151557

Toxicological Profile

Chromium

Chemical Formula	Cr	HSDB, 1996
Molecular Weight	51.996 g/mol	Weast, 1985
Vapor Pressure	1 mmHg @ 1616°C	Weast, 1985
Boiling Point	2672°C	Weast, 1985
Melting Point	1857°C	Weast, 1985
Henry's Law Constant	Not applicable	ATSDR, 1991
Solubility	Insoluble	Weast, 1985
Partition Coefficient		
K_{oc}	Not applicable	ATSDR, 1991

Chromium is a naturally occurring metal. The three most stable valence states of chromium are 0, +3, and +6. The parameters listed above reflect the properties of chromium 0. Chromium is used in plating for corrosion resistance and decorative purposes in the manufacture of appliances, tools, and automobiles. It is also employed in the manufacture of alloys such as stainless steel and heat resistant alloys. In addition, chromium compounds are used in printing, leather tanning, pigments, photography, graphics, and other industrial applications (ATSDR, 1991).

In the atmosphere, chromium is present primarily in particulate form with a mass median diameter of approximately 1 μm (Milford and Davidson, 1985; Ondov et al., 1989). Chromium particles with a diameter of <20 μm may remain airborne for long periods of time and may be transported great distances. Chromium compounds in the atmosphere are generally removed via wet or dry deposition (ATSDR, 1991). Chromium VI in the atmosphere may be reduced to chromium III at a significant rate in the presence of vanadium compounds, Fe^{2+}, HSO^{3-}, or As^{3+}. Conversely, chromium III salts in the atmosphere may be oxidized to chromium VI in the presence of at least 1% manganese oxide (USEPA, 1987).

Chromium in soils is not very mobile since it is generally present as insoluble oxide (USEPA, 1984). Soluble compounds such as chromium VI and III, however, are more mobile in soil systems. The mobility of soluble chromium compounds in soils depends on the sorption characteristics of the soil such as clay content, Fe_2O_3 content, and organic matter content. Chromium compounds that are

soluble or unabsorbed may leach to groundwater as soil pH increases (ATSDR, 1991). In addition, Cary (1982) reported that chromium maintains a low mobility for translocation from roots to aboveground portions of plants. Generally, the fate of chromium in soils is dependent on the redox potential and the pH in the soil. The reduction of chromium VI to chromium III is likely to occur in aerobic soils with a low pH or sufficient organic energy sources to initiate the redox reaction (Cary, 1982; USEPA, 1987; Saleh et al., 1989).

Most chromium compounds released to aquatic systems are likely to be deposited in sediments. Most of the soluble chromium compounds in water exist as chromium VI or III, which constitute a relatively small percentage of total chromium in water. Chromium VI in water is ultimately reduced to chromium III by organic matter (ATSDR, 1991). The residence time of total chromium in lakes has been estimated at a range of 4.6 to 18 years (Fishbein, 1981; Schmidt and Andren, 1984). Saleh et al. (1989) estimated the oxidation half-life of chromium III to chromium VI in lake water to be approximately 9 years. The addition of manganese oxide reduced the oxidation half-life to about 2 years. The oxidation of chromium III to chromium VI during chlorination was reported to be highest at a pH ranging from 5.5-6.0. In addition, the reduction of chromium VI by organic sediments and soils is slow depending on the type and amount of organic material present and redox potential of the aquatic system and is generally faster under anaerobic conditions. The reduction half-life of chromium VI in water with sediment and soil has been estimated to range from 4 to 140 days (Saleh et al., 1989). Although chromium compounds in water systems are not expected to biomagnify within aquatic food chains, bioconcentration factors ranging from 1 to 192 have been estimated for chromium VI in rainbow trout, oysters, blue mussels, and soft shell clams (Fishbein, 1981; Schmidt and Andren, 1984; USEPA, 1980, 1984).

Inhalation is the primary exposure route of concern for chromium. Evidence from animal studies and human epidemiologic studies indicate that chromium is readily and rapidly absorbed by the lungs (ATSDR, 1991). Epidemiological studies of chromate industry workers have indicated that chronic inhalation of high levels of chromium have been associated with an increased risk of respiratory cancer (IARC, 1980; USEPA, 1984; IRIS, 1996). USEPA has estimated an inhalation unit risk of 1.2E-02 $\mu g/m^3$ for chromium VI based on a study by Mancuso (1975). The unit risks reported by Langard et al. (1980), Axelsson et al. (1980), and Pokrovskaya and Shabynina (1973) are 0.13, 0.035, and 0.092, respectively.

Certain chromium VI compounds are generally believed to be responsible for the increased cancer risk of airborne chromium. In contrast, studies of laboratory animals exposed to chromium VI and

chromium III via inhalation have not demonstrated an increased cancer risk (Baetjer et al., 1959; Steffee and Baetjer, 1965; Nettesheim et al., 1971; Laskin, 1972; USEPA, 1984). Two potential factors which may account for this are: (1) animals are less sensitive to inhaled chromium, or (2) the carcinogenic effects of chromium occur only when humans are co-exposed to other carcinogenic agents such cigarette smoke (ATSDR, 1991).

The USEPA has classified chromium VI as a Group A carcinogen, human carcinogen, based on the results of occupational epidemiologic studies of chromium-exposed workers. Although chromium-exposed workers are exposed to both chromium III and chromium VI compounds, only hexavalent chromium has been determined to be carcinogenic in animals and is, therefore, the only chromium compound to be classified as a human carcinogen (IRIS, 1996). Additionally, chromium VI is classified as a carcinogen based only on inhalation exposures.

The USEPA has not established an oral cancer slope factor for chromium VI. The Agency has developed an inhalation unit risk of 0.012 $\mu g/m^3$ based on the results of epidemiological investigations (Pokrovskaya and Shabynina, 1973; Mancuso, 1975; Axelsson et al., 1980; Langard et al., 1980). For the purpose of this evaluation, the unit risk value is converted to an inhalation slope factor of 42 $(mg/kg\text{-}day)^{-1}$ (USEPA, 1995). The results of these studies are consistent across investigators and study populations and dose-response relationships have been established for chromium exposure and lung cancer.

The USEPA has also established a chronic oral RfD value of 0.005 mg/kg-day for chromium VI based on observations of rats administered drinking water containing up to 25 ppm hexavalent chromium. No adverse effects were observed in lower treatment levels, although rats receiving 25 ppm chromium VI were reported to have an approximate 20% reduction in water consumption and increased tissue concentration (IRIS, 1996; MacKenzie et al., 1958). The USEPA has also published an oral RfD value of 1.0 mg/kg-day for chromium III based on a chronic feeding study in rats. Ivankovic and Preussman (1975) reported no adverse effects to male and female rats fed chromic oxide in the diet for 840 days corresponding to an average total dose of 1800 g/kg (IRIS, 1996). For the purposes of this assessment, however, the oral RfD value for chromium VI is used as the inhalation RfD value.

The Agency has not published an inhalation RfD for chromium VI. For the purposes of this assessment, a provisional RfD value of 5.7×10^{-7} is used as the inhalation RfD value.

References

ATSDR. 1991. *Toxicological Profile for Chromium: Draft.* Agency for Toxic Substances and Disease Registry, Atlanta, GA and U.S. Department of Health and Human Services, Public Health Service, Washington, D.C. October.

Axelsson, G., R. Rylander, and A. Schmidt. 1980. Mortality and incidence of tumors among ferrochromium workers. *Br. J. Ind. Med.* 37:121-127. (Cited in IRIS, 1996)

Baetjer, A.M., C. Damron, and V. Budacz. 1959. The distribution and retention of chromium in men and animals. *A.M.A. Arch. Ind. Hyg. Occup. Med.* 20:136-150.

Cary, E.E. 1982. Chromium in air, soil and natural. In: *Topics in Environmental Health 5: Biological and Environmental Aspects of Chromium.* New York, NY: Elsevier Science Publishers. p. 49-64. (Cited in ATSDR, 1991)

Fishbein, L. 1981. Sources, transport, and alterations of metal compounds: An overview. I. Arsenic, beryllium, cadmium, chromium, and nickel. *Environ. Health Perspect.* 40:43-64. (Cited in ATSDR, 1991)

HSDB. 1996. *Chromium.* Hazardous Substances DataBank. National Library of Medicine, Bethesda, MD.

IARC. 1980. *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans: Volume 23. Some Metals and Metallic Compounds.* International Agency for Research on Cancer, World Health Organization, Lyon, France. p. 205-223.

IRIS. 1996. *Chromium.* Integrated Risk Information System, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH.

Ivankovic, S. and R. Preussman. 1975. Absence of toxic and carcinogenic effects after administration of high doses of chromic oxide pigment in subacute and long-term feeding experiments in rats. *Food Cosmet. Toxicol.* 13:347-351. (Cited in IRIS, 1996)

Langard, S., A. Anderson, and B. Gylseth. 1980. Incidence of cancer among ferrochromium and ferrosilicon workers. *Br. J. Ind. Med.* 37:114-120. (Cited in IRIS, 1996)

Laskin, S. 1972. *Research in Environmental Sciences.* Institute of Environmental Medicine, Washington, D.C. p. 92-97.

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. (Cited in IRIS, 1996)

Mancuso, T.F. 1975. *Consideration of Chromium as an Industrial Carcinogen.* International Conference on Heavy Metals in the Environment, Toronto, Ontario, Canada, October 27-31. p. 343-356. (Cited in IRIS, 1996)

Milford, J.B. and C.I. Davidson. 1985. The sizes of particulate trace elements in the atmosphere - a review. *J. Air Poll. Control Assoc.* 35:1249-1260. (Cited in ATSDR, 1991)

Nettesheim, P., M.G. Hanna, G.G. Doherty, R.F. Newell, and A. Hellman. 1971. Effect of calcium chromate dust, influenza virus, and 100 R whole-body X-radiation on lung tumor incidence in mice. *J. Natl. Cancer Inst.* 47:1129-1144.

Ondov, J.M., C.E. Choquette, W.H. Zoller et al. 1989. Atmospheric behavior of trace elements on particles emitted from a coal-fired power plant. *Atmos. Environ.* 23:2193. (Cited in ATSDR, 1991)

Pokrovskaya, L.V. and N.K. Shabynina. 1973. Carcinogenic hazards in the production of chromium ferroalloys. *Gig. Tr. Prof. Zabol.* 10:23-26. (Cited in IRIS, 1996)

Saleh, F.Y., T.F. Parkerton, R.V. Lewis et al. 1989. Kinetics of chromium transformations in the environment. *Sci. Tot. Environ.* 86:25-41. (Cited in ATSDR, 1991)

Schmidt, J.A. and A.W. Andren. 1984. Deposition of airborne metals into the Great Lakes: An evaluation of past and present estimates. *Adv. Environ. Sci. Technol.* 14:81-103. (Cited in ATSDR, 1991)

Steffee, C.H. and A.M. Baetjer. 1965. Histopathological effects of chromate chemicals. Report of studies in rabbits, guinea pigs, and mice. *Arch. Environ. Health.* 11:66.

USEPA. 1980. *Ambient Water Quality Criteria for Chromium.* U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA-440/5-80-035. (Cited in ATSDR, 1991)

USEPA. 1984. *Health Assessment Document for Chromium.* U.S. Environmental Protection Agency, Environmental and Criteria Assessment Office, Research Triangle Park, NC. EPA-600/8-83-014f.

USEPA. 1987. *Addendum to the Health Assessment Document for Chromium: Noncarcinogenic Effects.* U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, Research Triangle Park, NC. ECAP-R-117. (Cited in ATSDR, 1991)

USEPA. 1995. *Health Effects Assessment Summary Tables, FY-1995 Annual.* U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, D.C. EPA/540/R-95/036. May.

Weast, R.C. (ed.). 1985. *CRC Handbook of Chemistry and Physics.* 66th Edition. Boca Raton, FL: CRC Press, Inc. (Cited in ATSDR, 1991)

Toxicological Profile

Cyanide

Chemical Formula	HCN	ATSDR, 1991
Molecular Weight	27.03 g/mol	Weast, 1985
Vapor Pressure	264.3 mmHg @ 0°C	Jenks, 1979
Boiling Point	25.7°C	Jenks, 1979
Melting Point	-13.24°C	Jenks, 1979
Henry's Law Constant	5.1×10^{-2} atm-m^3/mol	Yoo et al., 1986
Solubility	Miscible	Weast, 1985
Partition Coefficient		
K_{oc}	No data	

Cyanide is commonly used in certain rat and pest poisons, silver and metal polishes, electroplating processes, photographic solutions, and fumigating products (ATSDR, 1991). Hydrogen cyanide is typically used in the production of adiponitrile, methyl methacrylate, sodium cyanide, cyanuric chloride, and chelating agents. Miscellaneous applications of hydrogen cyanide includes the manufacture of ferrocyanides, acrylates, lactic acid, pharmaceuticals, and specialty chemicals (Jenks, 1979; Worthing, 1987). Cyanide salts are often associated with electroplating and metal treatment, road salts, gold and silver extraction from ores, chelating agents, and dyes and pigments (Sax and Lewis, 1987; Towill et al., 1978; Worthing, 1987).

The primary environmental fate process of hydrogen cyanide is volatilization. It enters the atmosphere via industrial emissions and volatilization from soil and water. Once in the atmosphere, hydrogen cyanide has a relatively slow degradation rate. The estimated residence time at different tropospheric altitudes varies between 0.5 and 14 years. Due to the rapid volatilization and slow degradation rate, the atmosphere acts as a sink for hydrogen cyanide. Metal cyanides may be removed via wet and dry deposition (ATSDR, 1991).

The environmental fate of cyanide in water varies among cyanide compounds. Free cyanide is defined as the sum of all cyanide present as HCN and CN- (USEPA, 1984). The most common form of cyanide in water is hydrogen cyanide, although it may also occur as the cyanide ion, alkali metal cyanides, or a variety of metallocyanide complexes (Callahan et al., 1979). The volatilization rate of

cyanide in water is a significant fate process and is pH-dependent, with a faster rate of volatilization occurring at a lower pH (Ludzack et al., 1951). Biodegradation is also a significant fate process for cyanide in natural water systems. Depending on the conditions, cyanide may be converted to nitrate or produce nitrogen (Richards and Shieh, 1989). Ludzack et al. (1951) reported a range of <10 to 24 days for biodegradation half-lives for cyanide in two natural river waters. Adsorption of cyanide in water systems, however, is a much less significant fate process than volatilization or biodegradation. Generally, the extent of adsorption increases with decreasing pH and increases with increasing iron oxide, clay, and organic content (Callahan et al., 1979).

The fate processes of cyanide in soils is similar to the mechanisms which affect cyanide in aquatic systems. The transformation and degradation of cyanide in soils is largely pH-dependent, may produce nitrate or nitrogen, and is generally present in the same variety of cyanide complexes as those found in aquatic systems. In soil, cyanide ions are not influenced by oxidation-reduction reactions but may be involved in complexation reactions with metal ions (Towill et al., 1978). Cyanide has a low soil sorption capability and is generally not detected in groundwater due to fixation by trace metals through complexation or transformation by soil microorganisms (Callahan et al., 1979; Towill et al., 1978). Volatilization of hydrogen cyanide, however, is a significant loss mechanism for cyanides from soil surfaces at a pH of 9.2 or greater (Callahan et al., 1979).

Cyanide is readily absorbed from all routes of exposure, including the skin. Death may occur with the ingestion of even small amounts of sodium or potassium cyanide, and can occur within minutes or hours, depending on the route of exposure. Death is caused by respiratory arrest of the central region (ATSDR, 1991). Inhalation of toxic fumes represents a potentially rapid fatal type of exposure. Hydrogen cyanide is the chemical agent used in gas chamber executions. Generally, an average fatal hydrogen cyanide concentration for humans is estimated at 546 ppm for an exposure period of ten minutes (ATSDR, 1991). A blood cyanide level of greater than 0.2 μg/mL is considered toxic (Sax, 1989).

Cyanide intoxication is characterized by respiratory rate alterations, dyspnea, and cardiovascular effects (ATSDR, 1991). Electroplates and others chronically exposed to cyanide solutions may develop a cyanide rash, characterized by itching and macular, papular, and vesicular eruptions. Frequently, secondary infections develop. Chronic exposures can also cause loss of appetite, headaches, weakness, nausea, dizziness, and symptoms of irritation in the upper respiratory tract and eyes (Sax, 1989).

Additionally, there are no available data which indicates that cyanide may have developmental or reproductive effects in humans (ATSDR, 1991; USEPA, 1984).

The USEPA has classified cyanide as a Group D carcinogen, not classifiable as a human carcinogen, based on the lack of evidence from human and animal data (IRIS, 1996). In addition, the USEPA has not proposed an oral or inhalation cancer slope factor for cyanide.

The USEPA has established an oral RfD value of 0.02 mg/kg-day based on the results of rat bioassays (IRIS, 1996). Howard and Hanzel (1955) observed no treatment related effects to rats administered 73 and 183 mg/kg cyanide for 2 years in the diet. Philbrick et al. (1979), however, reported decreased weight gain, decreased thyroxin levels, and myelin degeneration in rats receiving 30 mg/kg-day. The Agency has not published an inhalation RfD value for cyanide.

References

ATSDR. 1991. *Toxicological Profile for Cyanide: Draft.* Agency for Toxic Substances and Disease Registry, Atlanta, GA and U.S. Department of Health and Human Services, Public Health Service. October.

Callahan, M.A., M.W. Slimak, N.W. Gabel et al. 1979. *Water-Related Environmental Fate of 129 Priority Pollutants.* Volume 1. U.S. Environmental Protection Agency, Office of Water Planning and Standards, Office of Water and Waste Management, Washington, D.C. (Cited in ATSDR, 1991)

Howard, J.W. and R.F. Hanzel. 1955. Chronic toxicity to rats of food treated with hydrogen cyanide. *J. Agric. Food Chem.* 3:325-329. (Cited in IRIS, 1996)

IRIS. 1996. *Cyanide.* Integrated Risk Information System, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH.

Jenks, W.R. 1979. Cyanides. In: *Kirk-Othmer Encyclopedia of Chemical Technology.* Grayson, M. (ed). New York, NY: John Wiley and Sons, Inc. p. 307-334. (Cited in ATSDR, 1991)

Jury, W.A., W.F. Spencer, and W.J. Farmer. 1983. Behavior assessment model for trace organics in soil: I. Model description. *J. Environ. Qual.* 12(4):558-564.

Ludzack, F.J., W.A. Moore, H.L. Krieger et al. 1951. Effect of cyanide on biochemical oxidation in sewage and polluted water. *Sewage Ind. Wastes.* 23:1298-1307. (Cited in ATSDR, 1991)

Philbrick, D.J., J.B. Hopkins, D.C. Hill, J.C. Alexander, and R.G. Thomson. 1979. Effects of prolonged cyanide and thiocyanate feeding in rats. *J. Toxicol. Environ. Health.* 5:579-592. (Cited in IRIS, 1996)

Richards, D.J. and W.K. Shieh. 1989. Anoxic-toxic activated-sludge treatment of cyanides and phenols. *Biotechnol. Bioeng.* 33:32-38. (Cited in ATSDR, 1991)

Sax, N.I. and R.D. Lewis (eds). 1987. *Hawley's Condensed Chemical Dictionary.* 11th Edition. New York, NY: Van Nostrand Reinhold. (Cited in ATSDR, 1991)

Towill, L.E., J.S. Drury, B.L. Whitfield et al. 1978. *Reviews of the Environmental Effects of Pollutants: V. Cyanide.* U.S. Environmental Protection Agency, Office of Research and Development, Cincinnati, OH. (Cited in ATSDR, 1991)

USEPA. 1984. *Health Effects Assessment for Cyanide - 1984.* U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. EPA-440/5-84-028. (Cited in ATSDR, 1991)

Weast, R.C. (ed). 1985. *CRC Handbook of Chemistry and Physics.* 66th Edition. Boca Raton, FL: CRC Press, Inc. (Cited in ATSDR, 1991)

Worthing, C.R. (ed). 1987. *The Pesticide Manual: 8th Edition.* Thornton Health, UK: British Crop Protection Council. (Cited in ATSDR, 1991)

Yoo, K.P., S.Y. Lee, and W.H. Lee. 1986. Ionization and Henry's Law constants for volatile, weak electrolyte water pollutants. *Korean J. Chem. Eng.* 3:67-72. (Cited in ATSDR, 1991)

Toxicity Profile

Dieldrin

Chemical Formula	$C_{12}H_8C_{l6}0$	ATSDR, 1991
Molecular Weight	308.93	HSDB, 1995
Vapor Pressure	$1.4x10^{-4}$ mmHg @ 25°C	HSDB, 1995
Boiling Point	330°C	ChemFate, 1985
Melting Point	175-176°C	HSDB, 1995
Henry's Law Constant	$1.51x10^{-5}$ atm m3/mol	ChemFate, 1985
Solubility	0.18 mg/L	HSDB, 1995
Partition Coefficients		
log K_{ow}	4.55	ChemFate, 1985
log K_{oc}	3.87	HSDB, 1995

Dieldrin was once used extensively for the control of pests in the corn and citrus industries. It has also been utilized in timber preservation and as a termite control for plastic and rubber coatings of electrical and communications cables and for plywood and other boards (Worthing and Walker, 1983). The use of dieldrin as a pesticide has significantly decreased, however, due to the availability of more practical alternatives and the increased resistance of insects to the chemical (USEPA, 1980). The uses of dieldrin were cancelled voluntarily or by USEPA by 1989 (USEPA, 1990).

Dieldrin is persistent in the environment due to its resistance to transformation and degradation processes (ATSDR, 1991). In the atmosphere, dieldrin may be transported great distances and is generally removed via wet or dry deposition (Baldwin et al., 1977). Bidleman et al. (1990) calculated the estimated lifetime of dieldrin in the atmosphere to be 1 day in the presence of hydroxyl radicals.

The primary removal process of dieldrin in soils is volatilization (Elgar, 1975). This process is slow, however, due to the low vapor pressure of dieldrin (ATSDR, 1991). It absorbs strongly to soil particulates, and moves slowly through soils indicating that it is unlikely to contaminate groundwater (ATSDR, 1991). For instance, Jury et al. (1987) calculated the transport of dieldrin in soils to a depth of 3 meters to be approximately 270 years. In addition, the estimated half life of dieldrin in soils has been determined to be 868 days (Jury et al., 1987).

In water, dieldrin maintains a high bioaccumulation potential based on its log K_{ow} -value. Bioconcentration factors for dieldrin in fish and snails have been measured at 2,700 and 61,657, respectively (Metcalf et al., 1973). Furthermore, a biomagnification factor of 1.0 has been estimated for dieldrin in rainbow trout on a lipid weight basis (Connell, 1989). In addition, MacKay and Leinonen (1975) estimated an evaporation half-life of 723 days for dieldrin in a water column at a depth of 1 meter.

Convulsion and other central nervous symptoms are the principal toxic effects observed in occupational studies of workers exposed in the application or manufacture of dieldrin. Conclusive studies of the effects of dieldrin in humans, however, are limited (ATSDR, 1991). Conversely, oral LD_{50} values ranging from 37 to 46 mg/kg-day have been determined for adult rats administered dieldrin (Gaines, 1960; Lu et al., 1965). LD_{50} values of 168 and 25 mg/kg-day have also been calculated for newborn and 2-week old rats, respectively (Lu et al., 1965). Decreased survival has also been observed in rats administered doses ranging from 0.5 to 2.5 mg/kg-day for two years (Deichmann et al., 1970; Fitzhugh et al., 1964; Harr et al., 1970; Reuber, 1980). Likewise, decreased survival was also reported for dogs exposed to dieldrin at 0.5 mg/kg-day for 25 months (Fitzhugh et al., 1964). Studies have also indicated similar effects in mice exposed to 1.3 mg/kg-day (Thorpe and Walker, 1973; Walker et al., 1972).

A number of chronic oral exposure studies have produced a variety of hepatic effects in rats. Several studies report increases in liver weights, cytoplasmic vacuoles, and smooth endoplasmic reticulum (Wright et al., 1972; Deichmann et al., 1967, 1970; Fitzhugh et al., 1964). An increased incidence of hepatocellular carcinomas has also been reported for mice administered 0.65 mg/kg-day for 80 weeks (NCI, 1978).

The USEPA has classified dieldrin as a Group B2 carcinogen, probable human carcinogen, based on inadequate data in humans and sufficient data in animals (IRIS, 1996). The Agency has established an oral cancer slope factor of 16.0 $(mg/kg\text{-}day)^{-1}$ based on liver carcinomas observed in mice exposed to dieldrin in their diets. This value represents the geometric mean of 13 slope factors calculated from data given in 7 studies (IRIS, 1996). The USEPA has also published an inhalation cancer slope factor of 16 $(mg/kg\text{-}day)^{-1}$ based on the same evidence used to derive the oral cancer slope factor (USEPA, 1995). Additionally, the Agency has derived an inhalation unit risk of 0.0046 $\mu g/m^3$ for dieldrin (IRIS, 1996).

In addition, the USEPA has established a chronic oral RfD value of 0.00005 mg/kg-day for dieldrin based on a 2-year rat feeding study in which animals were administered dietary concentrations of 0, 0.1, 1.0, or 10 ppm (IRIS, 1996). Females fed 1 and 10 ppm dieldrin were reported to have increased liver weights and liver-to-body weight ratios (Walker et al., 1969). The concentration of 0.1 ppm was identified as a NOAEL and converted to a dose estimate of 0.005 mg/kg-day. An uncertainty factor of 100 was applied to the NOAEL to account for interspecies extrapolation and interindividual variability. The Agency has not determined an inhalation RfD value for dieldrin (IRIS, 1996).

References

ATSDR. 1991. *Toxicological Profile for Aldrin/Dieldrin: Draft.*. Agency for Toxic Substances and Disease Registry, Atlanta, GA and U.S. Department of Health and Human Services, Public Health Service. October.

Baldwin, M.K., D. Bennett, and K.I. Benyon. 1977. The concentrations of aldrin and dieldrin and their photoisomers in the atmosphere. *Pestic. Sci.* 8:431-445. (Cited in ATSDR, 1991)

ChemFate. 1985. *Dieldrin*. Syracuse Research Corporation, Syracuse, New York. (Cited in ATSDR, 1991)

Connell, D.W. 1989. Biomagnification by aquatic organisms - A proposal. *Chemosphere*. 19:1573-1784. (Cited in ATSDR, 1991)

Deichmann, W.B., W.E. MacDonald, E. Blum et al. 1970. Tumorigenicity of aldrin, dieldrin, and endrin in the albino rat. *Ind. Med. Surg.* 40:10-20. (Cited in ATSDR, 1991)

Deichmann, W.B., M. Keplinger, F. Sala et al. 1967. Synergism among oral carcinogens: IV. the simultaneous feeding of four tumorigens to rats. *Toxicol. Appl. Pharmacol.* 11:88-103. (Cited in ATSDR, 1991)

Elgar, K.E. 1975. The dissipation and accumulation of aldrin and dieldrin residues in soil. *The Environ. Qual. Safety*. 3(suppl):250-257. (Cited in ATSDR, 1991)

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. (Cited in ATSDR, 1991)

Gaines, T.B. 1960. The acute toxicity of pesticides to rats. *Toxicol. Appl. Pharmacol.* 2:88-99. (Cited in ATSDR, 1991)

Harr, J.R., R.R. Claeys, J.F. Boone et al. 1970. Dieldrin toxicosis: Rat reproduction. *Am. J. Vet. Res.* 31:181-189. (Cited in ATSDR, 1991)

HSDB. 1995. *Dieldrin.* Hazardous Substances DataBank, National Library of Medicine, Bethesda, MD.

IRIS. 1996. *Dieldrin.* Integrated Risk Information System, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH.

Jury, W.A., D.D. Focht, and W.J. Farmer. 1987. Evaluation of pesticide groundwater pollution potential from standard indices of soil-chemical adsorption and biodegradation. *J. Environ. Qual.* 16:422-428. (Cited in ATSDR, 1991)

Lu, F.C., D.C. Jessup, and A. Lavallee. 1965. Toxicity of pesticide in young versus adult rats. *Food Cosmet. Toxicol.* 3:591-596. (Cited in ATSDR, 1991)

MacKay, D. and P.J. Leinonen. 1975. Rate of evaporation of low-solubility contaminants from water bodies to atmosphere. *Environ. Sci. Technol.* 9:1178-1180. (Cited in ATSDR, 1991)

Metcalf, R.L., I.P. Kapoor, C.K. Lu et al. 1973. Model ecosystem studies of environmental fate of six organochlorine pesticides. *Environ. Health Perspect.* 4:35-44. (Cited in ATSDR, 1991)

NCI. 1978. *Bioassay of Aldrin and Dieldrin for Possible Carcinogenicity.* U.S. Department of Health and Human Services, National Cancer Institute, Carcinogenesis Program, Bethesda, MD. DHEW 78-821. (Cited in ATSDR, 1991)

Reuber, M.D. 1980. Significance of acute and chronic renal disease in Osborne-Mendel rats ingesting dieldrin or aldrin. *Clin. Toxicol.* 17:75-79. (Cited in ATSDR, 1991)

Thorpe, E. and A.I.T. Walker. 1973. The toxicity of dieldrin (HEOD). II. Comparative long-term oral toxicology studies in mice with dieldrin, DDT, phenobarbitone, beta-BHC, and gamma-BHC. *Food. Cosmet. Toxicol.* 11:443-441. (Cited in ATSDR, 1991)

USEPA. 1980. *Ambient Water Quality Criteria for Aldrin/Dieldrin.* U.S. Environmental Protection Agency, Criteria and Standards Division, Washington, D.C. PB81-11730/OWRS. (Cited in ATSDR, 1991)

USEPA. 1990. *Suspended, Cancelled, and Restricted Pesticides.* U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances, Office of Compliance Monitoring, Washington, D.C. EPA/2OT-1002. (Cited in ATSDR, 1991)

USEPA. 1995. *Health Effects Assessment Summary Tables, FY-1995 Annual.* U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, D.C. May. EPA/540/R-95/036.

