PORSF
11.3.1b.1 v11

PART C-2
SOIL VAPORS (INDOOR AIR)

USEPA SF
1288878

Table C-2.1
Johnson and Ettinger Model Data Entry Printout for Benzene in Soil

CALCULATE RISK-BASED SOIL CONCENTRATION (enter "X" in "YES" box)

SL-ADV
Version 2.3; 03/01

YES [ ]

OR

CALCULATE INCREMENTAL RISKS FROM ACTUAL SOIL CONCENTRATION (enter "X" in "YES" box and initial soil conc. below)

YES [X]

ENTER Chemical CAS No. (numbers only, no dashes)	ENTER Initial soil conc., C_R (µg/kg)	Chemical	Final finite source bldg. conc., Cbuilding (ug/m3)
71432	3.10E+02	Benzene	1.517213681

MORE ↓

ENTER Average soil temperature, T_S (°C)	ENTER Depth below grade to bottom of enclosed space floor, L_F (cm)	ENTER Depth below grade to top of contamination, L_t (cm)	ENTER Depth below grade to bottom of contamination, (enter value of 0 if value is unknown) L_b (cm)	ENTER Totals must add up to value of L_t (cell D28) Thickness of soil stratum A, h_A (cm)	ENTER Thickness of soil stratum B, (Enter value or 0) h_B (cm)	ENTER Thickness of soil stratum C, (Enter value or 0) h_C (cm)	ENTER Soil stratum A SCS soil type (used to estimate soil vapor permeability)	OR	ENTER User-defined stratum A soil vapor permeability, k_v (cm²)
10	15	15	488	15	0	0	s		

MORE ↓

ENTER Stratum A soil dry bulk density, ρ_b^A (g/cm³)	ENTER Stratum A soil total porosity, n^A (unitless)	ENTER Stratum A soil water-filled porosity, θ_w^A (cm³/cm³)	ENTER Stratum A soil organic carbon fraction, f_{oc}^A (unitless)	ENTER Stratum B soil dry bulk density, ρ_b^B (g/cm³)	ENTER Stratum B soil total porosity, n^B (unitless)	ENTER Stratum B soil water-filled porosity, θ_w^B (cm³/cm³)	ENTER Stratum B soil organic carbon fraction, f_{oc}^B (unitless)	ENTER Stratum C soil dry bulk density, ρ_b^C (g/cm³)	ENTER Stratum C soil total porosity, n^C (unitless)	ENTER Stratum C soil water-filled porosity, θ_w^C (cm³/cm³)	ENTER Stratum C soil organic carbon fraction, f_{oc}^C (unitless)
1.66	0.375	0.054	0.006	1.35	0.489	0.167	0.006	1.7	0.34	0.26	0.002

MORE ↓

ENTER Enclosed space floor thickness, L_{crack} (cm)	ENTER Soil-bldg. pressure differential, ΔP (g/cm-s²)	ENTER Enclosed space floor length, L_B (cm)	ENTER Enclosed space floor width, W_B (cm)	ENTER Enclosed space height, H_B (cm)	ENTER Floor-wall seam crack width, w (cm)	ENTER Indoor air exchange rate, ER (1/h)
15	40	3048	1341	305	0.1	2

ENTER Averaging time for carcinogens, AT_C (yrs)	ENTER Averaging time for noncarcinogens, AT_{NC} (yrs)	ENTER Exposure duration, ED (yrs)	ENTER Exposure frequency, EF (days/yr)	ENTER Target risk for carcinogens, TR (unitless)	ENTER Target hazard quotient for noncarcinogens, THQ (unitless)
70	30	30	350	1.0E-06	1

Used to calculate risk-based soil concentration.

