IREF - None
CREF - Amano, M., K. Yagi, H. Nakajima, R. Takehara, H. Sakai and G. Umeda. 1984. Statistical observations about the causes of the death of patients with oil poisoning. Japan Hygiene. 39(1): 1-5.
CREF - Bahn, A.K., I. Rosenwaike, N. Herrmann, P. Grover, J. Stellman and K. O'Leary. 1976. Melanoma after exposure to PCB's. New Engl. J. Med. 295: 450.
CREF - Bahn, A.K., P. Grover, I. Rosenwaike, K. O'Leary and J. Stellman. 1977. Reply to letter from C. Lawrence entitled, "PCB? and melanoma". New Engl. J. Med. 296: 108.
CREF - Bertazzi, P.A., L. Riboldi, A. Pesatori, L. Radice and C. Zacchetti.

1987. Cancer mortality of capacitor manufacturing workers. Am. J. Ind. Med. 11(2): 165-176.

CREF - Brown, D.P. 1987. Mortality of workers exposed to polychlorinated biphenyls – An update. Arch. Environ. Health. 42(6): 333-339.

CREF - Brown, D.P. and M. Jones. 1981. Mortality and industrial hygiene study of workers exposed to polychlorinated biphenyls. Arch. Environ. Health. 36(3): 120-129.

CREF - Green, S., J.V. Carr, K.A. Palmer and E.J. Oswald. 1975. Lack of cytogenetic effects in bone marrow and spermatogonial[sic] cells in rats treated with polychlorinated biphenyls (Aroclors 1242 and 1254). Bull. Environ. Contam. Toxicol. 13(1): 14-22.

CREF - Heddle, J.A. and W.R. Bruce. 1977. Comparison of tests for mutagenicity or carcinogenicity using assays for sperm abnormalities, formation of micronuclei and mutations in Salmonella. In: Origins of Human Cancer, H.H. Hiatt et al., Ed. Cold Spring Harbor Conf. Cell Prolif., Cold Spring Harbor Lab., Cold Spring Harbor, NY. 4: 1549-1557.

CREF - Ito, N., H. Nagasaki, M. Arai, S. Makiura, S. Sugihara and K. Hirao. 1973. Histopathologic studies on liver tumorigenesis induced in mice by technical polychlorinated biphenyls and its promoting effect on liver tumors induced by benzene hexachloride. J. Natl. Cancer Inst. 51(5): 1637-1646.

CREF - Ito, N., H. Nagasaki, S. Makiura and M. Arai. 1974. Histopathological studies on liver tumorigenesis in rats treated with polychlorinated biphenyls. Gann. 65: 545-549.

CREF - Kimbrough, R.D. 1987. Human health effects of polychlorinated biphenyls (PCBs) and polybrominated biphenyls (PBBs). Ann. Rev. Pharmacol. Toxicol. 27: 87-111.

CREF - Kimbrough, R.D. and R.E. Linder. 1974. Induction of adenofibrosis and hepatomas in the liver of BALB/cJ mice by polychlorinated biphenyls (Aroclor 1254). J. Natl. Cancer Inst. 53(2): 547-552.

CREF - Kimbrough, R.D., R.A. Squire, R.E. Linder, J.D. Strandberg, R.J. Montali and V.W. Burse. 1975. Induction of liver tumors in Sherman strain female rats by polychlorinated biphenyl Aroclor 1260. J. Natl. Cancer Inst. 55(6): 1453- 1459.

CREF - Kimura, N.T. and T. Baba. 1973. Neoplastic changes in the rat liver induced by polychlorinated biphenyl. Gann. 64: 105-108.

CREF - NCI (National Cancer Institute). 1978. Bioassay of Aroclor (trademark) 1254 for possible carcinogenicity. CAS No. 27323-18-8. NCI Carcinogenesis Tech. Rep. Ser. No. 38.

CREF - NIOSH (National Institute for Occupational Safety and Health). 1977. Criteria for a Recommended Standard . . . Occupational Exposure to Polychlorinated Biphenyls (PCBs). U.S. DHEW, PHS, CDC, Rockville, Md. Publ. No. 77-225.

CREF - Norback, D.H. and R.H. Weltman. 1985. Polychlorinated biphenyl induction of hepatocellular carcinoma in the Sprague-Dawley rat. Environ. Health Perspect. 60: 97-105.

CREF - Peakall, D.B., J.L. Lincer and S.E. Bloom. 1972. Embryonic mortality and chromosomal alterations caused by Aroclor 1254 in ring doves. Environ. Health Perspect. 1: 103-104.

CREF - Schaeffer, E., H. Greim and W. Goessner. 1984. Pathology of chronic polychlorinated biphenyl (PCB) feeding in rats. Toxicol. Appl. Pharmacol. 75: 278-288.

CREF - Schoeny, R. 1982. Mutagenicity testing of chlorinated biphenyls and chlorinated dibenzofurans. Mutat. Res. 101: 45-56.

CREF - Schoeny, R.S., C.C. Smith and J.C. Loper. 1979. Non-mutagenicity for Salmonella of the chlorinated hydrocarbons Aroclor 1254, 1,2,4-trichlorobenzene, mirex and kepone. Mutat. Res. 68: 125-132.

CREF - U.S. EPA. 1988. Drinking Water Criteria Document for Polychlorinated Biphenyls (PCBs). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.

CREF - Wyndham, C., J. Devenish and S. Safe. 1976. The in vitro metabolism, macromolecular binding and bacterial mutagenicity of 4-chlorobiphenyl, a model PCB substrate. Res. Commun. Chem. Pathol. Pharmacol. 15: 563-570.

HAREF- None

Toluene

1 - IRIS
NAME - Toluene
RN - 108-88-3
IRSN - 115
DATE - 940406
UPDT - 04/06/94, 1 field
STAT - Oral RfD Assessment (RDO) on-line 04/01/94
STAT - Inhalation RfC Assessment (RDI) on-line 08/01/92
STAT - Carcinogenicity Assessment (CAR) on-line 02/01/94
STAT - Drinking Water Health Advisories (DWHA) on-line 09/01/90
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 04/01/92
IRH - 03/01/88 RDO Text revised
IRH - 09/07/88 CAR Carcinogen summary on-line
IRH - 02/01/89 CARDR Secondary contact's phone number corrected
IRH - 07/01/89 RDI Inhalation RfD now under review
IRH - 03/01/90 REFS Bibliography on-line
IRH - 04/01/90 CREF Combs et al., 1973 citation corrected
IRH - 06/01/90 CAA Area code for EPA contact corrected
IRH - 06/01/90 RCRA EPA contact changed
IRH - 07/01/90 RDO Withdrawn; new RfD verified (in preparation)
IRH - 07/01/90 OREF Oral RfD references withdrawn
IRH - 08/01/90 RDO Oral RfD summary replaced; RfD changed
IRH - 08/01/90 CAR Text edited
IRH - 08/01/90 OREF Oral RfD references revised
IRH - 09/01/90 HADV Health Advisory on-line
IRH - 09/01/90 HAREF Health Advisory references added
IRH - 08/01/91 CREF Litton Bionetics, Inc., 1981 reference title clarified
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 04/01/92 CAA CAA regulatory action withdrawn
IRH - 08/01/92 RDI Inhalation RfC on-line
IRH - 08/01/92 IREF Inhalation references on-line
IRH - 02/01/94 CARDR Secondary contact's phone number changed
IRH - 04/01/94 RDO Primary contact changed
RLEN - 73299
SY - ANTISAL 1a
SY - BENZENE, METHYL
SY - METHACIDE
SY - METHYL-BENZENE
SY - METHYLBENZOL
SY - NCI-C07272
SY - PHENYL-METHANE
SY - RCRA WASTE NUMBER U220
SY - TOLUEEN
SY - TOLUEN
SY - Toluene
SY - TOLUOL
SY - TOLUOLO
SY - TOLU-SOL
SY - UN 1294

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Changes in liver and kidney weights	NOAEL: 312 mg/kg converted to 223 mg/kg/day	1000	1	2E-1 mg/kg/day
13-Week Rat Gavage Study NTP, 1989	LOAEL: 625 mg/kg converted to 446 mg/kg/day			

*Conversion Factors: Dose adjusted for gavage schedule of 5 days/week.

o ORAL RFD STUDIES :

NTP (National Toxicology Program). 1989. Toxicology and Carcinogenesis Studies of toluene in F344/N rats and B6C3F1 mice. Technical Report Series No. 371. Research Triangle Park, NC.

The oral toxicity of toluene was investigated in this subchronic gavage study in F344 rats. Groups of 10 rats/sex/group were administered toluene in corn oil at dosage levels of 0, 312, 625, 1250, 2500, or 5000 mg/kg for 5 days/week for 13 weeks. All animals receiving 5000 mg/kg died within the first week. One female and 8 males in the 2500 mg/kg group died, but 2 of these were due to gavage errors. No deaths occurred at lower doses. Several toxic effects were noted at doses greater than or equal to 2500 mg/kg, including prostration, hypoactivity, ataxia, piloerection, lacrimation, excessive salivation, and body tremors. No signs of biologic significance were seen in groups receiving less than or equal to 1250 mg/kg. The only significant change in body weight was a decrease ($p<0.05$) for males in the 2500 mg/kg group. There were no toxicologically significant changes in hematology or urinalysis for any group of animals. Biochemical changes, including a significant increase ($p<0.05$) in SGOT in 2500 males and a dose-related increase in cholinesterase in females receiving 2500 and 5000 mg/kg, were not considered to be biologically significant. There were several pathologic findings and organ weight changes in the liver, kidney, brain, and urinary bladder. In males, absolute and relative weights of both the liver and kidney were significantly increased ($p<0.05$) at doses greater than or equal to 625 mg/kg. In females, absolute and relative weights of the liver, kidney, and heart were all significantly increased at doses greater than or equal to 1250 mg/kg ($p<0.01$ for all comparisons except $p<0.05$ for absolute kidney and heart weights at 1250 mg/kg). Histopathologic lesions in the liver consisted of hepatocellular hypertrophy, occurring at greater than or equal to 2500 mg/kg. Nephrosis was observed in rats that died, and damage to the tubular epithelia of the kidney occurred in terminally sacrificed rats. Histopathologic changes were also noted in the brain and urinary bladder. In the brain, mineralized foci and necrosis of neuronal cells were observed in males and females at 2500 mg/kg and males at 1250 mg/kg. In the bladder, hemorrhage of the muscularis was seen in males and females at 5000 mg/kg and males at 2500 mg/kg. The NOAEL for this study is 312 mg/kg/day based on liver and kidney weight changes in male rats at 625 mg/kg. The toxicologic significance of these organ weight changes is strengthened by the occurrence of histopathologic changes in both the liver and kidney at higher doses. Because the exposure was for 5 days/week, this dose is converted to 312 x 5/7 = 223 mg/kg/day. The LOAEL is 625 mg/kg, which is 446 mg/kg/day when converted.

NTP (1989) also conducted a 13-week gavage study in B6C3F1 mice, following the same regimen described above. All mice receiving 5000 mg/kg died and 8/20 receiving 2500 mg/kg also died. Signs of toxicity seen in animals receiving greater than or equal to 2500 mg/kg included subconvulsive jerking, prostration, impaired grasping reflex, bradypnea, hypothermia, ataxia, and hypoactivity. By week 13, the mean body weight of 2500 mg/kg males was significantly ($p<0.05$) lower than controls. No other significant changes were reported for any group, including macroscopic observation, organ weight means, or clinical pathology parameters. The NOAEL for mice in this study was 1250 mg/kg.

The subchronic study by Wolf et al. (1956) is supportive of the NTP studies. Groups of 10 female Wistar rats were administered gavage doses of 0, 118, 354, or 590 mg/kg toluene dissolved in olive oil. A total of 138 doses were administered over 193 days, resulting in average doses of approximately 0, 84, 253, or 422 mg/kg/day. Hematologic, behavioral, gross and histopathologic examinations were conducted with no toxic effects being reported at any dose. Therefore, the highest dose of 422 mg/kg/day is considered to be the NOAEL for this study. However, this study is not used as the basis for the RfD because the LOAEL of 446 mg/kg/day identified by NTP (1989) is too close to the NOAEL identified by Wolf et al. (1956). Also, the NTP study indicated that male rats are more sensitive to toluene and the Wolf study utilized only female rats.

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o ORAL RFD UNCERTAINTY :

UF -- An uncertainty factor of 1000 was applied to account for inter- and intraspecies extrapolations, for subchronic-to-chronic extrapolation and for limited reproductive and developmental toxicity data.

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o ORAL RFD MODIFYING FACTOR :

MF -- None

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o ORAL RFD COMMENTS :

Kostas and Hotchin (1981) exposed NYLAR mice pre- and post-natally to toluene provided in the drinking water at concentrations of 0, 16, 80, or 400 ppm. Effects were noted in all dosed groups on rotorod performance, measured at 45 to 55 days of age, but there was an inverse dose-response relationship. No effects of toluene exposure were seen on maternal fluid consumption, offspring mortality rate, development of eye or ear openings, or surface-righting response. This study is not suitable for use in risk assessment because only 6 to 9 pregnancies/dose group were obtained, and because the dose-response relationship was inverse.

In an abstract providing limited information, Nawrot and Staples (1979) reported an increase in embryonic lethality in mice exposed to toluene from days 6 to 15 of gestation. Pregnant CD-1 dams were administered 0.3, 0.5, or 1.0 mL/kg bw, 3 times/day (equivalent to approximately 780, 1300, or 2600 mg/kg/day). Maternal toxicity was not observed at any dose level, but toluene was shown to be teratogenic at the high dose and embryolethal at the low dose.

These levels are higher than the NOAEL demonstrated by the NTP (1989) study.

Several subchronic and chronic inhalation studies have been performed on toluene but are not considered to be suitable for deriving an oral RfD. These studies are summarized nicely in the introduction to the 2-year inhalation bioassay by NTP, 1989. The studies identify the following potential target organs: kidney (male rat); hematologic effects (mice); central nervous system (rats, mice, primates); developmental toxicity (rats, rabbits). It is beyond the scope of this oral RfD summary sheet to describe each of these studies, but the two chronic (2 year) inhalation studies are summarized briefly below.

In a 2-year inhalation study by NTP (1989), F344 rats (60/sex/group) were exposed to 0, 600, or 1200 ppm toluene and B6C3F1 mice (60/sex/group) to 0, 120, 600, or 1200 ppm toluene for 6.5 hours/day, 5 days/week. Ten animals/group (except male mice) were removed at 15 months for toxicologic evaluation. At 15 months, there was an increased incidence and severity of nonneoplastic lesions of the nasal cavity of exposed rats. Minimal hyperplasia of the bronchial epithelium was seen in 4/10 female mice at 1200 ppm. There were no significant differences in survival among any group of animals during the 2-year study. Mean body weights were generally similar for all groups throughout the study. Nephropathy was seen in almost all rats with the severity somewhat increased in exposed rats. There were also effects on the olfactory and respiratory epithelia of exposed rats. No biologically important lesions were seen in any groups of mice. There was no evidence of carcinogenicity for any group of animals in this study.

A chronic inhalation study in rats performed by CIIT (1980) failed to produce an adverse effect. Groups of 40 F344 rats/sex were exposed to 30, 100, or 300 ppm toluene for 6 hours/day, 5 days/week for 24 months. An unexposed group of 120 rats/sex served as a control. Clinical chemistry, hematology, and urinalysis testing were conducted at 18 and 24 months. All parameters measured at the termination of the study were normal except for a dose-related reduction in hematocrit values in females exposed to 100 and 300 ppm toluene. The highest dose of 300 ppm was considered to be a NOAEL.

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o ORAL RFD CONFIDENCE :

Study: High
Data Base: Medium
RfD: Medium

Confidence in the principal study is high because a sufficient number of animals/sex were tested in each of six dose groups (including vehicle controls) and many parameters were studied. The same protocol was tested in both mice and rats, with rats being identified as the more sensitive species. The data base is rated medium because it is supported by a 6-month oral study.

It is not higher than medium because there is no reproductive study. Also, the oral studies are all subchronic, with the critical study being only 13 weeks in duration. Medium confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

Source Document -- This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation -- None

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o REVIEW DATES : 05/20/85, 08/05/85, 08/05/86, 05/17/90, 06/20/90

o VERIFICATION DATE : 06/20/90

o EPA CONTACTS :

Harlal Choudhury / OHEA -- (513)569-7553

Krishan Khanna / OST -- (202)260-7588

RDI -

o INHALATION RFD SUMMARY :

Critical Effect	Exposures*	UF	MF	RfC
Neurological effects	NOAEL: None	300	1	4E-1 mg/cu.m
Occupational Study	LOAEL: 332 mg/cu.m (88 ppm)			
	LOAEL(ADJ): 119 mg/cu.m			
Foo et al., 1990	LOAEL(HEC): 119 mg/cu.m			
Degeneration of nasal epithelium	NOAEL: None			
	LOAEL: 2261 mg/cu.m (600 ppm)			
2-Year Rat Chronic	LOAEL(ADJ): 437 mg/cu.m			
Inhalation Study	LOAEL(HEC): 79 mg/cu.m			
NTP, 1990				

*Conversion Factors: MW = 92.15.

Foo et al., 1990: Assuming 25 C and 760 mmHg, LOAEL (mg/cu.m) = 88 ppm x 92.15/24.45 = 332 mg/cu.m. This is an extrarespiratory effect of a soluble vapor. The LOAEL is based on an 8-hour TWA occupational exposure. MVho = 10 cu.m/day, MVh = 20 cu.m/day. LOAEL(HEC) = LOAEL(ADJ) = 332 x MVho/MVh x 5 days/7 days = 119 mg/cu.m.

NTP, 1990: Assuming 25 C and 760 mmHg, LOAEL (mg/cu.m) = 600 ppm x 92.15/24.45 = 2261 mg/cu.m. LOAEL(ADJ) = LOAEL (mg/cu.m) x 6.5 hours/24 hours x 5 days/7 days = 437 mg/cu.m. The LOAEL(HEC) was calculated for a gas:respiratory effect in the extrathoracic region. MVa = 0.24 cu.m/day, MVh = 20 cu.m/day, Sa (ET) = 11.6 sq.cm, Sh (ET) = 177 sq.cm. RGDR = (MVa/Sa) / (MVh/Sh) = 0.18. LOAEL(HEC) = 437 x RGDR = 79 mg/cu.m.

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o INHALATION RFD STUDIES :

Foo, S., J. Jeyaratnam and D. Koh. 1990. Chronic neurobehavioral effects of toluene. Br. J. Ind. Med. 47(7): 480-484.

NTP (National Toxicology Program). 1990. Toxicology and carcinogenesis studies of toluene in F344/N rats and B6C3F1 mice (inhalation studies). NTP-TR-371. 253 p.

In humans, toluene is a known respiratory irritant with central nervous system (CNS) effects. Because available studies could not provide subthreshold (NOAEL) concentrations for either of these effects, the LOAELs for both effects need to be considered in developing the RfC. Consequently, the study of Foo et al. (1990) was used for the CNS effects, and that of the National Toxicology Program (NTP, 1990) for the irritant effects. Because the CNS effect was judged to be a more severe and relevant endpoint, the LOAEL for this effect was used for deriving the RfC. Further, this effect is supported by a number of other occupational studies that show effects around 100 ppm.

Foo et al. (1990) conducted a cross-sectional study involving 30 exposed female workers employed at an electronic assembly plant where toluene was emitted from glue. Toluene levels reported in the study were from personal sample monitoring and reported as an 8-hour TWA, although the number of samples taken and the actual sampling period were not given. No historical exposure values were given. Co-exposure to other solvents was not addressed in the study. The exposed and control cohorts were matched for age, ethnicity, and use of medications. Members of these cohorts did not use alcohol and were nonsmokers. Medical histories were taken to eliminate any histories of central or peripheral nervous system disorders. The average number of years (+/- SD) worked by the exposed population was 5.7 +/- 3.2 and by the controls was 2.5 +/- 2.7. Exposed workers breathed toluene air levels of 88 ppm (332 mg/cu.m) as a TWA and control workers 13 ppm (49 mg/cu.m) (TWA); both of which are averages of the individual personal samples. A battery of eight neurobehavioral tests were administered to all exposed and control workers. The tests were performed midweek, before the workers reported to their stations for the day. Group means revealed statistically significant differences in 6/8 tests; all tests showed that the exposed workers performed poorly compared with the control cohort. When individual test results were linearly regressed against personal exposure concentrations, poor concentration-response relationships resulted for the six tests, with correlation coefficients ranging from 0.44 to 0.30. Irritation effects were not evaluated in this study, and no clinical signs or symptoms were reported. The paucity of exposure information, coupled with the small size of the cohort, limits the interpretation of this study, although the results were essentially confirmed in a clinical study in which the toluene concentrations were carefully controlled (Echeverria et al., 1989) at levels bracketing 88 ppm. Although the data in Echeverria et al. (1989) were generated from short-term exposures (3-7 hours over a period of 142 days), the results may be considered relevant to longer-term exposures as several studies indicate the absence of a duration-response relationship in toluene-induced symptomatology.

Fornazzari et al. (1983) noted the absence of a duration-effect relationship among toluene abusers when they were segregated into neurologically impaired vs. unimpaired ($p = 0.65$). The human studies of Iregren (1982), Cherry et al. (1985), Baelum et al. (1985), and the principal study of Foo et al. (1990) all report this lack of a duration-response relationship and confirm the occurrence of CNS effects. Foo et al. (1990) indicate a LOAEL of 88 ppm toluene (332 mg/cu.m) for neurobehavioral changes from chronic exposure to toluene.

In a 2-year bioassay. Fischer 344 rats (60/sex/group) were exposed to 0, 600, or 1200 ppm (0, 2261, or 4523 mg/cu.m, respectively) toluene vapors, 6.5 hours/day, 5 days/week (duration-adjusted to 0, 437, and 875 mg/cu.m, respectively) for 103 weeks (NTP, 1990). To generate toluene vapor, the liquid material was heated, and the vapor diluted with nitrogen and mixed with the chamber ventilation air. An interim sacrifice was carried out at 15 months on control and 1200-ppm groups (10/sex/group) to conduct hematology and histopathology of the brain, liver, and kidney. Body weights were measured throughout the study. Gross necropsy and micropathology examinations were performed at the end of the study on all major organs including the nasal passage tissues (three sections), lungs, and mainstem bronchi. Mean body weights in both exposed groups were not different from controls for either sex. No exposure-related clinical signs were reported, and survival rate was similar for all groups. At the interim sacrifice, there was a mild-to-moderate degeneration in the olfactory and respiratory epithelium of the nasal cavity in 39/40 rats of the 600- and 1200-ppm groups compared with 7/20 controls. At the end of 2 years, there was a significant ($p<0.05$) increase in the incidence of erosion of the olfactory epithelium (males: 0/50, 3/50, and 8/49; females: 2/49, 11/50, and 10/50; at 0, 600, and 1200 ppm, respectively) and of degeneration of the respiratory epithelium (males: 15/50, 37/50, and 31/49; females: 29/49, 45/50, and 39/50; at 0, 600, and 1200 ppm, respectively) in the exposed animals. The females exposed to 600 and 1200 ppm also exhibited a significant increase in inflammation of the nasal mucosa (27/49, 42/50, and 41/50 at 0, 600, and 1200 ppm, respectively) and respiratory metaplasia of the olfactory epithelium (0/49, 2/50, and 6/50 at 0, 600, and 1200 ppm, respectively). A LOAEL of 600 ppm toluene was determined for the concentration-dependent increase in erosion of the olfactory epithelium in male rats and the degeneration of the respiratory epithelium in both sexes. No NOAEL could be derived from this study.