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. (Cited in IRIS, 1996)

Walker, A.I.T., E. Thorpe, and D.E. Stevenson. 1972. The toxicology of dieldrin (HEOD). Long-term oral toxicity studies in mice. *Food Cosmet. Toxicol.* 11:415-432. (Cited in ATSDR, 1991)
Worthing, C.R. and S.B. Walker (eds). 1983. *The Pesticide Manual: A World Compendium, 7th Edition.* Suffolk, Great Britain: The Lavenham Press Limited, 6, 191. (Cited in ATSDR, 1991)

Wright, A.S., D. Potter, M.F. Wooder et al. 1972. The effects of dieldrin on the subcellular structure and function of mammalian liver cells. *Food Cosmet. Toxicol.* 10:311-332. (Cited in ATSDR, 1991)

Toxicity Profile

Heptachlor Epoxide

Chemical Formula	$C_{10}H_5Cl_7O$	ATSDR, 1991
Molecular Weight	389.40	HSDB, 1995
Vapor Pressure	2.6x10- mmHg @ 25°C	OHM/TADS, 1985
Boiling Point	No data	ATSDR, 1991
Melting Point	160-161°C	HSDB, 1995
Henry's Law Constant	3.2x10-5 atm m3/mol	USEPA, 1987
Solubility	0.275 mg/L	USEPA, 1987
Partition Coefficients		
log K_{ow}	5.40	Mackay, 1982
log K_{oc}	3.34-4.37	Lyman et al., 1982

Heptachlor and heptachlor epoxide are no longer commercially produced in the United States. Historically, heptachlor was manufactured as an insecticide for the treatment of agricultural products such as corn, grains, and sorghum (ATSDR, 1991). It has also been used as a nonagricultural insecticide for the control of termites and household insects (Worthing and Walker, 1987). Heptachlor is currently used only for the control of fire ants in power transformers (ATSDR, 1991).

In the atmosphere, heptachlor epoxide is subject to long-range transport and removal via wet deposition (ATSDR, 1991). It undergoes direct photolysis in sunlight, and may be converted to intermediate and final photoproducts (Graham et al., 1973). The atmospheric half-life of heptachlor epoxide in the presence of hydroxyl radicals has been estimated at about 1.5 days (HSDB, 1995).

Based on its low log K_{oc} value, heptachlor epoxide is not mobile in soils and has a low potential to leach to groundwater. It is even less likely to leach in soils with high organic matter content (ATSDR, 1991). On the soil surface, heptachlor epoxide photodegrades slowly at a rate of about 1% per month during the summer season (HSDB, 1995).

In water, heptachlor epoxide adsorbs strongly to bottom and suspended sediments (ATSDR, 1991). Based on it Henry's law constant, heptachlor epoxide is likely to partition slowly to the atmosphere from surface water (Lyman et al., 1982). The log K_{ow} for heptachlor epoxide suggests a high

potential for bioconcentration within the aquatic food chain (ATSDR, 1991). Estimated bioconcentration factors for heptachlor epoxide in mussels, oysters, and Asiatic clams are 1,698, 851, and 2,330, respectively (Hawker and Connell, 1986; Hartley and Johnston, 1983; Geyer et al, 1982). Likewise, biomagnification of heptachlor epoxide within both aquatic and terrestrial food chains is significant based on its lipophilicity (ATSDR, 1991). A half-life of at least 4 years has been estimated for heptachlor epoxide in river systems (Eichelberger and Lichtenberg, 1971).

A study conducted by Davis (1965) in which 200 mice were administered 0 and 10 ppm heptachlor epoxide for 2 years resulted in a 2-fold increase of benign liver lesions over the control group. In a similar study, a test group of 200 mice received a 75:25 mixture of heptachlor epoxide:heptachlor at 0, 1, 5, and 10 ppm for 18 months. Results of this study indicate hyperplasia and an increase in hepatic carcinomas within the 5 and 10 ppm dose groups (Velsicol Chemical Co., 1973). Additionally, Witherup et al. (1959) reported a significant increase in hepatic carcinomas in a study of rats administered 5 and 10 ppm heptachlor epoxide for 108 weeks.

Acute oral LD_{50}s for rats, mice, and rabbits administered heptachlor epoxide range from 39 to 144 mg/kg (Podowski et al., 1979; Eisler, 1968). Buck et al. (1959) reported lethality within 3 days to six calves receiving 5, 10, 15, or 25 mg/kg heptachlor epoxide. An increase in liver-to-body weight was reported in beagle dogs fed diets of 0.013 to 0.19 mg/kg/day for 60 weeks. The increase, however, was not accompanied by histological changes (University of Cincinnati, 1958).

The USEPA has classified heptachlor epoxide as a Group B2 carcinogen, probable human carcinogen, based on inadequate carcinogenic data in humans and sufficient carcinogenic evidence in animals. The Agency has established an oral cancer slope factor of 9.1 $(mg/kg\text{-}day)^{-1}$ for heptachlor epoxide based on the results of long-term carcinogenesis bioassays conducted by Davis (1965) and Witherup et al. (1955). This value is based on the numbers of observed liver carcinomas in the exposed group of two strains of mice of both sexes and in female rats (IRIS, 1996). The USEPA has also established an inhalation cancer slope factor for this compound of 9.1 $(mg/kg\text{-}day)^{-1}$.

The USEPA has established an oral RfD value of 0.000013 mg/kg-day for heptachlor epoxide (IRIS, 1996). This value is based on the University of Cincinnati (1958) study which reported increased liver-to-body weight ratios in beagles administered 0.013 to 0.19 mg/kg/day for 60 weeks. The Agency has not derived an inhalation RfD value for heptachlor epoxide (IRIS, 1996). For the purposes of this assessment, the oral RfD value is used as the inhalation RfD value.

References

ATSDR. 1991. *Toxicological Profile for Heptachlor/Heptachlor Epoxide: Draft.*. Agency for Toxic Substances and Disease Registry, Atlanta, GA and U.S. Department of Health and Human Services, Public Health Service. October.

Buck, W.B., R.D. Radeleff, J.B. Jackson et al. 1959. Oral toxicity studies with heptachlor and heptachlor epoxide in young calves. *J. Econ. Entomon.* 52(6):1127-1129. (Cited in ATSDR, 1991)

Davis, K.J. 1965. *Pathology Report on Mice Fed Aldrin, Dieldrin, Heptachlor, and Heptachlor Epoxide for Two Years.* Internal FDA Memorandum to Dr. A.J. Lehman. July 19. (Cited in IRIS, 1996)

Eichelberger, J.W. and J.J. Lichtenberg. 1971. Persistence of pesticides in river water. *Environ. Sci. Technol.* 5:541-544. (Cited in ATSDR, 1991)

Eisler, M. 1968. Heptachlor: Toxicology and safety evaluation. *Ind. Med. Surg.* 37(11):840-844. (Cited in ATSDR, 1991)

Geyer, H., P. Sheehan, D. Kotzias et al. 1982. Prediction of ecotoxicological behavior of chemicals: Relationship between physicochemical properties and bioaccumulation of organic chemicals in the mussel *Mytilus edulis*. *Chemosphere*. 11:1121-1134. (Cited in ATSDR, 1991)

Graham, R.E., K.R. Burson, C.F. Hammer et al. 1973. Photochemical decomposition of heptachlor epoxide. *J. Agric. Food Chem.* 21:824-834. (Cited in ATSDR, 1991)

Hartley, D.M. and J.B. Johnston. 1983. Use of the fresh water clam *Corbicula manilensis* as a monitor for organochlorine pesticides. *Bull. Environ. Contam. Toxicol.* 31:33-40. (Cited in ATSDR, 1991)

Hawker, D.W. and D.W. Connell. 1986. Bioconcentration of lipophilic compounds by some aquatic organisms. *Ecotox. Environ. Safety.* 11:184-197. (Cited in ATSDR, 1991)

HSDB. 1995. *Heptachlor Epoxide.* Hazardous Substances DataBank, National Library of Medicine, Bethesda, MD.

IRIS. 1996. *Heptachlor Epoxide*. Integrated Risk Information System. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH.

Lyman, W.J., W.F. Reehl, and D.H. Rosenblatt. (eds). 1982. *Handbook of Chemical Property Estimation Methods*. New York, NY: McGraw-Hill Book Company. (Cited in ATSDR, 1991)

Mackay, D. 1982. Correlation of bioconcentration factors. *Environ. Sci. Technol.* 16:274-278. (Cited in ATSDR, 1991)

OHM/TADS. 1985. Heptachlor Epoxide. *Oil and Hazardous Materials/Technical Assistance Data System*. Chemical Information Systems, Inc. December. (Cited in ATSDR, 1991)

Podowski, A.A., B.C. Banerjee, M. Feroz et al. 1979. Photolysis of heptachlor and cis-chlordane and toxicity of their photoisomers to animals. *Arch. Environ. Contam. Toxicol.* 8:509-518. (Cited in ATSDR, 1991)

University of Cincinnati. 1958. *A Summary of the Observations Pertaining to the Physiological Responses of Beagle Dogs When They Were Fed Diets Containing Various Concentrations of Heptachlor Epoxide Daily for a Period of Sixty Weeks*. Preliminary Report. U.S. Environmental Protection Agency, Freedom of Information Office, Washington, D.C. MRID No. 00061912. (Cited in ATSDR, 1991)

USEPA. 1987. Determination of Henry's Law Constants of Selected Priority Pollutants. U.S. Environmental Protection Agency, Cincinnati, OH. EPA-600/D-87/229. (Cited in ATSDR, 1991)

Verschueren, K. 1983. *Handbook of Environmental Data on Organic Chemicals, Second Edition*. New York, NY: Van Nostrand Reinhold Company. (Cited in ATSDR, 1991)

Velsicol Chemical Co. 1973. *MRID No. 00061912*. (Cited in IRIS, 1996)

Weast, R.C. (ed). 1985. *CRC Handbook of Chemistry and Physics, 66th Edition*. Boca Raton, FL: CRC Press, Inc. (Cited in ATSDR, 1991)

Windholz, M. ed. 1983. *The Merck Index: An Encyclopedia of Chemicals, Drugs, and Biologicals, 10th Edition*. Rahway, NJ: Merck and Company, Inc. (Cited in ATSDR, 1991)

Witherup, S., F.P. Cleveland, F.E. Shaffer et al. 1955. *The Physiological Effects of the Introduction of Heptachlor into the Diet of Experimental Animals in Varying Levels of Concentration: Report of Experiment No. 2.* U.S. Environmental Protection Agency, Freedom of Information Office, Washington, D.C. MRID No. 00062599. (Cited in ATSDR, 1991)

Toxicity Profile

Manganese

Chemical Formula	Mn	ATSDR, 1990
Molecular Weight	54.94	Sax and Lewis, 1987
Vapor Pressure	1 mmHg @ 1,292°C	USEPA, 1984
Boiling Point	1,962°C	Weast, 1985
Melting Point	1,244°C	Weast, 1985
Henry's Law Constant	No data	ATSDR, 1990
Solubility	Decomposes	Sax and Lewis, 1987
Partition Coefficients		
$\log K_{ow}$	No data	ATSDR, 1990
$\log K_{oc}$	No data	ATSDR, 1990

Manganese, which comprises about 0.1% of the earth's crust, is a common component of numerous minerals (ATSDR, 1990). Most manganese in the U.S. is used to produce ferromanganese, which is subsequently used in steel production (ATSDR, 1990). Other manganese compounds are used in the production of batteries, porcelain, and fireworks, as catalysts, in glazes and varnishes, as a fungicide, and as a nutritional supplement (ATSDR, 1990).

Manganese may be released to the atmosphere as industrial emissions (especially from iron and steel foundries) or with the combustion of fossil fuels (ATSDR, 1990). Natural sources of airborne manganese include erosion of soils and volcanic eruptions. Releases of manganese to water may result from industrial facilities or as leachate from soils and landfills. Soils may contain naturally occurring manganese or may contain elevated levels associated with waste disposal (ATSDR, 1990).

Manganese exists primarily adsorbed to particulates in the atmosphere; removal is largely by dry deposition, with lesser removal by rain washout (ATSDR, 1990). In water, manganese transport and partitioning depends upon the solubility of the compound containing manganese, as well as pH and redox potential. In soils and sediments, manganese partitioning is likewise dependent upon cation exchange capacity and organic composition (ATSDR, 1990). Significant bioconcentration of manganese by lower aquatic organisms is possible; however there is some evidence that biomagnification in the food chain is unlikely (ATSDR, 1990).

The primary target for manganese toxicity by all exposure routes in humans appears to be the central nervous system. Humans with very high occupational inhalation exposures have developed a neurological syndrome resembling Parkinson's disease; similar symptoms have been reported in a few cases of high oral exposure (ATSDR, 1990).

The USEPA has classified manganese in Group D, not classifiable as to human carcinogenicity, based on inadequate human and animal carcinogenicity data (IRIS, 1996). The Agency has not derived an oral or inhalation cancer slope factor for manganese.

Because the oral bioavailability of manganese varies depending on the medium of exposure, the USEPA has derived two separate chronic oral RfDs for manganese, one for use in dietary exposures and one for use in drinking water exposures. The chronic oral RfD for dietary exposure to manganese is based on three studies of human consumption of manganese in the diet or as a dietary supplement. In these studies, a NOAEL of 0.14 mg/kg-day for dietary exposures was identified. Uncertainty and modifying factors of 1 each were applied to the NOAEL to derive the oral RfD of 0.14 mg/kg-day (IRIS, 1996). Additionally, an oral RfD for manganese ingested via water has been estimated to be 0.023 mg/kg-day (IRIS, 1997). The Agency has not, however, developed an inhalation RfD value for manganese. In lieu of verified values, a provisional inhalation RfD value of 0.0000143 mg/kg-day was applied in this assessment. This value was obtained from the USEPA Region III (1996) guidance document for RCRA sites and is regarded as a provisional value.

References

ATSDR. 1990. *Toxicological Profile for Manganese and Compounds.* Agency for Toxic Substance and Disease Registry, Atlanta, GA and U.S. Department of Health and Human Services. PB93-110781. October.

IRIS. 1996. *Manganese.* Integrated Risk Information System, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH.

Sax, N.I. and R.J. Lewis. (eds). 1987. *Hawley's Condensed Chemical Dictionary, 11th Edition.* New York, NY: Van Nostrand Reinhold Company. 727-731. (Cited in ATSDR, 1990)

USEPA. 1984. *Health Effects Assessment for Manganese and Compounds.* U.S. Environmental Protection Agency, Office of Research and Development, Cincinnati, OH. EPA/540/1-86/057. (Cited in ATSDR, 1990)

USEPA. 1996. *Risk-Based Concentration Table, January - June 1996.* Dr. Roy L. Smith, U.S. Environmental Protection Agency, Region III, Office of RCRA, Technical and Program Support Branch, Philadelphia, PA. April 30.

Toxicological Profile

Methylene Chloride

Chemical Formula	CH_2Cl_2	Sax and Lewis, 1987
Molecular Weight	84.94 g/mol	Verschueren, 1983
Vapor Pressure	349 mmHg @ 20°C	Verschueren, 1983
Boiling Point	312.9°K	Howard, 1991
Melting Point	-95.1°C	Weast, 1985
Henry's Law Constant	2.68×10^{-3} atm-m^3/mol	Howard, 1991
Solubility	16,700 mg/L @ 25°C	Verschueren, 1983
Liquid Diffusion Coefficients	1.02×10^{-5} cm^2/sec	Lyman et al., 1990
Gaseous Diffusion Coefficients	1.11×10^{-1} cm^2/sec	Lyman et al., 1990
Partition Coefficient		
K_{oc}	47.9	Howard, 1991

Methylene chloride is widely used in industrial solvents, as well as in a number of other products. One-fourth of the methylene chloride produced in the United States is used as a solvent in paint strippers and removers. To a lesser extent, it is employed as a propellent in aerosols, such as spray paints, automotive products, and insect sprays, and as a process solvent in the manufacture of drugs, pharmaceuticals, and film coatings. It is also used as a blowing agent in urethane foam and in the manufacture of electronics (NTP, 1989). Additionally, the use of methylene chloride may be involved in food processing (ATSDR, 1991).

Because it is a readily volatile liquid, methylene chloride volatilizes to the atmosphere from water and soil (ATSDR, 1991). In the atmosphere, it is likely to react with photochemically generated hydroxyl radicals with typical reaction rates ranging from 1.0×10^{-13} to 1.0×10^{-15} cm^3/mol-sec (Cox et al., 1976; Davis et al., 1976; Crutzen and Fishman, 1977; Cuppitt, 1980, 1987; Atkinson et al., 1985). An average atmospheric half-life for methylene chloride has been estimated at about 130 days (Cox et al., 1976; Davis et al., 1976; Altshuller, 1980; Cuppitt, 1987).

Methylene chloride does not strongly sorb to soils or sediments and, based on its low K_{oc} value, is likely to be very mobile and ultimately leach from soils into groundwater (Dilling et al., 1975; Roy and Griffin, 1985; Bahnick and Doucette, 1988; Dobbs et al., 1989). Volatilization is likely to be the

primary fate process for methylene chloride in soils, while biodegradation by soil organisms and leaching to groundwater probably occur more extensively within subsurface soil systems (Sawhney et al., 1989).

The rate of hydrolysis of methylene chloride in water varies with changes in temperature and pH (ATSDR, 1991). Dilling et al. (1975) calculated an estimated hydrolytic half-life of approximately 18 months for methylene chloride in aquatic systems at 25°C. In acidic solutions at 80-150°C, however, the half-life is reduced to about 14 days (ATSDR, 1991). Additionally, biodegradation may be a significant fate process for methylene chloride in water systems (Brunner et al., 1980; Davis et al., 1981; Tabak et al., 1981; Stover and Kincannon, 1983; USEPA, 1985). Based on an estimated bioconcentration factor of 2.3, methylene chloride is unlikely to significantly biomagnify within aquatic food chains (EPA, 1980; ATSDR, 1991).

Inhalation is the primary route of human exposure to methylene chloride. In some cases, acute exposure has resulted in death (Stewart and Hake, 1976; Bonventre et al., 1977; Hall and Rumack, 1990). At chronic exposures of 30-120 ppm, humans occupationally exposed to methylene chloride did not experience an increased risk of mortality (Friedlander et al., 1978). However, studies have shown that exposure to 3,500 ppm for 14 to 104 weeks increased the risk of mortality in some animals (MacEwen et al., 1972; Burek et al., 1984). By contrast, oral exposure has not been observed to cause human mortality. Laboratory studies, however, have shown that methylene chloride can be toxic to animals (ATSDR, 1991; IRIS, 1996). Studies of acute oral exposures have reported mortality in rats dosed with greater than 2,100 mg/kg methylene chloride (Kimura et al., 1971; Ugazio et al., 1973).

Neurological complications, such as decreased auditory and visual functions, have been observed in humans following a single exposure of 300 ppm of methylene chloride (Fodor and Winneke, 1971; Stewart et al., 1972; Winneke, 1974). Psychomotor performance was impaired in subjects exposed to concentrations of 800 ppm for a duration of four hours (Winneke, 1974). Exposure to levels ranging from 515-986 ppm for 1-2 hours has resulted in alterations in visual evoked response (Stewart et al., 1972). Studies of individuals occupationally exposed to 75 to 100 ppm reported no signs of neurological or behavioral impairment (Cherry et al., 1981). A minimal risk level for acute inhalation exposure (≤14 days) of 0.4 ppm has been proposed for methylene chloride (ATSDR, 1991).

Methylene chloride does not appear to significantly affect reproduction or development in laboratory animals. In a study conducted by Schwetz et al. (1975), mice and rats exposed to 1,250 ppm methylene chloride vapor experienced minor skeletal malformations. In a similar study, behavioral changes and reduced fetal weight occurred in rat pups following the exposure of the dams to 4,500 ppm. The significance of these findings is questionable, however, since observed effect levels occurred at maternally toxic levels (ATSDR, 1991). Animal tests for reproductive toxicity were negative. Rats exposed to concentrations of 1,500 ppm for two generations did not experience changes in fertility or litter size (Nitschke et al., 1988), and male rats exposed to 200 ppm for 6 weeks failed to develop microscopic lesions in the testes (Raje et al., 1988).

In addition, acute exposure (6 hours to 7 days) of guinea pigs to 5,200 ppm methylene chloride resulted in biochemical changes in the liver (Morris et al., 1979). At levels of 75-100 ppm, a 110-day continuous exposure caused fatty changes in the liver of mice and rats (Weinstein and Diamond, 1972; Haun et al., 1972; Kjellstrand et al., 1986). One 2-year study reported that rats experienced an increased incidence of fatty changes at 500 ppm, but not at 200 ppm (Nitschke et al., 1988). Similarly, ingestion of methylene chloride has been associated with fatty changes at 55 mg/kg-day in rats and at 175 mg/kg-day in mice. Neither mice nor rats showed adverse effects at dose levels of 6 mg/kg-day (Serota et al., 1986a;b).

ATSDR (1991) reports that methylene chloride may be a weak mutagen in mammalian systems. While epidemiological studies have not linked human exposure to methylene chloride to increased incidence of cancer, animal studies have provided sufficient evidence of carcinogenicity (ATSDR, 1991). In both male and female rats, mammary gland tumors increased when animals were exposed to 500 ppm (Burek et al., 1984; Nitschke et al., 1988; NTP, 1989). Inhalation of 2,000 ppm caused an increased incidence in alveolar and bronchiolar neoplasms in mice (NTP, 1986), and oral administration of 50-250 mg/kg-day of methylene chloride in drinking water resulted in an increased incidence of liver tumors in male mice and female rats (Serota et al., 1986a;b).

The USEPA has classified methylene chloride as a Group B2 carcinogen, a probable human carcinogen based on inadequate human data and sufficient evidence of carcinogenicity in animals. This classification is based on various animal studies which indicate an increased incidence of hepatocellular neoplasms and alveolar/bronchiolar neoplasms in mice, and an increased incidence of benign mammary tumors, salivary gland sarcomas, and leukemia in rats (IRIS, 1996).

The USEPA has established an oral cancer slope factor of 0.0075 $(mg/kg\text{-}day)^{-1}$ for methylene chloride based on the results of a number of studies which reported adverse hepatic effects in laboratory animals exposed to methylene chloride (IRIS, 1996). The National Coffee Association (NCA, 1982) conducted a 2-year study of rats dosed at levels of 5, 50, 125, and 250 mg/kg-day. Effects such as histopathological changes in the liver were seen at the nominal dose of 50 mg/kg-day. The Agency has also published an inhalation unit risk of 4.7×10^{-7} $\mu g/m^3$ based on the presence of hepatocellular adenomas or carcinomas in female mice exposed to methylene chloride via inhalation (IRIS, 1996). In the absence of an inhalation cancer slope factor, a provisional inhalation value of $0.00164\ (mg/kg\text{-}day)^{-1}$ was applied in this assessment. This value was obtained from the USEPA Region III (1996) guidance document for RCRA sites and is regarded as a provisional value.

The USEPA has also determined a chronic oral RfD value of 0.06 mg/kg-day based on the low nominal dose for histological alterations in the liver of rats exposed to methylene chloride via drinking water (NCA, 1982). The low nominal dose of 5 mg/kg-day acts as the NOAEL (IRIS, 1996). The Agency has published an inhalation RfC of 3.0 mg/kg-day based on liver toxicity in rats following a 2-year intermittent inhalation study (USEPA, 1995). For the purposes of this assessment, a provisional inhalation RfD value of 0.857 mg/kg-day was applied. This value was also obtained from the USEPA Region III (1996) guidance document for RCRA sites and is regarded as provisional.

References

Altshuller. 1980. Lifetimes of organic molecules in the troposphere and lower stratosphere, *Adv. Environ. Sci. Technol.* 10:221-228 (Cited in ATSDR, 1991)

Atkinson R., Carter W.P., Aschmann, S.M. et al. 1985. *Atmospheric fates of organic chemicals: Prediction of ozone and hydroxyl radical reaction rates and mechanisms.* Report to U.S. Environmental Protection Agency, Atmospheric Sciences Research Laboratory, Research Triangle Park, NC, by University of California, Statewide Air Pollution Research Center, Riverside, CA. EPA/600/3-85/063. NTIS No. PB85-241529. (Cited in ATSDR, 1991)

ATSDR. 1991. *Toxicological Profile for Methylene Chloride: Draft.* Agency for Toxic Substances and Disease Registry, Atlanta, GA and U.S. Department of Health and Human Services, Public Health Service, Washington, D.C. October.

Bahnick, D.A. and W.J. Doucette, 1988. Use of molecular connectivity indices to estimate soil sorption coefficients for organic chemicals. *Chemosphere* 17:1703-1715. (Cited in ATSDR, 1991)

Bonventre, J., D. Brennan, D. Juson et al. 1977. Two deaths following accidental inhalation of dichloromethane and 1,1,1-trichloroethane. *J. Anal. Toxicol.* 1:158-160. (Cited in ATSDR, 1991)

Brunner, W.D., D., Staub, and T. Leisinger. 1980. Bacterial degradation of dichloromethane. *Appl. Environ. Microbiol.* 40:950-958.

Burek, J.D., K.D. Nitschke, T.J. Bell et al. 1984. Methylene chloride: A two-year inhalation toxicity and oncogenicity study in rats and hamsters. *Fund. Appl. Toxicol.* 4:30-47. (Cited in ATSDR, 1991)

Cherry, H., H. Venables, H.A. Waldron et al. 1981. Some observations on workers exposed to methylene chloride. *Br. J. Ind. Med.* 38:351-355. (Cited in ATSDR, 1991)

Cox, R.A., R.C. Denwent, A.E.J. Eggleton et al. 1976. Photochemical oxidation of halocarbons in the troposphere, *Atmos. Environ.* 10:305-308. (Cited in ATSDR, 1991)

Crutzen, P.J. and J. Fishman. 1977. Average concentrations of OH in the troposphere and the budgets of CH_4, CO, H_2 and CH_3CCl_3. *Geophys. Res. Lett.* 4:321-324. (Cited in ATSDR, 1991)

Cuppit, L.T. 1980. *Fate of toxic and hazardous materials in the air environment.* U.S. Environmental Protection Agency, Environmental Sciences Research Lab. Research Triangle Park, NC. NTIS no. PB80-221948. (Cited in ATSDR, 1991)

Cuppit, L.T. 1987. *Atmospheric persistence of eight air toxics. Project summary.* U.S. Environmental Protection Agency, Atmospheric Sciences Reserch Laboratory, Research Triangle Park, NC. EPA/600/53-87/004. (Cited in ATSDR, 1991)

Davis E.M., H.E. Murray, J.G. Liehr et al. 1981. Basic microbial degradation rates and chemical byproducts of selected organic compounds. *Water Res.* 15:1125-1127. (Cited in ATSDR, 1991)

Davis, D.D., G. Machado, B. Conaway et al. 1976. A temperature-dependent kinetics study of the reaction of OH with CH_3Cl, CH_2Cl_2, $CHCL_3$, and CH_3. *Br. J. Chem. Phys.* 65:1268-1274. (Cited in ATSDR, 1991)

Dilling, W.L. N.B. Tefertiller, G.J. Kallos. 1975. Evaporation rates of methylene chloride, chloroform, 1,1,1-trichloroethane, trichloroethylene, tetrachloroethylene, and other chlorinated compounds in dilute aqueous solutions. *Environ. Sci. Technol.* 9:833-838. (Cited in ATSDR, 1991)

Dobbs, R.A., L. Wang, and R. Govind. 1989. Sorption of toxic organic compounds on wastewater solids: Correlation with fundamental properties. *Environ. Sci. Technol.* 23:1092-1097. (Cited in ATSDR, 1991)

Fodor, G.G. and G. Winneke. 1971. *Nervous system disturbances in men and animals experimentally exposed in industrial solvent vapors in England.* Proceedings of the 2nd International Clean Air Congress. New York, NY: Academic Press.

Friedlander, B.R., F.T. Hearne, and S. Hall. 1978. Epidemiologic investigation of employees chronically exposed to methylene chloride--mortality analysis. *J. Occup. Med.* 20:657-666. (Cited in ATSDR, 1991)

Hall, A.H. and B.H. Rumack. 1990. Methylene chloride exposure in furniture-stripping shops: Ventilation and respirator use practices. *J. Occup. Med.* 32:33-41. (Cited in ATSDR, 1991)

Hansch, C. and A. Leo. 1979. *Substituent constants for correlation analysis in chemistry and biology.* New York, NY: John Wiley and Sons. (Cited in ATSDR, 1991)

Haun, C.C., E.H. Vernot, K.I. Karmer et al. 1972. Continuous animal exposure to low levels of dichloromethane. In: *Proceedings of the Third Annual Conference on Environmental Toxicology.* Wright Patterson Air Force Base, OH: Aerospace Medical Research Laboratory, 199-208. AMRL-TR-72-130. (Cited in ATSDR, 1991)

Howard, P.H. (ed). 1991. *Handbook of Environmental Fate and Exposure Data for Organic Chemicals. Volume II: Solvents.* Celsea, MI: Lewis Publishers, Inc.

IRIS. 1996. *Methylene Chloride.* Integrated Risk Information System, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH.

Kimura, E.T., D.M. Ebert, and P.W. Doge. 1971. Acute toxicity and limits of solvent residue for sixteen organic solvents. *Toxicol. Appl. Pharmacol.* 19:699-704. (Cited in ATSDR, 1991)

Kjellstrand, P., M. Bjerkemo, M. Adler-Maihofer et al. 1986. Effects of methylene chloride on body and organ weight and plasma butyrylcholinesterase activity in mice. *Acta. Pharmacol. Toxicol. (Copenh).* 59:73-79. (Cited in ATSDR, 1991)

Lyman, W.J., W.F. Reehl, and D.H. Rosenblatt. 1990. *Handbook of Chemical Property Estimation Methods: Environmental Behavior of Organic Compounds.* Washington, DC: American Chemical Society.