END

Table C-2.2
Johnson and Ettinger Model Data Entry Printout for Naphthalene in Soil

CALCULATE RISK-BASED SOIL CONCENTRATION (enter "X" in "YES" box)

SL-ADV
Version 2.3; 03/01

YES [ ]

OR

CALCULATE INCREMENTAL RISKS FROM ACTUAL SOIL CONCENTRATION (enter "X" in "YES" box and initial soil conc. below)

YES [X]

ENTER Chemical CAS No. (numbers only, no dashes)	ENTER Initial soil conc., C_R (µg/kg)	Chemical	Final finite source bldg. conc., Cbuilding (ug/m3)
91203	1.83E+04	Naphthalene	3.000105036

MORE ↓

ENTER Average soil temperature, T_S (°C)	ENTER Depth below grade to bottom of enclosed space floor, L_F (cm)	ENTER Depth below grade to top of contamination, L_1 (cm)	ENTER Depth below grade to bottom of contamination, (enter value of 0 if value is unknown) L_b (cm)	ENTER Totals must add up to value of L_1 (cell D28) Thickness of soil stratum A, h_A (cm)	ENTER Thickness of soil stratum B, (Enter value or 0) h_B (cm)	ENTER Thickness of soil stratum C, (Enter value or 0) h_C (cm)	ENTER Soil stratum A SCS soil type (used to estimate soil vapor permeability)	OR	ENTER User-defined stratum A soil vapor permeability, k_v (cm^2)
10	15	15	488	15	0	0	s		

MORE ↓

ENTER Stratum A soil dry bulk density, ρ_b^A (g/cm^3)	ENTER Stratum A soil total porosity, n^A (unitless)	ENTER Stratum A soil water-filled porosity, θ_w^A (cm^3/cm^3)	ENTER Stratum A soil organic carbon fraction, f_{oc}^A (unitless)	ENTER Stratum B soil dry bulk density, ρ_b^B (g/cm^3)	ENTER Stratum B soil total porosity, n^B (unitless)	ENTER Stratum B soil water-filled porosity, θ_w^B (cm^3/cm^3)	ENTER Stratum B soil organic carbon fraction, f_{oc}^B (unitless)	ENTER Stratum C soil dry bulk density, ρ_b^C (g/cm^3)	ENTER Stratum C soil total porosity, n^C (unitless)	ENTER Stratum C soil water-filled porosity, θ_w^C (cm^3/cm^3)	ENTER Stratum C soil organic carbon fraction, f_{oc}^C (unitless)
1.66	0.375	0.054	0.006	1.35	0.489	0.167	0.006	1.7	0.34	0.26	0.002

MORE ↓

ENTER Enclosed space floor thickness, L_{crack} (cm)	ENTER Soil-bldg. pressure differential, ΔP ($g/cm\text{-}s^2$)	ENTER Enclosed space floor length, L_B (cm)	ENTER Enclosed space floor width, W_B (cm)	ENTER Enclosed space height, H_B (cm)	ENTER Floor-wall seam crack width, w (cm)	ENTER Indoor air exchange rate, ER (1/h)
15	40	3048	1341	305	0.1	2

ENTER Averaging time for carcinogens, AT_C (yrs)	ENTER Averaging time for noncarcinogens, AT_{NC} (yrs)	ENTER Exposure duration, ED (yrs)	ENTER Exposure frequency, EF (days/yr)	ENTER Target risk for carcinogens, TR (unitless)	ENTER Target hazard quotient for noncarcinogens, THQ (unitless)
70	30	30	350	1.0E-06	1

Used to calculate risk-based soil concentration.

END

Appendix D
Chemical Toxicity Profiles

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ARSENIC D-1
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APPENDIX D
CHEMICAL TOXICITY PROFILES

Toxic effects of the chemicals of concern are summarized in the following subsections. In general, the information has been summarized from the latest available Agency for Toxic Substances and Disease Registry (ATSDR) profile for each chemical.

ARSENIC

Arsenic trioxide is the most commercially important form of arsenic and is produced primarily from flue dust that is generated at copper and lead smelters. The principal use of arsenic (as arsenic trioxide) is in wood preservatives and a smaller proportion is used in the production of agricultural chemicals such as insecticides, herbicides, algaecides, and growth stimulants for plants and animals. The use of many arsenical pesticides has been phased out because of concerns about human health risks during production or use. Arsenic trioxide is no longer produced in the United States. Smaller amounts of arsenic are used in the production of glass and nonferrous alloys and in the semiconductor industry. The source of arsenic in EGDY soils is not known.

Arsenic has been shown to be toxic to human populations in areas of the world where it is present at naturally elevated concentrations in groundwater and to populations in certain occupations such as workers in copper smelters and chemical plants. There is strong evidence that arsenic is carcinogenic in humans by both oral and inhalation routes. Arsenic occurs in soil and rock along with other minerals such as copper, lead, iron, and nickel. It is typically found in soil in the form of an insoluble sulfide. Naturally occurring arsenic concentrations in soil range from 1 to 40 mg/kg, with a mean concentration of approximately 5 mg/kg (ATSDR 1993a).