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o INHALATION RFD UNCERTAINTY :

UF -- An uncertainty factor of 10 is used to account for intraspecies variability and another factor of 10 for the use of a LOAEL. An additional factor of 3 is applied for data base deficiencies, including the lack of data and well-characterized laboratory animal exposures evaluating neurotoxicity and respiratory irritation.

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o INHALATION RFD MODIFYING :

MF -- None
FACTOR

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o INHALATION RFD COMMENTS :

Toluene-induced neurotoxicity has been documented in humans over a broad spectrum of severity that correlates well with concentration. Numerous case studies on chronic toluene abusers [repeatedly exposed to greater than 30,000 ppm (113,000 mg/cu.m)] have demonstrated functional deficits of the CNS accompanied by abnormal morphology of cerebellar and cortical areas of the brain. Under acute exposure conditions [short exposures to greater than 10,000 ppm (37,690 mg/cu.m)], toluene produces CNS narcosis [American Conference of Governmental Industrial Hygienists (ACGIH), 1991]. Lower concentrations, i.e., 800-400 ppm (3015-1508 mg/cu.m), have been associated

with worker complaints of CNS-related effects (ACGIH, 1991). Clinical studies using controlled exposure to toluene have demonstrated concentration-related occurrence of complaints such as drowsiness, ataxia, visual impairment, and headache. A number of occupational studies indicate that these same effects are present in exposed worker populations at concentrations lower than 400 ppm (1508 mg/cu.m) although deficiencies in most of these studies preclude confirming this finding unequivocally. Descriptions of a number of these studies follow. The preponderence of the literature showing CNS effects and the well-known proclivity for solvents to affect CNS processes in humans leave little doubt that the brain is a principal target organ for toluene toxicity in humans.

In cases of inhalation abuse of toluene, Rosenberg et al. (1988) demonstrated diffuse cerebral, cerebellar, and brainstem atrophy in 3 of 11 toluene abusers who also had neurological abnormalities. Filley et al. (1990) were able to correlate neuropsychological impairment with the degree of white matter abnormality ($p<0.01$). Cerebellar and cortical functions were classified as impaired in 15/24 individuals who had abused toluene daily (425 +/- 366 mg/day) for extended periods (6.3 +/- 3.9 years) (Fornazzari et al., 1983). In a limited case study, Metrick and Brenner (1982) demonstrated brainstem atrophy through computerized tomographic scans and abnormal brainstem auditory-evoked potentials in 2/2 chronic toluene abusers (12-16 years of admitted, continuous abuse). These studies confirm the occurrence of severe CNS damage in response to highly abusive concentrations of toluene.

Several studies that have investigated the occurrence of neurotoxicity at lesser concentrations, such as occupational situations, have not demonstrated significant neurological or other effects. Hanninen et al. (1987) performed a battery of 11 psychological tests on 43 printing workers who had been occupationally exposed to approximately 117 ppm (441 mg/cu.m) toluene for an average of 22 years and found only mildly adverse effects in 2/11 tests. The control and exposed cohorts in this study were, however, mismatched in several areas, most notably alcohol use. Iregren (1982) examined the psychological performance of 38 printers who had been occupationally exposed to 50-150 ppm (188-565 mg/cu.m) toluene for an average of 16.3 years (range 3-32 years). No effects were seen, although the cohorts in this study were apparently matched only by age. In a cohort study, Cherry et al. (1985) attempted to better match the control and exposed cohorts and considered alcohol use. Although no differences between the cohorts were statistically significant, the exposed workers performed worse than the nonexposed workers on 10/13 psychological tests. The 52 workers in this study were not, however, rigorously matched, and the concentrations listed in the study ranged up to greater than 500 ppm (1884 mg/cu.m). The cohorts in the study of Foo et al. (1990) were well matched for a number of confounders, including alcohol use; and statistically significant psychological effects were seen.

In the occupational study conducted by Yin et al. (1987), 94 solvent workers (38 men and 56 women; average employment duration, 6.8 years) and 138 controls (48 men and 90 women) were examined for exposure using diffusion dosimeters, subjective symptoms by questionnaire, hematology, and urinalysis. Exposure concentration (7-hour mean TWA) in the workers was estimated at 42.8 ppm (161 mg/cu.m) toluene with a maximum measurement of 123 ppm (464 mg/cu.m). Workers were co-exposed to 1.3 ppm benzene. No exposure-related effects were noted in any of the biochemical tests examined. In considering the prevalence

of subjective symptoms (sore throat, headaches, and dizziness) workers were subgrouped into low (6-39 ppm, n = 28) and high (40-123 ppm, n = 29) categories. Although the prevalence of subjective symptoms was significantly higher in the exposed workers compared with the control cohort ($p<0.01$), a concentration-response relationship was not discernable among the groups. No other treatment-related effects were reported. The study was limited because the exposed and unexposed groups were not matched to control for confounding effects (e.g., age, smoking, alcohol consumption, exposure duration). Based on these results, exposure to an average of approximately 42.8 ppm toluene produced no biochemical abnormalities, although neither respiratory irritation nor psychological performance was directly evaluated in these workers.

In the occupational study by Lee et al. (1988), prevalence of subjective symptoms was categorized with respect to exposure levels. The study population (193 women and 65 controls) completed a questionnaire. The exposures were reported as 8-hour TWAs, and workers were grouped in exposure categories of nonexposed, 1-50 ppm, 51-100 ppm, 101-150 ppm, and more than 151 ppm (duration of exposures was not reported). A concentration-dependent increase in prevalence was reported for 25/67 symptoms with increases in complaints over controls occurring at around 100 ppm (348 mg/cu.m). Similar to the Yin et al. study (1987) reported above, symptomatology included headaches, sore throats, and dizziness. Although an effect level in humans of around 100 ppm is indicated by this study, no objective measures of toxicity were examined.

A number of acute human studies have focused on toluene effects. In general, these studies corroborate subjective CNS effects such as headaches and dizziness reported in other longer-term occupational studies (Yin et al., 1987; Lee et al., 1988) and also document irritation effects. The study of Echeverria et al. (1989) correlates the occurrence of these subjective effects with substantial neurological symptoms.

Forty-two college students (21 female and 21 male) were exposed to 0, 74 ppm (279 mg/cu.m), or 151 ppm (569 mg/cu.m) toluene for 7 hours over 3 days (Echeverria et al., 1989). This exposure sequence was repeated for a total of 42 exposures over a 3-month period. The odor of toluene was masked. A battery of performance tests was administered to each participant prior to starting the exposures and again at 4 and 7 hours during the exposure; the initial test served as a control for those tests performed during the exposure. A 5-10% decrement in performance was considered significant if consistent with a linear trend. Test results for visual perception differed from control values for both exposure levels. Results of a manual dexterity test differed from control values at the higher but not the lower exposure level. Psychomotor test results were unaffected by toluene exposure. Subjective symptomatology increased with exposure with increasing numbers of complaints of eye irritation, headache, and somnolence. A NOAEL of 74 ppm (279 mg/cu.m) is indicated for these results. The duration-adjusted value is 122 mg/cu.m for these acute effects.

Andersen et al. (1983) exposed 16 subjects (average age of 24 years) to 0, 10, 40, or 100 ppm (0, 38, 151, or 377 mg/cu.m) toluene for 6 hours on each of 4 consecutive days. Individuals were tested for nasal mucous flow, lung function, subjective response, and psychometric performance. At 100 ppm, irritation was experienced in the eyes and nose, but no effect on nasal mucous

flow or lung function was observed. The subjects frequently reported headaches, dizziness, and a feeling of intoxication. These effects were not reported by the 10- or 40-ppm exposure groups. No effects were seen in performance tests. This study indicates an effect level of 100 ppm, and a NOAEL of 40 ppm (151 mg/cu.m).

The acute study by Baelum et al. (1990) evaluated 32 males and 39 females exposed to 0 or 100 ppm (0 or 377 mg/cu.m), or to varying exposures of 50-300 ppm (188-1131 mg/cu.m) (TWA = 102 ppm), for 7 hours. Volunteers exercised on an ergometer cycle for 3 periods of 15 minutes each during the exposure. No significant differences were found in the performances between the exposed and control groups in a battery of tests for performance, visual attention, and reaction times. Exposed subjects reported an increase over nonexposed subjects ($p<0.1$) in nose and lower respiratory irritation, feelings of intoxication, dizziness, increased coughing, and headaches. Differences were not noted between the group exposed to a constant level (100 ppm) and the group exposed to the same TWA, but with peaks of up to 300 ppm.

Baelum et al. (1985) investigated the effects of a 6.5-hour toluene exposure to 43 printers with a long-term occupational exposure to a mixture of solvents including toluene and 43 controls with no history of exposure to solvents or other chemicals. The duration of employment for the workers ranged from 9-25 years. Each individual was exposed only once to either 0 or 100 ppm (0 or 377 mg/cu.m) toluene during a 6.5-hour exposure period, preceded by a 1-hour acclimatization period. These subjects were then subgrouped into printers exposed to toluene (n = 20), printers exposed to air (n = 23), controls exposed to toluene (n = 21), and controls exposed to air (n = 22). All subjects carried out a battery of tests for psychometric performance, visual perception, and vigilance evaluation. Both printers and controls complained of nasal and eye irritation, unacceptable air quality, and unacceptable odor level during the toluene exposure. Signs of neurotoxicity, including moderate fatigue, sleepiness, headaches, and a feeling of intoxication, were likewise similarly reported for both groups. A significant decrease in performance was found for the pegboard visual motor function test in the exposed printers, but not in the controls exposed to 100 ppm toluene. A decrease in psychometric performance, primarily in visual perception and accuracy, was observed in toluene-exposed individuals. Acute exposure to toluene resulted in a lower performance in 4/10 tests conducted, 3 of these 4 evaluated visual perception. The most profound difference between subjects exposed to 100 ppm toluene and those exposed to clean air was observed in the color discrimination test; this difference was seen in both exposed vs. nonexposed printers and exposed vs. nonexposed controls. This study indicates that little tolerance develops to the irritative and central effects in humans exposed to toluene and that 100 ppm (377 mg/cu.m) is the effect level for these symptoms.

Von Oettingen et al. (1942) exposed 3 humans to 100 or 200 ppm (377 or 754 mg/cu.m) toluene vapors for 8 hours. At 200 ppm, the subjects experienced muscular weakness, confusion, impaired coordination, and dilated pupils, with after-effects including fatigue, general confusion, and moderate insomnia. In 1 subject exposed to 100 ppm toluene, moderate fatigue, sleepiness, and headaches were reported.

Hepatotoxicity has also been examined as a toxicologic endpoint of toluene

exposure in humans. Fornazzari et al. (1983) described moderate elevation of serum AP levels in 13/24 (and SGOT in 7/24) toluene abusers upon admission to a clinic. These elevated levels were normal after 2 weeks of solvent abstinence, although the accompanying CNS effects were only minimally improved. In a cross-sectional study of 181 printing workers in which toluene exposures were less than 200 mg/cu.m, no adverse effects were apparent as judged from serum liver enzymes (Boewer et al., 1988). In another cross-sectional occupational study conducted by Guzelian et al. (1988) that involved 289 printing factory employees, 8 workers were found who had an increase described as "marked" in the ratio of ALT/AST enzyme serum activity. Biopsies revealed mild pericentral fatty livers in each of the eight cases. Based on environmental data (probably area monitors) the levels of toluene to which these workers were exposed was less than 200 mg/cu.m., 2-8 hours/day.

Fischer 344 rats (120/sex/group) inhaled 0, 30, 100, or 300 ppm (0, 113, 377, or 1130 mg/cu.m, respectively) toluene (99.9% purity), 6 hours/day, 5 days/week (duration-adjusted to 0, 20, 67, or 202 mg/cu.m, respectively) for 106 weeks (CIIT, 1980; Gibson and Hardisty, 1983). Vapor, generated by bubbling clean air through toluene, was passed through the air supply duct and mixed with air by turbulent flow to produce the desired concentration. Hematology, blood chemistry, and urinalysis were conducted in all groups at 6 (5/sex), 17 (5/sex), 18 (10-20/sex), and 24 months (10/sex). Histopathology was evaluated only in the control and 300-ppm groups at 6 (5/sex), 12 (5/sex), and 18 months (20/sex). At 24 months, histopathological examinations were conducted in organs of all surviving animals, including the respiratory system and sections through the nasal turbinates (number not indicated). No treatment-related non-neoplastic effects were observed in the exposed animals. Although the male rats exposed to 300 ppm had a significant increase in body weight compared to controls, no concentration-response was evident. At the end of the exposure period, the female rats exposed to 100 or 300 ppm exhibited a slight but significant reduction in hematocrit; an increase in the mean corpuscular hemoglobin concentration was also noted but only in the females exposed to 300 ppm. The highest concentration examined in this study, 300 ppm, is designated as a NOAEL for toxicity remote from the respiratory tract in rats. CIIT (1980) reported that the technical and raw data were not audited by their quality assurance group during the study period, although CIIT did conduct a quality assessment procedure to review the data. The available pathology reports containing these data indicate that at least the lower respiratory tract was examined. Communication with the testing sponsor has provided information indicating that only one section was examined from the nasal cavity of these test animals. It is not clear whether this single section would have been sufficient to elucidate the areas of lesions noted in the NTP (1990) study. Consequently, the designation of the 300-ppm exposure level as a NOAEL for respiratory lesions (see NTP,1990) is problematic.

Fischer 344/N rats (10/sex/group) were exposed to toluene vapors at 0, 100, 625, 1250, 2500, and 3000 ppm (0, 377, 2355, 4711, 9422, and 11,307 mg/cu.m, respectively) 6.5 hours/day, 5 days/week (duration-adjusted to 0, 73, 455, 911, 1823, and 2187 mg/cu.m, respectively) for 15 weeks (NTP, 1990). Organ weights were measured and histological examinations were performed only on controls, 2500- and 3000-ppm groups, and animals that died before the end of the study. Eight of 10 males exposed to 3000 ppm died, all during the 2nd exposure week. No females died at any exposure level. Compared to the controls, final body weights were 15 and 25% lower in the males and 15 and 14%

lower in the females of the 2500- and 3000-ppm groups, respectively. There was a concentration-related increase in the relative liver weight, significant at 1250, 2500, and 3000 ppm in males and at 2500 and 3000 ppm in females. The relative weights of the heart, lung, kidney, and right testis were also significantly elevated in the 2500- and 3000-ppm animals compared to those of the controls, although no histopathology was observed in any exposure group. Toxic effects noted in a concurrently conducted gavage study (urinary bladder hemorrhages in the two highest exposure groups) were not noted in this subchronic inhalation study. A LOAEL of 2500 ppm [LOAEL(HEC) = 1823 mg/cu.m] was determined for the decrease in body weight gain in both males and females, and the NOAEL for this effect was 1250 ppm [NOAEL(HEC) = 911 mg/cu.m].

Toluene has been suspected to cause congenital defects in infants born to mothers who were exposed to or who abused toluene during pregnancy. In a case report study, Hersh et al. (1985) describes clinical and morphometric characteristics common to 3 children whose mothers had abused toluene (but apparently not alcohol or any other substance) for a period of 4-5 years including during their pregnancies with the affected children. Clinical findings common to these three children included microcephaly, CNS dysfunction, attention deficits, and developmental delay/mental deficiency. Phenotypic similarities included a small midface, deep-set eyes, micrognathia (smallness of the jaws), and blunting of the fingertips. A retrospective cohort study was conducted by McDonald et al. (1987) who examined the history of exposure to chemicals of 301 women who had recently given birth to an infant with an important congenital defect. An identical number of women (referents) who had given birth to normal children were matched with respect to age, employment (hours/week), date of delivery, and educational level. In initial matched-pair analysis, chemical exposure was higher in the cases than in the referents (63 cases:47 referents) due to excess cardiac and miscellaneous defects. In further analysis by chemical categories, only exposure to aromatic solvents showed a clear excess of defects, mostly in the urinary tract. Details of these cases (n = 19) showed that toluene was identified as the solvent in 11 of these cases.

Hudak and Ungvary (1978) exposed three groups of pregnant CFY rats to toluene during different periods of gestation and for different durations of exposure. Two of the groups had their own control group exposed to air only and matched for period and daily duration. The first of these (n = 19) was exposed to 1500 mg/cu.m for 24 hours/day during gestational days 9 to 14. Two dams died during these exposures. No details on the deaths are given but no other maternal toxicity was observed. Fetotoxicity was also in evidence as sternebral alterations (6% vs. 1% in controls), extra ribs (22% vs. 0% in controls), and the presence of fetuses with missing tails (2/213, none observed in 315 controls) were recorded. Under these exposure conditions, 1500 mg/cu.m is a LOAEL for fetotoxicity and a frank effect level (FEL) for maternal toxicity. The second group (n = 14) received this same concentration continuously but on days 1-8 of gestation. Five dams died under these exposure conditions although toxicity parameters of the surviving dams were identical with the controls from the first group (gestational days 9-14). Slight hydrocephaly was noted in 4 fetuses (all from the same litter), and 17% growth retardation was noted vs. 7% in the controls. Thus these exposure conditions are a FEL for maternal toxicity and a LOAEL for fetotoxicity. A third group was exposed to 1000 mg/cu.m for 8 hours/day from the 1st to the 21st day of gestation. No maternal deaths or toxicity occurred. Minor skeletal

retardation was present in the exposed fetuses at a higher incidence rate (25%) than in concurrent controls (0%). These results indicate that 1000 mg/cu.m is a LOAEL for developmental effects under these exposure conditions. This concentration is also a NOAEL for maternal effects. These workers also exposed groups of pregnant CFLP mice (n = 11-15) to either air or 1500 or 500 mg/cu.m toluene continuously during days 6-13 of pregnancy. All mice exposed to the high concentration died within 24 hours of the beginning of exposure. No dams died in the lower exposure group. In this group, the average fetal weight decreased to 0.96 g from the average control weight of 1.07 g, and the percentage of weight-retarded fetuses (less than 0.9 g) increased to 27.6% from 6.5% in the controls. No difference in incidence of skeletal malformations or anomalies was noted between these and control fetuses. For mice, 1500 mg/cu.m is an FEL and 500 mg/cu.m is a mild LOAEL. Since duration adjustment is not performed for developmental effects, this concentration is also the LOAEL(HEC).

B6C3F1 mice (60/sex/group) were exposed to 0, 120, 600, or 1200 ppm (0, 452, 2261, or 4523 mg/cu.m, respectively) toluene 6.5 hours/day, 5 days/week (duration-adjusted to 0, 87, 47, and 875 mg/cu.m, respectively) for 2 years (NTP, 1990). Mean body weights were not significantly different among groups and no treatment-related clinical signs were observed. Deaths (moribund and natural) occurred in all exposure groups but were not related to exposure and were not greater than the control rates. An excess incidence of non-neoplastic inflammatory lesions of the urinary and genital system was observed in all the groups of male mice. At the 15-month interim sacrifice, minimal hyperplasia in the bronchial epithelium was observed in 4/10 females exposed to 1200 ppm. At the end of the study, there was a concentration-dependent increase in the incidence of splenic pigmentation in the exposed males (9/60, 11/60, and 18/59 at 120, 600, and 1200 ppm, respectively) compared to controls (4/60). In the females, the incidence was 37/50, 33/50, 34/49, and 28/47 at 0, 120, 600, and 1200 ppm, respectively. The occurrence of endometrial hyperplasia was present in 14% of the animals exposed to the highest concentration but only in 4% in the low-exposure groups and controls. No differences were noted between the exposed and control mice of either sex in the incidence of degeneration of either the olfactory or respiratory epithelium. No other non-neoplastic lesions were observed in exposed mice. As no adverse effects were noted in this study, the highest concentration, 1200 ppm was designated as a NOAEL in mice for this chronic study [NOAEL(HEC) = 875 mg/cu.m].

Sprague-Dawley rats (15/sex/group) were exposed to cumulative mean exposures of 0, 100, or 1481 ppm (0, 377, or 5653 mg/cu.m) toluene vapors, 6 hours/day, 5 days/week (duration-adjusted to 0, 67, and 1009 mg/cu.m, respectively) for 26 weeks (API, 1981). On weeks 9, 18, and 27, neurohistopathological examinations were conducted in 3-5 rats/sex/group. Hematology, clinical chemistry, and urinalysis parameters were evaluated after 13 and 26 weeks of exposure. Body weights were measured weekly. No significant treatment-related effects were reported. Therefore, a NOAEL of 1481 ppm [NOAEL(HEC) = 1009 mg/cu.m] toluene was determined for systemic effects in rats. The study was limited because there were no other neurohistopathological examinations or organ weight measurements conducted on the animals.

Inhalation exposure to toluene has been shown to result in irreversible high-frequency hearing loss in rats. Pryor et al. (1984) exposed young male

Fischer 344 rats to a variety of exposure concentrations and durations. Hearing loss was evaluated by a behavioral technique (avoidance response elicited to an auditory signal) or brainstem auditory-evoked responses (elicited by tone pips of differing loudness and frequency and detected by subdural scalp electrodes). Hearing loss, as measured by both techniques, was observed after as few as 2 weeks exposure to 1000 ppm toluene for 14 hours/day. Lower concentrations of 700 ppm for 14 hours/day were without effect after 16 weeks of exposure. Intermittent exposure to 3000 ppm for 30 minutes/hour for 8 hours/day caused hearing loss within 2 weeks, whereas a similar exposure schedule for only 4 hours/day was without effect after 9 weeks. These data define a NOAEL for hearing loss in rats of 700 ppm [NOAEL(HEC) = 2638 mg/cu.m]. The duration-adjusted HEC (assumed 5 days/week) would be 14/24 hours x 5/7 days = 1100 mg/cu.m. Although these results clearly document hearing loss in young adult rats, their direct significance to humans remains unclear. Among chronic toluene abusers there is only a single report of adverse effects on hearing; Metrick and Brenner (1982) claimed that the abnormal auditory-evoked potentials recorded in two chronic toluene abusers was evidence of brainstem abnormalities.

Pregnant Wistar rats and hamsters (group size not indicated) inhaled 0 or 800 mg/cu.m toluene vapors 6 hours/day on gestational days 14-20 (rats) or gestational days 6 to 11 (hamsters) (DaSilva et al., 1990). In the exposed rats, there was a significant ($p<0.05$) increase in the number of litters with one or more low birth weight pups (less than 4.9 g), from 10% in the controls to 54% in the exposed dams. A decrease ($p<0.05$) in the number of live pups at birth was also noted in the litters of exposed dams. No evaluation of malformations or anomalies was performed. The neurobehavioral development of the offspring of the exposed rats was assessed using tests of spontaneous alternation, rim escape, and avoidance responses. The only effect noted in the rats, a shortened first trial latency in choosing one side of a maze, was minimal and its significance unclear. No comparable reproductive deficits occurred in the exposed hamsters. The only effect noted in the neurobehavioral tests of the hamster offspring was an equivocal effect in rota-rod performance.