MacEwen, J.D., E.H. Vernot, and C.C. Haun. 1972. *Continuous animal exposure to dichloromethane.* Aerospace Medical Research Laboratory, Wright-Patterson Air Force Base, OH. AMRL-TR-72-28. (Cited in ATSDR, 1991)

Morris, J.B., F.A. Smith, and R.H. Garman. 1979. Studies on methylene chloride-induced fatty liver. *Exp. Mol. Path.* 30::386-393. (Cited in ATSDR, 1991)

NCA. 1982. *24-Month Chronic Toxicity and Oncogenicity Study of Methylene Chloride in Rats. Final Report.* Prepared for the National Coffee Association by Hazleton Laboratories of America, Inc., Vienna, VA. (Unpublished). (Cited in IRIS, 1996)

Nitschke, K.D., J.D. Burek, T.J. Bell et al. 1988. Methylene chloride: A 2-year inhalation toxicity and oncogenicity study in rats. *Fundam. Appl. Toxicol.* 11:48-59.

NTP. 1986. *Toxicology and Carcinogenesis Studies of Dichloromethane (CAS No. 75-09-2) in F344/N Rats and B6C3F1 Mice (Inhalation Studies).* National Toxicology Program, National Institutes of Health, U.S. Department of Health and Human Services, Washington, D.C. NTP-TRS-306. (Cited in IRIS, 1996)

NTP. 1989. *Fifth Annual Report on Carcinogens: Summary 1989.* National Toxicology Program, Report to the National Institute of Environmental Health Sciences, Research Triangle Park, NC, by Technical Resources, Inc., Rockville, MD. (Cited in ATSDR, 1991)

Raje, R., M. Basso, T. Tolen et al. 1988. Evaluation of *in vivo* mutagenicity of low-dose methylene chloride in mice. *J. Am. Coll. Toxicol.* 7:699-703. (Cited in ATSDR, 1991)

Roy, W.R. and R.A. Griffin. 1985. Mobility of organic solvents in water-saturated soil materials. *Environ. Geol. Water Sci.* 7:241-247. (Cited in ATSDR, 1991)

Sawhney, B.L. 1989. Movement of organic chemicals through landfill and hazardous waste disposal sites. In: *Reactions and movement of organic chemicals in soils.* Madison, WI: Soil Science Society of America and American and American jSociety of Agronomy, 447-474. (Cited in ATSDR, 1991)

Sax, N.I. and R. J. Lewis, Sr. 1987. *Hawley's Condensed Chemical Dictionary. 11th Ed.* New York, NY: Van Nostrand Reinhold Company, 768. (Cited in ATSDR, 1991)

Schwetz, B.A., B.J. Leong, and P.J. Gehring. 1975. The effect of maternally inhaled trichloroethylene, perchloroethane, methyl chloroform, and methylene chloride on embryonal and fetal development in mice and rats. *Toxicol. Appl. Pharmacol. 32:84-96.* (Cited in ATSDR, 1991)

Serota, D.G., A.K. Thakur, B.M. Ulland et al. 1986a. A two year drinking-water study of dichloromethane in rodents. I. Rats. *Food. Chem. Toxicol.* 24:951-958. (Cited in ATSDR, 1991)

Serota, D.G., A.K. Thakur, B.M. Ulland et al. 1986b. A two year drinking-water study of dichloromethane in rodents. II. Mice. *Food. Chem. Toxicol.* 24:959-963. (Cited in ATSDR, 1991)

Stewart, R.D., T.N. Fischer, M.J. Hosko et al. 1972. Experimental human exposure to methylene chloride. *Arch. Environ. Health.* 25:342-348. (Cited in ATSDR, 1991)

Stewart, R.D. and C.L. Hake. 1976. Paint remover hazard. *J. Med. Assoc.* 235:398-401. (Cited in ATSDR, 1991)

Stover, E.L. and D.F. Kincannon. 1983. Biological treatability of specific organic ompounds found in chemical industry wastewaters. *J. Water Pollut. Control. Fed.* 55:978-109. (Cited in ATSDR, 1991)

USEPA. 1980. *Ambient Water Quality Criteria for Halomethanes.* U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Washington, D.C. EPA-44/5-80. (Cited in ATSDR, 1991)

USEPA. 1985. *Health Assessment Document for Dichloromethane.* Final Report. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Washington, D.C. EPA/600/8-82/004f. (Cited in ATSDR, 1991)

USEPA. 1996. *Risk-Based Concentration Table, January - June 1996.* Dr. Roy L. Smith, U.S. Environmental Protection Agency, Region III, Office of RCRA, Technical and Program Support Branch, Philadelphia, PA. April 30.

Tabak, H.H., S.A. Quave, C.I. Mashni et al., 1981. Biodegradability studies with organic priority pollutant compounds. *J. Water Pollut. Control Assoc.* 53:1503-1518. (Cited in ATSDR, 1991)

Ugazio, G. E. Burdino, O. Danni et al. 1973. Hepatoxicity and lethality of halogenoalkanes. *Biochem. Soc. Trans.* 1:968-972. (Cited in ATSDR, 1991)

Verschueren, K. 1983. *Handbook of Environmental Data on Organic Chemicals. 2nd Ed.* New York: Van Nostrand Reinhold, 848-849. (Cited in ATSDR, 1991)

Weast, R.C. (ed). 1985. *CRC Handbook of Chemistry and Physics. 66th Ed.* Boca Raton, Fl: CRC Press, C-349. (Cited in ATSDR, 1991)

Weinstein, R.S. and S.S. Diamond. 1972. Hepatotoxicity of dichloromethane (methylene chloride) with continuous inhalation exposure at a low dose level. In: *Proceedings of the 3rd Annual Conference on Environmental Toxicology.* Aerospace Medical Research Laboratory, Wright-Patterson Air Force Base, OH. 209-220. AMRL-TR-72-130. (Cited in ATSDR, 1991)

Winneke, G. 1994. Behavioral effects of methylene chloride and carbon monixide as assessed by sensory and psychomotor performance. IN: Xintaras C., B.L. Johnson, and I. de Groot, (eds).

Behavioral Toxicology. Washington, DC: U.S. Government Printing Office, 130-144. (Cited in ATSDR, 1991)

Toxicity Profile

Polychlorinated Biphenyls (PCBs)

Polychlorinated biphenyls (PCBs) are a class of compounds comprised of 209 individual congeners. Each chlorinated biphenyl molecule contains from one to ten chlorine atoms. Commercially used PCBs are mixtures of individual polychlorinated biphenyl congeners that contain specified percentages of chlorine; for example, Aroclor 1260 contains 60% chlorine by weight. Commercial PCBs were manufactured in the United States by Monsanto Chemical Company and sold under the industrial trade name, Aroclor® (ATSDR, 1993). Analytical methods for environmental samples of PCBs are available for quantification of total PCBs, Aroclor mixtures, and PCB congeners (ATSDR, 1993).

In general, PCBs have low water solubility, high solubility in organic solvents, and are known for their insulating properties, thermal stability and resistance to oxidation and various chemical agents. Additional chemical and physical properties for several Aroclors are listed in Table G-1. PCBs do not occur naturally in the environment. Until their ban in July 1979, PCBs were largely used in electrical capacitors and transformers. Additionally, PCBs were used as electrical insulators, lubricants, hydraulic fluids, diffusion pump oils, cutting oils, plasticizers, liquid seals, and paint additives (ATSDR, 1993).

The environmental fate and transport of PCBs involves absorption to particulate and organic matter, volatilization, biodegradation, and photolysis. The low solubility, strong sorption to soils/sediments, and hydrophobicity of PCBs cause them to be extremely persistent and virtually immobile in soils (ATSDR, 1993). Several studies indicate that both aerobic and anaerobic biodegradation are significant removal mechanisms for lower chlorinated PCBs (Bedard et al., 1987; Kohler et al., 1988; Hill et al., 1989). Higher chlorinated Aroclors tend to resist aerobic biodegradation but are susceptible to anaerobic dechlorination (Brown et al., 1987; Abramowicz and Brennan, 1991). Reported half-lives in soil for PCBs range from 2 months for lower chlorinated congeners to 2 to 6 years for higher chlorinated congeners (Eduljee, 1987; McClure, 1976; Iwata et al., 1973). PCBs in the atmosphere tend to exist primarily in the vapor phase (ATSDR, 1993). However, the tendency of PCBs to adsorb to airborne particulates increases as the degree of chlorination increases (ATSDR, 1993). PCBs in the atmosphere are physically removed by both wet and dry deposition (Eisenreich et al., 1981). Vapor-phase reaction of PCBs with hydroxyl radicals is the dominant transformation process in the atmosphere (Atkinson, 1987).

A subgroup of PCB congeners, known as "coplanar PCBs", are considered chemically and toxicologically similar to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) (Safe, 1990). These "dioxin-like" congeners are so named because the pattern of chlorine substitution of the molecule allows the biphenyl rings to rotate in the same plane. The coplanar PCBs consist of a group of twelve congeners which have 4 or more chlorine atoms with no more than one substitution in the ortho- positions (USEPA, 1994). Due to their similarity to 2,3,7,8-substituted dioxins and furans, the coplanar PCBs are sometimes evaluated separately for risk assessment purposes.

Rats exposed to PCBs via oral exposure for acute durations have been reported to result in hepatotoxicity (Carter, 1984, 1985; Carter and Koo, 1984; Kato and Yoshida, 1980; Kling et al., 1978; Price et al., 1988). Intermediate-duration studies with several species indicate that liver, kidneys, and skin are the primary toxicity targets (Allen, 1975; Bleavins et al., 1980; Byrne et al., 1987; Kimbrough et al., 1972; Treon et al., 1956; Tryphonas et al., 1986a; Vos and Beems, 1971; Vos and Notenboom-Ram, 1972). Available chronic investigations with animals exposed to PCBs via inhalation or dermal exposures are inconclusive. Results from oral exposure studies, however, resemble intermediate-duration exposures (ATSDR, 1993).

Among humans, epidemiological studies of occupational exposures to PCBs show a variety of impacts including chromosomal aberrations, developmental effects, immunological effects, and neurotoxicity (Fein et al., 1984b; Fischbein et al., 1979; Humphrey, 1983; Kalina et al., 1991; Rogan et al., 1986; Taylor et al., 1984; Tryphonas et al., 1991a, 1991b).

Several laboratory studies have shown PCB mixtures containing an average of 60% chlorine substitution to be carcinogenic in rats (Kimbrough et al., 1975; Schaeffer et al., 1984; Norback and Weltman, 1985). In its weight-of-evidence determination of PCB carcinogenicity, the USEPA (1995) categorizes all PCB mixtures in Group B2 (probable human carcinogen) based on sufficient evidence of carcinogenicity in rodents. In its evaluation of the carcinogenic potency of PCB mixtures, USEPA (1988) considered the results of three positive rat studies (Kimbrough et al., 1975; Schaeffer et al., 1984; Norback and Weltman, 1985). Based on the female rat bioassay of Norback and Weltman (1985), the USEPA developed an oral cancer slope factor of 7.7 $(mg/kg\text{-}day)^{-1}$ (USEPA, 1988). This slope factor is based on the combined incidence of hepatocellular neoplastic nodules, trabecular carcinomas, and adenocarcinomas reported for female rats; scaling from rats to humans based on body weight raised to the 2/3s power; and adjusting the dose by the ratio of the exposure duration to the animals' expected lifespan. An inhalation cancer slope factor has not been established for

PCBs. For the purposes of this assessment, however, the oral value is used as the inhalation cancer slope factor.

The rat liver slides from these studies have recently been reevaluated by a Pathology Working Group (PWG) of independent expert pathologists, utilizing the current National Toxicology Program (NTP) guidelines for histopathological examination and diagnosis of proliferative rat liver lesions (IEHR, 1991; Moore et al., 1994). This reevaluation confirmed the findings of the original studies that PCB mixtures with 60% chlorination are carcinogenic in rats, but showed somewhat lower incidences of tumors than originally reported. The results of this reevaluation have been reported (IEHR, 1991; Moore et al., 1994) and submitted to the USEPA for review (Moore, 1991).

The USEPA has classified Aroclor 1248 and Aroclor 1254 as Group B2 carcinogens, probable human carcinogens, based on insufficient data in humans and adequate carcinogenic data in animals. To date, the USEPA has published RfDs for Aroclor 1016 and Aroclor 1254 (IRIS, 1996). In this assessment, the more conservative oral RfD for Aroclor 1254 was used to evaluate noncarcinogenic effects of Aroclor mixtures (IRIS, 1996). The RfD for Aroclor 1254 is based on dermal/ocular and immunologic effects in rhesus monkeys (Tryphonas et al., 1989; 1991a,b; Arnold et al., 1993a,b). For the purposes of developing the Aroclor 1254 RfD, USEPA (IRIS, 1996) identified a LOAEL of 0.005 mg/kg-day, and applied a total uncertainty factor of 300 to derive the RfD of 0.00002 mg/kg-day (20 ng/kg-day). The uncertainty factor applied included factors of 3 each for interspecies extrapolation, use of a LOAEL, and subchronic exposure. In addition, a factor of 10 was applied for interindividual variability, and the total uncertainty factor of 270 was rounded to 300. The USEPA has not established an inhalation RfD value for PCBs. For the purposes of this assessment, however, the oral RfD value is used as the inhalation RfD value.

"LOAEL" — "Lowest Observed Actual Effects Level"

References

Abramowicz, D.A. and M.J. Brennan. 1990. Anaerobic and aerobic biodegradation of endogenous PCBs. Biological Remediation of Contaminated Sediments, with Special Emphasis on the Great Lakes, Manitowoc, WI.

Allen, J.R. 1975. Response of the nonhuman primate to polychlorinated biphenyl exposure. *Fed Proc.* 34:1675-1679. (Cited in ATSDR, 1993)

Arnold, D.L., F. Bryce, R. Stapley et al. 1993a. Toxicological consequences of Aroclor 12454 ingestion by female rhesus (*Mucaca mulatta*) monkeys, Part 1A: Prebreeding phase - clinical health findings. *Fd. Chem. Toxicol.* 31:799-810.

Arnold, D.L., F. Bryce, K. Karpinski et al. 1993b. Toxicological consequences of Aroclor 12454 ingestion by female rhesus (*Mucaca mulatta*) monkeys, Part 1B: Prebreeding phase - clinical and analytical laboratory findings. *Fd. Chem. Toxicol.* 31:811-824.

Atkinson, R. 1987. Estimation of OH radical reaction rate constants and atmospheric lifetimes for polychlorobiphenyls, dibenzo-p-dioxins, and dibenzofurans. *Environ. Sci. Technol.* 21(3):305-307.

ATSDR. 1993. *Selected PCBs (Aroclor -1260, 1254, -1248, -1242, -1232, -1221, and -1016).* Agency for Toxic Substances and Disease Registry, Atlanta, GA and U.S. Department of Health and Human Services, Public Health Service. April.

Ballschmiter, K., C. Rappe, and H.R. Buser. 1989. Chemical properties, analytical methods and environmental levels of PCBs, PETs, PCNs, and PBBs. In: Kimbrough, R.D. and A.A. Jensen (eds). *Halogenated Biphenyls, Terphenyls, Naphthalenes, Dibenzodioxins, and Related Products. 2nd Edition.* Amsterdam: Elsevier Science Publishers. 47-102. (Cited in ATSDR, 1993)

Bedard, D.L., M.L. Haberl, R.J. May, and M.J. Brennan. 1987. Evidence for novel mechanisms of polychlorinated biphenyl metabolism in Alcaligenes eutrophus H850. *Appl. Environ. Microbiol.* 53(5):1103-1112.

Bleavins, M.R., R.J. Aulerich, and R.K. Ringer. 1980. Polychlorinated biphenyls (Aroclors 1016 and 1242): Effects on survival and reproduction in mink and ferrets. *Arch. Environ. Contam. Toxicol.* 9:627-635. (Cited in ATSDR, 1993)

Brown, J.F., D.L. Bedard, M.J. Brennan, J.C. Carnahan, H. Feng, and R.E. Wagner. 1987. Polychlorinated biphenyl dechlorination in aquatic sediments. *Science* 236(May 8):709-712.

Byrne, J.J., J.P. Carbone, and E.A. Hanson. 1987. Hypothyroidism and abnormalities in the kinetics of thyroid hormone metabolism in rats treated chronically with polychlorinated biphenyl and polybrominated biphenyl. *Endocrinology.* 121:520-527. (Cited in ATSDR, 1993)

Callahan, M.A., M.W. Slimak, N.W. Gabel et al. 1979. *Water-Related Environmental Fate of 129 Priority Pollutants, Volume I., Chapter 36.* U.S. Environmental Protection Agency, Washington, D.C. EPA-440/4-79-029a. (Cited in ATSDR, 1993)

Carter, J.W. 1984. Hypercholesterolemia induced by PCBs (Aroclor 1254) in Fischer rats. *Bull. Environ. Contam. Toxicol.* 33:78-83. (Cited in ATSDR, 1993)

Carter, J.W. 1985. Effects of dietary PCBs (Aroclor 1254) on serum levels of lipoprotein cholesterol in Fischer rats. *Bull. Environ. Contam. Toxicol.* 34:427-431. (Cited in ATSDR, 1993)

Carter, J.W. and S.I. Koo. 1984. Effects of dietary Aroclor 1254 (PCBs) on serum levels of lipoprotein cholesterol and tissue distribution of zinc, copper, and calcium in Fischer rats. *Nutr. Rep. Int.* 29:223-232. (Cited in ATSDR, 1993)

Eduljee, G. 1987. Volatility of TCDD and PCB from soil. *Chemosphere* 16(4):907-920.

Eisenreich, S.J., B.B. Looney, and J.D. Thornton. 1981. Airborne organic contaminants in the Great Lakes ecosystem. *Environ. Sci. Technol.* 15(1):30-38.

Fein, G.G., J.L. Jacobson, S.W. Jacobson et al. 1984. Prenatal exposure to polychlorinated biphenyls: Effects on birth size and gestation age. *J. Pediatr.* 105:315-320. (Cited in ATSDR, 1993)

Fischbein, A., M.S. Wolff, R. Lilis et al. 1979. Clinical findings among PCB-exposed capacitor manufacturing workers. *Ann. NY. Acad. Sci.* 320:703-715. (Cited in ATSDR, 1993)

Hill, T.A. and R.C. Wands. 1989. Serial allometric factor extrapolation: Compartmental and physiological pharmacokinetic approaches. *Health Phys.* 57(Supp. 1):395-401.

Hollifiled, H.C. 1979. Rapid nephelometric estimate of water solubility of highly insoluble organic chemicals of environmental interest. *Bull. Environ. Contam. Toxicol.* 23:579-586. (Cited in ATSDR, 1993)

Humphrey, H.E.B. 1983. Population studies of PCBs in Michigan residents. In: *PCBs: Human and Environmental Hazards.* D'itri, F.M. and M. Kamrin (eds). Boston, MA: Butterworth. 299-310. (Cited in ATSDR, 1993)

IEHR. 1991. *Reassessment of Liver Findings in Five PCB Studies in Rats.* Institute for Evaluating Health Risks, Washington, D.C. July 1.

IRIS. 1996. *Polychlorinated Biphenyls/Aroclors.* Integrated Risk Information System, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH.

Iwata, Y., W.E. Westlake and F.A. Gunther. 1973. Varying persistence of polychlorinated biphenyls in six California soils under laboratory conditions. *Bull. Environ. Contam. Toxicol.* 9(4):204-211.

Kalina, I., R.J. Sram, H. Konecna et al. 1991. Cytogenic analysis of peripheral blood lymphocytes in workers occupationally exposed to polychlorinated biphenyls. *Teratogenesis Carcinog. Mutagen.* 11:77-82. (Cited in ATSDR, 1993)

Kato, N. and A. Yoshida. 1980. Effect of dietary PCB on hepatic cholesterogenesis in rats. *Nutr. Rep. Int.* 21:107-112. (Cited in ATSDR, 1993)

Kimbrough, R.D., R.E. Linder, T.B. Gaines et al. 1972. Morphological changes in livers of rats fed polychlorinated biphenyls. *Arch. Environ. Health.* 25:354-364.

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.

Kling, D., J. Kunkle, A.S. Roller et al. 1978. Polychlorinated biphenyls: *In vivo* and *in vitro* modifications of cholesterol and fatty acid biosynthesis. *J. Environ. Pathol. Toxicol.* 1:813-828. (Cited in ATSDR, 1993)

Kohler, H.-P.E., D. Kohler-Staub, and D.D. Focht. 1988. Cometabolism of polychlorinated biphenyls: Enhanced transformation of Aroclor 1254 by growing bacterial cells. *Appl. Environ. Microbiol.* 54(8):1940-1945.

Lyman, W.J., W.F. Reehl, and D.H. Rosenblatt. 1990. *Handbook of Chemical Property Estimation Methods: Environmental Behavior of Organic Compounds.* Washington, D.C.: American Chemical Society.

Mabey, W.R., J.H. Smith, R.T. Podoll, H.L. Johnson, T. Mill, T.W. Chou, J. Gate, I. Waight-Partridge, H. Jaber, and D. Vandenberg (eds). 1982. *Aquatic Fate Process for Organic Priority Pollutants.* U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Washington, D.C. EPA-440/4-81-014. (Cited in Mackay et al., 1992)

Mackay, D., W.Y. Shiu, and K.C. Ma. 1992. *Illustrated Handbook of Physical-Chemical Properties and Environmental Fate for Organic Chemicals. Volume I: Monoaromatic Hydrocarbons, Chlorobenzenes, and PCBs.* Chelsea, MI: Lewis Publishers, Inc.

McClure, V.E. 1976. Transport of heavy chlorinated hydrocarbons in the atmosphere. *Environ. Sci. Technol.* 10(13):1223-1229.

Moore, R.W., C.R. Jefcoate, and R.E. Peterson. 1991. 2,3,7,8-Tetrachlorodibenzo-p-dioxin inhibits steroidogenesis in the rat testis by inhibiting the mobilization of cholesterol to cytochrome P450. *Toxicol. Appl. Pharmacol.* 109:85-97.

Moore, D.W. and T.M. Dillon. 1994. *Quality Assurance/Quality Control (QA/QC) Guidance for Laboratory Dredged Material Bioassays.* U.S. Army Corps of Engineers, Waterways Experiment Station, Vicksburg, MS. Miscellaneous Paper D-94-3. May.

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.

Paris, D.F., W.C. Steen, and G.L. Baughman. 1978. Role of the physicochemical properties of Aroclor 1016 and 1242 in determining their fate and transport in aquatic environments. *Chemosphere.* 7:319-325. (Cited in ATSDR, 1993)

Price, S.C., S. Ozalp, R. Weaver et al. 1988. Thyroid hyperactivity caused by hypolipodaemic compounds and polychlorinated biphenyls: The effect of coadministration in the liver and thyroid. In: *The Target Organ and The Toxic Process: Proceedings of the European Society of Toxicology.*

Strasbourg, September 17-19. Chambers, L. (ed). New York: Springer-Verlag. 85-92. (Cited in ATSDR, 1993)

Rogan, W.J., B.C. Gladen, J.D. McKinney et al. 1986. Neonatal effects of transplacental exposure to PCBs and DDE. *J. Pediatr.* 109:335-341. (Cited in ATSDR, 1993)

Safe, S. 1990. Polychlorinated biphenyls (PCBs), dibenzo-p-dioxins (PCDDs), dibenzofurans (PCDFs), and related compounds: Environmental and mechanistic considerations which support the development of toxic equivalency factors (TEFs). *CRC Crit. Rev. Toxicol.* 21(1):51-88.

Schaeffer, E., H. Greim, and W. Goessner. 1984. Pathology of chronic polychlorinated biphenyl (PCB) feeding in rats. *Toxicol. Appl. Pharm.* 75:278-288.

Taylor, P.R., C.E. Lawrence, H.L. Hwang et al. 1984. Polychlorinated biphenyls: Influence on birthweight and gestation. *Am. J. Public Health.* 74:1153-1154. (Cited in ATSDR, 1993)

Treon, J.F., F.P. Cleveland, J.W. Chappel et al. 1956. The toxicity of the vapours of Aroclor 1242 and Aroclor 1254. *Am. Ind. Hyg. Assoc. Q.* 17:204-213. (Cited in ATSDR, 1993)

Tryphonas, L., S. Charbonneau, H. Tryphonas et al. 1986. Comparative aspects of Aroclor 1254 toxicity in adult cynomolgus and rhesus monkeys: A pilot study. *Arch. Environ. Contam. Toxicol.* 15:159-169. (Cited in ATSDR, 1993)

Tryphonas, H., S. Hayward, L. O'Grady, J.C.K. Loo, D.L. Arnold, F. Bryce, and Z.Z. Zawidzka. 1989. Immunotoxicity studies of PCB (Aroclor 1254) in the adult rhesus (*Macaca mulatta*) monkey - Preliminary report. *Int. J. Immunoph.* 11(2):199-206.

Tryphonas, H., M.I. Luster, K.L. White et al. 1991a. Effects of PCB (Aroclor 1254) on non-specific immune parameters in rhesus monkeys. *Int. J. Immunopharmacol.* 13:639-648.

Tryphonas, H., M.I. Luster, K.L. White et al. 1991b. Effect of chronic exposure of PCB (Aroclor 1254) or specific and low specific immune parameters in the rhesus monkey. *Fund. Appl. Toxicol.* 16:773-786.

USEPA. 1988. *Final: Drinking Water Criteria Document for Polychlorinated Biphenyls (PCBs).* U.S. Environmental Protection Agency, Cincinnati, OH, Prepared by Office of Health and Environmental Assessment. Prepared for Office of Drinking Water. April.

USEPA. 1994. *Estimating Exposure to Dioxin-Like Compounds, Volume 1: Executive Summary.* U.S. Environmental Protection Agency, Office of Research and Development, Washington, DC. EPA/600/6-88/005Ca. June.

Vos, J.G. and R.B. Beems. 1971. Dermal toxicity studies of technical polychlorinated biphenyls and fractions thereof in rabbits. *Toxicol. Appl. Pharmacol.* 19:617-633. (Cited in ATSDR, 1993)

Vos, J.G. and E. Notenboom-Ram. 1972. Comparative study of 2,4,5,2',4',5'-hexachlorobiphenyl and a polychlorinated biphenyl mixture in rabbits. *Toxicol. Appl. Pharmacol.* 23:563-578. (Cited in ATSDR, 1993)

Toxicity Profile

Polychlorinated Dibenzo-p-dioxins and Dibenzofurans (PCDD/Fs)

Polychlorinated dibenzodioxins and dibenzofurans (PCDD/Fs) constitute a class of compounds comprised of 210 different congeners. Table G-2 lists the chemical/physical properties of all PCDD/F congeners. They have been found in incinerator emissions, pesticides, commercial PCB mixtures, technical products such as chlorophenol and chlorophenoxy acids, car exhaust, and as by-products in many industrial manufacturing processes (Rappe, 1987). Reports in the scientific literature have identified a growing number of other processes and vectors which can potentially lead to PCDD and PCDF emissions to the environment, including municipal solid waste (MSW) and hazardous waste incinerators, motor vehicles using diesel fuels, large and small combustion engines, residential and commercial fuel oil and wood heating systems, pigment and dye manufacture, dry cleaning operations, municipal sewage sludge, forest fires, chlorination of wood pulp, petroleum refining, scrap iron smelting, and secondary copper smelting (USEPA, 1994a). Many researchers have suggested that all incomplete combustion processes as well as any thermal treatment of organic material above 400-500°C (752-932°F) in combination with a chlorine source can lead to the production of PCDDs and PCDFs (Ballschmiter et al., 1986; Czuczwa and Hites, 1986; Konheim, 1986; Marklund et al., 1987; Rappe et al., 1987).

The scientific evidence collected over the past decade strongly indicates that PCDDs and PCDFs are transported in the atmosphere and are ubiquitous in the environment (Sheffield, 1985; Czuczwa and Hites, 1986; Rappe and Kjeller, 1987, 1988; Southerland et al., 1987). For example, investigators have identified the presence of PCDDs and PCDFs in the sediments of a pristine lake on a Lake Superior island (Czuczwa et al., 1985). Because of the isolated location of the lake, the only significant anthropogenic source of these chemicals is atmospheric deposition. The presence of PCDDs and PCDFs in airborne particulate samples from major cities in the United States and abroad also confirm that transport by wind is the predominant mechanism in the distribution of PCDDs and PCDFs (Czuczwa and Hites, 1986; Rappe and Kjeller, 1988). PCDDs and PCDFs have been detected in precipitation and in ground fog (Czuczwa et al., 1989), as well as in locations generally considered to be isolated and pristine (Czuczwa et al., 1985).

Once deposited, further migration of dioxin is limited (United Kingdom, 1989). The extremely low water solubility, low volatility, and high affinity for organic material causes dioxin compounds to partition out of the aqueous and gaseous phases and adsorb strongly to organic materials. In surface

water, dioxin tends to be removed from the aqueous phase and deposited in organic bottom sediments. On land, PCDD/Fs bind strongly to the soil matrix; therefore there is little potential for leaching to the groundwater. The estimated half-life of tetraCDD (TCDD) in soils has been estimated to be as long as 3.3 to 100 years (di Dominico et al., 1980; Young, 1983; Martin and Thiel, 1990; Gough, 1991).

Travis and Hattemer-Frey (1987) addressed the relationship of TCDD levels in air, water, soil, and sediment. In this study, the authors estimated that approximately 80% of the TCDD released into the environment is via air emissions, 15% via water, and 5% via soil application. The pattern of environmental residue levels from the U.S. and Europe clearly shows that the affinity for organic carbon material promotes the transfer of TCDD into the soil and sediments.