Inorganic arsenic (the form typically found in soil or water) is often in a form that is readily absorbed either by ingestion or by inhalation. Following absorption, it is distributed throughout the body. Studies with laboratory animals have shown that the bioavailability of arsenic in soil is lower than that of arsenic ingested in solution. The issue of arsenic bioavailability is especially important at mining, milling, and smelting sites because the arsenic at these sites often exists, at least in part, as a poorly soluble sulfide and may also occur in particles of inert or insoluble material. These factors all tend to reduce the bioavailability of arsenic.

Arsenic is metabolized in the liver by methylation (the metabolic addition of methyl groups to inorganic arsenic ions), converting inorganic arsenic into less toxic methyl- and dimethylarsenic compounds. Absorbed organic and inorganic arsenic compounds are principally excreted in the urine. Methylation followed by urinary excretion is considered a detoxification mechanism for inorganic arsenic. Several organic arsenicals have been found to accumulate in fish and shellfish. These derivatives (mainly arsenobetaine and arsenocholine, also referred to as "fish arsenic" have been studied by several researchers and have been found to be essentially nontoxic.

The distinguishing adverse effects associated with chronic ingestion of arsenic are skin lesions (hyperkeratoses and hyperpigmentation) and skin cancer. Other adverse effects due to ingestion exposure include cancer of the internal organs (liver, bladder, and kidney) and a vascular disease known as "blackfoot disease" (blackfoot disease has been observed only in an area of Taiwan where there are naturally elevated arsenic concentrations in drinking water). Occupational

exposure (principally copper smelter workers) has been associated with an increased incidence of lung cancer. The EPA has given arsenic a carcinogenicity weight-of-evidence classification of A (human carcinogen) based on sufficient evidence of cancer mortality from both ingestion and inhalation exposures in human populations. The oral SF for arsenic is 1.5 $(mg/kg\text{-}day)^{-1}$ and the inhalation SF is 15 $(mg/kg\text{-}day)^{-1}$ based on human data.

The oral RfD is 0.0003 mg/kg-day, based on human chronic oral exposure. The RfD is based on the (NOAEL) of 0.0008 mg/kg-day, a LOAEL value of 0.014 mg/kg-day, an uncertainty factor of 3, and a modifying factor of 1. The overall confidence in the RfD was rated medium because doses were not well-characterized (USEPA 2001b).

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BENZENE

Under the proposed revised Carcinogen Risk Assessment Guidelines (USEPA 1996c), benzene is characterized as a known human carcinogen for all routes of exposure based upon convincing human evidence from occupational epidemiological studies as well as supporting evidence from animal studies (USEPA 2001b). Significantly increased risk of leukemia, primarily acute myelogenous leukemia (AML), have been reported in benzene-exposed workers in the chemical industry, shoemaking, and oil refineries (USEPA 2001b). EPA's IRIS file on benzene summarizes several key studies that support the weight-of-evidence classification that exposure to benzene is causally related to an increase in the risk of cancer, specifically leukemia. Included in these studies are Aksoy et al. (1974) where the effects of benzene exposure among 28,500 Turkish shoe industry workers were reported; a retrospective cohort mortality study by Infante et al. (1977) where leukemogenic effects of benzene exposure in 748 white male workers in a rubber products manufacturing plant were examined; and two cohort studies by Rinsky et al. (1981, 1987) where an extension and elaboration for the analysis done by Infante et al. (1977) was performed. These studies (Rinsky et al. 1981, 1987), based upon the original cohort of rubber workers studied by Infante et al. (1977), were selected by the EPA as the critical studies for dose-response analysis and for the quantitative estimation of cancer risk to humans (USEPA 2001b).