No neurobehavioral effect levels were designated from this study, although it appears that the rat developmental processes are more sensitive than those of the hamster, exhibiting adverse effects at 800 mg/cu.m.

Ungvary and Tatrai (1985) exposed New Zealand rabbits (8-10/group) to 0, 500, or 1000 mg/cu.m toluene, 24 hours/day, on gestational days 7-20, and CFLP mice (15 females/group) to 0, 500, 1000, or 1500 mg/cu.m toluene, also continuously, on gestational days 6-15. The control groups consisted of 115 mice and 60 rabbits. All the female mice exposed to 1500 mg/cu.m died. In the mice exposed to 1000 mg/cu.m, there was an increase in fetuses with retarded weight (29%, level of retardation not indicated) and in fetuses with skeletal retardation (12%) compared to 7% and 5%, respectively, in the controls, which did not differ from the animals exposed to 500 mg/cu.m. Of the 8 pregnant rabbits exposed to 1000 mg/cu.m, 2 died, 4 had spontaneous abortions, and the remaining 2 had total litter resorption. No deaths occurred in the 10 rabbits exposed to 500 mg/cu.m but 1/10 rabbits had a spontaneous abortion (as compared to 0/60 reported for the controls). A NOAEL(HEC) of 500 mg/cu.m toluene was determined for reproductive effects in mice. For rabbits, the 500 mg/cu.m concentration is designated as a LOAEL. These results indicate that pregnant mice may be a sensitive population to the

effects of toluene.

Pregnant Charles River CD-1 mice (15-16 females/group) inhaled filtered air or 200 or 400 ppm (754 and 1508 mg/cu.m) toluene 7 hours/day on gestational days 7-16 (Courtney et al., 1986). The relative liver weight in the exposed dams was reported to be significantly lower in the two exposed groups compared to the controls, although no data were presented. A statistically significant increase in lactate dehydrogenase activity in the brain of the dams exposed to 400 ppm was also reported. The exposed pregnant mice did not exhibit any significant differences in the number of implantation sites, number of live fetuses, fetal deaths, or fetal body weight compared to the control values. A statistically significant increase over controls in the incidence (both per litter and per fetus) of enlarged renal pelves was noted in dams exposed to 200 ppm but not 400 ppm. A statistically significant alteration from controls in the rib profile (percentage of fetuses with 1 or 2 additional/fewer ribs) was reported for fetuses from dams exposed to 400 ppm but not 200 ppm. The toxicological significance of this finding is not clear.

As no clearly significant toxicological effects were observed, the highest concentration used, 400 ppm [NOAEL(HEC) = 1508 mg/cu.m] is designated as a NOAEL for reproductive and developmental effects in mice.

A 2-generation inhalation reproductive study was conducted in CD rats (10-40 males, 20-80 females/group) (API, 1985). Animals were exposed by whole-body inhalation to toluene at 0, 100, 500, or 2000 ppm (0, 377, 1885, or 7538 mg/cu.m, respectively) 6 hours/day, 7 days/week for 80 days and a 15-day mating period. The mated females were then exposed to the same concentrations during days 1-20 of gestation and days 5-20 of lactation. After weaning, the pups in this generation (F1) were exposed 80 times and then randomly mated with members of the same exposure group (2 females/1 male) to produce the second generation (F2). Mean male body weights were slightly reduced (maximum of 10%) in the first 2 weeks of the exposure in the animals exposed to 500 and 2000 ppm, although the size of the reduction was not related to exposure. No differences were observed in male or female fertility indices, length of gestation, mean numbers of viable and nonviable pups at birth, or pup survival indices during lactation. No abnormal histopathology was noted in organs examined. A significant decrease ($p<0.05$) in weight relative to controls was observed in the first generation offspring. The decrease was maintained throughout the lactation period in the pups from dams exposed to the highest exposure and in those from the ancillary group in which females exposed to the 2000 ppm concentration were mated with males having no exposure. No data were available in the report about the F2 generation. Based on the effects on the pups of the first generation (F1), a LOAEL of 2000 ppm [LOAEL(HEC) = 7538 mg/cu.m] is designated, the NOAEL being 500 ppm [NOAEL(HEC) = 1885 mg/cu.m].

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o INHALATION RFD CONFIDENCE : Study -- Medium Data Base -- Medium RfC -- Medium The study of Foo et al. (1990) indicates adverse neurological effects of toluene in a small worker population. These effects are consistent with more severe CNS effects occurring at abusive concentrations of toluene and could not have been confounded by alcohol as the control and exposed populations did not use alcohol. However, the

paucity of exposure information and identification of only a LOAEL is not sufficient to warrant a higher confidence than medium for this study. Other studies indicate that irritation may occur at around the same concentration, 100 ppm (Baelum et al., 1985; Echeverria et al., 1989). In regard to this effect, the NTP (1990) rat chronic inhalation study was well conducted, established the rat as the most sensitive species, examined an adequate number of animals, and performed histopathology on all major organs, including the brain and the respiratory tract. The sensitive endpoint was the concentration-dependent degeneration of the nasal epithelium characterized by the erosion of the olfactory epithelium and degeneration of the respiratory epithelium in male rats. The NTP study is also given medium confidence, however, as it did not establish a NOAEL. Although this data base has a complement of chronic laboratory animal studies, long-term data in humans are not available for either the neurotoxicity or irritation endpoints. The reproductive/developmental studies in three species were not comprehensive in endpoint evaluation but do identify the rabbit as the most sensitive species. The data base is thus given a medium confidence rating. A medium confidence rating for the RfC follows.

o INHALATION RFD SOURCE :

Source Document – This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation – U.S. EPA, 1984, 1985
DOCUMENT

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o REVIEW DATES : 04/21/88, 05/26/88, 02/16/89, 03/21/89, 05/18/89, 08/15/91, 12/11/91
o VERIFICATION DATE : 05/18/89, 12/11/91
o EPA CONTACTS :

Gary L. Foureman / OHEA – (919)541-1183

Annie M. Jarabek / OHEA – (919)541-4847

CAREV-
o CLASSIFICATION : D; not classified
o BASIS FOR CLASSIFICATION : No human data and inadequate animal data. Toluene did not produce positive results in the majority of genotoxic assays.

o HUMAN CARCINOGENICITY DATA :

None.

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o ANIMAL CARCINOGENICITY DATA :

A chronic (106-week) bioassay of toluene in F344 rats of both sexes reported no carcinogenic responses (CIIT, 1980). A total of 960 rats were exposed by inhalation for 6 hours/day, 5 days/week to toluene at 0, 30, 100, or 300 ppm. Groups of 20/sex/dose were sacrificed at 18 months. Gross and microscopic examination of tissues and organs identified no increase in neoplastic tissue or tumor masses among treated rats when compared with controls. The study is considered inadequate because the highest dose administered was well below the MTD for toluene and because of the high incidence of lesions and pathological changes in the control animals.

Several studies have examined the carcinogenicity of toluene following repeated dermal applications. Toluene (dose not reported) applied to shaved interscapular skin of 54 male mice (strains A/He, C3HeB, SWR) throughout their lifetime (3 times weekly) produced no carcinogen1c response (Poel, 1963). One drop of toluene (about 6 mL) applied to the dorsal skin of 20 random-bred albino mice twice weekly for 50 weeks caused no skin papillomas or carcinomas after a 1-year latency period was allowed (Coombs et al., 1973). No increase in the incidence of skin or systemic tumors was demonstrated in male or female mice of three strains (CF, C3H, or CBaH) when toluene was applied to the back of 25 mice of each sex of each strain at 0.05-0.1 mL/mouse, twice weekly for 56 weeks (Doak et al., 1976). One skin papilloma and a single skin carcinoma were reported among a group of 30 mice treated dermally with one drop of 0.2% (w/v) solution toluene twice weekly, administered from droppers delivering 16-20 uL per drop for 72 weeks (Lijinsky and Garcia, 1972). It is not reported whether evaporation of toluene from the skin was prevented during these studies.

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o SUPPORTING DATA :

Toluene was found to be nonmutagenic in reverse mutation assays with S. typhimurium (Mortelmans and Riccio, 1980; Nestmann et al., 1980; Bos et al., 1981; Litton Bionetics, Inc., 1981; Snow et al., 1981) and E. coli (Mortelmans and Riccio, 1980), with and without metabolic activation. Toluene did not induce mitotic gene conversion (Litton Bionetics, Inc., 1981; Mortelmans and Riccio, 1980) or mitotic crossing over (Mortelmans and Riccio, 1980) in S. cerevisiae. Although Litton Bionetics, Inc. (1981) reported that toluene did not cause increased chromosomal aberrations in bone marrow cells, several Russian studies (Dobrokhotov, 1972; Lyapkalo, 1973) report toluene as effective in causing chromosal damage in bone marrow cells of rats. There was no evidence of chromosomal aberrations in blood lymphocytes of workers exposed to toluene only (Maki-Paakkanen et al., 1980; Forni et al., 1971), although a slight increase was noted in workers exposed to toluene and benzene (Forni et al., 1971; Funes-Craviota et al., 1977). This finding is supported by studies of cultured human lymphocytes exposed to toluene in vitro; no elevation of chromosomal aberrations or sister chromatid exchanges was observed (Gerner-Smidt and Friedrich, 1978).

CARO - NO DATA
CARI - NO DATA
CARDR-
o CARCINOGENICITY SOURCE :

Source Document – U.S. EPA, 1987

The values in the 1987 Drinking Water Criteria Document for Toluene have received peer and administrative review.
DOCUMENT

o REVIEW DATES : 09/15/87
o VERIFICATION DATE : 09/15/87
o EPA CONTACTS :

Dharm V. Singh / OHEA – (202)260-5958

Robert E. McGaughy / OHEA – (202)260-5889

HAONE-

One-day HA – 2E+1 mg/L

NOAEL – 21.5 mg/kg/day
UF – 10 (allows for intrahuman variability with the use of a NOAEL from a human study)
Assumptions – 1 L/day water consumption for a 10-kg child

Principal Study – Gamberale and Hultengren, 1972

This study reported that a 20-minute exposure to 100 ppm toluene was a no-effect level when determined by perceptual speed and reaction time tests in human volunteers. At 200 ppm, toluene was noted as clearly causing toxic effects such as incoordination, exhilaration, and prolonged reaction time. These and other data support the selection of 100 ppm (377 mg/cu.m) toluene as the NOAEL in humans exposed for up to 8 hours. Based on the conditions of exposure and an assumed absorption rate of 60%, this level is equivalent to 21.5 mg/kg/day.

HATEN-

No information was found in the available literature that was suitable for determination of a Ten-day HA value. It is, therefore, recommended that the DWEL, adjusted for a 10-kg child (3 mg/L) be used as the Ten-day HA value.

HALTC-

No information was found in the available literature that was suitable for determination of a Longer-term HA value. It is, therefore, recommended that

the DWEL, adjusted for a 10-kg child (3 mg/L) be used as the Longer-term HA value for a child.

HALTA-

No information was found in the available literature that was suitable for determination of a Longer-term HA value. It is, therefore, recommended that the DWEL, adjusted for a 70-kg adult (10 mg/L) be used as the Longer-term HA value for an adult.

HALIF-

Drinking Water Equivalent Level (DWEL) -- 7E-0 mg/L

Assumptions -- 2 L/day water consumption for a 70-kg adult

RfD Verification Date -- 06/20/90

Lifetime HA -- 1E-0 mg/L

Assumptions -- 20% exposure by drinking water

Principal Study -- NTP, 1989 (This study was used in the derivation of the chronic oral RfD; see RDO)

OLEP -

Taste threshold in water is reported as 0.04 and 1 mg/L. Odor threshold in water is reported as 0.04 and 1 mg/L.

ALAB -

Analysis of toluene is by a purge-and-trap gas chromatographic procedure used for the determination of volatile aromatic and unsaturated organic compounds in water.

TREAT-

Treatment options for removing toluene form drinking water sources include air stripping and adsorption onto granular activated carbon.

HADR -
o HEALTH ADVISORY SOURCE :

U.S. EPA. 1990. Final Draft of the Drinking Water Criteria Document for Toluene. Office of Drinking Water, Washington, DC.
DOCUMENT

o HEALTH ADVISORY REVIEW

EPA review of HAs in 1986.

Public review of HAs in 1987.

Science Advisory Board review to be determined.

o EPA DRINKING WATER CONTACT

Krishan Khanna / OST – (202)260-9568

Edward V. Ohanian / OST – (202)260-7571

CAA - NO DATA

WQCHU-

Water and Fish Consumption: 1.43E+4 ug/L

Fish Consumption Only: 4.24E+5 ug/L

Considers technological or economic feasibility? – NO

Discussion – The WQC of 1.43E+4 ug/L is based on consumption of contaminated aquatic organisms and water. A WQC of 4.24E+5 ug/L has also been established based on consumption of contaminated aquatic organisms alone.

Reference – 45 FR 79318 (11/28/80)

EPA Contact – Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute LEC – 1.75E+4 ug/L
Chronic LEC – none

Marine:

Acute LEC – 6.3E+3 ug/L
Chronic LEC – 5.0E+3 ug/L

Considers technological or economic feasibility? – NO

Discussion – The values that are indicated as "LEC" are not criteria, but

are the lowest effect levels found in the literature. LEC's are given when the minimum data required to derive water quality criteria are not available.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value (status) -- 1 mg/L (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion -- EPA has set a MCLG for toluene based on its potential adverse effects reported in a 13-week oral study in rats. The MCLG is based upon a DWEL of 7 mg/L and an assumed drinking water contribution of 20 percent.

Reference -- 54 FR 22062 (05/22/89)

EPA Contact -- Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value -- 1 mg/L (Final, 1991)

Considers technological or economic feasibility? -- YES

Monitoring requirements -- All systems initially monitored for four consecutive quarters; repeat monitoring dependent upon detection, vulnerability status and system size.

Analytical methodology -- Gas chromatography (EPA 502.2, 503.1); gas chromatograpny/mass spectrometry (EPA 524.1, 524.2): PQL= 0.005 mg/L.

Best available technology -- Granular activated carbon; packed tower aeration

Reference -- 56 FR 3526 (01/30/91); 56 FR 30266 (07/01/91)

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

Value -- 0.04 mg/L (Proposed, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- SMCLs are non-enforceable and establish limits for contaminants which may affect the aesthetic qualities (e.g. taste and odor) of drinking water. It is recommended that systems monitor for these contaminants every three years. More frequent monitoring for contaminants such as pH, color, odor or others may be appropriate under certain circumstances. The SCML for toluene is based on odor detection. Promulgation deferred following public comment (56 FR 3526).

Reference -- 54 FR 22062 (05/22/89); 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

SMCL - NO DATA

FISTD- NO DATA

FIREV- NO DATA

CERC -

Value (status) -- 1000 pounds (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ is based on aquatic toxicity, as established under Section 311(b)(4) of the Clean Water Act, ignitability, and chronic toxicity. Available data indicate that the aquatic 96-Hour Median Threshold Limit for Toluene is between 10 and 100 ppm. Its closed-cup flash point is less than 100F and its boiling point is >100F. RQ assignments based on chronic toxicity reflect two primary attributes of the hazardous substance, the minimum effective dose (MED) levels for chronic exposure (mg/day for a 70-kg person) and the type of effect (liver necrosis, teratogenicity, etc). A composite score is determined from an evaluation of these two attributes. Toluene was determined to have a composite score between 6 and 20, corresponding to a chronic toxicity RQ of 1000 pounds.

Reference -- 50 FR 13456 (04/04/85); 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline (800)424-9346 / (202)260-3000 / FTS 260-3000

SARA - NO DATA

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

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Vanadium

1 - IRIS
NAME - Vanadium
RN - 7440-62-2
IRSN - 499
DATE - 910805
UPDT - 08/05/91, 52 fields
STAT - Oral RfD Assessment (RDO) pending
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) no data
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) no data
IRH - 08/01/91 RDO Oral RfD now under review
RLEN - 521
SY - HSDB 1022
SY - VANADIUM
SY - VANADIUM-51

RDO -
o ORAL RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

RDI - NO DATA
CAREV- NO DATA
CARO - NO DATA
CARI - NO DATA
CARDR- NO DATA

HAONE- NO DATA

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA - NO DATA

WQCHU- NO DATA

WQCAQ- NO DATA

MCLG - NO DATA

MCL - NO DATA

SMCL - NO DATA

FISTD- NO DATA

FIREV- NO DATA

CERC - NO DATA

SARA - NO DATA

RCRA - NO DATA

TSCA - NO DATA
OREF - NO DATA
IREF - NO DATA
CREF - NO DATA
HAREF- NO DATA

Vinyl Chloride

1 - IRIS
NAME - Vinyl chloride
RN - 75-01-4
IRSN - 520
DATE - 940706
UPDT - NO DATA
STAT - Oral RfD Assessment (RDO) no data
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) pending 07/01/94
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) no data
IRH - 08/01/91 CAR Carcinogenicity assessment now under review
IRH - 07/01/94 CAR Work group review date added
RLEN - 774
SY - CHLORETHENE
SY - CHLORETHYLENE
SY - CHLOROETHENE
SY - CHLOROETHYLENE
SY - CHLORURE DE VINYLE [FRENCH]
SY - CLORURO DE VINILO [SPANISH]
SY - CLORURO DI VINILE [ITALIAN]
SY - ETHENE, CHLORO-
SY - ETHYLENE, CHLORO-
SY - ETHYLENE MONOCHLORIDE
SY - HSDB 169
SY - MONOCHLOROETHENE
SY - MONOCHLOROETHYLENE
SY - RCRA WASTE NUMBER U043
SY - UN 1086
SY - VC
SY - VCM
SY - VINILE (CLORURO DI) [ITALIAN]
SY - VINYL CHLORIDE
SY - VINYL CHLORIDE, INHIBITED
SY - VINYL CHLORIDE MONOMER
SY - VINYLCHLORID [GERMAN]
SY - VINYL C MONOMER
SY - VINYLE(CHLORURE DE) [FRENCH]
SY - WINYLU CHLOREK [POLISH]

RDO - NO DATA

RDI - NO DATA
CAREV- NO DATA
CARO - NO DATA
CARI - NO DATA
CARDR- NO DATA

HAONE- NO DATA

HATEN- NO DATA

HALTC- NO DATA

HALTA- NO DATA

HALIF- NO DATA

OLEP - NO DATA

ALAB - NO DATA

TREAT- NO DATA

HADR - NO DATA

CAA - NO DATA

WQCHU- NO DATA

WQCAQ- NO DATA

MCLG - NO DATA

MCL - NO DATA

SMCL - NO DATA

FISTD- NO DATA

FIREV- NO DATA

CERC - NO DATA

SARA - NO DATA

RCRA - NO DATA

TSCA - NO DATA
OREF - NO DATA
IREF - NO DATA
CREF - NO DATA
HAREF- NO DATA

Appendix 6-G

Risk Characterization Methodologies

6-G1.0 Introduction

The risk characterization evaluates and quantitatively estimates the risks associated with each of the chemicals of potential concern (COPC) in each exposure pathway for each exposure scenario, given the assumptions of the exposure assessment and toxicity criteria. Cancer risks and noncancer risks are addressed separately. The methods for estimating Media Protection Standards (MPS) is given in Section 6-G4.0. The MPS estimates are based on those scenarios showing the highest risk.

6-G2.0 Methods for Characterizing Noncancer Risks

The noncancer risk associated with a given chemical in an exposure pathway is evaluated in terms of the hazard quotient (HQ). The HQ of chemical "*a*" via the ingestion pathway is calculated as follows:

$$HQ_{Ing}(a) = \frac{IN_{Ing}(a)}{RfD_{aR}fD_a}$$

If the IN_{Ing} is less than the RfD, then the HQ_{Ing} is less than a value of one and the IN_{Ing} is regarded as being unlikely to result in any adverse health effects even to the most susceptible members of a population. HQ values for the other exposure pathways are estimated similarly. The HQ does not define a particular level of risk. One reason for this is that the RfD is an estimate of a threshold exposure level, and below the threshold essentially no risk is assumed.

The sum of HQ values for the identified exposure pathways represents an estimate of the total noncancer risk associated with a given chemical, referred to as the hazard index (HI) of that chemical. The HI of chemical "*a*" via the ingestion, dermal absorption, and inhalation pathways is as follows:

$$HI(a) = HQ_{Ing}(a) + HQ_{Der}(a) + HQ_{Inh}(a)$$

The total hazard index (THI) represents the overall noncancer risks posed by the COPC in a given exposure scenario, and is the sum of the individual HI values:

$$THI = HI(a) + HI(b) + HI(c) + \ldots + HI(n)$$

The THI is compared to a target value of 1. If the THI is less than 1, then it is unlikely, given the exposure scenario assumptions, that the COPC represent a health risk. If the THI exceeds 1, then the effects of the COPC will be broken down by target organs. If any of the target organ-specific THI values exceed 1, then a potential for adverse health effects may be indicated. If all target organ-specific THI values are less than 1, then adverse noncarcinogenic health effects are not considered likely.

6-G3.0 Methods for Characterizing Cancer Risks

With regard to carcinogenic effects, the calculated cancer risk of a given compound in an exposure pathway is simply referred to as the cancer risk (CR). The CR of chemical "*a*" via the ingestion pathway is calculated as follows:

$$CR_{Ing}(a) = 1 - e^{(-CSF(a) \times IN_{Ing})}$$

The CR for other exposure pathways are estimated similarly. The cancer risk of a given compound, considering all exposure pathways, is referred to here as the chemical cancer risk (CCR) and is calculated as follows:

$$CCR(a) = CR_{Ing}(a) + CR_{Der}(a) + CR_{Inh}(a)$$

The estimated incremental lifetime cancer risk (ILCR) represents the overall risks posed by all COPC in a given exposure scenario, and is the sum of all the CCR values:

$$ILCR = CCR(a) + CCR(b) + \ldots + CCR(n)$$

The ILCR is compared to a target risk range that is considered protective of human health, generally between 10^{-6} and 10^{-4}.

6-G4.0 Estimation of Media Protection Standards

Media Protection Standards (MPSs) are concentrations of individual chemicals considered to be health protective for specific medium and land-use combinations. The calculations of MPS presented here are derived from the exposure scenarios and equations described in Section

6.4.2 of the text and Appendix 6-C. By setting a target hazard index at 1, it is possible to solve for the concentration term (i.e., the MPS). Media protection standards for PCBs are based on noncarcinogenic effects because noncarcinogenic effects were shown to drive the risks from PCBs at the site. MPSs are calculated for an on-site worker scenario for the Production Area and an on-site resident scenario for the Warwick Area. A risk assessment for a general worker scenario was also evaluated in the PHERE for the Production Area. Because the risk for that scenario was lower than that for the on-site worker, the MPS for the Production Area was conservatively based on the on-site worker scenario.