Of the 210 PCDD/Fs, the toxicological database is most extensive for TCDD. The USEPA (1985) has classified TCDD as a probable (B2) human carcinogen based on sufficient evidence of carcinogenicity from animal studies, but insufficient evidence from human studies. The USEPA does not currently have any published toxicity values for TCDD on IRIS. Virtually all risk estimates for humans have been based on extrapolations from a two-year chronic toxicity and oncogenicity study of Sprague-Dawley rats conducted by Kociba et al. (1978). The researchers conducted a dietary study in which rats were administered 0.001, 0.01, or 0.1 μg TCDD/kg-day. Female rats receiving 0.1 μg TCDD/kg-day exhibited a significant increase in liver tumors. The USEPA (1985) used the linearized multistage nonthreshold dose-response model and an animal-to-human scaling factor based on surface area to extrapolate human dose-response relationships from the results reported by Kociba et al. (1978) to estimate a cancer slope factor (CSF) of 156,000 $(mg/kg\text{-}day)^{-1}$ (USEPA, 1994). This CSF is being reevaluated. Currently, reported CSFs range from 9,000 to 100,000 $(mg/kg\text{-}day)^{-1}$ (USEPA, 1994).

In 1990 (Keenan et al., 1991), members of the Maine Scientific Advisory Panel (SAP) requested that the Kociba et al. (1978) study be reevaluated by an independent Pathology Working Group (PWG) using the recently updated National Toxicology Program (NTP) tumor classification system. In contrast to the previous system, the updated classification system distinguishes between hyperplasia and adenoma, a distinction which is critical to the cancer dose-response assessment of TCDD. The current protocol is now accepted by U.S. federal agencies, including the USEPA (1986).

Using the new classification protocol, the PWG (1990) concluded that there were substantially fewer cancerous tumors observed in the study than had been originally reported by Kociba et al. (1978).

Based on the results of the reanalysis, USEPA (1991) calculated revised CSFs of 51.000 (mg/kg-day)$^{-1}$ for liver tumors only, and 75,000 (mg/kg-day)$^{-1}$ for pooled liver, lung, or nasal turbinate/hard palate tumors. In the interim of finalizing those reports, the HEAST (1997) value of 150,000 was used as the oral CSF.

The USEPA has not developed an oral or inhalation RfD value for TCDD or any other PCDD/F. Furthermore, the USEPA (1994a) has stated that, given elevated background exposures to "dioxin-like compounds", it would be inappropriate to derive an RfD for these compounds.

Relative Toxicity of Dioxin and Furan Congeners

In most, if not all cases, environmental exposure to PCDD/F involves exposure to a variety of PCDD/F congeners, rather than solely TCDD. While little toxicity data exist regarding most of these congeners, it is widely acknowledged in the scientific and regulatory communities that only the 17 congeners with the 2,3,7,8 substitution pattern can be expected to exhibit "dioxin-like" toxicity. In an effort to assess the toxicity of PCDD/F congeners for which little or no scientific data are available, a rating scheme involving the assignment of Toxicity Equivalency Factors (TEFs) for each of the 17 congeners was devised (Safe, 1990; USEPA, 1986). Of the seventeen 2,3,7,8-substituted congeners, 2,3,7,8-TCDD is considered to be the most toxic and has been the most thoroughly examined. Therefore, 2,3,7,8-TCDD was selected as the basis for predicting the toxicity of the other sixteen 2,3,7,8-substituted congeners and assigned a TEF value of 1. TEF values ranging from 0 to 1 were assigned to each of these 2,3,7,8-substituted congeners based on an estimate of toxicity relative to that of 2,3,7,8-TCDD.

The USEPA (1989) proposed interim guidelines for estimating risks associated with mixtures of PCDDs and PCDFs following the International Toxicity Equivalency Factor (I-TEF) scheme developed by an international project under the auspices of the North Atlantic Treaty Organization's Committee on Challenges of Modern Society (NATO/CCMS). For each congener, one or more of the following endpoints were used to derive I-TEFs: enzyme induction, thymic atrophy, body weight gain, potential bioaccumulation, teratogenicity, immunotoxicity, and lethality. The I-TEF scheme has been used by the USEPA for carcinogenic evaluation of PCDD/Fs. Consistent with USEPA practice, the I-TEFs have been used in this assessment to evaluate the carcinogenic potential of PCDD/Fs.

References

Ballschmiter, K., H. Buchert, R. Niemczyk, A. Munder, and M. Swerev. 1986. Automobile exhausts versus municipal-waste incineration as sources of the polychloro-dibenzodioxins (PCDD) and -furans (PCDF) found in the environment. *Chemosphere* 15(7):901-915.

Czuczwa, J., V. Katona, G. Pitts, M. Zimmerman, F. DeRoos, P. Capel, and W. Giger. 1989. Analysis of fog samples for PCDD and PCDF. *Chemosphere* 18(1-6):847-850.

Czuczwa, J.M., F. Niessen, and R.A. Hites. 1985. Historical record of polychlorinated dibenzo-p-dioxins and dibenzofurans in Swiss lake sediments. *Chemosphere* 14(9):1175-1179.

Czuczwa, J.M. and R.A. Hites. 1986. Airborne dioxins and dibenzofurans: Sources and fates. *Environ. Sci. Technol.* 20(2):195-200.

Di Domenico, A. , V. Silano, G. Viviano, and G. Zapponi. 1980. Accidental release of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) at Seveso, Italy. V. Environmental persistence of TCDD in soil. *Ecotoxic. Environ. Saf.* 4:339-345.

Gough, M. 1991. Human health effects: What the data indicates. *Sci. Tot. Environ.* 104:129-158.

Keenan, R.E., R.J. Wenning, A.H. Parsons, and D.J. Paustenbach. 1991. A re-evaluation of the tumor histopathology of Kociba et al. (1978) using 1990 criteria: Implications for the risk assessment of 2,3,7,8-TCDD using the linearized multistage model. Dioxin '91 Conference Proceedings, University of North Carolina, NC.

Kociba, R.J., D.G. Keyes, J.E. Beyer, R.M. Carreon, C.E. Wade, D.A. Dittenber, R.P. Kalnins, L.E. Frauson, C.N. Park, S.D. Barnard, R.A. Hummel, and C.G. Humiston. 1978. Results of a two-year chronic toxicity and oncogenicity study of 2,3,7,8-tetrachlorodibenzo-p-dioxin in rats. *Toxicol. Appl. Pharmacol.* 46:279-303.

Konheim, C.S. 1986. Dioxin exposure and motor vehicle emissions: An eye-opening perspective. *Waste Age* (November):69-76.

Mackay, D., W.Y. Shiu. and K.C. Ma (eds). 1992. *Illustrated Handbook of Physical-Chemical Properties and Environmental Fate for Organic Chemicals. Volume II: Polynuclear Aromatic Hydrocarbons, Polychlorinated Dioxins, and Dibenzofurans.* Chelsea, MI: Lewis Publishers, Inc.

Marklund, S., C. Rappe, M. Tysklind, and K.-E. Egeback. 1987. Identification of polychlorinated dibenzofurans and dioxins in exhausts from cars run on leaded gasoline. *Chemosphere* 16(1):29-36.

Martin, S.G. and D.A. Thiel. 1990. Memorandum from S.G. Martin, President and Principal Consultant, S.G. Martin & Associates, Inc., Wellington, CO and D.A. Thiel, Manager, Environmental Research, Georgia-Pacific Corporation, Port Edwards, WI to W.S. Rabert, Office of Toxic Substances, U.S. Environmental Protection Agency, Washington, D.C. RE: Comments on the Revision of Appendix F, Entitled "Environmental Risk Assessment for TCDD and TCDF-Contaminated Pulp Sludges on Terrestrial and Aquatic Wildlife." Attachments: Appendix A; Revisions of Tables 9 and 10 from the W.S. Rabert Risk Assessment of October 4, 1990. S.G. Martin & Associates, Inc. December 3.

PWG. 1990. *Pathology Working Group Report on 2,3,7,8-Tetrachlorodibenzo-p-dioxin Chronic Toxicity/Carcinogenicity Study in Sprague-Dawley Rats.* Prepared by R.M. Sauer, PWG Chairperson, PATHCO, INC., Ijamsville, MD and submitted to R.A. Michaels, Chairperson, Maine Scientific Advisory Panel. March 13.

Rappe, C. and L.-O. Kjeller. 1987. PCDDs and PCDFs in environmental samples: Air, particulates, sediments and soil. *Chemosphere* 16(8/9):1775-1780.

Rappe, C. and L.-O. Kjeller. 1988. Identification and quantification of PCDDs and PCDFs in urban air. *Chemosphere* 17(1):3-20.

Rappe, C., R. Andersson, P.-A. Bergqvist, C. Brohede, M. Hansson, L.-O. Kjeller, G. Lindstrom, S. Marklund, M. Nygren, S.E. Swanson, M. Tysklind, and K. Wiberg. 1987. Sources and relative importance of PCDD and PCDF emissions. *Waste Mgt. Res.* 5:225-237.

Safe, S. 1990. Polychlorinated biphenyls (PCBs), dibenzo-p-dioxins (PCDDs), dibenzofurans (PCDFs), and related compounds: Environmental and mechanistic considerations which support the development of toxic equivalency factors (TEFs). *CRC Crit. Rev. Toxicol.* 21(1):51-88.

Sheffield, A. 1985. Sources and releases of PCDD's and PCDF's to the Canadian environment. *Chemosphere* 14(6/7):811-814.

Southerland, J.H., W.B. Kuykendal, W.H. Lamason, A. Miles, and D.A. Oberacker. 1987. Assessment of combustion sources as emitters of chlorinated dioxin compounds: A report on the results of tier 4 of the National Dioxin Study. *Chemosphere* 16(8/9):2161-2168.

Travis, C.C. and H.A. Hattemer-Frey. 1987. Human exposure to 2,3,7,8-TCDD. *Chemosphere* 16(10-12):2331-2342.

UK. 1989. *Dioxins in the Environment: Report of an Interdepartmental Working Group on polychlorinated dibenzo-para-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs).* Department of the Environment, Central Directorate of Environmental Protection, United Kingdom. Pollution Paper No. 27.

USEPA. 1985. *Health Assessment Document for Polychlorinated Dibenzo-p-dioxins; Final Report.* U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH. PB86-122546. September.

USEPA. 1986. *Guidelines for Carcinogen Risk Assessment.* U.S Environmental Protection Agency, Washington, D.C., 51 Federal Register 185: 33992-34003. September 24.

USEPA. 1989. *Interim Procedures for Estimating Risks Associated wirth Exposures to Mixtures of Chlorinated Dibenzo-p-Dioxins and -Dibenzofurans (CDDs and CDFs) and 1989 Uptake.* U.S. Environmentasl Protection Agency, Risk Assessment Forum, Washington, D.C. EPA/625/3-89/016. March.

USEPA. 1991. *Great Lakes Initiative Criteria for Protection of Wildlife.* U.S. Environmental Protection Agency, Washington, D.C.

USEPA. 1991. *Risk Assessment Guidance for Superfund: Volume I - Human Health Evaluation Manual Supplemental Guidance "Standard Default Exposure Factors".* U.S. Environmental Protection Agency, Office of Emergency and Remedial Response, Toxics Integration Branch,Washington, DC. EPA 540/1-89/002. March 25.

USEPA. 1993. *Derivation of Proposed Human Health and Wildlife Bioaccumulation Factors for the Great Lakes Initiative; Draft Report.* U.S. Environmental Protection Agency, Environmental Research Laboratory, Office of Research and Development. Duluth, MN. PB93-154672. March.

USEPA. 1994a. *Estimating Exposure to Dioxin-Like Compounds. Volume I: Executive Summary.* U.S. Environmental Protection Agency, Office of Research and Development. Washington, DC. EPA/600/6-88/005Ca. June.

USEPA. 1994b. *Health Effects Assessment Summary Tables FY-1994 Annual.* U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response. Washington. D.C. PB94-921199. March.

Young, A.L. 1983. Long-term studies on the persistence and movement of TCDD in a natural ecosystem. *Environ. Sci. Res.* 26:173-190.

References [Chemical/Physical Table]

Broman, D., C. Naf, C. Rolff, and Y. Zebuhr. 1991. Occurence and dynamics of polychlorinated dibenzo-p-dioxins and dibenzofurans and polycyclic aromatic hydrocarbons in mixed surface layer of remote coastal and offshore waters of the Baltic. *Environ. Sci. Technol.* 25(11):1850-1864. (Cited in Mackay et al., 1992)

Burkhard, L.P. and D.W. Kuehl. 1986. N-octanol/water partition coefficients by reverse phase liquid chromatography/mass spectrometry for eight tetrachlorinated planar molecules. *Chemosphere.* 15(2):163-167. (Cited in USEPA, 1994)

Dunnivant, F.M., A.W. Eizerman, P.C. Jurs, and M.N. Hasan. 1992. Quantitative structure-property relationships for aqueous solubilities and Henry's Law constants of polychlorinated biphenyls. *Environ. Sci. Technol.* 26(8):1567-1573. (Cited in USEPA, 1994)

Friesen, K.J., L.P. Sarna, and G.R.B. Webster. 1985. Aqueous solubility of polychlorinated dibenzo-p-dioxins determined by high pressure liquid chromatography. *Chemosphere.* 14(9):1267-1274. (Cited in USEPA, 1994)

Friesen, K.J., J. Vilk, and D.C.G. Muir. 1990. Aqueous solubility of selected 2,3,7,8-substituted polychlorinated dibenzofurans (PCDFs). *Chemosphere.* 20(1-2):27-32. (Cited in USEPA, 1994)

Lyman, W.J., W.F. Reehl, ans D.H. Rosenblatt. 1990. *Handbook of Chemical Property Estimation Methods: Environmental Behavior of Organic Compounds.* Washington, D.C.: American Chemical Society.

Mackay, D., W.Y. Shiu, and K.C. Ma (eds). 1992. *Illustrated Handbook of Physical-Chemical Properties and Environmental Fate for Organic Chemicals. Volume II: Polynuclear Aromatic Hydrocarbons, Polychlorinated Dioxins, and Dibenzofurans.* Chelsea, MI: Lewis Publishers, Inc.

Marple, L., R. Brunck, B. Berridge, and L. Throop. 1987. Experimental and calculated physical constants for 2,3,7,8-tetrachlorodibenzo-p-dioxin. In: *Solving Hazardous Waste Problems.* Exner, J.H. (eds). ACS Symposium Series 338. Developed from a symposium sponsored by the Division of Environmental Chemistry at the 191st meeting of the American Chemical Society, New York, NY, April 13-18, 1986. pp. 105-113. (Cited in USEPA, 1994)

Marple, L., R. Brunk, and L. Throop. 1986a. Water solubility of 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Environ. Sci. Technol.* 20(2):180-182. (Cited in USEPA, 1994)

Marple, L., R. Brunk, and L. Throop. 1986b. Measurement of the water-octanol partition coefficient of 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Environ. Sci. Technol.* 20(4):397-399. (Cited in USEPA, 1994)

Podoll, R.T., H.M. Jaber, and T. Mill. 1986. Tetrachlorodibenzodioxin: Rates of volatilization and photolysis in the environment. *Environ. Sci. Technol.* 20:490-492. (Cited in USEPA, 1994)

Rordorf, B.F. 1987. Prediction of vapor pressures, boiling points, and enthalpies of fusion for twenty-nine halogenated dibenzo-p-dioxins. *Thermochimica Acta.* 112:117-122. (Cited in USEPA, 1994)

Rordorf, B.F. 1989. Prediction of vapor pressures, boiling points, and enthalpies of fusion for twenty-nine halogenated dibenzo-p-dioxins and fifty-five dibenzofurans by a vapor pressure correlation method. *Chemosphere.* 18(1-6):783-788. (Cited USEPA, 1994)

Shiu, W.Y., W. Doucette, F.A.P.C. Gobas, A. Andren, and D. Mackay. 1988. Physical-chemical properties of chlorinated dibenzo-p-dioxins. *Environ. Sci. Technol.* 22(6):651-658. (Cited in USEPA, 1994)

Sijm, D.T.H.M., H. Wever, P.J. de Vries, and A. Opperhuizen. 1989. Octan-1-ol/water partition coefficients of polychlorinated dibenzo-p-dioxins and dibenzofurans: Experimental values determined with a stirring method. *Chemosphere.* 19(1-6):263-266. (Cited in USEPA, 1994)

USEPA. 1994. *Draft: Estimating Exposure to Dioxin-Like Compounds. Volume II: Properties, Sources, Occurrence and Background Exposures.* U.S. Environmental Protection Agency, Office of Research and Development, Washington, D.C. EPA/600/6-88/005Cb. June.

USEPA. 1990. *Background Document to the Integrated Risk Assessment for Dioxins and Furans from Chlorine Bleaching in Pulp and Paper Mills.* U.S. Environmental Protection Agency, Office of Toxic Substances, Washington, D.C. EPA-560/5-90-014. (Cited in USEPA, 1994)

Webster, G.R.B., D.H. Muldrew, J.J. Graham, L.P. Sarna, and D.C.G. Muir. 1986. Dissolved organic matter mediated aquatic transport of chlorinated dioxins. *Chemosphere.* 15:9-12. (Cited in Mackay et al., 1992)

Toxicity Profile

Polycyclic Aromatic Hydrocarbons (PAHs)

Polycyclic Aromatic Hydrocarbons (PAHs) are a class of compounds containing two or more benzene rings. The chemical/physical properties of selected PAHs are listed in Table G-3. Most PAHs in the environment are formed during the combustion of organic compounds. Major sources of PAHs include incomplete combustion of fuels for heat and manufacturing, burning of coal, gasoline, diesel exhaust, and burning of municipal and agricultural wastes. Cigarette smoke also contains PAHs. To a limited extent, these compounds also occur naturally. Plants and bacteria synthesize PAHs during growth, and naturally-caused brush and forest fires produce PAHs (ATSDR, 1995).

The physical-chemical properties of PAHs are roughly correlated with their size and molecular weight; thus, PAHs may be grouped by molecular weight for the purpose of describing their environmental fate (ATSDR, 1995). High molecular weight PAHs are: benz(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(g,h,i)perylene, benzo(a)pyrene, chrysene, dibenz(a,h)anthracene, and indeno(1,2,3-cd)pyrene. Medium molecular weight PAHs are fluoranthene and pyrene. Low molecular weight PAHs include acenaphthene, acenaphthylene, anthracene, fluorene, naphthalene, and phenanthrene.

The medium and high molecular weight PAHs have large K_{oc} values, indicating a strong tendency to bind to organic matter (ATSDR, 1995). In addition, these PAHs have limited solubility in water, Unless these PAHs encounter organic liquids in which they might be transported, they are essentially immobile in water. The low molecular weight PAHs have lower K_{oc} values and greater water solubility, indicating greater mobility in water.

In surface waters, PAHs may be removed by volatilization, binding to suspended solids or sediments, photodegradation, or accumulation in aquatic biota. Low molecular weight PAHs, which have higher Henry's Law contants, will tend to volatilize (ATSDR, 1995). Higher molecular weight PAHs have very low Henry's Law constants, high K_{oc} values, and a tendency to bioaccumulate (ATSDR, 1995); these PAHs preferentially sorb to sediments or accumulate in biota.

Benzo(a)pyrene

The USEPA has classified benzo(a)pyrene (B(a)P) as a Group B2 carcinogen, probable human carcinogen, based on sufficient data in animals and inadequate data in humans. B(a)P has been shown to be carcinogenic in experimental animals (rodent and non-rodent species) following administration by oral, intratracheal, inhalation, and dermal routes (ATSDR, 1995). Oral administration of B(a)P to rats and hamsters produces stomach tumors.

The USEPA has established an oral cancer slope factor of 7.3 $(mg/kg\text{-}day)^{-1}$ for B(a)P based on the geometric mean of four slope factors derived with the data from two studies. In the first study, dietary exposure to B(a)P doses between 1 and 250 ppm resulted in squamous cell papillomas and carcinomas of the forestomach in CFW mice (IRIS, 1996). In the second study, dietary doses of 3 and 39 mg B(a)P/kg diet/year produced papillomas and carcinomas of the forestomach, larynx, and esophagus in male and female Sprague-Dawley rats. Also, USEPA Region III (1996) has derived an inhalation cancer slope factor of 6.1 $(mg/kg\text{-}day)^{-1}$ for benzo(a)pyrene. Additionally, the USEPA has not derived an oral or inhalation RfD value for B(a)P (IRIS, 1996).

Benz(a)anthracene, Benzo(b)fluoranthene, Dibenz(a,h)anthracene, and Indeno(1,2,3-c,d)pyrene

The USEPA has classified benz(a)anthracene, benzo(b)fluoranthene, dibenz(a,h)anthracene, and indeno(1,2,3-c,d)pyrene as Group B2 carcinogens, probable human carcinogens based on sufficient data in animals and no data in humans. Although these compounds are well-studied as carcinogens, the data are insufficient for the purpose of developing cancer slope factors (USEPA, 1993). In the absence of compound-specific cancer slope factors, the Agency has developed guidance on "order of potential potency" (relative to the potency of B(a)P) for the quantitative risk assessment of the carcinogenic PAHs (USEPA, 1993). This interim guidance provides order of magnitude relative potency estimates which are multiplied by the slope factor for B(a)P to estimate slope factors for the carcinogenic PAHs. The relative potency factors proposed by USEPA (1993) were used in this assessment. An oral cancer slope factor of 7.3 $(mg/kg\text{-}day)^{-1}$ is used for dibenz(a,h)anthracene while a value of 0.73 $(mg/kg\text{-}day)^{-1}$ is used for benz(a)anthracene, benzo(b)fluoranthene, and indeno(1,2,3-c,d)pyrene. Likewise, an inhalation cancer slope factor of 6.1 $(mg/kg\text{-}day)^{-1}$ is used for dibenz(a,h)anthracene while a value of 0.61 $(mg/kg\text{-}day)^{-1}$ is used for benz(a)anthracene, benzo(b)fluoranthene, and indeno(1,2,3-c,d)pyrene (USEPA Region III, 1996). The Agency has not established oral or inhalation RfD values for these PAHs (IRIS, 1996).

Acenaphthylene, Benzo(g,h,i)perylene and Phenanthrene

The EPA has classified acenaphthylene, benzo(g,h,i)perylene and phenanthrene as Group D carcinogens, not classifiable as to human carcinogenicity, dut to an absence of data in humans and inadequate data in animals. The Agency has not developed oral or inhalation cancer slope factors or RfD values for these compounds.

References

ATSDR. 1995. *Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHs).* U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA. August.

IRIS. 1996. *Chemical Search for Polycyclic Aromatic Hydrocarbons (PAHs).* Integrated Risk Information System. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH.

USEPA. 1993. *Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons.* U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. ECAO-CN-842. March.

USEPA. 1996. *Risk-Based Concentration Table, January-June 1996.* Dr. Roy L. Smith, U.S. Environmental Protection Agency, Region III, Office of RCRA, Technical and Program Support Branch, Philadelphia, PA. April 30.

Toxicological Profile

Tetrachloroethylene

Chemical Formula	C_2Cl_4	ACGIH, 1991
Molecular Weight	165.85 g/mol	Lide, 1990
Vapor Pressure	18.47 mmHg @ 25°C	HSDB, 1994
Boiling Point	394°K	Howard, 1991
Melting Point	-19°C	Lide, 1990
Henry's Law Constant	1.49×10^{-2} atm-m³/mol	Howard, 1991
Solubility	150 mg/L @ 25°C	HSDB, 1994
Partition Coefficient		
K_{oc}	209	ATSDR, 1995

Tetrachloroethylene is used primarily as a chemical intermediate in metal cleaning, vapor degreasing, dry cleaning, and textile processing (ATSDR, 1995). It is also employed as an anthelmintic in the treatment of hookworm and some nematode infestations (Budavari, 1989; HSDB, 1995).

Tetrachloroethylene is expected to be transported long distances in the atmosphere and is likely to be slowly removed from the atmosphere via wet deposition (Class and Ballschmiter, 1986; Jung et al., 1992). Dry deposition, however, is not expected to be a significant removal process (Cupitt, 1987). In the atmosphere, the transformation of tetrachloroethylene occurs as a reaction with photochemically produced hydroxyl radicals (Singh et al., 1982). The atmospheric half-life of tetrachloroethylene has been calculated within the range of 70 to 251 days depending on temperatures and hydroxyl ion concentrations (Atkinson, 1985; Class and Ballschmiter, 1986; Cupitt, 1987).

In soils systems, tetrachloroethylene may be removed via volatilization, but this process is influenced by surface-to-volume ratio and soil type (Zytner et al., 1989b). Estimated soil sorption coefficients for tetrachloroethylene in soil range from 177 to 534, indicating relatively high mobility in soil systems (Kenaga, 1980; Wilson et al., 1981; Swann et al., 1983; Seip et al., 1986; Zytner et al., 1989a). Biodegradation of tetrachloroethylene in soil systems is limited and occurs only under specific conditions (ATSDR, 1995). Additionally, several studies indicate that tetrachloroethylene may leach to groundwater systems (Schwarzenbach et al., 1983; Barber et al., 1988; Doust and Huang, 1992).

Studies suggest that tetrachloroethylene is rapidly volatilized from aquatic systems (Dilling et al., 1975; Dilling, 1977; Roberts and Dandliker, 1983; Okouchi, 1986; Chodola et al., 1989; Zytner et al., 1989b). Depending on water characteristics such as temperature, movement, depth, air movement, and surface-to-volume ratios, volatilization half-lives for tetrachloroethylene have been estimated within the range of 4 hours to 25 days (Schwarzenbach et al., 1979; Thomas, 1982; Wakeham et al., 1983; Chodola et al., 1989; Zytner et al., 1989b). Tetrachloroethylene does not appear to transform readily in aquatic systems, and is not significantly influenced by hydrolytic or photolytic reactions (Chodola et al., 1989; ATSDR, 1995). Biodegradation, however, appears to be an important transformation process, although it does not occur rapidly (ATSDR, 1995). In addition, bioconcentration factors for tetrachloroethylene in fish have been measured ranging between 10 and 100 (Neely et al., 1974; Kawasaki, 1980; Kenaga, 1980; Veith et al., 1980). Biomagnification within the aquatic food chain, however, is unlikely to play an important role (Pearson and McConnell, 1975).

A number of epidemiology studies have been conducted with dry cleaning and laundry workers (Hoover et al., 1975; Blair et al., 1979; Brown and Kaplan, 1987; Lynge and Thygsen, 1990; Newcomb and Carbone, 1992). These studies, however, are generally complicated by potential exposures to petroleum solvents, tobacco use, or other carcinogens (ATSDR, 1995).

Tetrachloroethylene is currently under review by the USEPA. The carcinogen classification of tetrachloroethylene is between a Group B2 and Group C carcinogen, a probable human carcinogen and possible human carcinogen, respectively (ATSDR, 1995; IRIS, 1996). The Agency has not established an oral or inhalation cancer slope factor for tetrachloroethylene. In lieu of verified values, a provisional oral cancer slope factor of 0.052 $(mg/kg\text{-}day)^{-1}$ and a provisional inhalation cancer slope factor of 0.002 $(mg/kg\text{-}day)^{-1}$ were applied in this assessment. This value was obtained from the USEPA Region III (1996) guidance document for RCRA sites and is regarded as a provisional value.

The USEPA has established an oral RfD value of 0.01 mg/kg-day for tetrachloroethylene based on hepatotoxic effects reported for mice exposed to tetrachloroethylene via gavage (IRIS, 1996). Buben and O'Flaherty (1985) administered tetrachloroethylene to mice in doses up to 2,000 mg/kg at 5 days per week for a total of 6 weeks. Results of the study indicate that hepatotoxic effects may occur at doses greater than 100 mg/kg, including increased liver weight and liver triglycerides. Similar results were reported for rats administered doses of 14, 400, or 1,400 mg/kg-day tetrachloroethylene in drinking water. Rats receiving 400 mg/kg-day or greater exhibited increased liver and kidney weights, as well as depressed body weight (Hayes et al., 1986). Additionally, several other studies have reported results of comparable hepatotoxic effects due to gavage or inhalation exposure in

laboratory animals (Rowe et al., 1952; Schumann et al., 1980; Kjellstrand et al., 1984; NTP, 1985). Finally, the USEPA has not determined an inhalation RfD value for tetrachloroethylene.

References

ACGIH. 1991. *Documentation of the Threshold Limit Values and Biological Exposure Indices.* 6th Edition. American Conference of Governmental Industrial Hygienists, Cincinnati, OH. (Cited in ATSDR, 1995)

Atkinson, R. 1985. Kinetics and mechanisms of the gas-phase reactions of the hyroxyl radical with organic compounds under atmospheric conditions. *Chem. Rev.* 85:130-132. (Cited in ATSDR, 1995)

ATSDR. 1995. *Toxicological Profile for Tetrachloroethylene: Draft Update.* Agency for Toxic Substances and Disease Registry, Atlanta, GA and U.S. Department of Health and Human Services, Public Health Service, Washington, D.C. August.