The true cancer risk from exposure to inhaled benzene cannot be ascertained because of uncertainties in the low-dose exposure scenarios and lack of clear understanding of the mode of action. Therefore, "a range of estimates of risk is recommended, each having equal scientific plausibility. The range estimates are maximum likelihood values (i.e., best statistical estimates) and were derived from observable dose responses using a linear extrapolation model to estimate low environmental exposure risks...The use of a linear model is a default public health protective approach and an argument both for and against recognizing supra- and sublinear relationships at low doses and non-threshold or threshold modes of action on exposure to benzene. Therefore, the risk could be either higher or lower" (USEPA 2001b). Thus, the inhalation unit risk estimate for benzene is reported as a range, from 2.2E-06 to 7.8E-06 per $\mu g/m^3$. The inhalation SF can then be derived by applying standard inhalation exposure parameters, an air intake of 20 m^3/day, and an adult body weight of 70 kg. The benzene SF range is calculated as 0.0077 to 0.0273 $(mg/kg\text{-}day)^{-1}$. Risks from benzene inhalation evaluated in this assessment used the higher (more health protective) SF of 0.0273 $(mg/kg\text{-}day)^{-1}$. The oral SF for benzene is 0.0055 $(mg/kg\text{-}day)^{-1}$. This value is reported on the EPA's Region 9 PRG tables and is based on human data.

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Rinsky R., R.J. Young, and A.B. Smith. 1981. "Leukemia in Benzene Workers." *Am. J. Ind. Med.* 2:217-245, as cited in USEPA 1998a.

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N-PROPYLBENZENE

Little toxicity information exists for this chemical and there are no toxicity criteria available in EPA's online IRIS database. The NCEA-generated inhalation and oral reference dose of 0.01 mg/kg-day was used in this report (USEPA 2000). EPA's Region IX table, which provided the value does not report the target organ for these chemicals on which the RfDs were based (USEPA 2000).

EPA's Region IX Preliminary Remediation Goals Table lists an oral Reference Dose (RfD) of 0.01 mg/kg-day, and an inhalation RfD of 0.01 mg/kg-day for each of these chemicals. However, no further toxicity information (i.e., target organs, health effects, study results used to generate RfD values, etc.) are given.

REFERENCES

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CARCINOGENIC POLYCYCLIC AROMATIC HYDROCARBONS

(BENZO[a]PYRENE, BENZO[a]ANTHRACENE, BENZO[b]FLUORANTHENE, DIBENZ[a,h]ANTHRACENE, INDENO[1,2,3-cd]PYRENE)

Toxicity of all carcinogenic polycyclic aromatic hydrocarbons (PAHs) are based on benzo(a)pyrene and all of these chemicals have similar toxic endpoints. Thus, only benzo(a)pyrene is discussed in this profile.

Benzo(a)pyrene, a PAH, is a common and persistent environmental contaminant. It is produced as a mixture of PAHs during combustion reactions, and is a component of tobacco smoke, automobile exhaust and air pollution (Mauderly 1993).

Little or no data is available on the toxicity of benzo(a)pyrene in humans. Because exposure scenarios most commonly involve mixtures of PAHs, it is not possible for epidemiological studies to associate adverse health effects with exposure to benzo(a)pyrene alone. However, benzo(a)pyrene toxicity has been well documented in animals. For instance, benzo(a)pyrene has been identified as a vascular toxicant capable of initiating and/or promoting atherosclerosis (Ou and Ramos 1992). Benzo(a)pyrene has also been shown to affect reproductive function in both male and female animals. Mice fed high levels of benzo(a)pyrene during pregnancy had difficulty reproducing, and so did their offspring. Birth defects and decreased body weight were also observed in the offspring (ORNL 1982). Similar adverse health effects could occur in humans, but no data exists that allow a certain determination to be made (ATSDR 2000).

No non-carcinogenic toxicity values (i.e., reference dose, reference concentration) are listed for benzo(a)pyrene by the U.S. Environmental Protection Agency (EPA) Integrated Risk Information System (IRIS) or Oak Ridge National Laboratory (ORNL) Risk Assessment Information System (RAIS) online databases (USEPA 2002; ORNL 2002).

The primary health concern associated with benzo(a)pyrene exposure is its ability to induce and/or promote cancer. Animal studies reporting the formation of DNA adducts and tumors in the liver, lung, and stomach of animals, and human skin cells, exposed to benzo(a)pyrene (Culp

and Beland 1994; Zhang et al. 1990). The U.S. Department of Health and Human Services (DHHS) has determined that benzo[a]pyrene is a known animal carcinogen. Both the International Agency for Research on Cancer (IARC) and the EPA have determined that benzo[a]pyrene is also a probable human carcinogen (ATSDR 2000).