MPSs for the worker and resident scenarios are based on three exposure routes: soil ingestion, inhalation (of volatiles and dust), and dermal absorption as described in Section 6.4.2 and Appendix 6-C of the PHERE. Two PCB cogeners with potentially additive effects (PCB-1254 and PCB-1248) have been identified at the site. Total risk from PCBs in soil is expressed as:

$$\text{Total Hazard Index} = \text{Ingestion } HI_{PCB\text{-}1254} + \text{Inhalation } HI_{PCB\text{-}1254} + \text{Dermal } HI_{PCB\text{-}1254} + \text{Ingestion } HI_{PCB\text{-}1248} + \text{Inhalation } HI_{PCB\text{-}12248} + \text{Dermal } HI_{PCB\text{-}1248}$$

Adding the appropriate exposure parameters and rearranging the equation to solve for soil concentration results in equations G-6-1 for on-site workers and G-6-2 for on-site residents.

For the inhalation pathway it is necessary to relate air concentrations to soil concentrations. Air concentrations were modeled from soil concentrations (refer to Appendix 6-D). Air concentrations resulting from volatilization of chemicals were modeled using combined surface and subsurface soil concentrations. Air concentrations resulting from fugitive dust emissions were modeled from surface soil concentrations only. Air-to-soil conversion factors were calculated from the results of this modeling as:

Volatiles

$$ACF_V = (CA_V)/(CS_{CS})$$

and

Nonvolatiles

$$ACF_D = (CA_D)/(CS_{SS})$$

Where:

ACF = Air concentration factor for volatiles and dust (kg/mg^3)
CA = Air concentration (mg/m^3)
CS_{CS} = Concentration in combined surface and subsurface soil (mg/kg)
CS_{SS} = Concentration in surface soil (mg/kg)

As described in the risk characterization, slope factors and reference doses for the dermal absorption pathway are derived from oral values by applying an appropriate gastrointestinal absorption fraction.

6-G4.1 On-Site Worker

Media protection standards for the on-site worker at the Production Area are calculated using equation 6-G-1 and the chemical- and site-specific exposure parameters found in Section 6.4.2 of the PHERE. Media protection standards calculated by this method for PCBs are 51 mg/kg for PCB-1254 and 323 mg/kg for PCB-1248, assuming additive effects.

6-G4.2 On-Site Resident

Media protection standards for the on-site resident at the Waste Water Treatment and Warwick areas are calculated using Equation 6-G-2 and the chemical- and site-specific exposure parameters found in Section 6.4.2 of the PHERE.

The residential scenario assumes exposure of both a child and an adult. The exposure parameters which differ for children and adults (ingestion rate, inhalation rate, skin surface area, exposure duration, and body weight) are combined in age-adjusted intake factors (USEPA, 1991b). Intake factors are calculated as shown below:

Ingestion:

$$IF_{ing} = \frac{(IR_{child})(ED_{child})}{BW_{child}} + \frac{(IR_{adult})(ED_{adult})}{BW_{adult}}$$

Inhalation:

$$IF_{ing} = \frac{(IhR_{child})(ED_{child})}{BW_{child}} + \frac{(IhR_{adult})(ED_{adult})}{BW_{adult}}$$

Skin Surface Area:

$$SF = \frac{(SA_{child})(ED_{child})}{BW_{child}} + \frac{(SA_{adult})(ED_{adult})}{BW_{adult}}$$

Where:

IF_{ing}	=	Ingestion factor for soil (mg-yr/kg-day)
IR	=	Ingestion rate for soil (mg/day)
ED	=	Exposure duration (years)
BW	=	Body weight (kg)
IF_{inh}	=	Inhalation factor (m^3-yr/kg-hr)
IhR	=	Inhalation rate (m^3/hr)
SF	=	Skin factor (cm^2-yr/kg-day)
SA	=	Skin surface area available for contact (cm^2/day)

See Section 6.4.2 of the text and Appendix 6-C concerning input values for the above exposure input parameters.

MPSs calculated by this method for PCBs in Warwick Area soil are 5.4 mg/kg for PCB-1254 and 15.5 mg/kg for PCB-1248, assuming additive effects.

Equation 6-G-1
On-Site Resident Scenario

$$MPS_{1254} = \frac{(THI)(AT)}{(CF)(TF)(IF_{ing})(EF)[(1/RfD_{1254})+(1/RfD_{1248})(RC)]+(CF)(SF)(AF)(ABS)(EF)[(1/RfD_{1254})(GAF)+(1/RfD_{1248})(GAF)(RC)]+(ET)(IF_{INH})(EF_{INH})[(ACF_{V1254}+ACF_{D})(1/RfD_{1254})+(ACF_{V1248}+ACF_{D})(1/RfD_{1248})(RC)]} = 5.4 \text{ mg/kg}$$

Equation 6-G-2
On-Site Worker Scenario

$$MPS_{1254} = \frac{(THI)\ (BW)\ (AT)}{(EF)(ED)\{(CF)(IR)[(1/RfD_{1254})+(1/RfD_{1248})(RC)]+(CF)(SA_{s})(AF)(ABS)[(1/RfD_{1254})(GAF)+(1/RfD_{1248})(GAF)(RC)]+(ET)(IhR)[(ACF_{V1254}+ACF_{D})(1/RfD_{I1254})+(ACF_{V1248}+ACF_{D})(1/RfD_{I1248})(RC)]\}} = 51 \text{ mg/kg}$$

where:

ACF_D	=	Air concentration associated with fugitive dust (3.90×10^{-8} kg/m^3)
$ACF_{V\text{-}1254}$	=	Air concentration for volatile PCB 1254 (4.12×10^{-8} kg/m^3)
$ACF_{V\text{-}1248}$	=	Air concentration for volatile PCB 1248 (1.74×10^{-7} kg/m^3)
IF_{ING}	=	Age-adjusted intake factor for inhalation (114 mg-yr/kg-day)
IF_{INH}	=	Age-adjusted intake factor for ingestion (0.33 m^3-yr/kg-hr)
SF	=	Age-adjusted intake factor for skin sufrace area (1429 cm^2-yr/kg-day)
RC	=	Ratio of the concentration of PCB 1248 to PCB 1254. (On-site worker scenario--6.30; on-site resident scenario--2.88)
MPS_{1248}	=	Media Protection Standard (soil) for PCB 1248
MPS_{1254}	=	Media Protection Standard (soil) for PCB 1254

RfD_{1248} = Reference dose for PCB 1248
RfD_{I1248} = Inhalation route reference dose for PCB 1248
RfD_{I1254} = Inhalation route reference dose for PCB 1254

Other input parameters are found in Section 6.4.2 and Appendix 6-C of the PHERE.

Appendix 6-H

Ecological Risk Support Data

Table 6-H-1
Terrestrial Biota Inventory--March, 1992[a]

Family	Common Name	Scientific Name
Upland Vegetation		
Compositae	goldenrod thistle thickseed	*Solidago* spp. *Cirsium arvense* *Bidens* sp.
Umbelliferae	Queen Anne's lace	*Daucus carota*
Polygonaceae	Japanese knotweed	*Polygonum sagittatum*
Asclepiadaceae	milkweed	*Asclepias incarnata*
Anacardiaceae	poison-ivy staghorn sumac	*Toxicodenderon radicans* *Rhus typhina*
Rosaceae	black cherry common apple multiflora rose	*Prunus serotina* *Malus pumila* *Rosa multiflora*
Ericaceae	lowbush blueberry rosebay	*Vaccinium angustifolium* *Rhododendron maximum*
Araliaceae	English ivy	*Hedera helix*
Onagraceae	evening-primrose	*Oenothera biennis*
Scrophulariaceae	common mullein	*Verbascum thapsus*
Fagaceae	black oak northern red oak white oak	*Quercus velutina* *Quercus rubra* *Quercus alba*
Salicaceae	cottonwood	*Populus deltoides*
Cornaceae	flowering dogwood	*Cornus florida*
Betulaceae	gray birch	*Betula populifolia*
Myricaceae	northern bayberry	*Myrica pensylvanica*
Aceraceae	Norway maple	*Acer platanoides*
Plantaginaceae	plantain	*Plantago* spp.
Simarubaceae	Tree-of-Heaven	*Ailanthus altissima*

Table 6-H-1
Terrestrial Biota Inventory--March, 1992[a]

Family	Common Name	Scientific Name
Pinaceae	Austrian pine shortleaf pine white pine	*Pinus nigra* *Pinus echinata* *Pinus strobus*
Riparian / Wetland Vegetation		
Polypodiaceae	sensitive fern	*Onoclea sensibilis*
Gramineae	reed canary grass	*Phalaris canariensis*
Cyperaceae	soft rush tussock sedge	*Juncus effusus* *Carex stricta*
Typhaceae	cattail	*Typha latifolia*
Liliaceae	greenbrier	*Smilax rotundifolia*
Clethraceae	pepperbush	*Clethra alnifolia*
Rosaceae	black cherry	*Prunus serotina*
Salicaceae	black willow pussy willow	*Salix nigra* *Salix discolor*
Ericaceae	highbush blueberry	*Vaccinium corymbosum*
Fagaceae	bear oak northern red oak swamp white oak white oak	*Quercus ilicifolia* *Quercus rubra* *Quercus bicolor* *Quercus alba*
Cornaceae	black gum red-osier dogwood	*Nyssa sylvatica* *Cornus stolonifera*
Betulaceae	gray birch mockernut hickory speckled alder	*Betula populifolia* *Carya tomentosa* *Alnus rugosa*
Aceraceae	box elder red maple silver maple	*Acer negundo* *Acer rubrum* *Acer saccharinum*
Ulmaceae	American elm	*Ulmus americana*

Table 6-H-1
Terrestrial Biota Inventory--March, 1992[a]

Family	Common Name	Scientific Name
Lauraceae	sassafras	*Sassafras albidum*
Pinaceae	eastern hemlock	*Tsuga canadensis*
Birds		
Ardeidae	great blue heron	*Ardea herodias*
Anserinae	Canada goose	*Branta candensis*
Anatinae	American widgeon black duck mallard wood duck	*Anas americana* *Anas rubripes* *Anas platyrhynchos* *Aix sponsa*
Merginae	hooded merganser	*Lophodytes cucullatus*
Accipitrinae	Coopers' hawk	*Accipiter cooperii*
Buteoninae	red-tailed hawk	*Buteo jamaicensis*
Charadriidae	killdeer	*Charadrius vociferus*
Larinae	gulls (miscellaneous)	*Larus* spp.
Columbidae	mourning dove pigeon	*Zenaida macroura* *Columba livia*
Strigidae	great horned owl	*Bubo virginianus*
Alcedinidae	belted kingfisher	*Megaceryle alcyon*
Corvidae	American crow blue jay	*Corvus brachyrhynchos* *Cyanocitta cristata*
Paradae	black-capped chickadee	*Parus atricapillus*
Mimidae	mockingbird	*Mimus polyglottos*
Turdidae	American robin	*Turdus migratorius*
Ploceidae	house sparrow	*Passer domesticus*
Iceridae	red-winged blackbird	*Agelaius phoeniceus*

Table 6-H-1
Terrestrial Biota Inventory--March, 1992[a]

Family	Common Name	Scientific Name
Sturnidae	European starling	*Sturnus vulgaris*
Fringillidae	cardinal dark-eyed junco song sparrow	*Cardinalis cardinalis* *Junco hyemalis* *Melospiza melodia*
Mammals		
Lepopidae	eastern cottontail rabbit	*Sylvilagus floridanus*
Canidae	domestic dog	*Canis familiaris*
Sciuridae	eastern gray squirrel	*Sciurus carolinensis*
Cricetidae	muskrat	*Ondatra zibethicus*
Procyonidae	raccoon	*Procyon lotor*

a. This terrestrial/riparian survey included three localities: the Site itself, the region east and downstream of the Site boundary just west of the Warwick Avenue bridge to Rhodes-on-the-Pawtuxet, and the region west and upstream of the Site from its northern boundary near Atlantic Rubber and Tubing to the Elmwood Avenue bridge. Each locality included upland areas and riparian zones bordering the Pawtuxet River. The downstream locality included a wetland area.

Table 6-H-2
Screening Process for Chemicals of Potential Concern
Production Area

Chemical Name	Frequency of Detection	Selected Concentration	Statistically tested against background[a]	Above Background?[b]	Water Chemistry or Essential Nutrient?	COPC?
1,2-DICHLOROBENZENE	4.88	1.20E-01	No	NA	No	No
1,4-DICHLOROBENZENE	2.44	2.40E-01	No	NA	No	No
2,4,5-TP (SILVEX)	3.57	6.00E-03	No	NA	No	No
2,4-DIMETHYLPHENOL	2.44	1.10E-01	No	NA	No	No
2-METHYLNAPHTHALENE	9.76	3.80E-01	No	NA	No	Yes
2-NITROANILINE	19.51	8.90E-01	No	NA	No	Yes
3&4-METHYLPHENOL	31.25	2.52E-01	No	NA	No	Yes
4,4'-DDD	2.33	3.30E-03	No	NA	No	No
4-CHLOROANILINE	12.20	6.40E-01	No	NA	No	Yes
4-METHYLPHENOL	4.00	2.40E-01	No	NA	No	No
ACENAPHTHENE	24.39	2.10E-01	No	NA	No	Yes
ACENAPHTHYLENE	12.20	1.80E-01	No	NA	No	Yes
ACETOPHENONE	2.44	4.80E-02	No	NA	No	No
ALDRIN	2.33	3.50E-03	No	NA	No	No
ALPHA-BHC	4.65	9.90E-03	No	NA	No	No
ALPHA-CHLORDANE	4.65	9.70E-03	No	NA	No	No
AMMONIA AS N	36.00	1.12E+00	No	NA	Yes	No
ANILINE	2.44	2.30E-01	No	NA	No	No
ANTHRACENE	58.54	1.29E+00	No	NA	No	Yes
ARSENIC	90.63	1.54E+01	Yes	No	No	No
BARIUM	100.00	5.62E+01	Yes	No	No	No
BENZO(A)ANTHRACENE	68.29	1.52E+00	No	NA	No	Yes
BENZO(A)PYRENE	65.85	1.69E+00	Yes	No	No	No
BENZO(B)FLUORANTHENE	73.17	1.98E+00	No	NA	No	Yes
BENZO(G,H,I)PERYLENE	51.22	1.81E+00	No	NA	No	Yes
BENZO(K)FLUORANTHENE	65.85	1.95E+00	No	NA	No	Yes
BERYLLIUM	96.77	4.54E-01	No	NA	No	No
BICARBONATE ALKALINITY	80.00	1.96E+03	No	NA	Yes	No
BIS(2-CHLOROETHYL)ETHER	2.44	6.80E-01	No	NA	No	No
BIS(2-ETHYLHEXYL)PHTHALATE	39.02	1.49E+00	No	NA	No	No
BUTAZOLIDIN	4.00	5.20E+00	No	NA	No	No
BUTYLBENZYLPHTHALATE	31.71	3.22E+00	No	NA	No	No
CADMIUM	58.06	8.74E-01	No	NA	No	No

Table 6-H-2
Screening Process for Chemicals of Potential Concern
Production Area

Chemical Name	Frequency of Detection	Selected Concentration	Statistically tested against background[a]	Above Background?[b]	Water Chemistry or Essential Nutrient?	COPC?
CALCIUM	100.00	2.73E+04	No	NA	Yes	No
CARBONATE ALKALINITY	56.00	1.86E+03	No	NA	Yes	No
CATION EXCHANGE CAPACITY	100.00	7.29E+00	No	NA	Yes	No
CHLORIDE	44.00	4.89E+01	No	NA	Yes	No
CHLOROBENZENE	17.50	2.80E-01	No	NA	Yes	Yes
CHLOROFORM	2.50	3.40E-02	No	NA	No	No
CHROMIUM	93.55	1.37E+01	Yes	No	No	No
CHRYSENE	68.29	1.62E+00	No	NA	No	Yes
COBALT	93.55	3.44E+00	No	NA	No	No
COPPER	93.55	2.27E+01	No	NA	No	No
CYANIDE	32.35	1.98E+00	No	NA	No	Yes
DELTA-BHC	2.33	2.40E-03	No	NA	No	No
DI-N-BUTYLPHTHALATE	19.51	1.30E+00	No	NA	No	Yes
DIBENZ(A,H)ANTHRACENE	24.39	6.80E-01	No	No	No	No
DIBENZOFURAN	29.27	1.30E-01	No	NA	No	No
DIMETHYLPHTHALATE	2.44	2.50E-01	No	NA	No	No
DINOSEB	7.50	8.70E-03	No	NA	No	Yes
ENDRIN ALDEHYDE	2.33	1.00E-03	No	NA	No	No
ETHYLBENZENE	25.00	3.41E+00	No	NA	No	Yes
FAMPHUR	4.76	1.60E-02	No	NA	No	No
FLUORANTHENE	80.49	2.05E+00	No	NA	No	Yes
FLUORENE	29.27	1.80E-01	No	NA	No	Yes
GAMMA-BHC	4.65	3.30E-03	No	NA	No	No
GAMMA-CHLORDANE (d)	16.28	1.32E-01	No	NA	No	Yes
HEPTACHLOR	2.33	1.40E-02	No	NA	No	No
INDENO(1,2,3-CD)PYRENE	51.22	1.79E+00	No	NA	No	Yes
IRGASAN DP-300	11.54	4.20E+00	No	NA	No	Yes
IRON	100.00	1.18E+04	No	NA	Yes	No
KEPONE	2.33	1.50E-02	No	NA	No	No
LEAD	93.55	7.95E+01	No	NA	No	No
M&P-XYLENE	67.50	2.70E+01	No	NA	No	Yes
MAGNESIUM	100.00	2.54E+03	No	NA	Yes	No
MANGANESE	100.00	1.89E+02	No	NA	Yes	No

Table 6-H-2
Screening Process for Chemicals of Potential Concern
Production Area

Chemical Name	Frequency of Detection	Selected Concentration	Statistically tested against background[a]	Above Background?[b]	Water Chemistry or Essential Nutrient?	COPC?
MERCURY	73.33	6.10E-01	No	NA	No	No
METHOXYCHLOR	16.28	5.55E-01	No	NA	No	Yes
METHYL PARATHION	4.76	6.50E-03	No	NA	No	No
METHYLENE CHLORIDE	15.00	9.50E-03	No	NA	No	Yes
NAPHTHALENE	34.15	6.80E-01	No	NA	No	Yes
NICKEL	90.32	1.17E+01	Yes	Yes	No	Yes
NITRATE-NITRITE AS N	88.00	2.63E+00	No	NA	Yes	No
NITROBENZENE	7.32	1.40E-01	No	NA	No	Yes
O-XYLENE	47.50	8.12E+00	No	NA	No	Yes
OCDD	20.00	4.60E-04	No	NA	No	Yes
ORTHOPHOSPHATE	80.00	8.13E+00	No	NA	No	Yes
PCB-1248	38.78	4.40E-01	No	NA	No	Yes
PCB-1254	88.79	3.60E+00	No	NA	No	Yes
PCB-1260	13.46	6.10E+00	No	NA	No	Yes
PERCENT MOISTURE	100.00	1.01E+01	No	NA	Yes	No
PH	100.00	9.23E+00	No	NA	Yes	No
PHENANTHRENE	68.29	1.54E+00	No	NA	No	Yes
PHENOL	2.44	6.30E-01	No	NA	No	No
POTASSIUM	96.15	9.40E+02	No	NA	Yes	No
PYRENE	78.05	2.30E+00	No	NA	No	Yes
SODIUM	50.00	1.77E+02	No	NA	Yes	No
STYRENE	5.00	4.90E-02	No	NA	No	No
SULFATE	56.00	1.53E+02	No	NA	Yes	No
SULFIDE	20.00	2.70E+01	No	NA	Yes	No
SULFOTEPP	2.38	9.40E-03	No	NA	No	No
TCDF	60.00	1.77E-04	No	NA	No	Yes
TETRACHLOROETHENE	2.50	6.90E-02	No	NA	No	No
THALLIUM	3.45	2.14E-01	No	NA	No	No
TIN	3.85	6.60E+00	No	NA	No	No
TINUVIN 327	4.17	5.20E+00	No	NA	No	No
TINUVIN 328	100.00	5.90E+00	No	NA	No	No
TOLUENE	45.00	7.14E-01	No	NA	No	Yes
TOTAL ALKALINITY	96.00	3.65E+03	No	NA	Yes	No

Table 6-H-2
Screening Process for Chemicals of Potential Concern
Production Area

Chemical Name	Frequency of Detection	Selected Concentration	Statistically tested against background[a]	Above Background?[b]	Water Chemistry or Essential Nutrient?	COPC?
TOTAL ORGANIC CARBON	100.00	3.03E+03	No	NA	Yes	No
TRCDF	40.00	5.41E-01	No	NA	No	Yes
TRICHLOROFLUOROMETHANE	7.50	3.30E-01	No	NA	No	Yes
VANADIUM	1.00	1.13E+01	Yes	No	No	No
ZINC	1.00	3.84E+02	No	NA	No	No

a- statistically tested against background values using t-test
b- significant at the 95% confidence level

Table 6-H-3
Results of Analysis of Surface Soil for Chemicals of Potential Concern
Production Area