Barber, L.B., E.M. Thurman, and M.P. Schroeder. 1988. Long-term fate of organic micropollutants in sewage-contaminated groundwater. *Environ. Sci. Technol.* 22:205-211. (Cited in ATSDR, 1995)

Blair, A., P. Decooufle, and D. Grauman. 1979. Causes of death among laundry and dry cleaning workers. *Am. J. Public Health* 69:508-511. (Cited in ATSDR, 1995)

Brown, D.P. and S.D. Kaplan. 1987. Retrospective cohort mortality study of dry cleaning workers using perchloroethylene. *J. Occup. Med.* 29:535-541. (Cited in ATSDR, 1995)

Buben, J.A. and E.J. O'Flaherty. 1985. Delineation of the role of metabolism in the hepatotoxicity of trichloroethylene and perchloroethylene: A dose-effect study. *Toxicol. Appl. Pharmacol.* 78:105-122. (Cited in IRIS, 1996)

Budavari, S. 1989. *The Merck Index.* 11th Edition. Rahway, NJ: Merck and Co., Inc. (Cited in ATSDR, 1995)

Chodola, G.R., N. Biswas, J.K. Bewtra, et al. 1989. Fate of selected volatile organic substances in aqueous environment. *Wat. Poll. Res. J. Can.* 24:119-142. (Cited in ATSDR, 1995)

Class, T.H. and K. Ballschmiter. 1986. Chemistry of organic traces in air. VI: Distribution of chlorinated C1-C4 hydrocarbons in air over the northern and southern Atlantic Ocean. *Chemosphere* 15:413-427. (Cited in ATSDR, 1995)

Cupitt, L.T. 1987. *Atmospheric Persistence of Eight Air Toxics.* U.S. Environmental Protection Agency, Atmospheric Sciences Research Lab, Research Triangle Park, NC. EPA/600/3-87/004. (Cited in ATSDR, 1995)

Dilling, W.L. 1977. Interphase transfer processes. II. Evaporation rates of chloromethanes, ethanes, ethylenes, propanes, and propylenes from dilute aqueous solutions. Comparisons with theoretical predictions. *Environ. Sci. Technol.* 11:405-409. (Cited in ATSDR, 1995)

Dilling, W.L., N.B. Tefertiller, and G.J. Kallos. 1975. Evaporation rates and reactivities of methylene chloride, chloroform, 1,1,1-trichloroethane, trichloroethylene, tetrachloroethylene, and other chlorinated compounds in dilute aqueous solutions. *Environ. Sci. Technol.* 9:833-838. (Cited in ATSDR, 1995)

Doust, H.G. and J. Huang. 1992. The fate and transport of hazardous chemicals in the subsurface environment. *Wat. Sci. Technol.* 25:169-176. (Cited in ATSDR, 1995)

Hayes, J.R., L.W. Condie, and J.F. Borzelleca. 1986. The subchronic toxicity of tetrachloroethylene (perchloroethylene) administered in the drinking water of rats. *Fund. Appl. Toxicol.* 7:119-125. (Cited in IRIS, 1996)

Hoover, R., T.J. Mason, F.W. McKay, et al. 1975. Geographic patterns of cancer mortality in the United States. In: *Persons at High Risk of Cancer: An Approach to Cancer Ecology and Control.* New York, NY: Academic Press. p. 343-360. (Cited in ATSDR, 1995)

Howard, P.H. (ed). 1991. *Handbook of Environmental Fate and Exposure Data for Organic Chemicals. Volume II: Solvents.* Chelsea, MI: Lewis Publishers, Inc.

HSDB. 1995. *Tetrachloroethylene.* Hazardous Substances DataBank, National Library of Medicine, Bethesda, MD.

IRIS. 1996. *Tetrachloroethylene.* Integrated Risk Information System, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH.

Jung, W.T., M. Fujita, and D.H. Sohn. 1992. Levels of volatile halogenated hydrocarbons in Tokyo rain and their seasonal, time-series changes. *Jap. J. Toxicol. Environ. Health* 38:490-497. (Cited in ATSDR, 1995)

Kawasaki, M. 1980. Experiences with the test scheme under the chemical control law of Japan: An approach to structure-activity correlations. *Ecotoxicol. Environ. Saf.* 4:444-454. (Cited in ATSDR, 1995)

Kenaga, E.E. 1980. Predicted bioconcentration factors and soil sorption coefficients of pesticides and other chemicals. *Ecotoxicol. Environ. Saf.* 4:26-38. (Cited in ATSDR, 1995)

Kjellstrand, P., B. Holmquist, M. Kanje et al. 1984. Perchloroethylene: Effects on body and organ weights and plasma butyrylcholinesterase activity in mice. *Acta Pharmacol. Toxicol.* 54(5):414-424. (Cited in IRIS, 1996)

Lide, D.R. (ed.). 1990. *CRC Handbook of Chemistry and Physics.* 71st Edition. Ann Arbor, MI: CRC PRess, Inc. p. 3-242. (Cited in ATSDR, 1995)

Lynge, E. and L. Thygsen. 1990. Primary liver cancer among women in laundry and dry-cleaning work in Denmark. *Scand. J. Work Environ. Health* 16:108-112. (Cited in ATSDR, 1995)

Neely, W.B., D.R. Branson, and G.E. Blau. 1974. Partition coefficients to measure bioconcentration potential of organic chemicals in fish. *Environ. Sci. Technol.* 8:1113-1115. (Cited in ATSDR, 1995)

Newcomb, P.A. and P.P. Carbone. 1992. The health consequences of smoking: Cancer. *Med. Clin. North Am.* 76(2):305-331. (Cited in ATSDR, 1995)

NTP. 1985. *NTP Technical Report on the Toxicology and Carcinogenesis Studies of Tetrachloroethylene (Perchloroethylene).* National Toxicology Program, U.S. Department of Health and Human Services, National Institutes of Health, Washington, D.C. NIH Publication No. 85-2567. (Cited in IRIS, 1996)

Okouchi, S. 1986. Volatilization coefficient for stripping trichloroethylene, 1,1,1-trichloroethylene, and tetrachloroethylene from water. *Wat. Sci. Technol.* 18:137-138. (Cited in ATSDR, 1995)

Pearson, C.R. and G. McConnell. 1975. Chlorinated C1 and C2 hydrocarbons in the marine environment. *Proc. R. Soc. Lond.* 189:305-332. (Cited in ATSDR, 1995)

Roberts, P.V. and P.G. Dandliker. 1983. Mass transfer of volatile organic contaminants from aqueous solution to the atmosphere during surface aeration. *Environ. Sci. Technol.* 17:484-489. (Cited in ATSDR, 1995)

Rowe, V.K., D.D. McCollister, H.C. Spencer, E.M. Adams, and D.D. Irish. 1952. Vapor toxicity of tetrachloroethylene for laboratory animals and human subjects. *Arch. Ind. Hyg. Occup. Med.* 5:566-579. (Cited in IRIS, 1996)

Schumann, A.M., J.F. Quast, and P.G. Watanabe. 1980. The pharmacokinetics and macromolecular interaction of perchloroethylene in mice and rats as related to oncogenicity. *Toxicol. Appl. Pharmacol.* 55:207-219. (Cited in IRIS, 1996)

Schwarzenbach, R.P., E. Molnar-Kubica, W. Giger, et al. 1979. Distribution, residence time, and fluxes of tetrachloroethylene and 1,4-dichlorobenzene in Lake Zurich, Switzerland. *Environ. Sci. Technol.* 13:1367-1373. (Cited in ATSDR, 1995)

Schwarzenbach, R.P., W. Giger, E. Hoehn, and J.K. Schneider. 1983. Behavior of organic compounds during infiltration of river water to ground water: Field studies. *Environ. Sci. Technol.* 17:472-479. (Cited in ATSDR, 1995)

Seip, H.M., J. Alstad, G.E. Carlberg, et al. 1986. Measurement of mobility of organic compounds in soils. *Sci. Tot. Environ.* 50:87-101. (Cited in ATSDR, 1995)

Swann, R.L., D.A. Laskowski, P.J. McCall, et al. 1983. A rapid method for the estimation of the environmental parameters octanol/water partition coefficient, soil sorption constant, water to air ratio, and water solubility. *Res. Rev.* 85:17-28. (Cited in ATSDR, 1995)

Thomas, R.G. 1982. Volatilization from water. In: *Handbook of chemical property estimation methods: Environmental Behavior of Organic Compounds.* New York, NY: McGraw-Hill. (Cited in ATSDR, 1995)

USEPA. 1996. *Risk-Based Concentration Table, January - June 1996.* Dr. Roy L. Smith, U.S. Environmental Protection Agency, Region III, Office of RCRA, Technical and Program Support Branch, Philadelphia, PA. April 30.

Veith, G.D., K.J. Macek, S.R. Petrocelli, et al. 1980. An evaluation of using partition coefficients and water solubility to estimate bioconcentration factors for organic chemicals in fish. In: *Aquatic Toxicology.* Eaton, J.G., P.R. Parrish, and A.C. Hendricks (eds.). American Society for Testing MAterials. ASTM STP 707. (Cited in ATSDR, 1995)

Wakeham, S.G., A.C. Davis, and I.L. Karas. 1983. Mesocosm experiments to determine the fate and persistence of volatile organic compounds in coastal seawater. *Environ. Sci. Technol.* 17:611-617. (Cited in ATSDR, 1995)

Wilson, J.T., C.G. Enfield, W.J. Dunlap, et al. 1981. Transport and fate of selected organic pollutants in a sandy soil. *J. Environ. Qual.* 10:501-506. (Cited in ATSDR, 1995)

Zytner, R.G., N. Biswas, and J.K. Bewtra. 1989a. Adsorption and desorption of perchloroethylene in soils, peat moss, and granular activated carbon. *Can. J. Civil Eng.* 16:798-806. (Cited in ATSDR, 1995)

Zytner, R.G., N. Biswas, and J.K. Bewtra. 1989b. Volatilization of perchloroethylene from stagnant water and soil. In: *Proceedings of the 43rd Industrial Waste Conferences, Purdue University.* J.M. Bell (ed.). Chelsea, MI: Lewis Publishers, Inc. May 10-12. (Cited in ATSDR, 1995)

Toxicity Profile

Trichloroethylene

Chemical Formula	C_2HCl_3	SANSS, 1990
Molecular Weight	131.4	HSDB, 1990
Vapor Pressure	74 mmHg @ 25°C	Mackay and Shiu, 1981
Boiling Point	86.7°C	USEPA, 1985
Melting Point	-87.1°C	McNeill, 1979
Henry's Law Constant	0.011 atm-m^3/mol @ 25°C	Hine and Mookerjee, 1975
	0.020 atm-m^3/mol @ 20°C	Mackay and Shiu, 1981
Solubility	1.366 g/L @ 25°C	Tewari et al., 1982
	1.070 g/kg @ 20°C	McNeill, 1979
Liquid Diffusion Coefficient	8.16×10^{-6} cm^2/sec	Lyman et al., 1990
Gaseous Diffusion Coefficient	8.88×10^{-2} cm^2/sec	Lyman et al., 1990
Partition Coefficients		
$\log K_{ow}$	2.42	Hansch and Leo, 1985
$\log K_{oc}$	2.0	HSDB, 1995

The majority of trichloroethylene use is associated with vapor degreasing of fabricated metal parts, particularly among automotive and metal industries. A much smaller fraction is produced for chemical intermediates and export (CMR, 1983; Kimbrough et al., 1985). Trichloroethylene is used by textile industries as a solvent and to scour cotton and other fabrics (McNeill, 1979; Verschueren, 1983; Kuney, 1986). Trichloroethylene may also be employed as a component of adhesives, lubricants, paints, varnishes, paint strippers, pesticides, pharmaceuticals, polychlorinated aliphatics, and flame retardant chemicals (McNeill, 1979; Hawley, 1981; Windholz, 1983; Mannsville Chemical Products Corporation, 1985).

The high vapor pressure and Henry's Law constant suggest that trichloroethylene exists entirely in the vapor phase when released to the atmosphere (Eisenreich et al., 1981). Trichloroethylene may be removed from the atmosphere via wet deposition, but is quick to revolatilize. The relatively short half-life of trichloroethylene should not permit long-range global transport (Class and Ballschmiter, 1986).

Trichloroethylene released to surface water will volatilize rapidly to air. Volatilization, however, depends on temperature, water movement, water depth, and associated air movements (USEPA, 1985).

When released to land, trichloroethylene readily moves within soils to groundwater (Schwarzenbach et al., 1983). Once in groundwater, trichloroethylene is not expected to transform into other chemicals (ATSDR, 1991).

Trichloroethylene was once employed as a surgical anesthetic. Central nervous system depressant effects due to trichloroethylene exposure have been thoroughly studied (Vernon and Ferguson, 1969; Stewart et al., 1970; Salvini et al., 1971; Nomiyama and Nomiyama, 1977). Acute and chronic exposures to trichloroethylene among human subjects may result in neurological effects such as drowsiness, headache, dizziness, and alterations in behavioral performance. Laboratory animals exposed to trichloroethylene also display a variety of behavioral effects due to acute and chronic inhalation exposures (ATSDR, 1991).

Chronic effects of occupational exposures to trichloroethylene may result in gastrointestinal effects including anorexia, nausea, vomiting, and intolerance to fatty foods (Smith, 1966; Buxton and Hayward, 1967; Milby, 1968; Clearfield, 1970). Human subjects experiencing acute inhalation exposure may result in dry throats and mild eye irritation (Stewart et al., 1970). Humans exposed to higher inhalation doses of trichloroethylene may experience cranial nerve damage (Feldman et al., 1988). Human exposures to trichloroethylene resulting in death have occurred only in rare instances of accidental exposures to unusually high doses. Lethal concentrations of trichloroethylene to laboratory animals have been reported following acute, intermediate, and chronic inhalation exposures (ATSDR, 1991).

Results of reproductive or developmental studies with trichloroethylene have not produced significant adverse effects in humans or animals (ATSDR, 1991). Oral exposures to trichloroethylene combined with other volatile hydrocarbons, however, have resulted in instances of childhood leukemia (Lagakos et al., 1986a). Individuals reported to have consumed trichloroethylene and other solvents in drinking water for several years developed increased rates of childhood leukemia, as well as cardiac arrhythmias and immunological disorders (Byers et al., 1988; Lagakos et al., 1986b). Immunological effects of trichloroethylene exposure included increased incidence of auto-antibodies and increased infections. Inhalation and dermal exposures to trichloroethylene have not resulted in immunological disorders (ATSDR, 1991).

Humans experiencing dermal exposures to trichloroethylene may develop rashes, burns, or other irritations (Bauer and Rabens, 1974; Goh and Ng, 1988). There are no available studies which detail potential dermal effects to laboratory animals after inhalation exposure to trichloroethylene (ATSDR, 1991).

Genotoxicity studies of trichloroethylene in humans and animal investigations indicate sister chromatic exchange, chromosomal aberrations, single-strand breaks, and gene mutations. Results of similar genotoxicity studies, however, have shown negative results (ATSDR, 1991).

Adverse effects to both humans and animals may include renal dysfunction. However, supporting data for this effect is limited (ATSDR, 1991). Additionally, studies among laboratory animals have produced results indicating liver enlargement after inhalation exposure for acute or intermediate periods (Kjellstrand et al., 1981). The available literature does not indicate the presence of adverse hematological effects to humans. Various hematological effects, however, have been reported for laboratory animals (Fujita et al., 1984; Koizumi et al., 1984).

The carcinogenicity classification for trichloroethylene is currently under review by USEPA. The carcinogen assessment summary for trichloroethylene has been withdrawn following further review and a revised carcinogen summary is currently being prepared by the CRAVE Work Group. The Agency has not established an oral or inhalation cancer slope factor or RfD value for trichloroethylene (IRIS, 1996). In lieu of verified toxicity values, a provisional oral cancer slope factor of 0.011 $(mg/kg\text{-}day)^{-1}$ was applied in this assessment. Additionally, an inhalation cancer slope factor of 0.006 $(mg/kg\text{-}day)^{-1}$ and an oral RfD value of 0.006 mg/kg-day are also provisional values for trichloroethylene. These values were obtained from the USEPA Region III (1996) guidance document for RCRA sites and are regarded as provisional values.

References

ATSDR. 1991. *Toxicological Profile for Trichloroethylene: Draft.* Agency for Toxic Substances and Disease Registry, Atlanta, GA and U.S. Department of Health and Human Services, Public Health Service. October.

Bauer, M., and S.F. Rabens. 1974. Cutaneous manifestations of trichloroethylene toxicity. *Arch. Dermatol.* 110:886-890. (Cited in ATSDR, 1991)

Buxton, P.H. and M. Hayward. 1967. Polyneuritis cranials associated with industrial trichloroethylene poisoning. *J. Neurol. Neurosurg. Psychiat.* 30:511-518. (Cited in ATSDR, 1991)

Byers, V.S., A.S. Levin, D.M. Ozonoff et al. 1988. Association between clinical symptoms and lymphocyte abnormalities in a population with chronic domestic exposure to industrial solvent-contaminated domestic water supply and a high incidence of leukemia. *Cancer Immunol. Immunother.* 27:77-81. (Cited in ATSDR, 1991)

Class, T. and K. Ballschmiter. 1986. Chemistry of organic traces in air VI: Distribution of chlorinated C_1-C_4 hydrocarbons in air over the Northern and Southern Atlantic Ocean. *Chemosphere.* 15:413-427. (Cited in ATSDR, 1991)

Clearfield, H.R. 1970. Hepatorenal toxicity from sniffing spot remover (trichloroethylene). *Diges. Dis.* 15:851-856. (Cited in ATSDR, 1991)

CMR. 1983. *Chemical Profile - Trichloroethylene.* Chemical Marketing Reporter. February 14. (Cited in ATSDR, 1991)

Eisenreich, S.J., B.B. Looney, and J.D. Thornton. 1981. Airborne organic contaminants in the Great Lakes ecosystem. *Environ. Sci. Technol.* 15:30-38. (Cited in ATSDR, 1991)

Feldman, R.G., J. Chirico-Post, and S.P. Proctor. 1988. Blink reflex latency after exposure to trichloroethylene in well water. *Arch. Environ. Health.* 43:143-147. (Cited in ATSDR, 1991)

Fujita, H., A. Koizumi, M. Yamamoto et al. 1984. Inhibition of delta-aminolevulinate dehydratase in trichloroethylene-exposed rats, and the effects on heme regulation. *Biochem. Biophys. Acta.* 800:1-10. (Cited in ATSDR, 1991)

Goh, C.L. and S.K. Ng. 1988. A cutaneous manifestation of trichloroethylene toxicity. *Contact Dermatitis.* 18:59-61. (Cited in ATSDR, 1991)

Hansch, C. and A.J. Leo. 1985. *MedChem Project.* Issue No. 26. Claremont, CA: Pomona College. (Cited in ATSDR, 1991)

Hawley, G.G. 1981. *The Condensed Chemical Dictionary, 10th Edition.* New York, NY: Van Nostrand-Reinhold Co. 1041-1042. (Cited in ATSDR, 1991)

Hine, J. and P.K. Mookerjee. 1975. The intrinsic hydrophilic character of organic compounds. Correlations in terms of structural contributions. *J. Org. Chem.* 40:292-298. (Cited in ATSDR, 1991)

HSDB. 1990. *Chemical Search for Trichloroethylene.* Hazardous Substances Data Bank. National Library of Medicine, National Institutes of Health, Bethesda, MD. August. (Cited in ATSDR, 1991)

HSDB. 1995. *Trichloroethylene.* Hazardous Substances Data Bank. National Library of Medicine, National Institutes of Health, Bethesda, MD.

IRIS. 1996. *Trichloroethylene.* Integrated Risk Information System, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH.

Kimbrough, R.D., F.L. Mitchell, and V.N. Houk. 1985. Trichloroethylene: An update. *J. Toxicol. Environ. Health.* 15:369-383. (Cited in ATSDR, 1991)

Kjellstrand, P., M. Kanje, L. Mansson et al. 1981. Trichloroethylene: Effects on body and organ weights in mice, rats, and gerbils. *Toxicology.* 21:105-115. (Cited in ATSDR, 1991)

Koizumi, A., H. Fujita, T. Sadamoto et al. 1984. Inhibition of delta-aminolevulinic acid dehydratase by trichloroethylene. *Toxicol.* 30:93-102. (Cited in ATSDR, 1991)

Kuney, J.H. 1986. *Chemcyclopedia: Volume 5.* Washington, D.C.: American Chemical Society. (Cited in ATSDR, 1991)

Lagakos, S.W., B.J. Wessen, and M. Zelen. 1986a. Rejoinder. *J. Am. Statistical Assoc.* 81:611-614. (Cited in ATSDR, 1991)

Lagakos, S.W., B.J. Wessen, and M. Zelen. 1986b. An analysis of contaminated well water and health effects in Woburn, Massachusetts. *J. Am. Statistical Assoc.* 81:583-596. (Cited in ATSDR, 1991)

Lyman, W.J., W.F. Reehl, and D.H. Rosenblatt. 1990. *Handbook of Chemical Property Estimation Methods: Environmental Behavior of Organic Compounds.* Washington, D.C.: American Chemical Society.

Mackay, D. and W.Y. Shiu. 1981. A critical review of Henry's Law constants for chemicals of environmental interest. *J. Phys. Chem. Ref. Data.* 19:1175-1179. (Cited in ATSDR, 1991)

Mannsville Chemical Products Corporation. 1985. *Chemical Product Synopsis: Trichloroethylene.* Mannsville Chemical Products Corporation, Cortland, NY. (Cited in ATSDR, 1991)

McNeill, W.C. 1979. Trichloroethylene. In: *Kirk-Othmer Encyclopedia of Chemical Technology, 3rd Edition, Volume 5.* New York, NY: John Wiley and Sons. 745-753. (Cited in ATSDR, 1991)

Milby, T.H. 1968. Chronic trichloroethylene intoxication. *J. Occup. Med.* 10:252-254. (Cited in ATSDR, 1991)

Nomiyama, K., and H. Nomiyama. 1977. Dose-response relationship for trichloroethylenes in man. *Int. Arch. Occup. Environ. Health.* 39:237-248. (Cited in ATSDR, 1991)

Salvini, M., S. Binaschi, and M. Riva. 1971. Evaluation of the psychophysiological functions in humans exposed to trichloroethylene. *Br. J. Ind. Med.* 28:293-295. (Cited in ATSDR, 1991)

SANSS. 1990. *Structure and Nomenclature Search System.* Chemical Information System (CIS) Computer Database. August. (CIted in ATSDR, 1991)

Schwarzenbach, R.P., W. Giger, E. Hoehn, and J.K. Schneider. 1983. Behavior of organic compounds during infiltration of river water to ground water. Field studies. *Environ. Sci. Technol.* 17:472-479. (Cited in ATSDR, 1991)

Smith, G.F. 1966. Trichloroethylene: A review. *Br. J. Ind. Med.* 23:249-262. (Cited in ATSDR, 1991)

Stewart, R.D., H.C. Dodd, H.H. Gray et al. 1970. Experimental human exposure to trichloroethylene. *Arch. Environ. Health.* 20:64-71. (Cited in ATSDR, 1991)

Tewari, Y.B., M.M. Miller, S.P. Wasik et al. 1982. Aqueous solubility and octanol/water partition coefficient of organic compounds at 25°C. *J. Chem. Eng. Data.* 27:451-454. (Cited in ATSDR, 1991)

USEPA. 1985. *Health Assessment Document for Trichloroethylene.* U.S. Environmental Protection Agency, Office of Health Effects Assessment, Research Triangle Park, NC.

USEPA. 1996. *Risk-Based Concentration Table, January - June 1996.* Dr. Roy L. Smith, U.S. Environmental Protection Agency, Region III, Office of RCRA, Technical and Program Support Branch, Philadelphia, PA. April 30.

Vernon, R.J. and R.K. Ferguson. 1969. Effects of trichloroethylene on visual-motor performance. *Arch. Environ. Health.* 18:894-900. (Cited in ATSDR, 1991)

Verschueren, K. 1983. *Handbook of Environmental Data on Organic Chemicals.* New York, NY: Van Nostrand Reinhold. 1131-1135.

Windholz, M. (ed.) 1983. *The Merck Index, 10th Edition.* Rahway, NJ: Merck and Co., Inc. (Cited in ATSDR, 1991)

Toxicological Profile

Vinyl Chloride

Chemical Formula	C_2H_3Cl	HSDB, 1996
Molecular Weight	62.5 g/mol	Sax and Lewis, 1989
Vapor Pressure	2,660 mmHg @ 25°C	Sax and Lewis, 1989
Boiling Point	260°K	Mackay et al., 1992
Melting Point	-153.8°C	Cowfer and Magistro, 1983
Henry's Law Constant	5.68×10^{-2} atm-m^3/mol	Mackay et al., 1992
Solubility	2,763 mg/L @ 25°C	USEPA, 1985
Partition Coefficient		
K_{oc}	14.2	Howard, 1991

Vinyl chloride is used in a wide variety of applications. It is used primarily in the manufacture of polyvinyl chloride products such as pipes, packaging materials, automotive accessories, furniture, wire coatings, wall coverings, and other copolymer products (Cowfer and Magistro, 1985; Eveleth et al., 1990).

Based on its vapor pressure, vinyl chloride in the atmosphere is likely to exist in the vapor phase (Eisenriech et al., 1981; Verscheuren, 1983). In the atmosphere, the primary degradation process is reaction with photochemically produced hydroxyl radicals (Cox et al., 1974; Howard, 1976; Perry et al., 1977). Reactions with ozone or direct photolysis are unlikely to be significant degradation mechanisms for this compound (Zhang et al., 1983).

Vinyl chloride in soils volatilizes rapidly to the atmosphere from dry soil surfaces and has an estimated half-life of approximately 12 hours at 10 cm depth (Verscheuren, 1983; Jury et al., 1984). The K_{oc} of vinyl chloride suggests a very low sorption tendency with high mobility and sufficient potential to leach to groundwater (Lyman et al., 1982).

In aquatic systems, vinyl chloride partitions rapidly to the atmosphere (ATSDR, 1995). The half-life for vinyl chloride has been estimated at 43.3, 8.7, and 34.7 hours for volatilization from a pond, river, and lake, respectively (USEPA, 1982). Photolysis of vinyl chloride in water systems is relatively slow

process compared to volatilization. In addition, the hydrolytic half-life of vinyl chloride at 25°C has been estimated to be less than 10 years (USEPA, 1976).

Some evidence exists for the carcinogenic potential of vinyl chloride in humans via inhalation (ATSDR, 1995). Several studies report an increased incidence of hepatic angiosarcomas in workers occupationally exposed to vinyl chloride (Creech and Johnson, 1974; Monson et al., 1975; Byren et al., 1976; Infante et al., 1976a; Waxweiler et al., 1976; Fox and Collier, 1977; Weber et al., 1981; Jones et al., 1988; Rinsky et al., 1988; Wu et al., 1989; Piratsu et al., 1990; Teta et al., 1990; Simonato et al., 1991; Wong et al., 1991; Laplanche et al., 1992). Other studies with vinyl chloride workers report a statistically significant increase in cancer of the brain, central nervous system, lung and respiratory tract, and the lymphatic /hematopoietic system (Infante et al., 1976b; Waxweiler et al., 1976; Cooper, 1981; Weber et al., 1981; Wong et al., 1981; Belli et al., 1987; Rinsky et al., 1988; Smulevich et al., 1988).

No studies are available regarding the carcinogenic potential of vinyl chloride in humans due to oral exposure (ATSDR, 1995). In contrast, the results of studies with laboratory animals administered vinyl chloride in the diet and via gavage suggest a statistically significant increase in hepatic angiosarcomas of the liver (Feron et al., 1981; Maltoni et al., 1981; Til et al., 1983, 1991). In addition, no studies are available regarding the carcinogenic potential of vinyl chloride to humans or animals following dermal exposure (ATSDR, 1995).

The USEPA has classified vinyl chloride as a Class A human carcinogen (USEPA, 1995). The Agency has published an oral slope factor of 1.9 $(mg/kg\text{-}day)^{-1}$ for vinyl chloride based on the occurrence of lung and liver tumors in chronic feeding studies with rats (USEPA, 1995). Likewise, the presence of liver tumors in a 1-year inhalation study with rats has prompted USEPA to publish a unit risk factor of 8.4×10^{-5} mg/m^3, which corresponds to an inhalation slope factor of 0.3 $(mg/kg\text{-}day)^{-1}$ (USEPA, 1995). Other studies with laboratory animals exposed to vinyl chloride via inhalation indicate an increase in mammary gland carcinomas, Zymbal gland carcinomas, nephroblastomas, liver angiosarcomas, neuroblastomas, forestomach papillomas, and other tumors (Suzuki, 1978; Hong et al., 1981; Maltoni et al., 1981; Bi et al., 1985).

Finally, the Agency has not established an oral or inhalation RfD for vinyl chloride (IRIS, 1996).

References

Belli, S., P.A. Bertazzi, P. Comba et al. 1987. A cohort study on vinyl chloride manufacturers in Italy: Study desgin and preliminary results. *Cancer Lett.* 35:253-261. (Cited in ATSDR, 1995)

Bi, W., Y. Wang, M. Huang et al. 1985. Effect of vinyl chloride on testis in rats. Ecotoxicol. Environ. Saf. 10:281-289. (Cited in ATSDR, 1995)

Byren, D., G. Engholm, A. Englund et al. 1976. Mortality and cancer morbidity in a group of Swedish VCM and PCV production workers. *Environ. Health Perspect.* 17:167-170. (Cited in ATSDR, 1995)

ATSDR. 1995. *Toxicological Profile for Vinyl Chloride: Draft.* Agency for Toxic Substances and Disease Registry, Atlanta, GA and U.S. Department of Health and Human Services, Public Health Service. August.