EPA has classified benzo(a)pyrene as a probable human carcinogen (Group B2) based on data from three studies reporting increased incidence of forestomach tumors in mice and rats (Neal and Rigdon 1967; Rabstein et al. 1973; Brune et al. 1981). This classification is supported by positive results from mutagenicity assays (USEPA 2002). The oral slope factor (SF) for benzo(a)pyrene (calculated using data from the previously referenced mouse and rat studies) is 7.3 $(mg/kg\text{-}day)^{-1}$. A provisional inhalation SF of 3.1 $(mg/kg\text{-}day)^{-1}$ and dermal SF of 23.5 $(mg/kg\text{-}day)^{-1}$ have been developed for benzo(a)pyrene by the National Center for Environmental Assessment (NCEA 1995).

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1,2-DICHLOROETHANE

1,2-Dichloroethane is a clear, man-made liquid that evaporates quickly at room temperature. It has a pleasant smell and a sweet taste. 1,2-Dichloroethane is used primarily in the manufacture of vinyl chloride, as well as in the synthesis of tetrachloroethylene, trichloroethylene, 1,1,1-trichloroethane, vinylidene chloride, aziridines, and ethylenediamines. It is added to gasoline as a lead-scavenging agent, and, in the past, has been used as a metal degreasing agent; a solvent; and a fumigant for grain, upholstery, and carpets. It has also been used in paints, coatings, adhesives, varnishes, finish removers, soaps, and scouring agents (U.S. Air Force 1989; ATSDR 1999).

Bronchitis, hemorrhagic gastritis and colitis, hepatocellular damage, renal tubular necrosis, and histopathological changes in the brain have been reported in cases of acute oral poisoning of humans. Acute inhalation exposures to 1,2-dichloroethane (75-125 ppm) can result in irritation of the eyes, nose and throat; dizziness; nausea; vomiting; increasing stupor; cyanosis; rapid pulse; delirium; anesthesia; partial paralysis; loss of tactile sense; degenerative changes in the myocardium; liver and kidney damage; pulmonary edema; and systemic hemorrhaging. Chronic occupational exposure to 1,2-dichloroethane may result in central nervous system effects (i.e., irritability, sleeplessness, decreased heart rate), loss of appetite; nausea, vomiting, epigastric pain, and liver and kidney damage (NIOSH 1976, CEC 1986; ATSDR 1999; Nouchi et al. 1984). In animals, subchronic and chronic inhalation exposures to 1,2-dichloroethane resulted in pathological lesions in the kidney, liver, heart, lungs, and testes (NTP 1991; Cheever et al. 1990).

The U.S. Environmental Protection Agency (EPA) Integrated Risk Information System (IRIS) online database does not list an oral Reference Dose (RfD) for 1,2-dichloroethane (EPA 2002). However, a provisional RfD of 0.03 mg/kg-day has been derived by EPA (USEPA 1994) from a NOAEL of 26 mg/kg-day for rats tested in a subchronic gavage study (NTP 1991). The provisional oral RfD was calculated by applying an uncertainty factor of 1000 and a modifying factor of 1 to the reported NOAEL of 26 mg/kg-day. Use of the provisional RfD in risk assessment reports for specific sites must be approved by EPA.

An inhalation reference concentration (RfC) for 1,2-dichloroethane has not been established by EPA (USEPA 2002). However, EPA has also derived a provisional RfC of 0.005 mg/m^3 (EPA 1994) from a LOAEL (gastrointestinal disturbances and liver and gallbladder disease) of 10 mg/m^3 for occupationally exposed workers (Kozik 1957). Use of the provisional RfC in risk assessment reports for specific sites must be approved by EPA.

EPA has classified 1,2-dichloroethane as a probable human carcinogen (Group B2) based on induction of several tumor types in rats and mice treated by gavage, and lung papillomas in mice after topical application (NCI 1978). 1,2-Dichloroethane has also been shown to be mutagenic for Salmonella (Nestmann et al., 1980). Metabolites of 1,2-dichloroethane have been shown to form adducts with DNA after in vitro or in vivo exposures (USEPA 2002).