Chemical Name	Number of Detects	Number of Samples	Frequency of Detection	Mean	Standard Deviation	95% UCL	Maximum Concentration	Minimum Concentration	Selected Concentration
INORGANICS									
BERYLLIUM	30	31	96.77	4.00E-01	1.76E-01	4.54E-01	7.30E-01	9.00E-02	4.54E-01
CADMIUM	18	31	58.06	6.48E-01	7.42E-01	8.74E-01	3.90E+00	2.80E-01	8.74E-01
COBALT	29	31	93.55	3.04E+00	1.31E+00	3.44E+00	6.00E+00	3.80E-01	3.44E+00
COPPER[a]	29	31	93.55	1.17E+01	NA[a]	3.05E+01	7.61E+01	3.70E+00	3.05E+01
LEAD	29	31	93.55	5.43E+01	8.25E+01	7.95E+01	3.78E+02	3.60E+00	7.95E+01
MERCURY	22	30	73.33	4.60E-01	4.85E-01	6.11E-01	1.60E+00	1.10E-01	6.11E-01
NICKEL[a]	28	31	90.32	5.84E+00	NA[a]	1.16E+01	2.66E+01	1.50E+00	1.16E+01
THALLIUM	1	29	3.45	1.98E-01	5.29E-02	2.14E-01	2.60E-01	2.60E-01	2.14E-01
TIN	1	26	3.85	5.18E+00	4.21E+00	6.60E+00	2.56E+01	2.56E+01	6.60E+00
ZINC[a]	31	31	100.00	1.06E+02	NA[a]	3.68E+02	7.59E+02	1.30E+01	3.68E+02
CYANIDE	11	34	32.35	1.32E+00	2.26E+00	1.98E+00	1.26E+01	5.60E-01	1.98E+00
ORGANICS									
2-METHYLNAPHTHALENE	4	41	9.76	1.26E+00	1.83E+00	1.74E+00	3.80E-01	3.80E-02	3.80E-01
2-NITROANILINE	4	41	19.51	6.25E+00	9.06E+00	8.63E+00	8.90E-01	4.40E-02	8.90E-01
3&4-METHYLPHENOL	5	16	31.25	1.79E-01	1.66E-01	2.52E-01	7.70E-01	2.30E-02	2.52E-01
4-CHLOROANILINE	5	41	12.20	1.32E+00	1.79E+00	1.80E+00	6.40E-01	4.50E-02	6.40E-01
ACENAPHTHENE	10	41	24.39	1.24E+00	1.84E+00	1.72E+00	2.10E-01	5.70E-02	2.10E-01
ACENAPHTHYLENE	5	41	12.20	1.26E+00	1.83E+00	1.74E+00	1.80E-01	4.30E-02	1.80E-01
ANTHRACENE	24	41	58.54	8.81E-01	1.56E+00	1.29E+00	1.60E+00	3.40E-02	1.29E+00
BENZO(A)ANTHRACENE	28	41	68.29	1.14E+00	1.41E+00	1.52E+00	3.10E+00	1.50E-01	1.52E+00
BENZO(B)FLUORANTHENE	30	41	73.17	1.57E+00	1.56E+00	1.98E+00	4.30E+00	2.70E-02	1.98E+00
BENZO(G,H,I)PERYLENE	21	41	51.22	1.35E+00	1.75E+00	1.81E+00	2.90E+00	1.30E-01	1.81E+00
BENZO(K)FLUORANTHENE	27	41	65.85	1.50E+00	1.69E+00	1.95E+00	5.50E+00	7.40E-02	1.95E+00
BIS(2-ETHYLHEXYL)PHTHALATE	16	41	39.02	1.09E+00	1.49E+00	1.49E+00	4.10E+00	6.10E-02	1.49E+00
BUTYLBENZYLPHTHALATE	13	41	31.71	1.83E+00	5.29E+00	3.22E+00	3.30E+01	4.20E-02	3.22E+00
CHLOROBENZENE	7	40	17.50	2.03E-01	1.02E+00	4.77E-01	2.80E-01	3.20E-02	2.80E-01
CHRYSENE	28	41	68.29	1.24E+00	1.42E+00	1.62E+00	3.30E+00	1.50E-01	1.62E+00
DI-N-BUTYLPHTHALATE	8	41	19.51	9.99E-01	1.64E+00	1.43E+00	1.30E+00	4.50E-02	1.30E+00

Table 6-H-3
Results of Analysis of Surface Soil for Chemicals of Potential Concern
Production Area

Chemical Name	Number of Detects	Number of Samples	Frequency of Detection	Mean	Standard Deviation	95% UCL	Maximum Concentration	Minimum Concentration	Selected Concentration
DIBENZOFURAN	12	41	29.27	1.20E+00	1.86E+00	1.69E+00	1.30E-01	3.50E-02	1.30E-01
DINOSEB	3	40	7.50	6.50E-02	7.91E-02	8.62E-02	8.70E-03	2.40E-03	8.70E-03
ETHYLBENZENE	10	40	25.00	1.29E+00	7.90E+00	3.41E+00	5.00E+01	6.30E-03	3.41E+00
FLUORANTHENE	33	41	80.49	1.60E+00	1.73E+00	2.05E+00	8.40E+00	5.10E-02	2.05E+00
FLUORENE	12	41	29.27	1.21E+00	1.86E+00	1.70E+00	1.80E-01	4.80E-02	1.80E-01
GAMMA-CHLORDANE	7	43	16.28	6.59E-02	2.57E-01	1.32E-01	1.70E+00	7.50E-03	1.32E-01
INDENO(1,2,3-CD)PYRENE	21	41	51.22	1.33E+00	1.76E+00	1.79E+00	2.30E+00	4.50E-02	1.79E+00
IRGASAN DP-300	3	26	11.54	9.42E+00	1.01E+01	1.28E+01	4.20E+00	5.70E-01	4.20E+00
M&P-XYLENE	27	40	67.50	1.01E+01	6.32E+01	2.70E+01	4.00E+02	6.00E-03	2.70E+01
METHOXYCHLOR	7	43	16.28	3.54E-01	7.83E-01	5.55E-01	3.60E+00	1.20E-01	5.55E-01
METHYLENE CHLORIDE	6	40	15.00	2.69E-01	1.03E+00	5.46E-01	9.50E-03	5.40E-03	9.50E-03
NAPHTHALENE	14	41	34.15	8.30E-01	1.65E+00	1.27E+00	6.80E-01	3.30E-02	6.80E-01
NITROBENZENE	3	41	7.32	1.25E+00	1.83E+00	1.73E+00	1.40E-01	8.10E-02	1.40E-01
O-XYLENE	19	40	47.50	3.03E+00	1.90E+01	8.12E+00	1.20E+02	9.30E-03	8.12E+00
OCDD	1	5	20.00	5.06E-04	1.24E-04	6.24E-04	4.60E-04	4.60E-04	4.60E-04
ORTHOPHOSPHATE	20	25	80.00	5.15E+00	8.71E+00	8.13E+00	4.30E+01	7.10E-01	8.13E+00
PCB-1248	38	98	38.78	5.19E+01	4.49E+02	1.27E+02	4.50E+03	2.00E-02	1.27E+02
PCB-1254	95	107	88.79	1.58E+01	2.75E+01	2.03E+01	8.40E+01	4.30E-02	2.03E+01
PCB-1260	7	52	13.46	4.65E+00	2.77E+01	1.11E+01	6.10E+00	1.30E-01	6.10E+00
Total PCB's									1.45E+02
PHENANTHRENE	28	41	68.29	1.15E+00	1.49E+00	1.54E+00	5.00E+00	9.30E-02	1.54E+00
PYRENE	32	41	78.05	1.82E+00	1.84E+00	2.30E+00	6.70E+00	6.10E-02	2.30E+00
TCDF	3	5	60.00	1.30E-04	4.91E-05	1.77E-04	1.90E-04	9.50E-05	1.77E-04
TOLUENE	18	40	45.00	3.93E-01	1.20E+00	7.14E-01	4.60E+00	6.80E-03	7.14E-01
TRCDF	2	5	40.00	2.26E-01	3.31E-01	5.41E-01	7.30E-01	4.00E-01	5.41E-01
TRICHLOROFLUOROMETHANE	3	40	7.50	4.13E-01	2.12E+00	9.83E-01	3.30E-01	7.10E-02	3.30E-01

a- compound is log normally distributed. Mean concentration is the geometric mean.

Table 6-H-4
Exposure Due to Dermal Contact of Juvenile Shrews to COPCs

Chemical Name	Selected Concentration	TRV mammal dermal	Test Species	Reference	Baby Shrew Concentration	TQ Baby shrew
2-BUTANONE	1.22E-01	6.48E+01	rabbit	Lewis, 1992	3.61E-05	-6.25E+00
2-NITROANILINE	7.00E+00	2.00E+02	rabbit	Lewis, 1992	2.07E-03	-4.99E+00
3&4-METHYLPHENOL	2.51E-01	1.10E+01	rat	HSDB	7.40E-05	-5.17E+00
4-CHLOROANILINE	2.01E+00	3.60E+00	rabbit	Lewis, 1992	5.93E-04	-3.78E+00
4,4'-DDT	2.52E-01	1.93E+01	rat	Lewis, 1992	7.44E-05	-5.41E+00
ALDRIN	2.08E-01	9.80E-01	rat	Lewis, 1992	6.14E-05	-4.20E+00
BIS(2-CHLOROETHYL)ETHER	4.30E-01	7.20E+00	rabbit	Lewis, 1992	1.27E-04	-4.75E+00
BIS(2-ETHYLHEXYL)PHTHALATE	2.00E+01	2.50E+02	rabbit	Lewis, 1992	5.91E-03	-4.63E+00
DIELDRIN	1.64E-01	5.60E-01	rat	Lewis, 1992	4.84E-05	-4.06E+00
DISULFOTON	7.70E-03	6.00E-02	rat	Lewis, 1992	2.27E-06	-4.42E+00
ENDOSULFAN I	1.80E-02	3.40E-01	rat	Lewis, 1992	5.31E-06	-4.81E+00
ENDRIN	1.41E-01	6.00E-01	rabbit	Lewis, 1992	4.16E-05	-4.16E+00
GAMMA-BHC	1.70E-03	5.00E+00	rat	Lewis, 1992	5.02E-07	-7.00E+00
GAMMA-CHLORDANE	2.79E+00	6.90E+00	rat	Lewis, 1992	8.23E-04	-3.92E+00
HEPTACHLOR EPOXIDE	1.92E-01	1.19E+00	rat, heptachlor	Lewis, 1992	5.65E-05	-4.32E+00
M&P-XYLENE	2.70E+01	1.41E+02	rabbit	Lewis, 1992	7.97E-03	-4.25E+00
NITROBENZENE	2.13E+00	2.10E+01	rat	Lewis, 1992	6.27E-04	-4.52E+00
PCB-1248	1.52E+01	1.00E+02	rabbit	HSDB	4.47E-03	-4.35E+00
PCB-1254	5.19E+00	1.00E+02	rabbit	HSDB	1.53E-03	-4.81E+00
Total PCB's	1.80E+01	1.00E+02	rabbit	HSDB	5.31E-03	-4.27E+00
PHENOL	8.90E-01	6.69E+00	rat	HSDB	2.63E-04	-4.41E+00
TOLUENE	1.98E+00	1.21E+02	rabbit	HSDB	5.84E-04	-5.32E+00

Table 6-H-5
Exposure Due to Inhalation for Shrews

Chemical Name	Selected Concentration[a]	Henry's Law Constant[b]	TRV mammal (inhalation)	Test Species	Reference	Estimated vapor concentration	Shrew Daily Dose	TQ Shrew
INORGANICS								
CYANIDE	2.70E+00	1.22E-04	1.58E+00	rat	BEIA, 1989	8.07E+00	1.33E-02	-2.07E+00
ORGANICS								
2-BUTANONE	1.22E-01	4.35E-05	4.00E+02	mouse	BEIA, 1989	1.44E-03	2.37E-06	-8.23E+00
ALPHA-CHLORDANE	7.70E-02	3.67E-05	1.00E+00	cat	BEIA, 1989	7.86E-07	1.30E-09	-8.89E+00
BIS(2-ETHYLHEXYL)PHTHALATE	2.00E+01	1.38E-07	3.00E+02	mammal	BEIA, 1989	1.17E-07	1.94E-10	-1.22E+01
CHLOROBENZENE	6.68E-01	3.93E-03	1.50E+02	mouse	BEIA, 1989	5.89E-02	9.73E-05	-6.19E+00
DIELDRIN	1.61E-01	2.00E-07	1.30E-01	rat	Lewis, 1992	1.62E-07	2.67E-10	-8.69E+00
GAMMA-CHLORDANE	2.79E+00	3.67E-05	1.00E+00	cat	BEIA, 1989	4.65E-06	7.68E-09	-8.11E+00
M&P-XYLENE	2.70E+01	5.20E-03	5.00E+01	rat	BEIA, 1989	1.39E+00	2.30E-03	-4.34E+00
METHYLENE CHLORIDE	1.10E-01	3.19E-03	1.50E+02	mouse	Verschueren, 1983	2.57E-01	4.24E-04	-5.55E+00
O-XYLENE	8.12E+00	5.27E-03	5.00E+01	rat	BEIA, 1989	7.49E-01	1.24E-03	-4.61E+00
PHENOL	8.90E-01	4.54E-07	1.77E+00	mouse	BEIA, 1989	2.06E-04	3.40E-07	-6.72E+00
TETRACHLOROETHENE	2.52E-01	2.90E-02	5.20E+01	mouse	BEIA, 1989	3.31E-01	5.45E-04	-4.98E+00
TOLUENE	7.14E-01	6.68E-03	5.32E+01	mouse	BEIA, 1989	1.50E-01	2.48E-04	-5.33E+00
TRICHLOROETHENE	5.97E-02	9.10E-03	5.50E+01	mouse	BEIA, 1989	3.46E-02	5.70E-05	-5.98E+00

[a]Selected concentrations are the highest concentration from the Production, Warwick and Wastewater Treatment Areas.

[b]Sources include BEIA, 1992 and Lyman, et.al., 1990

Table 6-H-6
Screening Process for Chemicals of Potential Concern
Waste Water Treatment Area

Chemical Name	Frequency of Detection (%)	Selected Concentration	Statistically tested against background?[a]	Above Background?[b]	Water Chemistry or Essential Nutrient?	COPC?
1,2,4-TRICHLOROBENZENE	4	3.40E-02	No	NA	No	No
2,3,7,8-TCDF	50	4.58E-04	No	NA	No	Yes
2,4,5-T	20	3.11E-02	No	NA	No	Yes
2,4-D	5	1.20E-01	No	NA	No	No
2,4-DICHLOROPHENOL	39	8.39E-01	No	NA	No	Yes
2-METHYLNAPHTHALENE	17	5.00E-01	No	NA	No	Yes
2-METHYLPHENOL	4	7.76E-01	No	NA	No	No
2-NITROANILINE	9	7.30E-02	No	NA	No	Yes
3&4-METHYLPHENOL	18	2.51E-01	No	NA	No	Yes
4,4'-DDD	30	1.73E-01	No	NA	No	Yes
4,4'-DDE	22	1.55E-01	No	NA	No	Yes
4,4'-DDT	9	9.70E-03	No	NA	No	Yes
4-CHLOROANILINE	17	1.71E+00	No	NA	No	Yes
4 CHLOROPHENYL-PHENYLETHER	9	2.80E-01	No	NA	No	Yes
4-METHYLPHENOL	17	2.90E-01	No	NA	No	Yes
ACENAPHTHENE	9	1.00E+00	No	NA	No	Yes
ACENAPHTHYLENE	4	4.50E-02	No	NA	No	Yes
ACETONE	22	1.34E-01	No	NA	No	Yes
ACETOPHENONE	9	2.60E-02	No	NA	No	Yes
ALPHA-BHC	9	2.10E-03	No	NA	No	Yes
ALPHA-CHLORDANE	4	4.50E-03	No	NA	No	No
AMMONIA AS N	70	2.57E+01	No	NA	Yes	No
ANILINE	17	1.23E+00	No	NA	No	Yes
ANTHRACENE	13	1.16E+00	No	NA	No	Yes
ARSENIC	100	7.26E+00	Yes	No	No	No
BARIUM	100	3.52E+01	No	NA	No	Yes
BENZO(A)ANTHRACENE	39	1.36E+00	Yes	No	No	No
BENZO(A)PYRENE	35	1.36E+00	Yes	No	No	No
BENZO(B)FLUORANTHENE	70	1.51E+00	Yes	No	No	No
BENZO(G,H,I)PERYLENE	17	1.30E+00	Yes	No	No	No
BENZO(K)FLUORANTHENE	52	1.32E+00	Yes	No	No	No
BERYLLIUM	95	5.21E-01	Yes	No	No	No
BETA-BHC	4	1.60E-02	No	NA	No	No

Table 6-H-6
Screening Process for Chemicals of Potential Concern
Waste Water Treatment Area

Chemical Name	Frequency of Detection (%)	Selected Concentration	Statistically tested against background?[a]	Above Background?[b]	Water Chemistry or Essential Nutrient?	COPC?
BICARBONATE ALKALINITY	60	6.61E+02	No	NA	Yes	No
BIS(2-ETHYLHEXYL)PHTHALATE	39	1.78E+00	No	NA	No	Yes
BUTYLBENZYLPHTHALATE	9	1.50E-01	No	NA	No	Yes
CADMIUM	17	3.86E-01	No	NA	No	Yes
CALCIUM	100	1.05E+04	No	NA	Yes	No
CARBONATE ALKALINITY	60	6.07E+02	No	NA	Yes	No
CATION EXCHANGE CAPACITY	100	5.41E+00	No	NA	Yes	No
CHLORIDE	70	3.10E+02	No	NA	Yes	No
CHLOROBENZENE	48	2.62E+00	No	NA	No	Yes
CHROMIUM	100	1.12E+01	Yes	No	No	No
CHRYSENE	61	1.38E+00	Yes	No	No	No
COBALT	100	3.81E+00	No	NA	No	Yes
COPPER	100	5.37E+01	No	NA	No	Yes
CYANIDE	24	1.14E+00	No	NA	No	Yes
DCDF	50	2.02E+00	No	NA	No	Yes
DELTA-BHC	4	2.60E-02	No	NA	No	No
DI-N-BUTYLPHTHALATE	4	1.00E-01	No	NA	No	No
DI-N-OCTYLPHTHALATE	4	1.60E-01	No	NA	No	No
DIBENZ(A,H)ANTHRACENE	9	2.46E+00	Yes	NA	No	No
DIBENZOFURAN	13	7.03E-01	No	NA	No	Yes
DIELDRIN	17	1.50E-01	No	NA	No	Yes
DIETHYLPHTHALATE	17	7.36E-01	No	NA	No	Yes
DIMETHOATE	4	2.60E-02	No	NA	No	No
ENDRIN	9	7.00E-03	No	NA	No	Yes
ENDRIN ALDEHYDE	13	2.09E-01	No	NA	No	Yes
ETHYL PARATHION	9	5.40E-02	No	NA	No	Yes
ETHYLBENZENE	17	4.41E-01	No	NA	No	Yes
FAMPHUR	14	7.70E-02	No	NA	No	Yes
FLUORANTHENE	74	1.56E+00	No	NA	No	Yes
FLUORENE	9	5.10E-01	No	NA	No	Yes
GAMMA-CHLORDANE	35	2.58E+00	No	NA	No	Yes
HEPTACHLOR	9	7.79E-02	No	NA	No	Yes
INDENO(1,2,3-CD)PYRENE	26	1.24E+00	Yes	No	No	No

Table 6-H-6
Screening Process for Chemicals of Potential Concern
Waste Water Treatment Area

Chemical Name	Frequency of Detection (%)	Selected Concentration	Statistically tested against background?[a]	Above Background?[b]	Water Chemistry or Essential Nutrient?	COPC?
IRGASAN DP-300	46	8.61E+01	No	NA	No	Yes
IRON	100	1.27E+04	No	NA	Yes	No
KEPONE	4	5.70E-02	No	NA	No	No
LEAD	100	5.36E+01	No	NA	No	No
M&P-XYLENE	48	1.65E+00	No	NA	No	Yes
MAGNESIUM	100	1.48E+03	No	NA	Yes	No
MANGANESE	100	2.36E+02	Yes	No	Yes	No
MERCURY	46	1.93E-01	No	NA	No	No
METHYL PARATHION	9	2.13E-02	No	NA	No	Yes
METHYLENE CHLORIDE	30	1.10E-02	No	NA	No	Yes
NAPHTHALENE	43	8.27E-01	No	NA	No	Yes
NICKEL	85	8.97E+00	No	NA	No	No
NITRATE-NITRITE AS N	50	2.83E+00	No	NA	Yes	No
O-XYLENE	35	6.35E-01	No	NA	No	Yes
OCDD	25	3.00E-04	No	NA	Yes	Yes
ORTHOPHOSPHATE	100	6.15E+01	No	NA	No	Yes
PCB-1254	4	2.10E-01	No	NA	No	No
PERCENT MOISTURE	100	8.69E+00	No	NA	Yes	No
pH	100	1.03E+01	No	NA	Yes	No
PHENANTHRENE	83	1.44E+00	No	NA	No	Yes
PHENOL	13	7.54E-01	No	NA	No	Yes
PHORATE	9	1.70E-02	No	NA	No	Yes
POTASSIUM	100	8.08E+02	No	NA	Yes	No
PROPAZINE	8	9.10E+00	No	NA	No	Yes
PYRENE	91	2.25E+00	No	NA	No	Yes
SELENIUM	10	2.45E-01	No	NA	No	No
SILVER	35	1.19E+00	No	NA	No	No
SODIUM	50	2.10E+02	No	NA	Yes	No
STYRENE	4	1.90E-02	No	NA	No	No
SULFATE	80	2.23E+02	No	NA	Yes	No
SULFIDE	20	4.99E+01	No	NA	Yes	No
SULFOTEPP	23	6.31E-02	No	NA	No	Yes
TCDF	50	3.30E-03	No	NA	No	Yes

Table 6-H-6
Screening Process for Chemicals of Potential Concern
Waste Water Treatment Area

Chemical Name	Frequency of Detection (%)	Selected Concentration	Statistically tested against background?[a]	Above Background?[b]	Water Chemistry or Essential Nutrient?	COPC?
TETRACHLOROETHENE	4	6.40E-03	No	NA	No	No
THALLIUM	7	2.76E-01	No	NA	No	No
TINUVIN 327	33	8.67E+00	No	NA	No	Yes
TOLUENE	52	1.81E+00	No	NA	No	Yes
TOTAL ALKALINITY	90	1.34E+03	No	NA	Yes	No
TOTAL ORGANIC CARBON	100	7.65E+03	No	NA	Yes	No
TRCDF	67	4.31E+00	No	NA	No	Yes
VANADIUM	100	1.14E+01	Yes	No	No	No
ZINC	100	7.48E+02	No	NA	No	No

[a]Statistically tested against background values using t-test.
[b]Significant at 95% confidence interval.