Cooper, W.C. 1981. Epidemiologic study of vinyl chloride workers: Mortality through December 31, 1972. *Environ. Health Perspect.* 41:101-106. (Cited in ATSDR, 1995)

Cowfer, J.A. and A.J. Magistro. 1983. Vinyl chloride. In: *Kirk-Othmer Concise Encyclopedia of Chemical Technology.* New York, NY: Wiley Interscience. (Cited in ATSDR, 1995)

Cox, R.A., A.E.J. Enggleton, and F.J. Sandalls. 1974. Photochemical reactivity of vinyl chloride. Oxfordshire, England: Are Harwell. AERE-R7820. (Cited in ATSDR, 1995)

Creech, J.L. and M.N. Johnson. 1974. Angiosarcoma of liver in the manufacture of polyvinyl chloride. *J. Occup. Med.* 16:150-151. (Cited in ATSDR, 1995)

Eisenreich, S.J., B.B. Looney, and J.D. Thornton. 1981. Airborne organic contaminants in the Great Lakes ecosystem. *Environ. Sci. Technol.* 15:30-38. (Cited in ATSDR, 1995)

Eveleth, W.T., V. Kollonitsch, K. Slater (eds.). 1990. *Kline Guide to the U.S. Chemical Industry.* 5th Edition. Fairfield, NJ: Kline & Co. (Cited in ATSDR, 1995)

Feron, V.J., C.F.M. Hendriksen, A.J. Speek et al. 1981. Lifespan oral toxicity study of vinyl chloride in rats. *Fd. Cos. Toxicol.* 19:317-333. (Cited in ATSDR, 1995)

Fox, A.J. and P.F. Collier. 1977. Mortality experience of workers exposed to vinyl chloride monomer in the manufacture of polyvinyl chloride in Great Britain. *Brit. J. Ind. Med.* 34:1-10. (Cited in ATSDR, 1995)

Hong, C.B., J.M. Winston, L.P. Thornburg et al. 1981. Follow-up study on the carcinogencity of vinyl chloride and vinylidene chloride in rats and mice: Tumor incidence and mortality subsequent to exposure. *J. Toxicol. Environ. Health* 7:909-924. (Cited in ATSDR, 1995)

Howard, C.J. 1976. Rate constants for the gas-phase reactions of hydroxyl radicals with ethylene and halogenated ethylene compounds. *J. Chem. Phys.* 65:4771-4777. (Cited in ATSDR, 1995)

Howard, P.H. (ed). 1991. *Handbook of Environmental Fate and Exposure Data for Organic Chemicals. Volume II: Solvents.* Chelsea, MI: Lewis Publishers, Inc.

HSDB. 1996. *Vinyl Chloride.* Hazardous Substances Data Bank, National Library of Medicine, National Institutes of Health, Bethesda, MD.

Infante, P.F., J.K. Wagoner, A.J. McMichael et al. 1976a. Genetic risks of vinyl chloride. *Lancet* (April 3):734-735. (Cited in ATSDR, 1995)

Infante, P.F., J.K. Wagoner, and R.J. Waxweiler. 1976b. Carcinogenic, mutagenic, and teratogenic risks associated with vinyl chloride. *Mutat. Res.* 41:131-142. (Cited in ATSDR, 1995)

IRIS. 1996. *Vinyl Chloride.* Integrated Risk Information System, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH.

Jones, R.D., D.M. Smith, and P.G. Thomas. 1988. A mortality study of vinyl chloride monomer workers employed in the United Kingdom in 1940-1974. *Scand. J. Work Environ. Health* 14:153-160. (Cited in ATSDR, 1995)

Jury, W.A., W.F. Spencer, and W.J. Farmer. 1984. Behavior assessment model for trace organics in soil: III. Application of screening model. *J. Environ. Qual.* 13:573-579. (Cited in ATSDR, 1995)

Laplanche, A., F. Clavel, J.C. Contassot et al. 1992. Exposure to vinyl chloride monomer - results of a cohort study after a 7 year follow-up. *Brit. J. Ind. Med.* 49(2):134-137. (Cited in ATSDR, 1995)

Lyman, W.J., W.F. Reehl, and D.H. Rosenblatt. 1982. *Hanbook of Chemical Property Estimation Methods: Environmental Behavior of Organic Compounds.* New York, NY: McGraw Hill Book Co. (Cited in ATSDR, 1995)

Mackay, D., W.Y. Shiu, and K.C. Ma (eds). 1992. *Illustrated Handbook of Physical-Chemical Properties and Environmental Fate for Organic Chemicals. Volume II: Polynuclear Aromatic Hydrocarbons, Polychlorinated Dioxins, and Dibenzofurans.* Boca Raton, FL: Lewis Publishers, Inc.

Maltoni, C. and P. Lodi. 1981. Results of sputum cytology among workers exposed to vinyl chloride and to polyvinyl chloride. *Environ. Health Perspect.* 41:85-88. (Cited in ATSDR, 1995)

Monson, R.R., J.M. Peters, and M.N. Johnson. 1975. Proportional mortality among vinyl chloride workers. *Environ. Health Perspect.* 11:75-77. (Cited in ATSDR, 1995)

NIOSH. 1986. *Chemical Reviews Vinyl Chloride. Dangerous Properties of Industrial Materials Report 6:13-43.* National Institute for Occupational Safety and Health, U.S. Department of Health and Human Services, Washington, D.C. NIOSH Publication No. 00188891. (Cited in ATSDR, 1995)

Perry, R.A., R. Atkinson, J.N. Pitts Jr. 1977. Rate constants for the reaction of hydroxyl radicals with vinyl flouride, vinyl chloride, and vinyl bromide over the temperature range 229-426°K. *J. Chem. Phys.* 67:458-462. (Cited in ATSDR, 1995)

Piratsu, R., P. Comba, A. Reggiani, et al. 1990. Mortality from liver disease among Italian vinyl chlorine monomer/polyvinyl chloride manufacturers. *Am. J. Ind. Med.* 17:155-161. (Cited in ATSDR, 1995)

Rinsky, R.A., G. Ott, E. Ward, et al. 1988. Study of mortality among chemical workers in the Kanawha Valley of West Virginia. *Am. J. Ind. Med.* 13:429-438. (Cited in ATSDR, 1995)

Sax, N.I. and R.J. Lewis (eds.) 1987. *Hawley's condensed chemical dictionary*. 11th ed. New York, NY: Van Nostrand Reinhold Co., 1223.

Simonato, L., K.A. L'Abbe, A. Andersen, et al. 1991. A collaborative study of cancer incidence and mortality among vinyl chloride workers. *Scan J. Work Environ. Health* 17(3):159-169. (Cited in ATSDR, 1995)

Smulevich, V.B., I.V. Fedotova, V.S. Filatova. 1988. A soft tissue sarcoma case control study in a large multi-chemical manufacturing facility. *Chemosphere* 16:2095-2099. (Cited in ATSDR, 1995)

Suzuki, Y. 1978. Pulmonary tumors induced in mice by vinyl chloride monomer. *Environ. Res.* 16:285-301. (Cited in ATSDR, 1995)

Teta, M.J., A.R. Schnatter, M.G. Ott, et al. 1990. Mortality surveillance in a large chemical company: The Union Carbide Corporation experience, 1974-1983. *Am. J. Ind. Med.* 44:718-719. (Cited in ATSDR, 1995)

Til, H.P., H.R. Immel, V.J. Feron. 1983. Lifespan oral carcinogenicity study of vinyl chloride in rats. Final Report. Civo Institutes, TNO. Report No. V 93.285/291099. (Cited in ATSDR, 1995)

Til, H.P., V.J. Feron, H.R. Immel. 1991. Lifetime (149-weeks) oral carcinogenicity study of vinyl chloride in rats. *Food Chem. Toxicol.* 29(10):713-718. (Cited in ATSDR, 1995)

USEPA. 1976. *Dynamic Behavior of Vinyl Chloride in Aquatic Ecosystems.* United States Environmental Protection Agency, Atlanta, GA. Federal Register 40(248):59532-59552. (Cited in ATSDR, 1995)

USEPA. 1982. *Aquatic Fate Process Data for Organic Priority Pollutants.* United States Environmental Protection Agency, Office of Water Regulations and Standards, Wasington, DC. EPA 440/4-81-014. (Cited in ATSDR, 1995)

USEPA. 1985. *Drinking Water Criteria Document for Vinyl Chloride.* United States Environmental Protection Agency, Office of Drinking Water, Wasington, DC. NTIS PB86-118320. (Cited in ATSDR, 1995)

USEPA. 1995. *Health Effects Assessment Summary Tables, FY-1995 Annual.* U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, D.C. EPA/540/R-95/036. May.

Waxweiler, R.J., W. Stringer, J.K. Wagner, et al. 1976. Neoplastic risk among workers exposed to vinyl chloride. *Ann NY Acad. Sci.* 271:40-48. (Cited in ATSDR, 1995)

Weber, H., W. Reinl, E. Greiser. 1981. German investigations on morbidity and mortality of workers exposed to vinyl chloride. *Environ. Health Perspect.* 41:95-99. (Cited in ATSDR, 1995)

Wong, O., M.D. Whorton, D.E. Foliart, et al. 1991. An industry-wide epidemiologic study of vinyl chloride workers, 1942-1982. *Am. J. Ind. Med.* 20(3):317-334. (Cited in ATSDR, 1995)

Wu, W., K. Steenland, D. Brown, et al. 1989. Cohort and case-control analyses of workers exposed to vinyl chloride: An update. *J. Occup. Med.* 31:518-523. (Cited in ATSDR, 1995)

Zhang, J., S. Hatakeyama, H. Akimoto. 1983. Rate constants of the reaction of ozone with trans-1,2-dichloroethene and vinyl chlorine in air. *Int. J. Chem. Kinetics* 15:655-668. (Cited in ATSDR, 1995)

Toxicity Profile

Zinc

Chemical Formula	Zn	HSDB, 1995
Molecular Weight	65.38	Merck, 1983
Vapor Pressure	1 mmHg @ 487°C	Merck, 1983
Boiling Point	908°C	Merck, 1983
Melting Point	419.5°C	Merck, 1983
Henry's Law Constant	NA	ATSDR, 1988
Solubility	Insoluble	Weast, 1989
Partition Coefficients		
$\log K_{ow}$	NA	ATSDR, 1988
$\log K_{oc}$	NA	ATSDR, 1988

Zinc is used primarily as a protective coating of other metals. It is also employed in bronze and brass alloys, electrical materials, and organic chemical reductions and extractions (ATSDR, 1988). To a lesser extent, zinc is used as a component of pharmaceuticals, United States coins, and military and law enforcement smoke bombs (Lloyd and Showak, 1983; Merck, 1983; ATSDR, 1988).

In the environment, zinc occurs in the +2 oxidation state (Lindsay, 1979). In the atmosphere, the chemical interactions of zinc compounds may result in a change in the speciation of the compound. It is likely to be highly reactive in the atmosphere since most atmospheric zinc compound concentrations are present as submicron particles and which are probably short-lived (USEPA, 1980; Fishbein, 1981). However, there is no available estimated atmospheric lifetime for zinc (ATSDR, 1988). Additionally, volatilization does not appear to be an important fate process for zinc in the atmosphere (Callahan et al, 1979).

Transport of zinc in soils systems is affected by such factors as compound solubility, soil type, soil pH, and soil salinity (Kalbasi et al., 1978; USEPA, 1980; Clement Associates, 1985). It is strongly sorbed to soils and movement towards groundwater is expected to be slow unless the compound is soluble or accompanied by corrosive materials (USEPA, 1980). Furthermore, zinc may leach to groundwater if the soil matrix does not support the sorption of zinc to soil particles (ATSDR, 1988).

The primary fate process for zinc in aquatic systems is sorption. Zinc may partition to sediments via sorption to hydrous iron and manganese oxides, clay minerals, and organic material. The relative mobility of zinc in water is determined by the same factors as affecting its transport in soil systems. In surface waters, zinc may occur in both suspended and dissolved forms (ATSDR, 1988). Zinc may be degraded via hydrolysis, while photolysis of zinc is not expected to be significant. Zinc may be bioaccumulated by biota, but is unlikely to biomagnify (Callahan et al., 1979).

Zinc is an essential trace element necessary to enzyme functions, protein synthesis, and carbohydrate metabolism (USEPA, 1985). The Recommended Daily Allowance for zinc is 15 mg. Ingestion of excessive amounts of zinc may cause fever and gastrointestinal distress. It appears that zinc is absorbed via ingestion and inhalation. Inhalation exposure to zinc dust or fumes has been associated with pulmonary fibrosis and metal fume fever. Additionally, chronic exposure to zinc may produce anemia (USEPA, 1984).

Zinc and its compounds produce effects in experimental animals similar to those in humans. Excess zinc in rats can cause growth retardation, hypochromic anemia, and defective mineralization of bone. Chronic ingestion of zinc in experimental animals has also been reported to result in adverse effects to the brain, pancreas, and pituitary gland. Other effects resulting from exposure to zinc include adverse effects on hemoglobin, appetite, and weight gain at high exposure levels (USEPA, 1984, 1985).

Elevated fetal exposures to zinc have produced reduced copper levels in experimental studies. Premature deliveries and one stillbirth were observed in a small group of women administered zinc supplements late in pregnancy. An increased number of fetal resorptions were observed in studies with rats administered zinc supplements. It is possible, however, that zinc deficiencies caused the adverse fetal effects (USEPA, 1984, 1985).

Walters and Roe (1965) reported a significant increase of anemia in treated mice administered concentrations of 0, 1000, or 5000 ppm zinc as zinc sulfate or zinc oleate in drinking water. In another study, mice administered 500 mg/L zinc sulfate in drinking water exhibited hypertrophy of the adrenal cortex and pancreatic islets (Aughey et al., 1977).

The USEPA has classified zinc as a Group D carcinogen, not classifiable as to human carcinogenicity, based on inadequate evidence in humans and animals. Studies of occupational exposure to zinc compounds do not correlate exposure with cancer risk. Likewise, case reports of chronic therapeutic

exposures to zinc are also limited (IRIS, 1996). The Agency has not determined an oral or inhalation cancer slope factor for zinc (IRIS, 1996).

The USEPA has established an oral RfD value of 0.3 mg/kg-day for zinc (IRIS, 1996). This value is based on a 10-week clinical study of zinc supplementation in adult women administered gluconate supplements twice daily at a concentration of 1.0 mg/kg-day. The study results indicate a 47 percent decrease of erythrocyte superoxide dismutase (ESOD) activity (Yadrick et al., 1989). The Agency has not proposed an inhalation RfD value for zinc.

References

ATSDR. 1988. *Toxicological Profile for Zinc: Draft.* Agency for Toxic Substances and Disease Registry, Atlanta, GA and U.S. Department of Health and Human Services, Public Health Service, Washington, D.C. December.

Aughey, E., L. Grant, B.L. Furman, and W.F. Dryden. 1977. The effects of oral zinc supplementation in the mouse. *J. Comp. Pathol.* 87:1-14. (Cited in IRIS, 1996)

Callahan, M.A., M.W. Slimak, N.W. Gabel et al. 1979. *Water-Related Environmental Fate of 129 Priority Pollutants.* U.S. Environmental Protection Agency, Office of Water Planning and Standards, Washington, D.C. EPA 440/4-79-029a. (Cited in ATSDR, 1988)

Clement Associates. 1985. *Chemical, Physical, and Biological Properties of Compounds Present at Hazardous Waste Sites.* U.S. Environmental Protection Agency, Office of Waste Programs Enforcement, Office of Solid Waste and Emergency Response, Washington, D.C. (Cited in ATSDR, 1988)

Fishbein, L. 1981. Sources, transport, and alterations of metal compounds: An overview. *Environ. Health Perspect.* 40:43-64. (Cited in ATSDR, 1988)

HSDB. 1995. *Zinc.* Hazardous Substances DataBank, National Library of Medicine, National Institutes of Health, Bethesda, MD.

IRIS. 1996. *Zinc.* Integrated Risk Information System, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH.

Kalbasi, M., G.J. Racz, and L.A. Lewen-Rudgers. 1978. Reaction products and solubility of applied zinc compounds in some Manitoba soils. *Soil Sci.* 125:55-64. (Cited in ATSDR, 1988)

Lindsay, W.L. 1979. *Zinc: Chemical Equilibrium in Soils.* New York, NY: John Wiley & Sons. (Cited in ATSDR, 1988)

Lloyd, T.B. and W. Showak. 1983. Zinc and zinc alloys. In: *Kirk-Othmer Encyclopedia of Chemical Technology: Volume 24, 3rd Edition.* M. Grayson (ed.). New York, NY: John Wiley & Sons. (Cited in ATSDR, 1988)

Merck. 1983. *The Merck Index: 10th Edition.* Rahway, NJ: Merck & Co., Inc. (Cited in ATSDR, 1988)

USEPA. 1980. *Exposure and Risk Assessment for Zinc.* U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Washington, D.C. EPA 440/4-81-016. (Cited in ATSDR, 1988)

USEPA. 1984. *Health Effects Assessment for Zinc.* U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office, Cincinnati, OH.

USEPA. 1985. *Chemical, Physical, and Biological Properties of Compounds Present at Hazardous Waste Sites.* U.S. Environmental Protection Agency. Prepared by Clement Associates. September 27.

Walters, M. and F.J.C. Roe. 1965. A study of the effects of zinc and tin administered orally to mice over a prolonged period. *Food Cosmet. Toxicol.* 3:271-276. (Cited in IRIS, 1996)

Weast, R.C. 1989. *CRC Handbook of Chemistry and Physics, 70th Edition.* Boca Raton, FL: CRC Press, Inc. (Cited in ATSDR, 1988)

Windholz, E. ed. 1983. *The Merck Index, 10th Edition.* Rahway, NJ: Merck & Co., Inc. 48:50-53. (Cited in ATSDR, 1988)

Toxicological Profiles

The following toxicity profiles were developed with the intention to present only brief toxicological information and to omit any data pertaining to the chemical properties and the chemical fate and transport processes of the individual compounds. The toxicity profiles included herein were designed to be similar to the toxicity profiles presented in Appendix C of the USEPA Region III December 1995 report entitled *Draft Baseline Risk Assessment for Westinghouse Sharon Middle Sector Building*.

Toxicological Profile

1,1,1-Trichloroethane

1,1,1,-Trichloroethane (1,1,1-TCA) is used in a wide variety of applications. The primary use of 1,1,1-TCA is as an agent involved in vapor degreasing. It is also employed in cold cleaning or as an aerosol, adhesive, and chemical intermediate. In addition, 1,1,1-TCA may be used in the production of coatings, inks, textiles, and electronics (ATSDR, 1995).

1,1,1-TCA may be absorbed via the oral, inhalation, or dermal routes of exposure. Inhalation of 1,1,1-TCA is the predominant and most rapid route of absorption into the body. It is also rapidly absorbed through the gastrointestinal tract (USEPA, 1987). The primary effect resulting from acute exposure to 1,1,1-TCA is depression of the central nervous system, including anesthesia at high concentrations. At low concentrations, coordination, equilibrium, and judgement may be impaired. Other observed effects include cardiovascular effects, and slight adverse effects to the lungs, liver, and kidneys. Irritation of the skin, eyes, and mucous membranes may also occur upon exposure. Potential chronic effects include neurological and behavioral effects and, at very high concentrations, possible kidney and liver damage (USEPA, 1984a).

1,1,1-TCA has been shown to be weekly mutagenic in some studies (USEPA, 1984a,b). No information is available on the reproductive effects of 1,1,1-TCA in humans. Short- and long-term exposures have produced no reproductive or teratogenic effects in rats or mice. In both animals the only effect observed was a delay in fetal development, but this effect was considered reversible (USEPA, 1984b).

The USEPA has classified 1,1,1-TCA as a Group D carcinogen, not classifiable as to human carcinogenicity, based on the absence of human carcinogenicity data and inadequate animal carcinogenicity data (IRIS, 1996). The Agency has not established an oral or inhalation cancer slope factor for this compound. USEPA Region III has published an oral RfD value 0.035 mg/kg-day and an inhalation RfD value of 0.286 mg/kg-day for 1,1,1-TCA (USEPA, 1996).

References

ATSDR. 1995. *Toxicological Profile for 1,1,1-Trichloroethane (Update).* Agency for Toxic Substances and Disease Registry, Atlanta, GA and U.S. Department of Health and Human Services, Public Health Service. August.

IRIS. 1996. *1,1,1-Trichloroethane.* Integrated Risk Information System. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH.

USEPA. 1984a. *Health Assessment Document for 1,1,1-Trichloroethane (Methyl Chloroform).* U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Washington, D.C. EPA-600/8-82-003F. February.

USEPA. 1984b. *Health Effects Assessment for 1,1,1-Trichloroethane.* U.S. Environmental Protection Agency, Office of Environmental Criteria and Assessment, Cincinnati, OH. ECAO-CIN-H005. September.

USEPA. 1987. *Health Advisories for 25 Organics.* U.S. Environmental Protection Agency, Office of Drinking Water, Washington, D.C. March.

USEPA. 1996. *Risk-Based Concentration Table, January-June 1996.* U.S. Environmental Protection Agency, Region III, Dr. Roy L. Smith, Office of RCRA, Technical and Program Support Branch, Philadelphia, PA. April 30.

Toxicological Profile

Antimony

Antimony compounds are employed as constituents of metal alloys, flame retardants, batteries, textiles, chemicals, and glass. Some antimony compounds are also used in the treatment of parasitic diseases and infection (USEPA, 1980; Friberg et al. , 1986; ATSDR, 1990).

In humans, acute exposures to antimony via inhalation or ingestion may result in vomiting, nausea, and diarrhea (Friberg et al., 1986). Chronic exposures to antimony may result in myocardial changes, pneumoconiosis, tracheitis, laryngitis, bronchitis, or pustular skin eruptions (Seiler, 1988). Belyaeva (1967) reported a higher incidence of spontaneous abortions among women employed in a metallurgical plant. Effects in experimental animals are similar to humans including respiratory system effects, cardiovascular system effects, and effects on the liver, kidney, and spleen (USEPA, 1980).

The USEPA has not classified antimony as to potential human carcinogenicity. Likewise, the Agency has not determined an oral or inhalation cancer slope factor for antimony.

The USEPA has derived an oral RfD value of 0.0004 mg/kg-day for antimony based on the results of a chronic oral bioassay in which rats administered 0.35 mg/kg-day in drinking water exhibited decreased longevity, blood glucose, and cholesterol (Schroeder et al., 1970). The Agency has not established an inhalation RfD value for antimony.

References

ATSDR. 1990. *Toxicological Profile for Antimony: Draft.* Agency for Toxic Substances and Disease Registry, Atlanta, GA and U.S. Department of Health and Human Services, Public Health Service, Washington, D.C. October.

Belyaeva, A.P. 1967. The effect of antimony on reproduction. *Gig. Truda Prof. Zabol.* 11:32. (Cited in IRIS, 1996)

Friberg, L., G.F. Nordberg, and V.B. Vouk (eds). 1986. *Handbook on the Toxicology of Metals, Volume II.* Amsterdam: Elsevier Science Publishers.

HSDB. 1996. *Antimony*. Hazardous Substances DataBank, National Library of Medicine, Bethesda, MD.

IRIS. 1996. *Antimony*. Integrated Risk Information System, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH.

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. (Cited in IRIS, 1996)

Seiler. 1988. *Handbook of Toxicity of Inorganic Compounds*. p.71. (Cited in HSDB, 1996)

USEPA. 1980. *Ambient Water Quality Criteria for Antimony*. U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Washington, D.C. EPA 440/5-80-020.

Toxicological Profile

Bis(2-ethylhexyl)phthalate

Bis(2-ethylhexyl)phthalate (BEHP) is widely used in plastic products. BEHP is added to synthetic plastic resins to impart flexibility to the finished product, improve workability during fabrication, and extend or modify properties not present in the original resins. In addition, BEHP is often employed as an insect repellant and orchard acaricide (ATSDR, 1989).

Few studies have been conducted on the effects of BEHP in humans. The results of animal studies, however, have shown that BEHP administered either orally or intraperitoneally has a low level of acute toxicity (ATSDR, 1989). A number of studies in laboratory animals have shown that chronic oral exposure to BEHP results in adverse hepatic effects (ATSDR, 1989). At high oral doses, BEHP may cause functional hepatic damage characterized by morphological changes and alterations in the activity of hepatic enzyme systems. Additionally, BEHP is a developmental and reproductive toxin in rats and mice (Singhe et al., 1972; Shiota and Nishimura, 1982).

The USEPA has classified BEHP as a B2 carcinogen, probable human carcinogen, based on inadequate human carcinogenicity data and sufficient animal carcinogenicity data (IRIS, 1996).

The USEPA has published an oral cancer slope factor of 0.014 $(mg/kg\text{-}day)^{-1}$ based on the results of studies with laboratory animals (IRIS, 1996). In bioassays, orally administered BEHP produced significant dose-related increases in liver tumor responses in male and female rats and mice administered 0, 6000, or 12000 ppm BEHP and 0, 3000, or 6000 ppm BEHP, respectively, for 103 weeks. Histological examinations indicated a significant increase in the incidence of hepatocellular carcinomas and combined incidence of carcinomas and adenoma in treated animals (NTP, 1982). The Agency has not proposed an inhalation cancer slope factor for BEHP.

In addition, the USEPA (1996) has established an oral RfD value of 0.02 mg/kg-day for BEHP based on the results of a study by Carpenter et al. (1953). The results of this study indicated increased relative liver weights after a 1-year feeding study in which guinea pigs were administered BEHP at 0, 19, or 64 mg/kg-day. Statistically significant increases in relative liver weights were observed in both groups of treated females (Carpenter et al., 1953). The Agency has not established an inhalation RfD value for BEHP.

References

ATSDR. 1989. *Toxicological Profile for Bis(2-ethylhexyl)phthalate: Draft.* Agency for Toxic Substances and Disease Registry, Atlanta, GA and U.S. Department of Health and Human Services, Public Health Service. April.

Carpenter, C.P., C.S. Weil, and H.F. Smith. 1953. Chronic oral toxicity of di(2-ethylhexyl)phthalate for rats and guinea pigs. *Arch. Indust. Hyg. Occup. Med.* 8:219-226. (Cited in IRIS, 1996)

IRIS. 1996. *Bis(2-ethylhexyl)phthalate.* Integrated Risk Information System, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH.

NTP. 1982. *Carcinogenesis Bioassay of Di(2-ethylhexyl)phthalate (CAS No. 117-81-7) in F344 Rats and B6C3F1 Mice (Feed Study).* NTP Technical Report Series TR No. 217. National Toxicology Program, Research Triangle Park, NC. (Cited in IRIS, 1996)

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. (Cited in IRIS, 1996)

Singhe, A.R., W.H. Lawrence, and J. Autian. 1972. Teratogenicity of phthalate esters in rats. *J. Pharmacol. Sci.* 61:51. (Cited in IRIS, 1996)

Toxicological Profile

Copper

Copper is typically used in metal alloys, including brass, bronze, coins, and gun metal. Copper compounds are employed in a variety of industries including electrical, construction, machinery, transportation, and ordinance. Additionally, copper compounds may also be used as fungicides, algicides, or nutritional supplements in animal feed and fertilizers. Copper is also an essential nutrient for humans. (ATSDR, 1990).

The USEPA has determined copper to be a Group D carcinogen (not classified) due to the absence of human data, inadequate animal data from assays of copper compounds, and equivocal mutagenicity data (IRIS, 1996). The Agency has not derived an oral or inhalation cancer slope factor for this compound.

The USEPA has developed an oral RfD value of 0.04 mg/kg-day (USEPA, 1996). The Agency has not, however, established an inhalation RfD value for copper.

References

ATSDR. 1990. *Toxicological Profile for Copper.* Agency for Toxic Substances and Disease Registry, U.S. Public Health Service, Atlanta, GA. December.

IRIS. 1996. *Copper.* Integrated Risk Information System, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH.

USEPA. 1996. *Risk-Based Concentration Table, January-June 1996.* Dr. Roy L. Smith, U.S. Environmental Protection Agency, Region III, Office of RCRA, Technical and Program Support Branch, Philadelphia, PA. April 30.

Toxicological Profile

Endrin Aldehyde

Endrin aldehyde is a minor impurity of the insecticide endrin and may be released to the environment during its use and application. Endrin aldehyde is also an environmental metabolite of endrin (Sanborn et al, 1977; Callahan et al., 1979)

Toxicity data for human and animal exposures to endrin aldehyde are limited. Acute exposures to endrin aldehyde in various doses may result in gastrointestinal upset, sensory aberrations, central nervous system excitations, seizures, coma, and possibly liver necrosis (HSDB, 1996).

The USEPA has not classified endrin aldehyde as to human carcinogenicity. Also, the Agency has not established oral or inhalation cancer slope factors or RfD values for endrin aldehyde (USEPA, 1995). In lieu of these data, the oral cancer slope factor for endrin was used.

References

Callahan, M.A. et al. 1979. *Water-Related Environmental Fate of 129 Priority Pollutants, Volume I.* U.S. Environmental Protection Agency, Washington, D.C. EPA-440/4-79-029A. (Cited in HSDB, 1996)

HSDB. 1996. *Endrin Aldehyde.* Hazardous Substances DataBank, National Library of Medicine, Bethesda, MD.

Sanborn, J.R. et al. 1977. *The Degradation of Selected Pesticides in Soil: A Review of Published Literature.* U.S. Environmental Protection Agency, Washington, D.C. EPA-600/9-77-022. (Cited in HSDB, 1996)

USEPA. 1995. *Health Effects Assessment Summary Tables, FY-1995 Annual.* U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, D.C. EPA/540/R-95/036. May.

Toxicological Profile

Mercury

Mercury is best known for its use in thermometers, barometers, and other pressure-sensing devices, but it is also used in a wide variety of industrial applications (ATSDR, 1988). Mercury may be used as a component in batteries, radios, calculators, smoke alarms, watches, missiles, and spacecraft. Additionally, mercury lamps may be employed for street lamps, film projectors, and photographic equipment. It is often used in the production of vinyl chlorides, urethanes, anthraquinone, chlorines, and caustic sodas. To a lesser extent, mercury may also be found in pharmaceuticals, paints, pigments, lubrication oils, and dental amalgrams (Grayson, 1983; Merck, 1983).