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CHROMIUM

Chromium is a naturally occurring element found in rocks, soil, plants, animals, and in volcanic dust and gases. The most common environmental forms are Chromium (0), Chromium (III), and Chromium (VI). Chromium (0), the metal chromium, is a gray solid and has a high melting point. This form is primarily used to make steel and other alloys. Chromium (III) is used to line high-temperature industrial furnaces. Chromium-containing compounds are used in many industrial processes, such as, stainless steel welding, chrome plating, and leather tanning.

Chromium (III) is considered an essential nutrient that helps to maintain normal metabolism of glucose, cholesterol, and fat in humans. The minimum human daily requirement of chromium for optimal health is not known, but a daily ingestion of 50 to 200 μg/day (0.0007 to 0.003 mg/kg bw/day) has been estimated to be safe and adequate. The long-term effects of eating diets low in chromium are difficult to evaluate.

The three major forms differ in their effects on health. Chromium (VI) is irritating, and short-term, high-level exposure can result in adverse effects at the site of contact, such as ulcers of the skin, irritation of the nasal mucosa and perforation of the nasal septum, and irritation of the GI tract. Chromium (VI) may also cause adverse effects in the kidney and liver. Chromium (III) does not result in these effects and is the form that is an essential food nutrient when ingested in small amounts, although very large doses may be harmful. For example, ingesting large amounts can cause stomach upset and ulcers, convulsions, kidney and liver damage. Very limited data suggest that chromium (III) may have respiratory effects on humans. No data on chronic or subchronic effects of inhaled chromium (III) in animals can be found. Adequate reproductive and developmental toxicity data do not exist. Information on chromium (0) health effects is limited. Animal studies have found that inhalation exposure had increased frequencies of chromosomal aberrations and sister chromatid exchanges in peripheral lymphocytes (ATSDR 1993c).

Of the three forms of chromium of toxicological importance, chromium (VI) is the most toxic. Chromium (VI) is classified by the EPA as a Group A carcinogen by inhalation, based on evidence that indicates sufficient cancer data in both animals and humans. Several epidemiological studies found an association between chromium exposure and lung cancer. The inhalation cancer SF for total chromium (one-sixth ratio of chromium VI:III) is 42 $(mg/kg\text{-}day)^{-1}$ and is based on benign and malignant stomach tumor data in female mice (DTSC 1999; USEPA 2001b). This SF was used in this HHRA for assessing inhalation exposures to chromium at the site.

The overall confidence in this RfD assessment was rated low because of the lack of explicit detail on study protocol and results, the lack of high-dose supporting data, and the lack of an observed effect level. Thus, the RfD as given should be considered conservative (USEPA 2001b).

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LEAD

Lead is a soft, bluish-gray metal. Lead acetate and lead nitrate are soluble in water; lead chloride is slightly soluble; and lead sulfide, lead phosphate, and lead oxides are not soluble in water. Some primary uses of lead in the United States are in lead-acid storage batteries, ammunition, bearing metals, brass, bronze, cable covering, extruded products, sheet lead, solder, ceramics, type metal, ballast or weights, tubes or containers, oxides, and gasoline additives.

Substantial quantities of both human and animal data are available regarding the toxicity of lead. This toxicity profile relies primarily on human data. Adverse effects of lead in humans are most often related to the blood lead (PbB) level as an indicator of internal lead dose. Therefore, whenever possible, this text relates adverse effects to PbB levels rather than to external exposure.

Lead absorption is influenced by the route of exposure, the exposure medium, speciation and physiochemical characteristics of lead, and the age and physiological state of the exposed individual. Approximately 30 to 50 percent of airborne particulate lead is absorbed. Nonfasted adults may absorb less than 10 percent of soluble lead ingested in food or water and only 2.5 percent of lead ingested in soil. The amount of lead absorbed from the skin in humans is unknown.

Women with occupational exposures to lead during pregnancy have an increased rate of miscarriages and stillbirths. There is no evidence of teratogenic effects in humans or animals due to exposure to low levels of lead. There is conflicting information regarding the potential effects of lead on birth weight, gestational age, and growth in children. There is conflicting evidence regarding the potential effects of lead on human chromosomes. In men with occupational exposures some reproductive effects (e.g., decreased sperm count, abnormal sperm morphology, decreased sperm mobility, and hormonal changes) can occur.