Table 6-H-7

Results of Analysis of Surface Soil for Chemicals of Potential Concern[a]
Waste Water Treatment Area

Chemical Name	Number of Detects	Number of Samples	Frequency of Detection %	Mean Conc. (mg/kg)	Standard Deviation	95th% UCL (mg/kg)	Maximum Conc. (mg/kg)	Minimum Conc. (mg/kg)	Selected Conc. (mg/kg)
INORGANICS									
BARIUM	20	20	1.00	2.94E+01	1.50E+01	3.52E+01	7.52E+01	1.32E+01	3.52E+01
CADMIUM	2	12	0.17	2.96E-01	1.74E-01	3.86E-01	7.40E-01	5.70E-01	3.86E-01
COBALT	20	20	1.00	3.43E+00	9.78E-01	3.81E+00	5.30E+00	1.90E+00	3.81E+00
COPPER	23	23	1.00	3.31E+01	3.41E+01	4.53E+01	1.28E+02	3.70E+00	4.53E+01
LEAD	20	20	1.00	4.21E+01	2.97E+01	5.36E+01	1.03E+02	2.40E+00	5.36E+01
MERCURY	6	13	0.46	1.22E-01	1.43E-01	1.93E-01	4.70E-01	6.00E-02	1.93E-01
NICKEL	17	20	0.85	7.27E+00	3.30E+00	8.55E+00	1.32E+01	3.30E+00	8.55E+00
SILVER	8	23	0.35	8.42E-01	9.69E-01	1.19E+00	4.00E+00	6.60E-01	1.19E+00
THALLIUM	1	14	0.07	2.26E-01	1.07E-01	2.76E-01	5.06E-01	5.06E-01	2.76E-01
ZINC	20	20	1.00	2.54E+02	2.71E+02	3.59E+02	8.40E+02	1.52E+01	3.59E+02
CYANIDE	4	17	0.24	8.44E-01	6.96E-01	1.14E+00	1.70E+00	3.70E-01	1.14E+00
ORGANICS									
2-METHYLNAPHTHALENE	4	23	0.17	4.74E-01	4.26E-01	6.26E-01	5.00E-01	2.60E-01	5.00E-01
2-NITROANILINE	2	23	0.09	3.67E+00	5.57E+00	5.66E+00	7.30E-02	5.30E-02	7.30E-02
2,3,7,8-TCDF	4	8	0.50	1.82E-04	4.12E-04	4.58E-04	1.20E-03	2.00E-05	4.58E-04
2,4,5-T	4	20	0.20	2.28E-02	2.15E-02	3.11E-02	4.60E-02	3.10E-02	3.11E-02
2,4-DICHLOROPHENOL	9	23	0.39	6.30E-01	5.86E-01	8.39E-01	2.10E+00	1.30E-01	8.39E-01
3&4-METHYLPHENOL	2	11	0.18	2.11E-01	7.40E-02	2.51E-01	3.80E-01	8.40E-02	2.51E-01
4-CHLOROANILINE	4	23	0.17	1.19E+00	1.46E+00	1.71E+00	5.60E+00	1.20E+00	1.71E+00
4-CHLOROPHENYL-PHENYLETHER	2	23	0.09	7.00E-01	1.14E+00	1.11E+00	2.80E-01	1.90E-01	2.80E-01
4-METHYLPHENOL	2	12	0.17	1.15E+00	1.46E+00	1.91E+00	2.90E-01	2.90E-01	2.90E-01
4,4'-DDD	7	23	0.30	1.01E-01	2.00E-01	1.73E-01	3.20E-01	7.20E-03	1.73E-01
4,4'-DDE	5	23	0.22	8.62E-02	1.93E-01	1.55E-01	2.60E-01	1.10E-02	1.55E-01
4,4'-DDT	2	23	0.09	7.61E-02	1.88E-01	1.43E-01	9.70E-03	7.50E-03	9.70E-03
ACENAPHTHENE	2	23	0.09	7.63E-01	1.12E+00	1.16E+00	1.00E+00	6.00E-01	1.00E+00
ACENAPHTHYLENE	1	23	0.04	7.46E-01	1.13E+00	1.15E+00	4.50E-02	4.50E-02	4.50E-02
ACETONE	5	23	0.22	1.05E-01	8.24E-02	1.34E-01	2.00E-01	3.20E-02	1.34E-01
ACETOPHENONE	2	23	0.09	7.38E-01	1.13E+00	1.14E+00	2.60E-02	2.30E-02	2.60E-02
ALPHA-BHC	2	23	0.09	3.85E-02	9.70E-02	7.32E-02	2.10E-03	1.20E-03	2.10E-03

Table 6-H-7

Results of Analysis of Surface Soil for Chemicals of Potential Concern[a]
Waste Water Treatment Area

Chemical Name	Number of Detects	Number of Samples	Frequency of Detection %	Mean Conc. (mg/kg)	Standard Deviation	95th% UCL (mg/kg)	Maximum Conc. (mg/kg)	Minimum Conc. (mg/kg)	Selected Conc. (mg/kg)
ANILINE	4	23	0.17	8.24E-01	1.12E+00	1.23E+00	1.60E+00	4.20E-01	1.23E+00
ANTHRACENE	3	23	0.13	7.60E-01	1.12E+00	1.16E+00	1.00E+00	6.00E-01	1.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	9	23	0.39	1.17E+00	1.71E+00	1.78E+00	7.20E+00	1.20E-01	1.78E+00
BUTYLBENZYLPHTHALATE	2	23	0.09	7.28E-01	1.14E+00	1.14E+00	1.50E-01	1.50E-01	1.50E-01
CHLOROBENZENE	11	23	0.48	1.28E+00	3.74E+00	2.62E+00	1.30E+01	1.50E-02	2.62E+00
DCDF	3	6	0.50	9.33E-01	1.32E+00	2.02E+00	3.00E+00	4.00E-01	2.02E+00
DIBENZOFURAN	3	23	0.13	5.44E-01	4.45E-01	7.03E-01	1.10E+00	4.00E-01	7.03E-01
DIELDRIN	4	23	0.17	8.23E-02	1.90E-01	1.50E-01	1.70E-01	7.70E-03	1.50E-01
DIETHYLPHTHALATE	4	23	0.17	5.48E-01	5.25E-01	7.36E-01	1.70E+00	7.50E-02	7.36E-01
ENDRIN	2	23	0.09	6.74E-02	1.89E-01	1.35E-01	7.00E-03	2.00E-03	7.00E-03
ENDRIN ALDEHYDE	3	23	0.13	1.21E-01	2.46E-01	2.09E-01	8.20E-01	2.30E-01	2.09E-01
ETHYL PARATHION	2	22	0.09	5.96E-02	7.88E-02	8.86E-02	5.40E-02	2.50E-03	5.40E-02
ETHYLBENZENE	4	23	0.17	2.27E-01	5.98E-01	4.41E-01	2.10E+00	2.40E-02	4.41E-01
FAMPHUR	3	22	0.14	2.52E-01	2.46E-01	3.42E-01	7.70E-02	1.40E-02	7.70E-02
FLUORANTHENE	17	23	0.74	9.40E-01	1.74E+00	1.56E+00	6.90E+00	4.60E-02	1.56E+00
FLUORENE	2	23	0.09	7.25E-01	1.13E+00	1.13E+00	5.10E-01	1.10E-01	5.10E-01
GAMMA-CHLORDANE	9	26	0.35	1.30E+00	3.81E+00	2.58E+00	1.90E+01	3.40E-03	2.58E+00
HEPTACHLOR	2	23	0.09	4.27E-02	9.84E-02	7.79E-02	1.20E-01	4.50E-03	7.79E-02
IRGASAN DP-300	6	13	0.46	4.08E+01	9.17E+01	8.61E+01	3.40E+02	5.60E+00	8.61E+01
M&P-XYLENE	11	23	0.48	8.08E-01	2.35E+00	1.65E+00	8.20E+00	1.00E-02	1.65E+00
METHYL PARATHION	2	22	0.09	1.60E-02	1.46E-02	2.13E-02	3.20E-02	3.80E-03	2.13E-02
METHYLENE CHLORIDE	7	23	0.30	1.20E-01	1.56E-01	1.75E-01	1.10E-02	7.20E-03	1.10E-02
NAPHTHALENE	10	23	0.43	5.94E-01	6.50E-01	8.27E-01	2.50E+00	6.60E-02	8.27E-01
O-XYLENE	8	23	0.35	3.19E-01	8.82E-01	6.35E-01	3.10E+00	7.40E-03	6.35E-01
OCDD	2	8	0.25	2.45E-04	9.27E-05	3.07E-04	3.00E-04	2.00E-04	3.00E-04
ORTHOPHOSPHATE	10	10	1.00	3.10E+01	5.26E+01	6.15E+01	1.40E+02	9.10E-01	6.15E+01
PHENANTHRENE	19	23	0.83	8.94E-01	1.52E+00	1.44E+00	5.20E+00	4.80E-02	1.44E+00
PHENOL	3	23	0.13	5.66E-01	5.25E-01	7.54E-01	1.80E+00	2.70E-01	7.54E-01
PHORATE	2	22	0.09	5.50E-02	7.87E-02	8.39E-02	1.70E-02	1.70E-02	1.70E-02
PROPAZINE	1	13	0.08	5.57E+00	7.15E+00	9.10E+00	2.80E+01	2.80E+01	9.10E+00

Table 6-H-7

Results of Analysis of Surface Soil for Chemicals of Potential Concern[a]
Waste Water Treatment Area

Chemical Name	Number of Detects	Number of Samples	Frequency of Detection %	Mean Conc. (mg/kg)	Standard Deviation	95th% UCL (mg/kg)	Maximum Conc. (mg/kg)	Minimum Conc. (mg/kg)	Selected Conc. (mg/kg)
PYRENE	21	23	0.91	1.47E+00	2.18E+00	2.25E+00	6.80E+00	3.90E-02	2.25E+00
SULFOTEPP	5	22	0.23	4.20E-02	5.75E-02	6.31E-02	1.40E-01	4.00E-03	6.31E-02
TCDF	4	8	0.50	1.22E-03	3.11E-03	3.30E-03	8.90E-03	8.70E-05	3.30E-03
TINUVIN 327	4	12	0.33	4.92E+00	7.24E+00	8.67E+00	2.60E+01	2.30E-01	8.67E+00
TOLUENE	12	23	0.52	9.25E-01	2.48E+00	1.81E+00	8.70E+00	1.40E-02	1.81E+00
TRCDF	4	6	0.67	2.22E+00	2.54E+00	4.31E+00	5.80E+00	1.10E+00	4.31E+00

a- Includes results from sediment sampling in seep

Table 6-H-8
Screening Process for Chemicals of Potential Concern in Seep Sediment
Waste Water Treatment Area

Chemical Name	Frequency of Detection[a]	Selected Concentration	Statistically tested against background?[b]	Above Background?[c]	Essential Nutrient?	COPC?
2,3,7,8-TCDF	100	5.00E-05	No	NA	No	Yes
2-METHYLNAPHTHALENE	20	2.80E-01	No	NA	No	Yes
4,4'-DDD	60	1.34E-02	No	NA	No	Yes
4,4'-DDE	40	1.71E-02	No	NA	No	Yes
4,4'-DDT	20	9.70E-03	No	NA	No	Yes
4-CHLOROPHENYL-PHENYLETHER	40	2.80E-01	No	NA	No	Yes
ACENAPHTHENE	20	1.00E+00	No	NA	No	Yes
ACETONE	20	1.90E-01	No	NA	No	Yes
ALPHA-CHLORDANE	20	4.50E-03	No	NA	No	Yes
ANTHRACENE	20	1.43E+00	No	NA	No	Yes
ARSENIC	100	8.62E+00	Yes	No	No	No
BARIUM	100	4.13E+01	No	NA	No	Yes
BENZO(A)ANTHRACENE	60	2.14E+00	Yes	No	No	No
BENZO(A)PYRENE	60	1.96E+00	Yes	No	No	No
BENZO(B)FLUORANTHENE	100	2.24E+00	Yes	No	No	No
BENZO(G,H,I)PERYLENE	20	1.15E+00	Yes	No	No	No
BENZO(K)FLUORANTHENE	80	9.46E-01	Yes	No	No	No
BERYLLIUM	100	6.88E-01	Yes	No	No	No
BIS(2-ETHYLHEXYL)PHTHALATE	80	1.00E+00	No	NA	No	Yes
BUTYLBENZYLPHTHALATE	20	1.50E-01	No	NA	No	Yes
CADMIUM	33	5.70E-01	No	NA	No	Yes
CALCIUM	100	3.04E+03	No	NA	Yes	No
CHLOROBENZENE	20	1.05E-01	No	NA	No	Yes
CHROMIUM	100	1.01E+01	Yes	No	No	No
CHRYSENE	100	2.27E+00	Yes	No	No	No
COBALT	100	4.47E+00	No	NA	No	Yes
COPPER	100	2.62E+01	No	NA	No	Yes
DCDF	60	2.45E+00	No	NA	No	Yes

Table 6-H-8
Screening Process for Chemicals of Potential Concern in Seep Sediment
Waste Water Treatment Area

Chemical Name	Frequency of Detection[a]	Selected Concentration	Statistically tested against background?[b]	Above Background?[c]	Essential Nutrient?	COPC?
DIBENZ(A,H)ANTHRACENE	20	4.60E-01	Yes	No	No	No
DIBENZOFURAN	20	5.60E-01	No	NA	No	Yes
ENDRIN	40	7.00E-03	No	NA	No	Yes
FLUORANTHENE	100	3.64E+00	No	NA	No	Yes
GAMMA-CHLORDANE	40	1.31E-02	No	NA	No	Yes
INDENO(1,2,3-CD)PYRENE	40	1.25E+00	Yes	No	No	No
IRON	100	8.00E+03	No	NA	Yes	No
KEPONE	20	3.97E-02	No	NA	No	Yes
LEAD	100	9.09E+01	No	NA	No	Yes
M&P-XYLENE	20	1.00E-02	No	NA	No	Yes
MAGNESIUM	100	1.09E+03	No	NA	Yes	No
MANGANESE	100	1.76E+02	Yes	No	No	No
NAPHTHALENE	20	8.20E-01	No	NA	No	Yes
NICKEL	60	8.80E+00	No	NA	No	Yes
OCDD	100	3.00E-04	No	NA	No	Yes
PHENANTHRENE	100	3.32E+00	No	NA	No	Yes
POTASSIUM	100	4.41E+02	No	NA	Yes	No
PYRENE	100	3.57E+00	No	NA	No	Yes
SELENIUM	20	4.50E-01	No	NA	No	No
SODIUM	33	2.66E+02	No	NA	Yes	No
TCDF	40	4.00E-04	No	NA	No	Yes
THALLIUM	20	4.28E-01	No	NA	No	Yes
TINUVIN 327	67	5.10E-01	No	NA	No	Yes
TOLUENE	20	1.73E-01	No	NA	No	Yes
TRCDF	60	5.09E+00	No	NA	No	Yes
VANADIUM	100	1.37E+01	Yes	No	No	No
ZINC	100	2.21E+02	No	NA	No	Yes

a- due to the small number of samples, frequency of detection cannot be used to eliminate chemicals as COPC.

Table 6-H-8
Screening Process for Chemicals of Potential Concern in Seep Sediment
Waste Water Treatment Area

Chemical Name	Frequency of Detection[a]	Selected Concentration	Statistically tested against background?[b]	Above Background?[c]	Essential Nutrient?	COPC?

b- statistically tested against background values using t-test

c- significant at the 95% confidence interval

Table 6-H-9
Results of Analysis of Sediment in Seep
Waste Water Treatment Area

Chemical Name	Number of Detects	Number of Samples	Frequency of Detection(%)	Mean Conc. (mg/kg)	Standard Deviation	95% UCL (mg/kg)	Maximum Conc. (mg/kg)	Minimum Conc. (mg/kg)	Selected Conc. (mg/kg)
INORGANICS									
BARIUM	18	18	100.00	2.92E+01	1.53E+01	3.54E+01	7.52E+01	1.32E+01	3.54E+01
CADMIUM	1	3	33.33	3.70E-01	1.73E-01	6.62E-01	5.70E-01	5.70E-01	5.70E-01
COBALT	5	5	100.00	3.42E+00	1.10E+00	4.47E+00	5.30E+00	2.50E+00	4.47E+00
COPPER	5	5	100.00	1.84E+01	8.22E+00	2.62E+01	3.21E+01	1.11E+01	2.62E+01
LEAD	18	18	100.00	4.08E+01	2.94E+01	5.29E+01	1.03E+02	2.40E+00	5.29E+01
NICKEL	3	5	60.00	6.18E+00	2.75E+00	8.80E+00	1.06E+01	5.50E+00	8.80E+00
THALLIUM	1	5	20.00	2.48E-01	1.89E-01	4.28E-01	5.06E-01	5.06E-01	4.28E-01
ZINC	5	5	100.00	1.57E+02	6.68E+01	2.21E+02	2.36E+02	5.63E+01	2.21E+02
ORGANICS									
2-METHYLNAPHTHALENE	1	5	20.00	5.79E-01	3.10E-01	8.74E-01	2.80E-01	2.80E-01	2.80E-01
2,3,7,8-TCDF	2	2	100.00	3.50E-05	2.12E-05	1.30E-04	5.00E-05	2.00E-05	5.00E-05
4-CHLOROPHENYL-PHENYLETHER	2	5	40.00	3.43E-01	2.31E-01	5.63E-01	2.80E-01	1.90E-01	2.80E-01
4,4'-DDD	3	5	60.00	8.77E-03	4.83E-03	1.34E-02	1.40E-02	7.20E-03	1.34E-02
4,4'-DDE	2	5	40.00	1.00E-02	7.40E-03	1.71E-02	2.20E-02	1.10E-02	1.71E-02
4,4'-DDT	1	5	20.00	8.93E-03	6.28E-03	1.49E-02	9.70E-03	9.70E-03	9.70E-03
ACETONE	1	5	20.00	1.41E-01	7.16E-02	2.09E-01	1.90E-01	1.90E-01	1.90E-01
ALPHA-CHLORDANE	1	5	20.00	4.30E-03	3.25E-03	7.40E-03	4.50E-03	4.50E-03	4.50E-03
ACENAPHTHENE	1	5	20.00	7.23E-01	3.03E-01	1.01E+00	1.00E+00	1.00E+00	1.00E+00
ANTHRACENE	1	5	20.00	8.83E-01	5.75E-01	1.43E+00	1.80E+00	1.80E+00	1.43E+00
BIS(2-ETHYLHEXYL)PHTHALATE	4	5	80.00	7.66E-01	2.50E-01	1.00E+00	1.10E+00	4.80E-01	1.00E+00
BUTYLBENZYLPHTHALATE	1	5	20.00	5.53E-01	3.44E-01	8.81E-01	1.50E-01	1.50E-01	1.50E-01
CHLOROBENZENE	1	5	20.00	5.65E-02	5.12E-02	1.05E-01	1.20E-01	1.20E-01	1.05E-01
DCDF	3	5	60.00	1.12E+00	1.39E+00	2.45E+00	3.00E+00	4.00E-01	2.45E+00
DIBENZOFURAN	1	5	20.00	6.35E-01	2.64E-01	8.87E-01	5.60E-01	5.60E-01	5.60E-01
ENDRIN	2	5	40.00	5.23E-03	2.89E-03	7.98E-03	7.00E-03	2.00E-03	7.00E-03
FLUORANTHENE	5	5	100	1.40E+00	2.35E+00	3.64E+00	5.60E+00		3.64E+00
GAMMA-CHLORDANE	2	5	40.00	7.26E-03	6.13E-03	1.31E-02	1.70E-02	3.40E-03	1.31E-02
KEPONE	1	5	20.00	1.95E-02	2.12E-02	3.97E-02	5.70E-02	5.70E-02	3.97E-02

Table 6-H-9
Results of Analysis of Sediment in Seep
Waste Water Treatment Area

Chemical Name	Number of Detects	Number of Samples	Frequency of Detection(%)	Mean Conc. (mg/kg)	Standard Deviation	95% UCL (mg/kg)	Maximum Conc. (mg/kg)	Minimum Conc. (mg/kg)	Selected Conc. (mg/kg)
NAPHTHALENE	1	5	20.00	6.87E-01	2.71E-01	9.45E-01	8.20E-01	8.20E-01	8.20E-01
M&P-XYLENE	1	5	20.00	5.27E-02	4.27E-02	9.34E-02	1.00E-02	1.00E-02	1.00E-02
OCDD	2	2	100.00	2.50E-04	7.07E-05	5.66E-04	3.00E-04	2.00E-04	3.00E-04
PHENANTHRENE	5	5	100.00	1.18E+00	2.25E+00	3.32E+00	5.20E+00	4.80E-02	3.32E+00
PYRENE	5	5	100.00	1.35E+00	2.32E+00	3.57E+00	5.50E+00	1.14E-01	3.57E+00
TCDF	2	5	40.00	3.50E-04	7.07E-05	4.17E-04	4.00E-04	3.00E-04	4.00E-04
TINUVIN 327	2	3	66.67	1.75E+00	2.39E+00	5.77E+00	5.10E-01	2.30E-01	5.10E-01
TOLUENE	1	5	20.00	8.05E-02	9.65E-02	1.73E-01	2.40E-01	2.40E-01	1.73E-01
TRCDF	3	5	60.00	2.44E+00	2.78E+00	5.09E+00	5.80E+00	1.40E+00	5.09E+00

Table 6-H-10
Results of Surface Water Analysis in Seep
Waste Water Treatment Area

Chemical Name	Number of Detects	Number of Samples	Frequency of Detection(%)	Mean Conc. (mg/kg)	Standard Deviation	95% UCL (mg/kg)	Maximum Conc. (mg/kg)	Minimum Conc. (mg/kg)	Selected Conc. (mg/kg)
INORGANICS									
BARIUM	4	4	1	4.08E+01	2.22E+01	6.69E+01	7.30E+01	2.30E+01	6.69E+01
LEAD	3	4	0.75	1.12E+01	1.22E+01	2.55E+01	2.90E+01	6.40E+00	2.55E+01
ZINC	4	4	1	4.80E+01	1.31E+01	6.35E+01	6.50E+01	3.30E+01	6.35E+01
ORGANICS									
2-HEXANONE	1	2	0.5	6.00E+00	1.41E+00	1.23E+01	7.00E+00	7.00E+00	7.00E+00
4,4'-DDE	1	2	0.5	8.45E-03	5.02E-03	3.09E-02	1.20E-02	1.20E-02	1.20E-02
ALDRIN	1	2	0.5	1.05E-02	7.85E-03	4.55E-02	1.60E-02	1.60E-02	1.60E-02
KEPONE	1	2	0.5	4.20E-02	5.24E-02	2.76E-01	7.90E-02	7.90E-02	7.90E-02

Table 6-H-11
Screening Process for Chemicals of Potential Concern
Warwick Area

Chemical Name	Frequency of Detection (%)	Selected Concentration (mg/kg)	Statistically tested against background?[a]	Above Background?[b]	Water Chemistry or Essential Nutrient?	COPC?
1,1-BIPHENYL	50.00	1.90E+00	No	NA	No	Yes
1,1-DICHLOROETHANE	3.13	4.40E-02	No	NA	No	No
1,2-DICHLOROBENZENE	3.23	1.80E-01	No	NA	No	No
1,4-DICHLOROBENZENE	6.45	3.90E-02	No	NA	No	Yes
2,4,5-T	3.23	9.50E-03	No	NA	No	No
2,4,5-TP (SILVEX)	12.90	5.27E-02	No	NA	No	Yes
2-BUTANONE	6.25	1.22E-01	No	NA	No	Yes
2-METHYLNAPHTHALENE	19.35	3.60E-01	No	NA	No	Yes
2-NITROANILINE	6.45	7.00E+00	No	NA	No	Yes
3&4-METHYLPHENOL	16.67	2.20E-02	No	NA	No	Yes
3,3'-DIMETHYLBENZIDINE	3.23	4.23E+00	No	NA	No	No
4,4'-DDD	6.25	1.34E-01	No	NA	No	Yes
4,4'-DDE	18.18	1.49E-01	No	NA	No	Yes
4,4'-DDT	28.13	2.52E-01	No	NA	No	Yes
4-CHLOROANILINE	32.26	2.01E+00	No	NA	No	Yes
ACENAPHTHENE	9.68	1.60E-01	No	NA	No	Yes
ACENAPHTHYLENE	9.68	1.10E-01	No	NA	No	Yes
ACETONE	3.13	1.26E-01	No	NA	No	No
ALDRIN	9.09	2.08E-01	No	NA	No	Yes
ALPHA-BHC	18.18	1.83E-01	No	NA	No	Yes
ALPHA-CHLORDANE	9.38	7.70E-02	No	NA	No	Yes
AMMONIA AS N	20.00	1.42E+00	No	NA	Yes	No
ANTHRACENE	32.26	3.20E-01	No	NA	No	Yes
ANTIMONY	26.09	5.35E+00	All data rejected	Yes	No	Yes
ARSENIC	100.00	1.03E+01	Yes	No	No	No
BARIUM	100.00	1.83E+02	No	NA	No	Yes
BENZENE	3.13	3.40E-02	No	NA	No	No
BENZO(A)ANTHRACENE	48.39	1.36E+00	No	NA	No	Yes
BENZO(A)PYRENE	41.94	1.62E+00	Yes	No	No	No
BENZO(B)FLUORANTHENE	45.16	1.65E+00	No	NA	No	Yes
BENZO(G,H,I)PERYLENE	29.03	1.20E+00	No	NA	No	Yes
BENZO(K)FLUORANTHENE	41.94	1.76E+00	No	NA	No	Yes