It is thought that exposure to inorganic mercury compounds may affect kidney function. Experimental animal studies reproduced the central nervous system effects observed in humans, but only at very high concentrations. Animal experiments have, however, demonstrated kidney effects in animals administered inorganic salts of mercury (USEPA, 1984). Inhalation exposure to inorganic mercury compounds has been associated with increased numbers of spontaneous abortions and menstrual difficulties in exposed animals. No information is available on the potential reproductive and developmental effects of oral exposure to inorganic mercury (USEPA, 1984; ATSDR, 1988).

The USEPA has classified mercury as a Group D carcinogen, not classifiable as to human carcinogenicity, based on inadequate human and animal data. The available epidemiologic studies do not show a correlation between mercury exposure and carcinogenicity (Cragle et al., 1984; Ahlbom et al., 1986; Barregard et al., 1990). These studies are generally complicated by the presence of confounding factors such as concurrent exposures to other carcinogens or lifestyle variables (IRIS, 1996). Additionally, the USEPA has not published an oral or inhalation cancer slope factor for mercury (USEPA, 1995).

The USEPA has established an oral RfD for methylmercury of 0.0004 mg/kg-day based on neurological effects (IRIS, 1996). In the absence of an oral RfD for total mercury, the value published for methylmercury will be used in this assessment. The USEPA has established an inhalation reference concentration (RfC) of 0.0003 mg/m^3 for elemental mercury based on occupational studies (IRIS, 1996). For this evaluation, the RfC value is converted to an inhalation RfD of 0.0000857 mg/kg-day. Results of studies conducted by Fawer et al. (1983) indicate a significant incidence of tremors in workers exposed to low concentrations of mercury vapors. In a

similar investigation, Piikivi and Tolonen (1989) reported significantly slower and attenuated brain activity among exposed workers. In addition, other adverse effects as a result of mercury exposure to workers include increases in memory disturbances as well as subjective and objective evidence of autonomic dysfunction (Piikivi, 1989; Piikivi and Hanninen, 1989; Ngim et al., 1992; Liang et al., 1993).

References

Ahlbom, A., S. Norell, Y. Rodvall, and M. Nylander. 1986. Dentists, dental nurses, and brain tumors. *Br. Med. J.* 292:662. (Cited in IRIS, 1996)

ATSDR. 1988. *Toxicological Profile for Mercury: Draft.* Agency for Toxic Substances and Disease Registry, Atlanta, GA and U.S. Department of Health and Human Services, Public Health Service. December.

Barregard, L., G. Sallsten, and B. Jarvholm. 1990. Mortality and cancer incidence in chloralkali workers exposed to inorganic mercury. *Br. J. Ind. Med.* 47(2):99-104. (Cited in IRIS, 1996)

Cragle, D.L., D.R. Hollis, J.R. Qualters, W.G. Tankersley, and S.A. Fry. 1984. A mortality study of men exposed to elemental mercury. *J. Occup. Med.* 26(11):817-821. (Cited in IRIS, 1996)

Fawer, R.F., U. DeRibaupierre, M.P. Guillemin, M. Berode, and M. Lobe. 1983. Measurement of hand tremor induced by industrial exposure to metallic mercury. *J. Ind. Med.* 40:204-208. (Cited in IRIS, 1996)

Grayson, M. (ed.). 1983. *Kirk-Othmer Encyclopedia of Chemical Technology.* 3rd Edition. New York, NY: John Wiley & Sons. (Cited in ATSDR, 1988)

IRIS. 1996. *Mercury.* Integrated Risk Information System, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH.

Liang, Y-X., R-K. Sun, Y. Sun, Z-Q. Chen, and L-H. Li. 1993. Psychological effects of low exposure to mercury vapor: Application of a computer-administered neurobehavioral evaluation system. *Environ. Res.* 60:320-327. (Cited in IRIS, 1996)

Merck. 1983. *The Merck Index.* Windholz, M. (ed.). Rahway, NJ: Merck & Co., Inc.

Ngim, C.H., S.C. Foo, K.W. Boey, and J. Jeyaratnam. 1992. Chronic neurobehavioral effects of elemental mercury in dentists. *Br. J. Ind. Med.* 49:782-790. (Cited in IRIS, 1996)

Piikivi, L. 1989. Cardiovascular reflexes and low long-term exposure to mercury vapor. *Int. Arch. Occup. Environ. Health.* 61:391-395. (Cited in IRIS, 1996)

Piikivi, L. and H. Hanninen. 1989. Subjective symptoms and psychological performance of chlorine-alkali workers. *Scand. J. Work Environ. Health* 15:69-74. (Cited in IRIS, 1996)

Piikivi, L. and U. Tolonen. 1989. EEG findings in chlor-alkali workers subjected to low long term exposure to mercury vapor. *Br. J. Ind. Med.* 46:370-375. (Cited in IRIS, 1996)

USEPA. 1984. *Mercury Health Effects Update; Health Issue Assessment: Final Report.* U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Washington, D.C. EPA 600 8-84-019f. (Cited in ATSDR, 1988)

USEPA. 1995. *Health Effects Assessment Summary Tables, FY-1995 Annual.* U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, D.C. EPA/540/R-95/036. May.

Toxicological Profile

Nickel

Nickel is a silvery metal that is insoluble in water. Nickel and its compounds are used in nickel-plating, for coins, electrotypes, lightning rod tips, electrical contacts, as a catalyst, and in the manufacture of various metals and alloys (ATSDR, 1988).

In humans, acute dermal exposure to nickel commonly results in contact dermatitis, atopic dermatitis, and allergenic sensitization. Other signs and symptoms of exposure to nickel include nausea, vomiting, diarrhea, central nervous system depression, coughing, shortness of breath, chest pain, fever and weakness (ATSDR, 1988). Chronic occupational inhalation exposures may result in respiratory effects such as asthma and chronic respiratory tract infections (NAS, 1975). However, nickel may also be an essential human nutrient (ATSDR, 1988).

Studies with experimental animals suggest that nickel and nickel compounds have relatively low acute and chronic oral toxicity. Noncarcinogenic respiratory effects are found in animals exposed to nickel by inhalation (ATSDR, 1988). Ingested nickel may cause reproductive and developmental toxicity in animals (Smith et al., 1990). Reproductive effects in male rats include degenerative changes in the testes, epididymis and spermatozoa (ATSDR, 1988; Ambrose et al., 1976). Studies in female rats and hamsters suggest an effect on embryo viability and the implantation process. Animals exposed to nickel before implantation have shown delayed embryonic development and increased resorptions, although there are problems associated with the interpretation of these studies (ATSDR, 1988; Smith et al., 1990).

The USEPA has classified nickel as a Group A (human) carcinogen, based on sufficient evidence in humans (IRIS, 1996). The Agency has not determined an oral cancer slope factor for nickel. However, the USEPA has derived an inhalation unit risk value of 0.00024 $(\mu g/m^3)^{-1}$ and an inhalation cancer slope factor of 0.84 $(mg/kg\text{-}day)^{-1}$ based on human data from several epidemiologic studies which suggest that exposure to nickel refinery dust caused lung and nasal tumors in sulfide nickel matte workers (Chovil et al., 1981; Enterline and Marsh, 1982; Magnus et al., 1982; Peto et al., 1984). In addition, animal studies in which rats were exposed to nickel via inhalation and injection indicate the presence of carcinomas (Gilman and Ruckerbauer, 1962; Saknyn and Blohkin, 1978). The USEPA has established an oral RfD value for nickel of 0.02 mg/kg-day (USEPA, 1995).

References

Ambrose, A.M., P.S. Larson, J.R. Borzelleca, and G.R. Hennigar. 1976. Long-term toxicologic assessment of nickel in rats and dogs. J. Food Sci. Technol. 13:181-187. (Cited in IRIS, 1996)

ATSDR. 1988. *Toxicological Profile for Nickel.* Agency of Toxic Substances for Disease Registry, U.S. Public Health Service, Atlanta, GA.

Chovil, A., R.B. Sutherland, and M. Halliday. 1981. Respiratory cancer in a cohort of sinter plant workers. *Br. J. Ind. Med.* 38:327-333. (Cited in IRIS, 1996)

Enterline, P.E. and G.M. Marsh. 1982. Mortality among workers in a nickel refinery and alloy manufacturing plant in West Virginia. *J. Natl. Cancer Inst.* 68:925-933. (Cited in IRIS, 1996)

Gilman, J.P.W. and G.M. Ruckerbauer. 1962. Metal carcinogenesis. I. Observations on the carcinogenicity of a refinery dust, cobalt oxide, and colloidal thorium dioxide. *Cancer Res.* 22:152-157. (Cited in IRIS, 1996)

IRIS. 1996. *Nickel.* Integrated Risk Information System, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH.

Magnus, K., A. Andersen, and A. Hogetveit. 1982. Cancer of respiratory organs among workers at a nickel refinery in Norway. *Int. J. Cancer.* 30:681-685. (Cited in IRIS, 1996)

NAS. 1975. *Nickel.* National Academy of Sciences, Committee on Medical and Biologic effects of Environmental Pollutants, Washington, D.C. (Cited in ATSDR, 1988)

Peto, J., H. Cuckle, R. Doll, C. Hermon, and L.G. Morgan. 1984. Respiratory cancer mortality of Welsh nickel refinery workers. In: *Nickel in the Human Environment: Proceedings of a Joint Symposium, March, 1983.* IARC Scientific Publication No. 53. International Agency for Research on Cancer, Lyon, France. p. 36-46. (Cited in IRIS, 1996)

Saknyn, A.V. and V.A. Blohkin. 1978. On the development of malignant tumors in rats exposed to nickel containing aerosols. *Vopr. Onkol.* 24(4):44-48. (Cited in IRIS, 1996)

Smith, M.K., J.A. George, H.F. Stober, and G.L. Kimmel. 1990. Perinatal toxicity associated with nickel chloride exposure. *Fund. Appl. Toxicol.* Preliminary unpublished draft. (Cited in IRIS, 1996)

USEPA. 1995. *Health Effects Assessment Summary Tables, FY-1995 Annual.* U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, D.C. EPA/540/R-95/036. May.

Toxicological Profile

Silver

The primary use of silver and silver compounds is in the production of photographic materials. It is also utilized in electrical products such as contacts, paints, and batteries. Additionally, silver may be employed as a component in the manufacture of alloys, solders, and bearings. To a lesser extent, silver is used in electroplated ware, jewelry, mirrors, dental amalgram, medical supplies, chemical catalysts, and cloud seeding (Smith and Carson, 1977; Grayson, 1983; ATSDR, 1989; HSDB, 1996).

Limited data are available regarding the acute effects of exposure to silver. Reports of acute poisoning in humans include a case of respiratory distress in a worker who became ill 14 hours after working with molten silver (Forycki et al., 1983). Initial symptoms observed include rapid pulse, low oxygen content of capillary blood, and scattered thickening of the lungs observed in chest radiograms. Intravenous injection of a silver salt (50 mg or more) for therapeutic purposes has been reported to be lethal. Autopsy findings in such cases have included pulmonary edema, hemorrhage, and necrosis of bone marrow, liver, and kidney (Patein and Roblin, 1909; Hill and Pillsbury, 1939). Repeated exposure to silver salts or colloidal silver by inhalation or ingestion can cause a blue-gray discoloration of the skin known as argyria (Friberg et al., 1986). This discoloration is not known to be symptomatic of any other toxic effect. Occupational exposure to silver dusts has been associated with respiratory and gastrointestinal irritation (Rosenman et al., 1979).

The USEPA has classified silver as a Group D carcinogen, not classifiable as to human carcinogenicity, based on inadequate data in human and animal studies (IRIS, 1996). Local sarcomas have been induced in animals after implantation of silver foils and silver discs (Schmal and Steinhoff, 1960; Furst and Schlauder, 1977; Furst, 1981). Interpretation of these findings, however, is questionable due to the occurrence of solid-state carcinogenesis in which even insoluble solids such as plastic have been shown to result in local fibrosarcomas (IRIS, 1996). Consequently, the Agency has not established an oral or inhalation cancer slope factor for silver.

The USEPA has developed an oral RfD of 0.005 mg/kg-day based on a number of reported cases of subpopulations which have exhibited an increased propensity for the development of argyria following exposure to silver from dietary sources (IRIS, 1996). The USEPA has not proposed an inhalation RfD value for silver (USEPA, 1995). For the purposes of this evaluation, however, the oral RfD is used for the inhalation RfD value.

References

ATSDR. 1989. *Toxicological Profile for Silver: Draft.* Agency for Toxic Substances and Disease Registry, Atlanta, GA and U.S. Department of Health and Human Services, Public Health Service. October.

Forycki, Z., W. Zegarski, J. Bardzik et al. 1983. Acute silver poisoning through inhalation. *Bulletin of the Institute of Maritime and Tropical Medicine in Gdynia.* 34:199-203. (Cited in ATSDR, 1989)

Friberg, L., G.F. Nordberg, and V.B. Vouk (eds). 1986. *Handbook on the Toxicology of Metals.* Volume II. Amsterdam: Elsevier Science Publishers.

Furst, A. 1981. Bioassay of metals for carcinogenesis: Whole animals. *Environ. Health Perspect.* 40:83-92. (Cited in IRIS, 1996)

Furst, A. and M.C. Schlauder. 1977. Inactivity of two noble metals as carcinogens. *J. Environ. Pathol. Toxicol.* 1:51-57. (Cited in IRIS, 1996)

Grayson, M. (ed.). 1983. *Kirk-Othmer Encyclopedia of Chemical Technology.* 3rd Edition. New York, NY: John Wiley & Sons. (Cited in ATSDR, 1989)

Hill, W.R. and D.M. Pillsbury. 1939. *Argyria, The Pharmacolgy of Silver.* Baltimore, MD: William and Wilkins. (Cited in Friberg et al., 1986)

HSDB. 1996. *Silver.* Hazardous Substances Data Bank, National Library of Medicine, National Institutes of Health, Bethesda, MD.

IRIS. 1996. *Silver.* Integrated Risk Information System, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH.

Patein, G. and L. Roblin. 1909. *Bull. Gen. Ther.* (Paris) 158:898-912. (Cited in Friberg et al., 1986)

Rosenman, K.D., A. Moss, and S. Kon. 1979. Argyria: Clinical implications of exposure to silver nitrate and silver oxide. *J. Occup. Med.* 21:430-435. (Cited in ATSDR, 1989)

Schmal, D. and D. Steinhoff. 1960. Versuche zur Krebserzeugung mit kolloidalen Silber-und Goldlosungen an Ratten. *Z. Krebsforsch.* 63:586-591. (Cited in IRIS, 1996)

Smith, I.C. and B.L. Carson. 1977. *Trace Metals in the Environment; Volume 2: Silver.* Ann Arbor, MI: Ann Arbor Science Publishers, Inc. (Cited in ATSDR, 1989)

USEPA. 1995. *Health Effects Assessment Summary Tables, FY-1995 Annual.* U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, D.C. EPA/540/R-95/036. May.

Toxicological Profile

Thallium

Thallium occurs naturally as a trace compound in several minerals and occurs in small amounts in almost all living things (Seiler, 1988). Thallium is most commonly employed by the semiconductor industry in the production of switches and closures. To a lesser extent, it is used in the pharmaceutical industry and as a chemical intermediate for other thallium compounds (Windholz, 1983; U.S. Bureau of Mines, 1988).

In humans, various thallium exposures may cause neural, hepatic, and renal injury. Acute thallium toxicity to humans may be characterized by gastrointestinal irritation, acute ascending paralysis, and psychic disturbances. Chronic thallium exposures to humans may result in liver necrosis, nephritis, gastroenteritis, pulmonary edema, or degeneration of peripheral and central nervous system. Humans subjected to industrial exposures may experience loss of vision and other signs of thallium poisoning (Browning, 1969; Fowler, 1982; Amdur et al., 1991).

Acute toxicity studies in rats suggest that thallium toxicity may be significant. An oral LD_{50} of 30 mg/kg has been reported for rats (Downs et al., 1960). In addition, rat bioassays have reported renal lesions, and degenerative histologic and central nervous system changes (Amdur et al., 1991).

The USEPA has not classified thallium as to human carcinogenicity. Also, the Agency has not established oral or inhalation cancer slope factors or RfD values for thallium (USEPA, 1995). In the absence of data specific to thallium, the oral RfD for thallium chloride was used in the risk assessment.

References

Amdur, M.O., J. Doull, and C.D. Klaassen (eds). 1991. *Casarett and Doull's Toxicology: The Basic Science of Poisons, 4th Edition.* New York, NY: Pergamon Press.

ATSDR. 1990. Toxicological Profile for Thallium: Draft. U.S. Department of Health and Human Services, U.S. Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA. October.

Browning, E. 1969. *Toxicity of Industrial Metals, 2nd Edition.* London: Butterworths. (Cited in Amdur et al., 1991)

Downs, W.L., J.K. Scott, L.T. Steadman, and E.A. Maynard. 1960. Acute and subacute toxicity studies of thallium compounds. *Am. Ind. Hyg. Assoc. J.* 21:399-406. (Cited in Amdur et al., 1991)

Fowler, B.A. 1982. Indium and thallium in health. In: Rose, J. (Ed). *Trace Metals in Human Health.* London: Butterworths. Pp. 74-82. (Cited in Amdur et al., 1991)

HSDB. 1996. *Thallium.* Hazardous Substances Data Bank, National Library of Medicine, National Institutes of Health, Bethesda, MD.

Ludolph, A., C.E. Elger, R. Sennhenn et al. 1986. Chronic thallium exposure in cement plant workers: Clinical and electrophysiological data. *Trace Elem. Med.* 3:121-125. (Cited in ATSDR, 1990)

Seiler. 1988. *Hdbk Toxicity Inorganic Cpds.* P. 678. (Cited in HSDB, 1996)

U.S. Bureau of Mines. 1988. *Mineral Commodity Summaries.* U.S. Bureau of Mines, Washington, D.C. (Cited in ATSDR, 1990)

USEPA. 1995. *Health Effects Assessment Summary Tables, FY-1995 Annual.* U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, D.C. EPA/540/R-95/036. May.

Windholz, M. (Ed). 1983. *The Merck Index: An Encyclopedia of Chemicals, Drugs, and Biologicals, 10th Edition.* Rahway, NJ: Merck and Company, Inc. 1324-1325. (Cited in ATSDR, 1990)

Toxicological Profile

Vanadium

Vanadium is a naturally occurring element found in a variety of ores and fuel oil. It is used primarily in the steel industry as an alloy in the production of automotive parts, springs, ball bearings, and ferrovanadium alloys. In addition, vanadium compounds are widely used in the production of aircraft engines and as industrial catalysts. Other applications of vanadium compounds are utilized by the textile, ceramic, and printing industries (ATSDR, 1990).

Vanadium toxicity among humans is characterized by nasal bleeding combined with soreness of the chest and throat, coughing, and dyspnea. These symptoms usually cease, however, following removal from exposure (Browning, 1969). In animals, exposures to vanadium compounds have been reported to result in embryotoxicity, catarrhal gastritis, reduced blood glucose levels, reduced body weight, and metabolic effects (Friberg, 1986; Meyerovitch et al, 1987; Paternain et al, 1987; Zaporowska and Wasilewski, 1989).

The USEPA has not classified vanadium as to potential human carcinogenicity. The Agency has not derived an oral or inhalation cancer slope factor for this compound (IRIS, 1996).

The USEPA has developed a chronic oral RfD value of 0.0073 mg/kg-day based on the results of a lifetime drinking water study with rats (USEPA, 1995). The Agency has not, however, established an inhalation RfD value for vanadium.

References

ATSDR. 1990. *Toxicological Profile for Vanadium and Compounds: Draft.* Agency for Toxic Substances and Disease Registry, U.S. Public Health Service, Atlanta, GA. October.

Browning (ed). 1969. *Toxicity of Industrial Metals, 2nd Edition.* P. 344. (Cited in HSDB, 1996)

Friberg (ed). 1986. *Handbook of the Toxicity of Metals, 2nd Edition, Volumes I-II.* P. 648. (Cited in HSDB, 1996)

HSDB. 1996. *Vanadium.* Hazardous Substances DataBank, National Library of Medicine, National Institutes of Health, Bethesda, MD.

IRIS. 1996. *Vanadium.* Integrated Risk Information System, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH.

Meyerovitch, J. et al. 1987. *J. Biol. Chem.* 262(14):6658-6662. (Cited in HSDB, 1996)

Paternain, J. et al. 1987. *Rev. Esp. Fisiol.* 43(2):223-228. (Cited in HSDB, 1996)

USEPA. 1995. *Health Effects Assessment Summary Tables, FY-1995 Annual.* U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, D.C. EPA/540/R-95/036. May.

Zaporowska, H. and W. Wasilewski. 1989. 93(1):175-180. (Cited in HSDB, 1996)

Toxicity Profile

Lead

In the United States, lead is used primarily in lead acid batteries, gasoline additives, and other applications within the transportation industry. It is also employed as a product for construction, ammunition, electronics, television glass, and paint. To a lesser degree, lead is used in ceramics, type metal, ballasts and weights, and tubes. Due to its toxic nature, however, the use of lead has slowly decreased in recent years as alternative materials are developed due to its toxic nature (ATSDR, 1991).

Chemical Formula	Pb	ATSDR, 1991
Molecular Weight	207.20	HSDB, 1995
Vapor Pressure	1.77 mmHg @ 1,000°C	HSDB, 1995
Boiling Point	1,740°C	HSDB, 1995
Melting Point	327.4°C	HSDB, 1995
Water Solubility	Insoluble	HSDB, 1995
Partition Coefficients		
Henry's Law Constant (air/water)	No data	ATSDR, 1991
log K_{ow}	No data	ATSDR, 1991
log K_{oc}	No data	ATSDR, 1991

Lead in the atmosphere exists primarily in the particulate form. Smaller particulates may be transported thousands of kilometers, while particulates of 2 μm or more are generally deposited near emission sources. Ultimately, lead is removed from the atmosphere via wet or dry deposition (ATSDR, 1991). It is unclear as to the chemical composition changes of tetraethyl lead during dispersion. Studies suggest that it reacts with hydroxyl ions to form ionic trialkyl and dialkyl species which are more stable in the atmosphere (USEPA, 1986). Eventually it is transformed to inorganic lead via direct photolysis, hydroxyl radicals, and reaction with ozone. Adsorption of lead to atmospheric particles is not a significant fate process (ATSDR, 1991).

The fate of lead in soils is dependent on the soil pH, organic matter content, ion-exchange characteristics, and the presence of inorganic colloids and iron oxides (ATSDR, 1991). Ordinarily, lead is retained in soil, and is unlikely to be transported to groundwater or surface waters (NSF, 1977;

USEPA, 1986). Inorganic lead is essentially immobile in soil if bound into crystalline matrices. The transformation of lead complexes and precipitates depends on soil type. Soils with high organic matter content and a pH of 6-8 may form insoluble organic lead complexes, while soils with less organic matter and the same pH may form hydrous lead oxide complexes. Soils with a pH of 4-6, however, may permit the formation of organic lead complexes which are soluble and become subject to leaching or uptake by plants (USEPA, 1986). Conditions that may induce the leaching of lead within soils includes the presence of lead at concentrations greater than the cation exchange capacity of the soil, the presence of soil constituents capable of forming soluble chelates, and a decrease in the pH of the leaching solution (NSF, 1977).

The amount of lead in water systems is dependent upon the pH and the dissolved salt content. At a pH of less than 5.4 the total solubility of lead is estimated to be 30 μg/L and 500 μg/L in hard and soft water, respectively. Lead carbonates may limit the lead concentration, however, at a pH of less than 5.4 (USEPA, 1979). In river systems, lead may be present as sorbed ions or surface coatings on sediment mineral particles, or it may exist as part of suspended organic matter. Tetraalkyl lead compounds in water may be subject to photolysis and volatilization. Degradation of the more volatile lead compounds via evaporation occur as trialkyl lead to dialkyl lead to inorganic lead. In addition, tetraethyl lead may decomposed in water via photolysis (ATSDR, 1991).

Lead in humans may be stored in bone, kidney, and liver. The major adverse effects in humans caused by lead include alterations in the blood and nervous systems. The toxic effects are generally related to the concentration of this metal in blood. Toxic blood concentrations in children and in sensitive adults may cause severe, irreversible brain damage, encephalopathy, and possible death. Physiological and biochemical effects that occur even at low levels include enzyme inhibition, interference with vitamin D metabolism, cognitive dysfunction in infants, electrophysiological dysfunction, and reduced childhood growth (ATSDR, 1991).

Exposure to lead has been associated with developmental effects in humans. Effects include reduced birth weight, gestational age, and neurobehavioral deficits or delays. There is no positive association between lead exposure and congenital malformations, suggesting that lead is not teratogenic (ATSDR, 1991).

There is evidence that exposure to lead can cause genotoxic effects. Lead has been shown in a number of assays to affect processes associated with gene expression (IRIS, 1995). Lead exposure has also been associated with sister chromatic exchange in workers, and induction of chromosomal

aberrations in vivo (Grandjean et al., 1983). Lead acetate has been shown to induce cell transformation in Syrian Hamster embryo cells (DiPaolo et al., 1978).

The available studies of carcinogenicity of lead following ingestion by laboratory animals indicates that lead is carcinogenic and that renal tumors are the most common carcinogenic response (ATSDR, 1991; IRIS, 1995). Azar et al. (1973) reported renal tumors in rats administered lead for two years at doses of 27, 56.5, 105 mg/kg-day. Likewise, renal tubular carcinomas were observed in 81% of the study rats given lead in their drinking water at 37 mg/kg-day for 76 weeks (Koller et al., 1986).

The USEPA has classified lead as a Group B2 carcinogen, probable human carcinogen, based on inadequate carcinogenic evidence in humans and sufficient animal carcinogenic evidence (IRIS, 1995). The results of a number of animal studies, including ten rat bioassays and one mouse assay, show a statistically significant increase in renal tumors due to dietary and subcutaneous exposures to soluble lead salts (Van Esch and Kroes, 1969; Azar et al., 1973; Casto et al., 1979; DiPaolo et al., 1979; Grandjean et al., 1983; Kasprzak et al., 1985; Koller et al., 1986). The Agency has not established an oral or inhalation cancer slope factor or RfD value for lead (IRIS, 1995).

References

ATSDR. 1991. *Toxicological Profile for Lead.* Agency for Toxic Substance and Disease Registry, Atlanta, GA and U.S. Department of Health and Human Services. PB93-110781. October.

Azar, A., H.J. Trochimowicz, and M.E. Maxfield. 1973. Review of lead studies in animals carried out at Haskell Laboratory: Two year feeding study and response to hemorrhage study. In: Barth, D., A. Berlin, R. Engel et al. (eds). *Environmental Health Aspects of Lead: Proceedings, International Symposium, October 1972, Amsterdam, The Netherlands.* Luxembourg: Commission of the European Communities. 199-210. (Cited in ATSDR, 1991)

Casto, B.C., J. Meyers, and J.A. DiPaolo. 1978. Enhancement of viral transformation for evaluation of the carcinogenic or mutagenic potential of inorganic metal salts. *Cancer Res.* 39:193-198. (Cited in IRIS, 1995)

DiPaolo, J.A., R.L. Nelson, and B.C. Castro. 1978. In vitro neoplastic transformation of Syrian hamster cells by lead acetate and its relevance to environmental carcinogenesis. *Br. J. Cancer.* 38:452-455. (Cited in IRIS, 1995)

Grandjean, P., H.C. Wulf, and E. Niebuhr. 1983. Sister chromatic exchange in response to variations in occupational lead exposure. *Environ. Res.* 32:199-204.

HSDB. 1995. *Lead.* Hazardous Substances DataBank, National Library of Medicine, Bethesda, MD.

IRIS. 1995. *Lead.* Integrated Risk Information System. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH.

Kasprzak, K.S., K.L. Hoover, and L.A. Poirier. 1985. Effects of dietary calcium acetate on lead subacetate carcinogenicity in kidneys of male Sprague-Dawley rats. *Carcinogenesis.* 6(2):279-282.

Koller, L.D., N.I. Kerkvliet, and J.H. Exon. 1986. Neoplasia induced in male rats fed lead acetate, ethylurea, and sodium nitrite. *Toxicologic. Pathol.* 13:50-57.

NSF. 1977. *Lead in the Environment.* In: Boggess, W.R. (ed). Washington, DC: National Science Foundation. NSF/RA-770214. (Cited in ATSDR, 1991)

USEPA. 1979. *Water-Related Environmental Fate of 129 Priority Pollutants. Volume I: Introduction and Technical Background, Metals, and Inorganic Pesticides, and PCBs.* U.S. Environmental Protection Agency, Washington, D.C. EPA-440/4-79-029a, 13-1 to 13-19.

USEPA. 1986. *Air Quality Criteria for Lead.* U.S. Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA-600/8-83-028F.