Although lead is considered to be carcinogenic in animals, evidence of its carcinogenicity in humans is generally considered to be inadequate. EPA's IRIS database classifies lead as a probable human carcinogen (Group B2), based on sufficient evidence in animals, but inadequate evidence in humans.

Sensitive members of the population are children, women, and individuals with chronic neurological dysfunction or kidney disease. Children may be especially at risk because compared to adults they absorb more lead from the gastrointestinal tract; retain more absorbed lead; and have a greater prevalence of nutritional deficiencies (e.g., calcium, iron, and zinc). Women who are pregnant or have osteoporosis may be at greater risk due to lead because each of these conditions may intensify the mobilization of lead from bone.

Ingestion is the primary route of exposure for children and other nonoccupationally exposed receptors. However, dose-response data based on external ingestion dose (mg/kg-day) in humans were limited. Hematological effects were observed in adult humans who ingested 0.02 to 0.03 mg lead acetate/kg-day for 14 days or 0.01 to 0.02 mg lead acetate/kg-day for 3 to 7 weeks.

The adult EPA model (USEPA 1996b) was used at this site to evaluate risk posed from exposure of adults to environmental lead. The model is used rather than the toxicity criteria approach used for the other COPCs.

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NAPHTHALENE

Naphthalene is a white solid that evaporates easily. Naphthalene has an odor threshold in air of 84 ppb; that threshold is lowered to 21 ppb when water is applied. Naphthalene is present in cigarette smoke, wood smoke, fossil fuels, tar, and asphalt, and at some hazardous waste sites. Products containing naphthalene include moth repellents, toilet deodorant blocks, dyes, resins, leather tanning agents, and the insecticide, carbaryl (ATSDR 2000).

In humans, exposure to high concentrations of naphthalene is known to cause hemolytic anemia, nausea, vomiting, diarrhea, blood in the urine, and a yellow color to the skin. In animals, ingestion of naphthalene has been shown to cause rats to develop cataracts, and chronic inhalation has been shown to cause respiratory inflammation and irritation in mice. Evidence of naphthalene-induced reproductive toxicity is inconclusive. Evidence that naphthalene is a carcinogen is also inconclusive: female mice with daily lifetime exposures to naphthalene developed lung cancer, but a significant association between tumor development and naphthalene exposure was not established (ATSDR 2000).

EPA has established an oral Reference Dose (RfD) for naphthalene of 0.02 mg/kg-day, based on a study reporting decreased mean terminal body weight in rats exposed to 142.9 and 285.7 mg/kg-day naphthalene, 5 days/week for 13 weeks (BCL 1980). The oral RfD was calculated by applying an uncertainty factor of 3000 (to account for interspecies extrapolation, intraspecies extrapolation to protect sensitive human populations, subchronic to chronic extrapolation, and database deficiencies) and a modifying factor of 1 to the reported NOAEL of 71.4 mg/kg-day. The overall confidence in the RfD is rated low because of inadequate chronic oral data for naphthalene; the lack of any dose-response data for naphthalene-induced hemolytic anemia; and the lack of two-generation reproductive toxicity studies (USEPA 2002).

EPA has established an inhalation Reference Concentration (RfC) for naphthalene of 0.003 mg/m^3, based on a study reporting hyperplasia and metaplasia in respiratory and olfactory epithelium, respectively, in mice exposed to naphthalene concentrations as low as 9.3 mg/m^3 (NTP 1992). The inhalation RfC was calculated by applying an uncertainty factor of 3,000 (to account for interspecies extrapolation, protection of sensitive individuals, and extrapolation from a LOAEL to a NOAEL) and a modifying factor of 3 (to account for database deficiencies) to the reported LOAEL of 9.3 mg/m^3. The overall confidence in the RfC is rated low to medium (USEPA 2002).

EPA has determined that the human carcinogenic potential of naphthalene can not be determined (Group D) due to the lack of human carcinogencicity data and inadequate animal data. However, there is suggestive evidence that naphthalene is carcinogenic in animals. One study reported observations of benign respiratory tumors and one carcinoma in female mice only exposed to naphthalene by inhalation (NTP, 1992). Additional support includes increase in respiratory tumors associated with exposure to 1-methylnaphthalene (Murata 1993).

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