Table 6-H-11
Screening Process for Chemicals of Potential Concern
Warwick Area

Chemical Name	Frequency of Detection (%)	Selected Concentration (mg/kg)	Statistically tested against background?[a]	Above Background?[b]	Water Chemistry or Essential Nutrient?	COPC?
BERYLLIUM	100.00	8.18E-01	Yes	No	No	Yes
BETA-BHC	6.25	9.60E-03	No	NA	No	Yes
BICARBONATE ALKALINITY	68.00	3.07E+02	No	NA	Yes	No
BIS(2-CHLOROETHYL)ETHER	6.45	4.30E-01	No	NA	No	Yes
BIS(2-ETHYLHEXYL)PHTHALATE	45.16	2.00E+01	No	NA	No	Yes
BUTYLBENZYLPHTHALATE	19.35	7.80E-01	No	NA	No	Yes
CADMIUM	48.39	2.22E+00	Yes	Yes	No	Yes
CALCIUM	100.00	1.74E+03	No	NA	Yes	No
CATION EXCHANGE CAPACITY	100.00	8.81E+00	No	NA	Yes	No
CHLORIDE	52.00	1.01E+02	No	NA	Yes	No
CHLOROBENZENE	28.13	6.68E-01	No	NA	No	Yes
CHLOROFORM	3.13	4.80E-02	No	NA	No	No
CHROMIUM	100.00	1.01E+02	Yes	Yes	No	Yes
CHRYSENE	45.16	1.47E+00	No	NA	No	Yes
COBALT	100.00	5.01E+00	No	NA	No	Yes
COPPER	96.77	2.25E+02	No	NA	No	Yes
CYANIDE	30.00	2.70E+00	No	NA	No	Yes
DELTA-BHC	9.09	1.23E-01	No	NA	No	Yes
DI-N-BUTYLPHTHALATE	3.23	5.70E-02	No	NA	No	No
DI-N-OCTYLPHTHALATE	25.81	4.12E+00	No	NA	No	Yes
DIBENZ(A,H)ANTHRACENE	9.68	1.30E-01	No	NA	No	Yes
DIBENZOFURAN	9.68	2.00E-01	No	NA	No	Yes
DIELDRIN	15.63	1.61E-01	No	NA	No	Yes
DINOSEB	3.23	6.37E-02	No	NA	No	No
DISULFOTON	10.00	7.70E-03	No	NA	No	Yes
ENDOSULFAN I	9.38	1.80E-02	No	NA	No	Yes
ENDOSULFAN II	3.13	1.80E-02	No	NA	No	No
ENDOSULFAN SULFATE	9.38	2.90E-01	No	NA	No	Yes
ENDRIN	18.75	1.41E-01	No	NA	No	Yes
ENDRIN ALDEHYDE	15.63	4.44E-01	No	NA	No	Yes
ETHYL PARATHION	6.67	6.40E-03	No	NA	No	Yes
ETHYLBENZENE	3.13	2.70E-02	No	NA	No	No

Table 6-H-11
Screening Process for Chemicals of Potential Concern
Warwick Area

Chemical Name	Frequency of Detection (%)	Selected Concentration (mg/kg)	Statistically tested against background?[a]	Above Background?[b]	Water Chemistry or Essential Nutrient?	COPC?
FLUORANTHENE	54.84	1.58E+00	No	NA	No	Yes
FLUORENE	22.58	2.30E-01	No	NA	No	Yes
GAMMA-BHC	6.25	1.70E-03	No	NA	No	Yes
GAMMA-CHLORDANE	28.13	1.49E-01	No	NA	No	Yes
HEPTACHLOR	3.13	3.20E-03	No	NA	No	No
HEPTACHLOR EPOXIDE	21.88	1.92E-01	No	NA	No	Yes
INDENO(1,2,3-CD)PYRENE	25.81	8.60E-01	No	NA	No	Yes
IRON	100.00	1.36E+04	No	NA	Yes	No
ISODRIN	6.25	1.55E-01	No	NA	No	Yes
KEPONE	3.13	1.51E-01	No	NA	No	No
LEAD	96.30	1.18E+02	No	NA	No	Yes
M&P-XYLENE	25.00	5.46E-02	No	NA	No	Yes
MAGNESIUM	100.00	2.07E+03	No	NA	Yes	No
MANGANESE	100.00	2.45E+02	Yes	No	Yes	No
MERCURY	48.39	3.11E-01	No	NA	No	Yes
METHOXYCHLOR	38.24	2.32E+02	No	NA	No	Yes
METHYLENE CHLORIDE	9.38	2.50E-02	No	NA	No	Yes
N-NITROSO-DI-N-PROPYLAMINE	3.23	1.70E-01	No	NA	No	No
NAPHTHALENE	51.61	1.58E+00	No	NA	No	Yes
NICKEL	90.32	2.76E+01	No	NA	No	Yes
NITRATE-NITRITE AS N	88.00	2.09E+00	No	NA	Yes	No
NITROBENZENE	6.45	2.13E+00	No	NA	No	Yes
O-XYLENE	12.50	4.60E-02	No	NA	No	Yes
ORTHOPHOSPHATE	80.00	6.88E+00	No	NA	No	Yes
PCB-1248	8.82	1.52E+01	No	NA	No	Yes
PCB-1254	46.88	5.19E+00	No	NA	No	Yes
PERCENT MOISTURE	100.00	1.52E+01	No	NA	Yes	No
PH	100.00	6.40E+00	No	NA	Yes	No
PHENACETIN	3.23	1.00E+00	No	NA	No	No
PHENANTHRENE	54.84	1.13E+00	No	NA	No	Yes
PHENOL	9.68	8.90E-01	No	NA	No	Yes
PHORATE	3.33	9.60E-03	No	NA	No	No

Table 6-H-11
Screening Process for Chemicals of Potential Concern
Warwick Area

Chemical Name	Frequency of Detection (%)	Selected Concentration (mg/kg)	Statistically tested against background?[a]	Above Background?[b]	Water Chemistry or Essential Nutrient?	COPC?
POTASSIUM	100.00	1.06E+03	No	NA	Yes	No
PROPAZINE	4.00	1.20E+01	No	NA	No	No
PYRENE	58.06	1.60E+00	No	NA	No	Yes
SAFROLE	12.90	4.05E+00	No	NA	No	Yes
SODIUM	60.00	1.38E+02	No	NA	Yes	No
SULFATE	44.00	1.97E+02	No	NA	Yes	No
SULFIDE	20.00	2.07E+02	No	NA	Yes	No
SULFOTEPP	3.23	4.10E-03	No	NA	No	No
TETRACHLOROETHENE	28.13	2.52E-01	No	NA	No	Yes
THIONAZIN	3.33	5.80E-03	No	NA	No	No
TIN	19.35	9.75E+00	No	NA	No	Yes
TINUVIN 327	32.00	9.27E+00	No	NA	No	Yes
TOLUENE	56.25	3.55E-01	No	NA	No	Yes
TOTAL ALKALINITY	68.00	3.07E+02	No	NA	Yes	No
TOTAL ORGANIC CARBON	100.00	1.56E+04	No	NA	Yes	No
TRICHLOROETHENE	6.25	5.97E-02	No	NA	No	Yes
VANADIUM	70.37	1.22E+01	No	NA	No	Yes
ZINC	90.32	3.91E+03	Yes	Yes	No	Yes

a- statistically tested against background values using t-test

b- significant at the 95% confidence interval

Table 6-H-12
Results of Analysis of Surface Soil for Chemicals of Potential Concern
Warwick Area

Chemical Name	Number of Detects	Number of Samples	Frequency of Detection(%)	Mean Conc. (mg/kg)	Standard Deviation	95th% UCL (mg/kg)	Maximum Conc. (mg/kg)	Minimum Conc. (mg/kg)	Selected Conc. (mg/kg)
INORGANICS									
ANTIMONY	6	23	26.09	2.27E+00	8.63E+00	5.35E+00	4.18E+01	8.60E-01	5.35E+00
BARIUM	31	31	100.00	1.14E+02	2.28E+02	1.83E+02	1.27E+03	7.30E+00	1.83E+02
BERYLLIUM[a]	31	31	100.00	6.22E-01	NA(a)	8.18E-01	2.00E+00	3.20E-01	8.18E-01
CADMIUM	15	31	48.39	1.56E+00	2.17E+00	2.22E+00	6.90E+00	2.80E-01	2.22E+00
CHROMIUM	31	31	100.00	6.82E+01	1.08E+02	1.01E+02	3.57E+02	1.30E+00	1.01E+02
COBALT	27	27	100.00	4.43E+00	1.78E+00	5.01E+00	7.80E+00	1.00E+00	5.01E+00
COPPER	30	31	96.77	1.18E+02	3.52E+02	2.25E+02	1.96E+03	2.90E+00	2.25E+02
LEAD	26	27	96.30	8.40E+01	1.03E+02	1.18E+02	4.28E+02	2.80E+00	1.18E+02
MERCURY	15	31	48.39	2.26E-01	2.79E-01	3.11E-01	9.40E-01	1.30E-01	3.11E-01
NICKEL[a]	28	31	90.32	8.92E+00	NA(a)	2.63E+01	1.99E+02	2.70E+00	2.63E+01
TIN	6	31	19.35	7.42E+00	7.65E+00	9.75E+00	3.78E+01	1.47E+01	9.75E+00
VANADIUM	19	27	70.37	1.05E+01	5.27E+00	1.22E+01	2.20E+01	1.70E+00	1.22E+01
ZINC	28	31	90.32	2.54E+03	4.51E+03	3.91E+03	1.61E+04	2.48E+01	3.91E+03
CYANIDE	9	30	30.00	1.86E+00	2.71E+00	2.70E+00	9.40E+00	8.00E-01	2.70E+00
ORGANICS									
1,1-BIPHENYL	1	2	50.00	1.04E+00	1.22E+00	6.48E+00	1.90E+00	1.90E+00	1.90E+00
1,4-DICHLOROBENZENE	2	31	6.45	1.39E+00	2.13E+00	2.04E+00	3.90E-02	3.20E-02	3.90E-02
2-BUTANONE	2	32	6.25	1.03E-01	6.37E-02	1.22E-01	2.30E-01	1.80E-01	1.22E-01
2-METHYLNAPHTHALENE	6	31	19.35	1.34E+00	2.15E+00	2.00E+00	3.60E-01	1.40E-02	3.60E-01
2-NITROANILINE	2	31	6.45	7.15E+00	1.07E+01	1.04E+01	7.00E+00	9.80E-01	7.00E+00
2,4,5-TP (SILVEX)	4	31	12.90	3.14E-02	6.98E-02	5.27E-02	3.40E-01	2.00E-02	5.27E-02
3&4-METHYLPHENOL	1	6	16.67	1.52E-01	6.40E-02	2.05E-01	2.20E-02	2.20E-02	2.20E-02
4-CHLOROANILINE	10	31	32.26	1.44E+00	1.86E+00	2.01E+00	7.40E+00	3.10E-01	2.01E+00
4,4'-DDD	2	32	6.25	6.54E-02	2.29E-01	1.34E-01	4.10E-01	1.80E-02	1.34E-01
4,4'-DDE	6	33	18.18	7.74E-02	2.41E-01	1.49E-01	6.50E-01	4.00E-03	1.49E-01
4,4'-DDT	9	32	28.13	1.19E-01	4.46E-01	2.52E-01	5.10E-01	9.60E-04	2.52E-01
ALDRIN	3	33	9.09	1.12E-01	3.25E-01	2.08E-01	1.10E+00	1.30E-01	2.08E-01
ALPHA-BHC	6	33	18.18	9.49E-02	2.97E-01	1.83E-01	1.20E+00	1.00E-03	1.83E-01

Table 6-H-12
Results of Analysis of Surface Soil for Chemicals of Potential Concern
Warwick Area

Chemical Name	Number of Detects	Number of Samples	Frequency of Detection(%)	Mean Conc. (mg/kg)	Standard Deviation	95th% UCL (mg/kg)	Maximum Conc. (mg/kg)	Minimum Conc. (mg/kg)	Selected Conc. (mg/kg)
ALPHA-CHLORDANE	3	32	9.38	5.50E-02	2.20E-01	1.21E-01	7.70E-02	4.00E-03	7.70E-02
ACENAPHTHENE	3	31	9.68	1.37E+00	2.13E+00	2.02E+00	1.60E-01	1.60E-02	1.60E-01
ACENAPHTHYLENE	3	31	9.68	1.39E+00	2.13E+00	2.04E+00	1.10E-01	6.10E-02	1.10E-01
ANTHRACENE	10	31	32.26	1.31E+00	2.16E+00	1.97E+00	3.20E-01	3.10E-02	3.20E-01
BENZO(A)ANTHRACENE	15	31	48.39	9.72E-01	1.26E+00	1.36E+00	1.60E+00	1.40E-01	1.36E+00
BENZO(B)FLUORANTHENE	14	31	45.16	1.23E+00	1.36E+00	1.65E+00	2.80E+00	4.20E-02	1.65E+00
BENZO(G,H,I)PERYLENE	9	31	29.03	1.45E+00	2.10E+00	2.09E+00	1.20E+00	6.40E-02	1.20E+00
BENZO(K)FLUORANTHENE	13	31	41.94	1.30E+00	1.51E+00	1.76E+00	3.60E+00	6.20E-02	1.76E+00
BETA-BHC	2	32	6.25	5.23E-02	2.20E-01	1.18E-01	9.60E-03	9.10E-03	9.60E-03
BIS(2-CHLOROETHYL)ETHER	2	31	6.45	1.38E+00	2.13E+00	2.03E+00	4.30E-01	3.30E-01	4.30E-01
BIS(2-ETHYLHEXYL)PHTHALATE	14	31	45.16	1.05E+01	3.14E+01	2.00E+01	1.40E+02	1.00E-01	2.00E+01
BUTYLBENZYLPHTHALATE	6	31	19.35	1.39E+00	2.13E+00	2.04E+00	7.80E-01	4.60E-02	7.80E-01
CHLOROBENZENE	9	32	28.13	4.07E-01	8.70E-01	6.68E-01	3.60E+00	5.70E-03	6.68E-01
CHRYSENE	14	31	45.16	1.08E+00	1.29E+00	1.47E+00	2.30E+00	1.20E-01	1.47E+00
DELTA-BHC	3	33	9.09	5.82E-02	2.20E-01	1.23E-01	2.60E-01	2.00E-03	1.23E-01
DIBENZ(A,H)ANTHRACENE	3	31	9.68	1.38E+00	2.13E+00	2.03E+00	1.30E-01	8.30E-02	1.30E-01
DI-N-OCTYLPHTHALATE	8	31	25.81	2.52E+00	5.25E+00	4.12E+00	2.30E+01	4.00E-02	4.12E+00
DIBENZOFURAN	3	31	9.68	1.36E+00	2.14E+00	2.01E+00	2.00E-01	5.20E-02	2.00E-01
DIELDRIN	5	32	15.63	8.06E-02	2.67E-01	1.61E-01	9.10E-01	1.90E-03	1.61E-01
DISULFOTON	3	30	10.00	5.69E-02	2.46E-02	6.45E-02	7.70E-03	5.90E-03	7.70E-03
ENDOSULFAN I	3	32	9.38	5.24E-02	2.20E-01	1.18E-01	1.80E-02	1.00E-02	1.80E-02
ENDOSULFAN SULFATE	3	32	9.38	2.46E-01	1.06E+00	5.64E-01	2.90E-01	7.50E-03	2.90E-01
ENDRIN	6	32	18.75	7.02E-02	2.36E-01	1.41E-01	5.40E-01	3.10E-03	1.41E-01
ENDRIN ALDEHYDE	5	32	15.63	2.21E-01	7.45E-01	4.44E-01	3.50E+00	2.10E-03	4.44E-01
ETHYL PARATHION	2	30	6.67	3.59E-02	1.29E-02	3.99E-02	6.40E-03	5.60E-03	6.40E-03
FLUORANTHENE	17	31	54.84	1.23E+00	1.15E+00	1.58E+00	3.70E+00	3.80E-02	1.58E+00
FLUORENE	7	31	22.58	1.34E+00	2.15E+00	2.00E+00	2.30E-01	3.50E-02	2.30E-01
GAMMA-BHC	2	32	6.25	5.18E-02	2.20E-01	1.18E-01	1.70E-03	1.40E-03	1.70E-03
GAMMA-CHLORDANE	9	32	28.13	7.83E-02	2.34E-01	1.49E-01	5.00E-01	4.00E-03	1.49E-01

Table 6-H-12
Results of Analysis of Surface Soil for Chemicals of Potential Concern
Warwick Area

Chemical Name	Number of Detects	Number of Samples	Frequency of Detection(%)	Mean Conc. (mg/kg)	Standard Deviation	95th% UCL (mg/kg)	Maximum Conc. (mg/kg)	Minimum Conc. (mg/kg)	Selected Conc. (mg/kg)
HEPTACHLOR EPOXIDE	7	32	21.88	1.02E-01	3.00E-01	1.92E-01	1.20E+00	2.20E-03	1.92E-01
INDENO(1,2,3-CD)PYRENE	8	31	25.81	1.41E+00	2.12E+00	2.06E+00	8.60E-01	7.00E-02	8.60E-01
ISODRIN	2	32	6.25	7.69E-02	2.61E-01	1.55E-01	8.50E-01	7.20E-03	1.55E-01
M&P-XYLENE	8	32	25.00	4.56E-02	3.00E-02	5.46E-02	7.00E-02	6.50E-03	5.46E-02
METHOXYCHLOR	13	34	38.24	1.10E+02	4.17E+02	2.32E+02	2.20E+03	4.60E-02	2.32E+02
METHYLENE CHLORIDE	3	32	9.38	2.39E-01	3.91E-01	3.56E-01	2.50E-02	1.10E-02	2.50E-02
NAPHTHALENE	16	31	51.61	1.13E+00	1.48E+00	1.58E+00	3.50E+00	3.60E-02	1.58E+00
NITROBENZENE	2	31	6.45	1.48E+00	2.13E+00	2.13E+00	2.90E+00	4.80E-01	2.13E+00
O-XYLENE	4	32	12.50	4.45E-02	3.06E-02	5.37E-02	4.60E-02	1.70E-02	4.60E-02
ORTHOPHOSPHATE	20	25	80.00	4.57E+00	6.76E+00	6.88E+00	2.90E+01	3.60E-01	6.88E+00
PCB-1248	3	34	8.82	6.89E+00	2.84E+01	1.52E+01	1.60E+02	8.10E+00	1.52E+01
PCB-1254	15	32	46.88	2.86E+00	7.78E+00	5.19E+00	3.60E+01	3.20E-02	5.19E+00
Total PCB's									1.80E+01
PHENANTHRENE	17	31	54.84	8.67E-01	8.58E-01	1.13E+00	1.70E+00	1.80E-01	1.13E+00
PHENOL	3	31	9.68	1.18E+00	1.86E+00	1.75E+00	8.90E-01	3.50E-01	8.90E-01
PYRENE	18	31	58.06	1.28E+00	1.08E+00	1.60E+00	3.00E+00	5.30E-02	1.60E+00
SAFROLE	4	31	12.90	2.46E+00	5.21E+00	4.05E+00	2.80E+01	7.00E-01	4.05E+00
TETRACHLOROETHENE	9	32	28.13	1.26E-01	4.19E-01	2.52E-01	2.40E+00	9.00E-03	2.52E-01
TINUVIN 327	8	25	32.00	6.34E+00	8.56E+00	9.27E+00	1.80E+01	5.70E-01	9.27E+00
TOLUENE	18	32	56.25	2.32E-01	4.10E-01	3.55E-01	1.80E+00	6.90E-03	3.55E-01
TRICHLOROETHENE	2	32	6.25	4.97E-02	3.35E-02	5.97E-02	1.30E-01	4.10E-02	5.97E-02

[a]Compound is log normally distributed. Concentration is the geometric mean.

Table 6-H-13
Physical Properties of Analytes

Chemical Name	Octanol-Water Partition Coefficient (Log K_{ow})	Soil to Plant Transfer Coefficient (B_r)	Calculation of Log (BCF)	Bioconcentration Factor (BCF)
1,1-BIPHENYL	3.95		2.772	5.92E+02
1,4-DICHLOROBENZENE	3.39		2.3464	2.22E+02
2-METHYLNAPHTHALENE	3.86		2.7036	5.05E+02
2,3,7,8-TCDF	6.37		4.6112	4.09E+04
2,4,5-T	3.13		2.1488	1.41E+02
2,4,5-TP (SILVEX)	3.93		2.7568	5.71E+02
2,4-D	2.81		1.9056	8.05E+01
2,4-DICHLOROPHENOL	3.23		2.2248	1.68E+02
2-BUTANONE	1.74		1.0924	1.24E+01
2-HEXANONE	1.38		0.8188	6.59E+00
2-NITROANILINE	1.79		1.1304	1.35E+01
3&4-METHYLPHENOL	1.96		1.2596	1.82E+01
4,4'-DDD	5.91		4.2616	1.83E+04
4,4'-DDE	5.69		4.0944	1.24E+04
4,4'-DDT	5.98		4.3148	2.06E+04
4-CHLOROANILINE	1.83		1.1608	1.45E+01
4-CHLOROPHENYL-PHENYLETHER	4.08		2.8708	7.43E+02
ACENAPHTHENE	4.17		2.9392	8.69E+02
ACENAPHTHYLENE	3.92		2.7492	5.61E+02
ACETONE	-0.24		-0.4124	3.87E-01
ACETOPHENONE	1.68		1.0468	1.11E+01
ALDRIN	3.01		2.0576	1.14E+02
ALPHA-BHC	3.9		2.734	5.42E+02
ALPHA-CHLORDANE	4.78		3.4028	2.53E+03
ANILINE	0.9		0.454	2.84E+00
ANTHRACENE	4.4		3.114	1.30E+03
ANTIMONY		0.03		1.00E+00
BARIUM		0.015		no data
BENZO(A)ANTHRACENE	5.6		4.026	1.06E+04
BENZO(B)FLUORANTHENE	6.57		4.7632	5.80E+04
BENZO(G,H,I)PERYLENE	7.23		5.2648	1.84E+05
BENZO(K)FLUORANTHENE	6.84		4.9684	9.30E+04
BERYLLIUM		0.0015		1.90E+01
BETA-BHC	3.9		2.734	5.42E+02
BIS(2-CHLOROETHYL)ETHER	1.5		0.91	8.13E+00
BIS(2-ETHYLHEXYL)PHTHALATE	5.61		4.0336	1.08E+04
BUTYLBENZYLPHTHALATE	4.47		3.1672	1.47E+03
CADMIUM		0.15		8.10E+01
CHLOROBENZENE	2.84		1.9284	8.48E+01

Table 6-H-13
Physical Properties of Analytes

Chemical Name	Octanol-Water Partition Coefficient (Log K_{ow})	Soil to Plant Transfer Coefficient (B_r)	Calculation of Log (BCF)	Bioconcentration Factor (BCF)
CHROMIUM		.0045		1.60E+01
CHRYSENE	5.6		4.026	1.06E+04
COBALT		.007		no data
COPPER		0.25		2.00E+02
CYANIDE	-0.25		0	0.00E+00
DCDF	6.37		4.6112	4.09E+04
DELTA-BHC	4.1		2.886	7.69E+02
DI-N-BUTYLPHTHALATE	5.6		4.026	1.06E+04
DI-N-OCTYLPHTHALATE	5.22		3.7372	5.46E+03
DIBENZ(a,h)ANTHRACENE	5.97		4.3072	2.03E+04
DIBENZOFURAN	4.12		2.9012	7.97E+02
DIELDRIN	3.5		2.43	2.69E+02
DIETHYLPHTHALATE	2.46		1.6396	4.36E+01
DINOSEB	3.66		2.5516	3.56E+02
DISULFOTON	4.02		2.8252	6.69E+02
ENDOSULFAN I	3.55		2.468	2.94E+02
ENDOSULFAN SULFATE	3.89		2.7264	5.33E+02
ENDRIN	5.6		4.026	1.06E+04
ENDRIN ALDEHYDE	5.6		4.026	1.06E+04
ETHYL PARATHION	3.76		2.6276	4.24E+02
ETHYLBENZENE	3.15		2.164	1.46E+02
FAMPHUR	2.289		1.50964	3.23E+01
FLUORANTHENE	5.33		3.8208	6.62E+03
FLUORENE	4.18		2.9468	8.85E+02
GAMMA-BHC	3.71		2.5896	3.89E+02
GAMMA-CHLORDANE	5.58		4.0108	1.03E+04
HEPTACHLOR	5.19		3.7144	5.18E+03
HEPTACHLOR EPOXIDE	2.7		1.822	6.64E+01
INDENO(1,2,3-CD)PYRENE	7.66		5.5916	3.90E+05
IRGASAN DP-300	data gap			no data
ISODRIN	5.09		3.6384	4.35E+03
KEPONE	2		1.29	1.95E+01
LEAD		0.009		
M&P-XYLENE	3.2		2.202	1.59E+02
MERCURY		0.2		5.50E+03
METHOXYCHLOR	4.3		3.038	1.09E+03
METHYL PARATHION	1.91		1.2216	1.67E+01
METHYLENE CHLORIDE	1.3		0.758	5.73E+00
NAPHTHALENE	3.36		2.3236	2.11E+02

Table 6-H-13
Physical Properties of Analytes

Chemical Name	Octanol-Water Partition Coefficient (Log K_{ow})	Soil to Plant Transfer Coefficient (B_r)	Calculation of Log (BCF)	Bioconcentration Factor (BCF)
NICKEL		0.06		4.70E+01
NITROBENZENE	1.91		1.2216	1.67E+01
O-XYLENE	2.95		2.012	1.03E+02
OCDD	6.37		4.6112	4.09E+04
ORTHOPHOSPHATE	data gap			no data
PCB-1248	9.21		6.7696	5.88E+06
PCB-1254	9.21		6.7696	5.88E+06
PCB-1260	9.21		6.7696	5.88E+06
PHENANTHRENE	4.46		3.1596	1.44E+03
PHENOL	1.46		0.8796	7.58E+00
PHORATE	2.92		1.9892	9.75E+01
PROPAZINE	2.93		1.9968	9.93E+01
PYRENE	5.18		3.7068	5.09E+03
SAFROLE	2.53		1.6928	4.93E+01
SELENIUM		0.025		
SILVER		0.1		3.08E+03
SULFOTEPP	3.83		2.6808	4.80E+02
TCDF	6.37		4.6112	4.09E+04
TETRACHLOROETHENE	2.53		1.6928	4.93E+01
THALLIUM		0.004		no data
TINUVIN 327	6		4.33	2.14E+04
TOLUENE	2.69		1.8144	6.52E+01
TRCDF	6.37		4.6112	4.09E+04
TRICHLOROETHENE	2.38		1.5788	3.79E+01
TRICHLOROFLUOROMETHANE	2.53		1.6928	4.93E+01
ZINC		0.9		4.70E+01

Table 6-H-14
Toxicity Reference Values for Mammal and Avian Receptors

Chemical Name	Test Species	Species Type	Toxicity Endpoint *	Endpoint Value (mg/kg)	Uncertainty Factor	Toxicity Reference Value (mg/kg)	Reference
INORGANICS							
antimony	rat	mammal	LD-50	7.00E+03	100	7.00E+01	Lewis, 1992
barium	rat	mammal	NOEL	5.00E+00	1	5.00E+00	Schroeder and Mitchener, 1975
beryllium	rat	mammal	NOAEL	5.40E-01	1	5.40E-01	IRIS database
cadmium	mouse	mammal	LD-50	8.90E+02	100	8.90E+00	BEIA, 1989
	Anas platyrhynchos	bird	LOAEL	4.00E+00	10	4.00E-01	Heinz and Hazeltine, 1983
chromium	mouse	mammal	LD-50	1.27E+02	100	1.27E+00	BEIA, 1989
	Anas rubripes	bird	LOAEL	1.00E+01	10	1.00E+00	Eisler, 1986a
cobalt	rat	mammal	LD-50	1.50E+03	100	1.50E+01	EPA, 1985
copper	non-ruminant animals	mammal	NOEL	1.00E+02	1	1.00E+02	Suter, 1991
cyanide	domestic mouse	mammal	LD-50	8.50E+00	100	8.50E-02	Eisler, 1991
lead	*Falco sparvenius*	bird	LOAEL	5.00E+01	10	5.00E+00	EPA, 1980a, 1985b
mercury	mouse	mammal	LD-50	1.30E+01	100	1.30E-01	BEIA, 1989
	Anas platyrhynchos	bird	NOAEL	5.00E-01	1	5.00E-01	Heinz, 1974
nickel	rat	mammal	LD-50	3.50E+02	100	3.50E+00	NRC, 1980
silver	rat	mammal	NOEL	1.00E+02	1	1.00E+02	NRC, 1980
tin	rat	mammal	NOEL	1.00E+00	1	1.00E+00	Eisler, 1989
thallium						No data	
vanadium						No data	
zinc	mouse	mammal	NOEL	4.58E+02	1	4.58E+02	Maita, et.al., 1981
	Coturnix c. japonica	bird	NOEL	3.00E+01	1	3.00E+01	Eisler, 1993
ORGANICS							
1,1-biphenyl	rat	mammal	LD-50	4.00E+03	100	4.00E+01	Verschueren, 1983
1,4-dichlorobenzene	mouse	mammal	LD-50	2.95E+03	100	2.95E+01	Lewis, 1992
2-butanone	mouse	mammal	LD-50	4.05E+03	100	4.05E+01	BEIA, 1989
2-hexanone	mouse	mammal	LD-50	2.43E+03	100	2.43E+01	Lewis, 1992
2-nitroaniline	rat	mammal	LD50	3.56E+03	100	3.56E+01	Hazardous Substances Data Base
	Aeglaius phoeniceus	bird	LD50	7.50E+02	100	7.50E+00	Hazardous Substances Data Base
2,4-dichlorophenol	mouse	mammal	LD-50	1.13E+03	100	1.13E+01	Verschueren, 1983

Table 6-H-14
Toxicity Reference Values for Mammal and Avian Receptors

Chemical Name	Test Species	Species Type	Toxicity Endpoint *	Endpoint Value (mg/kg)	Uncertainty Factor	Toxicity Reference Value (mg/kg)	Reference
4-chloroaniline	rat	mammal	LOAEL	1.25E+01	10	1.25E+00	IRIS database
4-chlorophenyl-phenylether						No data	
4-methylphenol	rat	mammal	NOAEL	5.00E+01	1	5.00E+01	IRIS database
3&4-methylphenol	rat	mammal	NOAEL	5.00E+01	1	5.00E+01	IRIS database
acetone	mouse	mammal	LD-50	3.00E+03	100	3.00E+01	BEIA, 1989
acetophenone	rat	mammal	NOAEL	4.23E+02	1	4.23E+02	IRIS database
aniline						No data	
bis(2-chloroethyl)ether	mouse	mammal	LD-50	1.36E+02	100	1.36E+00	Hazardous Substances Data Base
chlorobenzene	mouse	mammal	LD-50	2.30E+03	100	2.30E+01	Lewis, 1992
ethylbenzene	rabbit	mammal	LD-50	3.50E+03	100	3.50E+01	Verschueren, 1983
Irgasan DP-300	rat	mammal	LD-50	3.70E+03	100	3.70E+01	Dr. Marty Bernstein (CIBA), pers. comm., 1994
methylene chloride	rat	mammal	NOAEL	5.85E+00	1	5.85E+00	IRIS database
nitrobenzene	mouse	mammal	LD-50	5.90E+02	100	5.90E+00	Lewis, 1992
phenol	rat	mammal	LD-50	2.70E+02	100	2.70E+00	BEIA, 1989
	Agelaius phoeniceus	bird	LD-50	1.13E+02	100	1.13E+00	Schafer et al., 1983
safrole	mouse	mammal	LD-50	2.35E+03	100	2.35E+01	Hazardous Substances Data Base
tetrachloroethene	mouse	mammal	LD-50	8.00E+03	100	8.00E+01	Verschueren, 1983
Tinuvin 327	rat	mammal	LD-50	7.00E+03	100	7.00E+01	Dr. Marty Bernstein (CIBA), pers. comm., 1994
toluene	rat	mammal	LD-50	5.00E+03	100	5.00E+01	Lewis, 1992
trichloroethene	mouse	mammal	LD-50	2.40E+03	100	2.40E+01	Lewis, 1992
trichlorofluoromethane	mouse	mammal	LOAEL	3.49E+02	10	3.49E+01	IRIS database
xylene (total)	rat	mammal	LD-50	4.30E+03	100	4.30E+01	Lewis, 1992
PESTICIDES							
2,4-D	mouse	mammal	LD-50	3.47E+02	100	3.47E+00	BEIA, 1989
	Phasianus colchicus	bird	LC-50	3.11E+02	100	3.11E+00	DDT value; Hill et al, 1975
2,4,5-T	mouse	mammal	LD-50	2.42E+02	100	2.42E+00	BEIA, 1989
	Phasianus colchicus	bird	LC-50	3.11E+02	100	3.11E+00	DDT value; Hill et al, 1975
2,4,5-TP (Silvex)	mouse	mammal	LD-50	2.76E+02	100	2.76E+00	BEIA, 1989
	Phasianus colchicus	bird	LC-50	3.11E+02	100	3.11E+00	DDT value; Hill et al, 1975
4,4'-DDD	rat	mammal	LD-50	3.40E+03	100	3.40E+01	Verschueren, 1983

Table 6-H-14
Toxicity Reference Values for Mammal and Avian Receptors

Chemical Name	Test Species	Species Type	Toxicity Endpoint *	Endpoint Value (mg/kg)	Uncertainty Factor	Toxicity Reference Value (mg/kg)	Reference
	Phasianus colchicus	bird	LD-50	4.45E+02	100	4.45E+00	BEIA, 1989
4,4'-DDE	rat	mammal	LD-50	8.80E+02	100	8.80E+00	BEIA, 1989
	Falco sparvenius	bird	LOAEL	2.80E+00	10	2.80E-01	Wiemeyer and Porter, 1970
4,4'-DDT	mouse	mammal	LD-50	2.00E+02	100	2.00E+00	BEIA, 1989
	Phasianus colchicus	bird	LC-50	3.11E+02	100	3.11E+00	Hill et al, 1975
aldrin	rat	mammal	LD-50	4.50E+01	100	4.50E-01	BEIA, 1989
	Phasianus colchicus	bird	LD-50	5.70E+01	100	5.70E-01	Hill et al, 1975
alpha-BHC	rat	mammal	LD-50	1.77E+02	100	1.77E+00	Lewis, 1992
	Agelaius phoeniceus	bird	LD-50	7.50E+01	100	7.50E-01	gamma-BHC value; Schafer, et.al.,1983
alpha-chlordane	rat	mammal	LD-50	4.57E+02	100	4.57E+00	chlordane value, Verschueren, 1983
	Tyto alba	bird	LD-50	7.50E+01	100	7.50E-01	Chlordane, value; Eisler, 1990
beta-BHC	rat	mammal	LD-50	1.04E+03	100	1.04E+01	Lewis, 1992; delta-BHC value
	Agelaius phoeniceus	bird	LD-50	7.50E+01	100	7.50E-01	gamma-BHC value; Schafer, et.al.,1983
delta-BHC	rat	mammal	LD-50	1.04E+03	100	1.04E+01	Lewis, 1992
	Agelaius phoeniceus	bird	LD-50	7.50E+01	100	7.50E-01	gamma-BHC value; Schafer, et.al.,1983
dieldrin	rat	mammal	LD-50	6.30E+01	100	6.30E-01	Verschueren, 1983
	Tyto alba	bird	NOAEL	5.00E-01	1	5.00E-01	Mendenhall et al., 1983
dinoseb	rat	mammal	LD-50	5.80E+01	100	5.80E-01	Verschueren, 1983
	Tyto alba	bird	NOAEL	5.00E-01	1	5.00E-01	Mendenhall, et.al., 1983, value for dieldrin
disulfoton	rat	mammal	LD-50	1.25E+01	100	1.25E-01	Verschueren, 1983
	Tyto alba	bird	NOAEL	5.00E-01	1	5.00E-01	Mendenhall, et.al, 1983; value for dieldrin
endosulfan I/II	rat	mammal	LD-50	1.75E+01	100	1.75E-01	Verschueren, 1983
	Tyto alba	bird	NOAEL	5.00E-01	1	5.00E-01	dieldrin value; Mendenhall et al., 1983
endosulfan sulfate	rat	mammal	LD-50	1.75E+01	100	1.75E-01	Verschueren, 1983; value for endosulfan
	Tyto alba	bird	NOAEL	5.00E-01	1	5.00E-01	dieldrin value; Mendenhall et al., 1983
endrin	mouse	mammal	LD-50	1.37E+00	100	1.37E-02	Lewis, 1992
	Phasianus colchicus	bird	LD-50	5.70E+01	100	5.70E-01	aldrin value, Hill et al, 1975
endrin aldehyde	mouse	mammal	LD-50	1.37E+00	100	1.37E-02	Lewis, 1992
	Phasianus colchicus	bird	LD-50	5.70E+01	100	5.70E-01	aldrin value; Hill et al, 1975
ethyl parathion	mouse	mammal	LD-50	5.00E+00	100	5.00E-02	value for parathion; Lewis, 1992

Table 6-H-14
Toxicity Reference Values for Mammal and Avian Receptors

Chemical Name	Test Species	Species Type	Toxicity Endpoint *	Endpoint Value (mg/kg)	Uncertainty Factor	Toxicity Reference Value (mg/kg)	Reference
famphur	mouse	mammal	LD-50	2.70E+01	100	2.70E-01	Hazardous Substances Data Base
	Agelaius phoeniceus	bird	LD-50	1.78E+00	100	1.78E-02	BEIA, 1989
gamma-BHC (Lindane)	mouse	mammal	LD-50	4.40E+01	100	4.40E-01	Lewis, 1992
	Agelaius phoeniceus	bird	LD-50	7.50E+01	100	7.50E-01	gamma-BHC value; Schafer, et.al.,1983
gamma-Chlordane	rat	mammal	LD-50	4.57E+02	100	4.57E+00	chlordane value; Verschueren, 1983
	Tyto alba	bird	LD-50	7.50E+01	100	7.50E-01	Chlordane, value; Eisler, 1990
heptachlor	mouse	mammal	LD-50	6.80E+01	100	6.80E-01	Lewis, 1992
	wild bird species	bird	LD-50	2.00E+01	100	2.00E-01	EPA, 1985
heptachlor epoxide	mouse	mammal	LD-50	6.80E+01	100	6.80E-01	heptachlor value; Lewis, 1992
	wild bird species	bird	LD-50	2.00E+01	100	2.00E-01	EPA, 1985
isodrin	*Tyto alba*	bird	NOAEL	5.00E-01	1	5.00E-01	dieldrin value; Mendenhall, et.al., 1983
kepone	rat	mammal	LD-50	9.50E+01	100	9.50E-01	Hazardous Substances Data Base
	Tyto alba	bird	NOAEL	5.00E-01	1	5.00E-01	dieldrin value; Mendenhall, et.al., 1983
methoxychlor	rabbit	mammal	NOEL	5.01E+00	1	5.01E+00	IRIS database
	Phasianus colchicus	bird	LC-50	5.00E+03	100	5.00E+01	Hazardous Substances Data Base
methyl parathion	mouse	mammal	LD-50	5.00E+00	100	5.00E-02	value for parathion; Lewis, 1992
orthophosphate						No data	
phorate	mouse	mammal	LD-50	2.25E+00	100	2.25E-02	Hazardous Substances Data Base
	Phasianus colchicus	bird	LD-50	7.12E+00	100	7.12E-02	Hazardous Substances Data Base
propazine	rat	mammal	NOEL	5.00E+00	1	5.00E+00	IRIS database
	Anas Platyrhyncus	bird	LC-50	1.00E+04	100	1.00E+02	Hazardous Substances Data Base
sulfotepp	rat	mammal	NOEL	5.00E-01	1	5.00E-01	IRIS database
PCBs							
PCB-1248	rat	mammal	LD-50	1.10E+04	100	1.10E+02	Lewis, 1992
	Phasianus colchicus	bird	LC-50	1.31E+03	100	1.31E+01	Hazardous Substances Data Base
PCB-1254	rat	mammal	LD-50	1.01E+03	100	1.01E+01	Lewis, 1992
	Phasianus colchicus	bird	LC-50	1.09E+03	100	1.09E+01	Hazardous Substances Data Base
PCB-1260	rat	mammal	LD-50	1.30E+03	100	1.30E+01	Hazardous Substances Data Base
	Phasianus colchicus	bird	LD-50	1.26E+03	100	1.26E+01	Hazardous Substances Data Base
PHTHALATE ESTERS							

Table 6-H-14
Toxicity Reference Values for Mammal and Avian Receptors

Chemical Name	Test Species	Species Type	Toxicity Endpoint *	Endpoint Value (mg/kg)	Uncertainty Factor	Toxicity Reference Value (mg/kg)	Reference
bis(2-ethylhexyl)phthalate	mouse	mammal	LD-50	3.00E+04	100	3.00E+02	Lewis, 1992
butylbenzylphthalate	mouse	mammal	LD-50	4.17E+03	100	4.17E+01	Lewis, 1992
diethylphthalate	mouse	mammal	LD-50	6.17E+03	100	6.17E+01	Lewis, 1992
di-n-butylphthalate	mouse	mammal	LD-50	5.29E+03	100	5.29E+01	Lewis, 1992
di-n-octylphthalate	mouse	mammal	LD-50	6.51E+03	100	6.51E+01	Lewis, 1992
PAHs							
2-methylnaphthalene	rat	mammal	LD-50	5.00E+01	1.00E+02	5.00E-01	lowest PAH LD-50; Suter, 1991
acenaphthene	rat	mammal	LD-50	5.00E+01	1.00E+02	5.00E-01	lowest PAH LD-50; Suter, 1991
acenaphthylene	rat	mammal	LD-50	5.00E+01	1.00E+02	5.00E-01	lowest PAH LD-50; Suter, 1991
anthracene	rat	mammal	LD-50	2.00E+04	1.00E+02	2.00E+02	Lewis, 1992
benzo(a)anthracene	rat	mammal	LD-50	5.00E+01	1.00E+02	5.00E-01	lowest PAH LD-50; Suter, 1991
benzo(b)fluoranthene	rat	mammal	LD-50	5.00E+01	1.00E+02	5.00E-01	lowest PAH LD-50; Suter, 1991
benzo(g,h,i)perylene	rat	mammal	LD-50	5.00E+01	1.00E+02	5.00E-01	lowest PAH LD-50; Suter, 1991
benzo(k)fluroanthene	rat	mammal	LD-50	5.00E+01	1.00E+02	5.00E-01	lowest PAH LD-50; Suter, 1991
chrysene	rat	mammal	LD-50	5.00E+01	1.00E+02	5.00E-01	lowest PAH LD-50; Suter, 1991
dibenz(a,h)anthracene	rat	mammal	LD-50	5.00E+01	1.00E+02	5.00E-01	lowest PAH LD-50; Suter, 1991
fluoranthene	rat	mammal	LD-50	2.00E+03	1.00E+02	2.00E+01	Lewis, 1992
fluorene	rat	mammal	LD-50	5.00E+01	1.00E+02	5.00E-01	lowest PAH LD-50; Suter, 1991
indeno(1,2,3-cd)pyrene	rat	mammal	LD-50	5.00E+01	1.00E+02	5.00E-01	lowest PAH LD-50; Suter, 1991
naphthalene	mouse	mammal	LD-50	5.33E+02	1.00E+02	5.33E+00	Lewis, 1992
phenanthrene	mouse	mammal	LD-50	7.00E+02	1.00E+02	7.00E+00	Lewis, 1992
pyrene	mouse	mammal	LD-50	8.00E+02	1.00E+02	8.00E+00	Lewis, 1992
DIOXINS/FURANS							
Dibenzofuran	*Agelaius phoeniceus*	bird	LD-50	1.02E+02	100	1.02E+00	Schafer, et.al., 1983
2,3,7,8-TCDD	mouse	mammal	LD-50	1.14E-01	100	1.14E-03	2,3,7,8-TCDD value; BEIA, 1989
2,3,7,8-TCDF	mouse	mammal	LD-50	1.14E-01	100	1.14E-03	2,3,7,8-TCDD value; BEIA, 1989
DCDF	rat	mammal	LD-50	5.00E+03	100	5.00E+01	Dr. Marty Bernstein (CIBA), pers. comm., 1994
OCDD	mouse	mammal	LD-50	1.00E+00	100	1.00E-02	Lewis, 1992
TCDF	mouse	mammal	LD-50	1.00E+00	100	1.00E-02	OCDD value; Lewis, 1992
TRCDF	rat	mammal	LD-50	5.00E+03	100	5.00E+01	Dr. Marty Bernstein (CIBA), pers. comm., 1994

* ENDPOINT TYPES:

Table 6-H-14
Toxicity Reference Values for Mammal and Avian Receptors

Chemical Name	Test Species	Species Type	Toxicity Endpoint *	Endpoint Value (mg/kg)	Uncertainty Factor	Toxicity Reference Value (mg/kg)	Reference

NOAEL = no observed adverse effect level

NOEL = no observed effect level

LD50 = dose that is lethal to 50% of test organisms

LC50 = concentration that is lethal to 50 % of test organisms

LOAEL = lowest observed adverse effect level

LOEL = lowest observed effect level

NOEC = no observed effect concentration

LD-100= dose that is lethal to 100% of test organisms

no data = no suitable toxicity data available