Van Esch, G.J. and R. Kroes. 1969. The induction of renal tumors by feeding of basic lead acetate to mice and hamsters. *Br. J. Cancer.* 23:265-271. (Cited in IRIS, 1995)

Appendix H

Carcinogenic and Noncarcinogenic Risk Calculations

Table H-1

Sector: River
Scenario: Wading
Pathway: Incidental Ingestion of Surface Sediment
Receptor: Child (7 to 12 years)

CARCINOGENIC
CDI = Cs x IgR x IgRF x ROA x EF x ED x CF x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	Cs Surface Sediment Concentration (mg/kg)	IgR Sediment Ingestion Rate (mg/d)	IgRF Fraction Ingested Attributable to Site (unitless)	ROA Relative Oral Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals												
Acenaphthylene	0.091	200	0.5	1.0	82	6	1.00E-06	33	25,550	5.3E-09	NA	
Benzo(a)anthracene	1.0	200	0.5	1.0	82	6	1.00E-06	33	25,550	6.0E-08	7.30E-01	4E-08
Benzo(a)pyrene	0.94	200	0.5	1.0	82	6	1.00E-06	33	25,550	5.5E-08	7.30E+00	4E-07
Benzo(b)fluoranthene	1.1	200	0.5	1.0	82	6	1.00E-06	33	25,550	6.5E-08	7.30E-01	5E-08
Benzo(g,h,i)perylene	0.44	200	0.5	1.0	82	6	1.00E-06	33	25,550	2.6E-08	NA	
Phenanthrene	1.3	200	0.5	1.0	82	6	1.00E-06	33	25,550	7.7E-08	NA	
Pesticides/PCBs												
Aroclor-1242	4.4	200	0.5	1.0	82	6	1.00E-06	33	25550	2.57E-07	2.00E+00	5E-07
Aroclor-1260	3.1	200	0.5	1.0	82	6	1.00E-06	33	25550	1.81E-07	2.00E+00	4E-07
Inorganic Chemicals												
Aluminum	19600	200	0.5	1.0	82	6	1.00E-06	33	25550	1.14E-03	NA	
Antimony	16.535	200	0.5	1.0	82	6	1.00E-06	33	25550	9.65E-07	NA	
Arsenic	29.549	200	0.5	1.0	82	6	1.00E-06	33	25550	1.72E-06	1.50E+00	3E-06
Beryllium	1.2924	200	0.5	1.0	82	6	1.00E-06	33	25550	7.54E-08	4.30E+00	3E-07
Cadmium	6.6672	200	0.5	1.0	82	6	1.00E-06	33	25550	3.89E-07	NA	
Chromium	467.00	200	0.5	1.0	82	6	1.00E-06	33	25550	2.73E-05	NA	
Iron	215513	200	0.5	1.0	82	6	1.00E-06	33	25550	1.26E-02	NA	
Lead	786.19	200	0.5	1.0	82	6	1.00E-06	33	25550	4.59E-05	NA	
Manganese	2720.0	200	0.5	1.0	82	6	1.00E-06	33	25550	1.59E-04	NA	
Vanadium	53.938	200	0.5	1.0	82	6	1.00E-06	33	25550	3.15E-06	NA	
Zinc	28200	200	0.5	1.0	82	6	1.00E-06	33	25550	1.65E-03	NA	
											Sum	4E-06

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Table H-2

Sector: River
Scenario: Wading
Pathway: Incidental Ingestion of Surface Sediment
Receptor: Child (7 to 12 years)

NONCARCINOGENIC
CDI = Cs x IgR x IgRF x ROA x EF x ED x CF x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	Cs Surface Sediment Concentration (mg/kg)	IgR Sediment Ingestion Rate (mg/d)	IgRF Fraction Ingested Attributable to Site (unitless)	ROA Relative Oral Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals												
Acenaphthylene	0.091	200	0.5	1.0	82	6	1.00E-06	33	2,190	6.2E-08	NA	
Benzo(a)anthracene	1.0	200	0.5	1.0	82	6	1.00E-06	33	2,190	6.9E-07	NA	
Benzo(a)pyrene	0.94	200	0.5	1.0	82	6	1.00E-06	33	2,190	6.4E-07	NA	
Benzo(b)fluoranthene	1.1	200	0.5	1.0	82	6	1.00E-06	33	2,190	7.6E-07	NA	
Benzo(g,h,i)perylene	0.44	200	0.5	1.0	82	6	1.00E-06	33	2,190	3.0E-07	NA	
Phenanthrene	1.3	200	0.5	1.0	82	6	1.00E-06	33	2,190	9.0E-07	NA	
Pesticides/PCBs												
Aroclor-1242	4.4	200	0.5	1.0	82	6	1.00E-06	33	2,190	3.0E-06	NA	
Aroclor-1260	3.1	200	0.5	1.0	82	6	1.00E-06	33	2,190	2.1E-06	NA	
Inorganic Chemicals												
Aluminum	19600	200	0.5	1.0	82	6	1.00E-06	33	2,190	1.3E-02	1.00E+00	1.3E-02
Antimony	16.535	200	0.5	1.0	82	6	1.00E-06	33	2,190	1.1E-05	4.00E-04	2.8E-02
Arsenic	29.549	200	0.5	1.0	82	6	1.00E-06	33	2,190	2.0E-05	3.00E-04	6.7E-02
Beryllium	1.2924	200	0.5	1.0	82	6	1.00E-06	33	2,190	8.8E-07	5.00E-03	1.8E-04
Cadmium	6.6672	200	0.5	1.0	82	6	1.00E-06	33	2,190	4.5E-06	5.00E-04	9.1E-03
Chromium	467.00	200	0.5	1.0	82	6	1.00E-06	33	2,190	3.2E-04	5.00E-03	6.4E-02
Iron	215513	200	0.5	1.0	82	6	1.00E-06	33	2,190	1.5E-01	3.00E-01	4.9E-01
Lead	786.19	200	0.5	1.0	82	6	1.00E-06	33	2,190	5.4E-04	NA	
Manganese	2720.0	200	0.5	1.0	82	6	1.00E-06	33	2,190	1.9E-03	2.30E-02	8.1E-02
Vanadium	53.938	200	0.5	1.0	82	6	1.00E-06	33	2,190	3.7E-05	7.00E-03	5.2E-03
Zinc	28200	200	0.5	1.0	82	6	1.00E-06	33	2,190	1.9E-02	3.00E-01	6.4E-02
											Sum	8.2E-01

Table H-3

Sector: River
Scenario: Wading
Pathway: Dermal Contact with Surface Sediment
Receptor: Child (7 to 12 years)

CARCINOGENIC

CDI = Cs x DAF x SA x FSA x RDAsc x EF x ED x CF x 1/BW x 1/ATc

Risk = CDI x CSF

Chemical	Cs Surface Sediment Concentration (mg/kg)	DAF Dermal Adherence Factor (mg/cm^2-d)	SA Total Body Surface Area (cm^2)	FSA Fraction SA Exposed (unitless)	RDA Relative Dermal Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals													
Acenaphthylene	0.091	0.2	10,500	0.3	0.10	82	6	1.00E-06	33	25,550	3.3E-09	NA	
Benzo(a)anthracene	1.0	0.2	10,500	0.3	0.10	82	6	1.00E-06	33	25,550	3.8E-08	7.30E-01	3E-08
Benzo(a)pyrene	0.94	0.2	10,500	0.3	0.10	82	6	1.00E-06	33	25,550	3.5E-08	7.30E+00	3E-07
Benzo(b)fluoranthene	1.1	0.2	10,500	0.3	0.10	82	6	1.00E-06	33	25,550	4.1E-08	7.30E-01	3E-08
Benzo(g,h,i)perylene	0.44	0.2	10,500	0.3	0.10	82	6	1.00E-06	33	25,550	1.6E-08	NA	
Phenanthrene	1.3	0.2	10,500	0.3	0.10	82	6	1.00E-06	33	25,550	4.9E-08	NA	
Pesticides/PCBs													
Aroclor-1242	4.4	0.2	10,500	0.3	0.06	82	6	1.00E-06	33	25,550	9.7E-08	2.00E+00	2E-07
Aroclor-1260	3.1	0.2	10,500	0.3	0.06	82	6	1.00E-06	33	25,550	6.8E-08	2.00E+00	1E-07
Inorganic Chemicals													
Aluminum	19600	0.2	10,500	0.3	0.01	82	6	1.00E-06	33	25,550	7.2E-05	NA	
Antimony	16.535	0.2	10,500	0.3	0.01	82	6	1.00E-06	33	25,550	6.1E-08	NA	
Arsenic	29.549	0.2	10,500	0.3	0.032	82	6	1.00E-06	33	25,550	3.5E-07	1.50E+00	5E-07
Beryllium	1.2924	0.2	10,500	0.3	0.25	82	6	1.00E-06	33	25,550	1.2E-07	4.30E+00	5E-07
Cadmium	6.6672	0.2	10,500	0.3	0.01	82	6	1.00E-06	33	25,550	2.5E-08	NA	
Chromium	467.00	0.2	10,500	0.3	0.01	82	6	1.00E-06	33	25,550	1.7E-06	NA	
Iron	215513	0.2	10,500	0.3	0.01	82	6	1.00E-06	33	25,550	7.9E-04	NA	
Lead	786.19	0.2	10,500	0.3	0.02	82	6	1.00E-06	33	25,550	5.8E-06	NA	
Manganese	2720.0	0.2	10,500	0.3	0.20	82	6	1.00E-06	33	25,550	2.0E-04	NA	
Vanadium	53.938	0.2	10,500	0.3	0.01	82	6	1.00E-06	33	25,550	2.0E-07	NA	
Zinc	28200	0.2	10,500	0.3	0.01	82	6	1.00E-06	33	25,550	1.0E-04	NA	
												Sum	2E-06

Table H-4

Sector: River
Scenario: Wading
Pathway: Dermal Contact with Surface Sediment
Receptor: Child (7 to 12 years)

NONCARCINOGENIC
CDI = Cs x DAF x SA x FSA x RDAsnc x EF x ED x CF x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	Cs Surface Sediment Concentration (mg/kg)	DAF Dermal Adherence Factor (mg/cm^2-d)	SA Total Body Surface Area (cm^2)	FSA Fraction SA Exposed (unitless)	RDA Relative Dermal Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals													
Acenaphthylene	0.091	0.2	10,500	0.3	0.10	82	6	1.00E-06	33	2,190	3.9E-08	NA	
Benzo(a)anthracene	1.0	0.2	10,500	0.3	0.10	82	6	1.00E-06	33	2,190	4.4E-07	NA	
Benzo(a)pyrene	0.94	0.2	10,500	0.3	0.10	82	6	1.00E-06	33	2,190	4.0E-07	NA	
Benzo(b)fluoranthene	1.1	0.2	10,500	0.3	0.10	82	6	1.00E-06	33	2,190	4.8E-07	NA	
Benzo(g,h,i)perylene	0.44	0.2	10,500	0.3	0.10	82	6	1.00E-06	33	2,190	1.9E-07	NA	
Phenanthrene	1.3	0.2	10,500	0.3	0.10	82	6	1.00E-06	33	2,190	5.7E-07	NA	
Pesticides/PCBs													
Aroclor-1242	4.4	0.2	10,500	0.3	0.06	82	6	1.00E-06	33	2,190	1.1E-06	NA	
Aroclor-1260	3.1	0.2	10,500	0.3	0.06	82	6	1.00E-06	33	2,190	8.0E-07	NA	
Inorganic Chemicals													
Aluminum	19600	0.2	10,500	0.3	0.01	82	6	1.00E-06	33	2,190	8.4E-04	1.00E+00	8.4E-04
Antimony	16.535	0.2	10,500	0.3	0.01	82	6	1.00E-06	33	2,190	7.1E-07	4.00E-04	1.8E-03
Arsenic	29.549	0.2	10,500	0.3	0.032	82	6	1.00E-06	33	2,190	4.1E-06	3.00E-04	1.4E-02
Beryllium	1.2924	0.2	10,500	0.3	0.25	82	6	1.00E-06	33	2,190	1.4E-06	5.00E-03	2.8E-04
Cadmium	6.6672	0.2	10,500	0.3	0.01	82	6	1.00E-06	33	2,190	2.9E-07	5.00E-04	5.7E-04
Chromium	467.00	0.2	10,500	0.3	0.01	82	6	1.00E-06	33	2,190	2.0E-05	5.00E-03	4.0E-03
Iron	215513	0.2	10,500	0.3	0.01	82	6	1.00E-06	33	2,190	9.2E-03	3.00E-01	3.1E-02
Lead	786.19	0.2	10,500	0.3	0.02	82	6	1.00E-06	33	2,190	6.7E-05	NA	
Manganese	2720.0	0.2	10,500	0.3	0.20	82	6	1.00E-06	33	2,190	2.3E-03	2.30E-02	1.0E-01
Vanadium	53.938	0.2	10,500	0.3	0.01	82	6	1.00E-06	33	2,190	2.3E-06	7.00E-03	3.3E-04
Zinc	28200	0.2	10,500	0.3	0.01	82	6	1.00E-06	33	2,190	1.2E-03	3.00E-01	4.0E-03
												Sum	1.6E-01

Table H-5

Sector: Middle Buildings
Scenario: Future Employee
Pathway: Inhalation of Indoor Vapor
Receptor: Worker (>18 years)

CARCINOGENIC
CDI = Cv x InR x RIA x EF x ED x CF x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	Cv Vapor Concentration (mg/m^3)	InR Inhalation Rate (m^3/day)	RIA Relative Inhalation Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	BW Body Weight (kg)	ATc Averaging Time Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor $(mg/kg-d)^{-1}$	Risk
Volatile Organics										
1,1-Dichloroethane	0.0039	10.8	1.0	250	25	70	25,550	1.5E-04	1.75E-01	3E-05
Freon 113	0.021	10.8	1.0	250	25	70	25,550	7.9E-04	NA	
1,2-Dichloroethane	0.0400	10.8	1.0	250	25	70	25,550	1.5E-03	9.10E-02	1E-04
Benzene	0.0040	10.8	1.0	250	25	70	25,550	1.5E-04	2.90E-02	4E-06
Trichloroethene	0.0068	10.8	1.0	250	25	70	25,550	2.6E-04	6.00E-03	2E-06
Tetrachloroethene	0.0071	10.8	1.0	250	25	70	25,550	2.7E-04	2.03E-03	5E-07
PCBs/Pesticides										
Aroclor-1242	0.00020	10.8	1.0	250	25	70	25,550	7.5E-06	2.00E+00	2E-05
									Sum =	2E-04

Table H-6

Sector: Middle Buildings
Scenario: Future Employee
Pathway: Inhalation of Indoor Vapor
Receptor: Worker (>18 years)

NONCARCINOGENIC
CDI = Cv x IhR x RIA x EF x ED x CF x 1/BW x 1/ATc
HQ = CDI/RfD

Chemical	Cw Vapor Concentration (mg/m^3)	IhR Inhalation Rate (cm^3/day)	RIA Relative Inhalation Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Volatile Organics										
1,1-Dichloroethene	0.0039	10.8	1.0	250	25	70	9,125	4.1E-04	NA	
Freon 113	0.021	10.8	1.0	250	25	70	9,125	2.2E-03	NA	
1,2-Dichloroethene	0.0400	10.8	1.0	250	25	70	9,125	4.2E-03	2.86E-03	1.5E+00
Benzene	0.0040	10.8	1.0	250	25	70	9,125	4.2E-04	1.71E-03	2.5E-01
Trichloroethene	0.0068	10.8	1.0	250	25	70	9,125	7.2E-04	NA	
Tetrachloroethene	0.0071	10.8	3.0	250	25	70	9,125	2.3E-03	NA	
PCBs/Pesticides										
Aroclor-1242	0.00020	10.8	1.0	250	25	70	9,125	2.1E-05	NA	
									Sum =	1.7E+00

Table H-7

Sector: Middle Buildings
Scenario: Future Indoor Construction
Pathway: Dermal Contact with Subsurface Soil
Receptor: Construction Worker (>18 years)

CARCINOGENIC

CDI = Cs x DAF x SA x FSA x RDA x EF x ED x CF x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	Cs Subsurface Soil Concentration (mg/kg)	DAF Dermal Adherence Factor (mg/cm^2-d)	SA Total Body Surface Area (cm^2)	FSA Fraction SA Exposed (unitless)	RDA Relative Dermal Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals													
Acenaphthylene	0.13	0.2	18,000	0.22	0.10	65	2	1.00E-06	70	25,550	7.5E-10	NA	
Benzo(g,h,i)perylene	0.25	0.2	18,000	0.22	0.10	65	2	1.00E-06	70	25,550	1.4E-09	NA	
Phenanthrene	2.3	0.2	18,000	0.22	0.10	65	2	1.00E-06	70	25,550	1.3E-08	NA	
Pesticides/PCBs													
Aroclor-1242	2.4	0.2	18,000	0.22	0.06	65	2	1.00E-06	70	25,550	8.3E-09	2.00E+00	2E-08
Aroclor-1254	8.3	0.2	18,000	0.22	0.06	65	2	1.00E-06	70	25,550	2.9E-08	2.00E+00	6E-08
Aroclor-1260	11	0.2	18,000	0.22	0.06	65	2	1.00E-06	70	25,550	3.8E-08	2.00E+00	8E-08
Inorganic Chemicals													
Arsenic	18	0.2	18,000	0.22	0.03	65	2	1.00E-06	70	25,550	3.4E-08	1.50E+00	5E-08
Beryllium	1.4	0.2	18,000	0.22	0.25	65	2	1.00E-06	70	25,550	2.1E-08	4.30E+00	9E-08
Iron	31,804	0.2	18,000	0.22	0.01	65	2	1.00E-06	70	25,550	1.8E-05	NA	
Manganese	3,447	0.2	18,000	0.22	0.20	65	2	1.00E-06	70	25,550	4.0E-05	NA	
												Sum =	3E-07

Table H-8

Sector: Middle Buildings
Scenario: Future Indoor Construction
Pathway: Dermal Contact with Subsurface Soil
Receptor: Construction Worker (>18 years)

NONCARCINOGENIC
CDI = Cs x DAF x SA x FSA x RDA x EF x ED x CF x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	Cs Soil Concentration (mg/kg)	DAF Dermal Adherence Factor (mg/cm^2-d)	SA Total Body Surface Area (cm^2)	FSA Fraction SA Exposed (unitless)	RDA Relative Dermal Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals													
Acenaphthylene	0.13	0.2	18,000	0.22	0.10	65	2	1.00E-06	70	730	2.6E-08	NA	
Benzo(g,h,i)perylene	0.25	0.2	18,000	0.22	0.10	65	2	1.00E-06	70	730	5.0E-08	NA	
Phenanthrene	2.3	0.2	18,000	0.22	0.10	65	2	1.00E-06	70	730	4.6E-07	NA	
Pesticides/PCBs													
Aroclor-1242	2.4	0.2	18,000	0.22	0.06	65	2	1.00E-06	70	730	2.9E-07	NA	
Aroclor-1254	8.3	0.2	18,000	0.22	0.06	65	2	1.00E-06	70	730	1.0E-06	2.00E-05	5.0E-02
Aroclor-1260	11	0.2	18,000	0.22	0.06	65	2	1.00E-06	70	730	1.3E-06	NA	
Inorganic Chemicals													
Arsenic	18	0.2	18,000	0.22	0.03	65	2	1.00E-06	70	730	1.2E-06	3.00E-04	3.9E-03
Beryllium	1.4	0.2	18,000	0.22	0.25	65	2	1.00E-06	70	730	7.2E-07	5.00E-03	1.4E-04
Iron	31,604	0.2	18,000	0.22	0.01	65	2	1.00E-06	70	730	6.4E-04	3.00E-01	2.1E-03
Manganese	3,447	0.2	18,000	0.22	0.20	65	2	1.00E-06	70	730	1.4E-03	2.30E-02	6.0E-02
												Sum =	1.2E-01

Table H-9

Sector: Middle Buildings
Scenario: Future Indoor Construction
Pathway: Ingestion of Subsurface Soil
Receptor: Construction Worker (>18 years)

CARCINOGENIC
CDI = Cs x IgR x IgRF x ROA x EF x ED x CF x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	Cs Subsurface Soil Concentration (mg/kg)	IgR Soil Ingestion Rate (mg/d)	IgRF Fraction Ingested Attributable to Site (unitless)	ROA Relative Oral Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time Carcinogens (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals												
Acenaphthylene	0.13	163	1	1.0	65	2	1.00E-06	70	25,550	1.5E-09	NA	
Benzo(g,h,i)perylene	0.25	163	1	1.0	65	2	1.00E-06	70	25,550	3.0E-09	NA	
Phenanthrene	2.3	163	1	1.0	65	2	1.00E-06	70	25,550	2.7E-08	NA	
Pesticides/PCBs												
Aroclor-1242	2.4	163	1	1.0	65	2	1.00E-06	70	25,550	2.8E-08	2.00E+00	6E-08
Aroclor-1254	8.3	163	1	1.0	65	2	1.00E-06	70	25,550	9.8E-08	2.00E+00	2E-07
Aroclor-1260	11	163	1	1.0	65	2	1.00E-06	70	25,550	1.3E-07	2.00E+00	3E-07
Inorganic Chemicals												
Arsenic	18	163	1	1.0	65	2	1.00E-06	70	25,550	2.1E-07	1.50E+00	3E-07
Beryllium	1.4	163	1	1.0	65	2	1.00E-06	70	25,550	1.7E-08	4.30E+00	7E-08
Iron	31,604	163	1	1.0	65	2	1.00E-06	70	25,550	3.7E-04	NA	
Manganese	3,447	163	1	1.0	65	2	1.00E-06	70	25,550	4.1E-05	NA	
											Sum =	9E-07

Table H-10

Sector: Middle Buildings
Scenario: Future Indoor Construction
Pathway: Ingestion of Subsurface Soil
Receptor: Construction Worker (>18 years)

NONCARCINOGENIC
CDI = Cs x IgR x IgRF x ROA x EF x ED x CF x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	Cs Subsurface Soil Concentration (mg/kg)	IgR Soil Ingestion Rate (mg/d)	IgRF Fraction Ingested Attributable to Site (unitless)	ROA Relative Oral Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals												
Acenaphthylene	0.13	163	1	1.0	65	2	1.00E-06	70	730	5.4E-08	NA	
Benzo(g,h,i)perylene	0.25	163	1	1.0	65	2	1.00E-06	70	730	1.0E-07	NA	
Phenanthrene	2.3	163	1	1.0	65	2	1.00E-06	70	730	9.5E-07	NA	
Pesticides/PCBs												
Aroclor-1242	2.4	163	1	1.0	65	2	1.00E-06	70	730	1.0E-06	NA	
Aroclor-1254	8.3	163	1	1.0	65	2	1.00E-06	70	730	3.4E-06	2.00E-05	1.7E-01
Aroclor-1260	11	163	1	1.0	65	2	1.00E-06	70	730	4.5E-06	NA	
Inorganic Chemicals												
Arsenic	18	163	1	1.0	65	2	1.00E-06	70	730	7.5E-06	3.00E-04	2.5E-02
Beryllium	1.4	163	1	1.0	65	2	1.00E-06	70	730	5.9E-07	5.00E-03	1.2E-04
Iron	31,604	163	1	1.0	65	2	1.00E-06	70	730	1.3E-02	3.00E-01	4.4E-02
Manganese	3,347	163	1	1.0	65	2	1.00E-06	70	730	1.4E-03	2.30E-02	6.2E-02
											Sum =	3.0E-01

Table H-11

Sector: Middle Buildings
Scenario: Future Indoor Construction
Pathway: Inhalation of Dust and Vapor
Receptor: Construction Worker (>18 years)

CARCINOGENIC
CDI =((Cvs + Cvg) x IhR) + (Cs x Cp x CF1 x IhR x LDF x Ai)) x EF x ED x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	Cvs Vapor Concentration (mg/m^3)	Cvg Vapor Concentration (mg/m^3)	Cs Subsurface Soil Concentration (mg/kg)	Cp Particulate Concentration (mg/m^3)	IhR Inhalation Rate (m^3/day)	LDF Lung Deposition Fraction (unitless)	RIA Relative Inhalation Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals															
Acenaphthylene	1.0E-06	NA	0.13	5	20.0	0.125	1.0	65	2	1.00E-06	70	25,550	1.6E-09	NA	
Benzo(g,h,i)perylene	1.6E-10	NA	0.25	5	20.0	0.125	1.0	65	2	1.00E-06	70	25,550	2.3E-10	NA	
Phenanthrene	6.3E-06	NA	2.3	5	20.0	0.125	1.0	65	2	1.00E-06	70	25,550	1.1E-08	NA	
Pesticides/PCBs															
Aroclor-1242	8.2E-06	NA	2.4	5	20.0	0.125	1.0	65	2	1.00E-06	70	25,550	1.4E-08	2.00E+00	3E-08
Aroclor-1248	NA	7.6E-06	NA	5	20.0	0.125	1.0	65	2	1.00E-06	70	25,550	1.1E-08	2.00E+00	2E-08
Aroclor-1254	1.9E-05	8.5E-07	8.3	5	20.0	0.125	1.0	65	2	1.00E-06	70	25,550	3.6E-08	2.00E+00	7E-08
Aroclor-1260	9.3E-06	6.0E-07	11	5	20.0	0.125	1.0	65	2	1.00E-06	70	25,550	2.4E-08	2.00E+00	5E-08
Inorganic Chemicals															
Arsenic	NA	NA	18	5	20.0	0.125	1.0	65	2	1.00E-06	70	25,550	1.7E-08	1.50E+01	2E-07
Beryllium	NA	NA	1.4	5	20.0	0.125	1.0	65	2	1.00E-06	70	25,550	1.3E-09	8.40E+00	1E-08
Iron	NA	NA	31604	5	20.0	0.125	1.0	65	2	1.00E-06	70	25,550	2.9E-05	NA	
Manganese	NA	NA	3447	5	20.0	0.125	1.0	65	2	1.00E-06	70	25,550	3.1E-06	NA	
														Sum =	4E-07

Table H-12

Sector: Middle Buildings
Scenario: Future Indoor Construction
Pathway: Inhalation of Dust and Vapor
Receptor: Construction Worker (>18 years)

NONCARCINOGENIC
CDI =((Cvs + Cvg) x IhR) + (Cs x Cp x CF1 x IhR x LDF x Ai)) x EF x ED x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	Cvs Vapor Concentration (mg/m^3)	Cvg Vapor Concentration (mg/m^3)	Cs Subsurface Soil Concentration (mg/kg)	Cp Particulate Concentration (mg/m^3)	IhR Inhalation Rate (m^3/day)	LDF Lung Deposition Fraction (unitless)	RIA Relative Inhalation Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals															
Acenaphthylene	1.0E-06	NA	0.13	5	20.0	0.125	1.0	65	2	1.00E-06	70	730	5.8E-08	NA	
Benzo(g,h,i)perylene	1.6E-10	NA	0.25	5	20.0	0.125	1.0	65	2	1.00E-06	70	730	8.0E-09	NA	
Phenanthrene	6.3E-06	NA	2.3	5	20.0	0.125	1.0	65	2	1.00E-06	70	730	3.9E-07	NA	
Pesticides/PCBs															
Aroclor-1242	8.2E-06	NA	2.4	5	20.0	0.125	1.0	65	2	1.00E-06	70	730	4.9E-07	NA	
Aroclor-1248	NA	7.6E-06	NA	5	20.0	0.125	1.0	65	2	1.00E-06	70	730	3.9E-07	NA	
Aroclor-1254	1.9E-05	8.5E-07	8.3	5	20.0	0.125	1.0	65	2	1.00E-06	70	730	1.3E-06	NA	
Aroclor-1260	9.3E-06	6.0E-07	11	5	20.0	0.125	1.0	65	2	1.00E-06	70	730	8.5E-07	NA	
Inorganic Chemicals															
Arsenic	NA	NA	18	5	20.0	0.125	1.0	65	2	1.00E-06	70	730	5.8E-07	NA	
Beryllium	NA	NA	1.4	5	20.0	0.125	1.0	65	2	1.00E-06	70	730	4.5E-08	NA	
Iron	NA	NA	31604	5	20.0	0.125	1.0	65	2	1.00E-06	70	730	1.0E-03	NA	
Manganese	NA	NA	3447	5.0	20.000	0.1	1	65	2	1.00E-06	70	730	1.1OE-04	1.4E-05	7.7E+00
														Sum =	7.7E+00

Table H-13

Sector: Middle Buildings
Scenario: Future Outdoor Construction
Pathway: Dermal Contact with Subsurface Soil
Receptor: Construction Worker (>18 years)

CARCINOGENIC

CDI = Cs x DAF x SA x FSA x RDA x EF x ED x CF x 1/BW x 1/ATc

Risk = CDI x CSF

Chemical	Cs Subsurface Soil Concentration (mg/kg)	DAF Dermal Adherence Factor (mg/cm^2-d)	SA Total Body Surface Area (cm^2)	FSA Fraction SA Exposed (unitless)	RDA Relative Dermal Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals													
Acenaphthylene	0.13	0.2	18,000	0.22	0.10	195	1	1.00E-06	70	25,550	1.1E-09	NA	
Benzo(g,h,i)perylene	0.25	0.2	18,000	0.22	0.10	195	1	1.00E-06	70	25,550	2.2E-09	NA	
Phenanthrene	2.3	0.2	18,000	0.22	0.10	195	1	1.00E-06	70	25,550	2.0E-08	NA	
Pesticides/PCBs													
Aroclor-1242	2.4	0.2	18,000	0.22	0.06	195	1	1.00E-06	70	25,550	1.2E-08	2.00E+00	2E-08
Aroclor-1254	8.3	0.2	18,000	0.22	0.06	195	1	1.00E-06	70	25,550	4.3E-08	2.00E+00	9E-08
Aroclor-1260	11	0.2	18,000	0.22	0.06	195	1	1.00E-06	70	25,550	5.7E-08	2.00E+00	1E-07
Inorganic Chemicals													
Arsenic	18	0.2	18,000	0.22	0.032	195	1	1.00E-06	70	25,550	5.0E-08	1.50E+00	8E-08
Beryllium	1.4	0.2	18,000	0.22	0.25	195	1	1.00E-06	70	25,550	3.1E-08	4.30E+00	1E-07
Iron	31,604	0.2	18,000	0.22	0.01	195	1	1.00E-06	70	25,550	2.7E-05	NA	
Manganese	3,447	0.2	18,000	0.22	0.20	195	1	1.00E-06	70	25,550	6.0E-05	NA	
												Sum =	4E-07

Table H-14

Sector: Middle Buildings
Scenario: Future Outdoor Construction
Pathway: Dermal Contact with Subsurface Soil
Receptor: Construction Worker (>18 years)

NONCARCINOGENIC
CDI = Cs x DAF x SA x FSA x RDA x EF x ED x CF x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	Cs Subsurface Soil Concentration (mg/kg)	DAF Dermal Adherence Factor (mg/cm^2-d)	SA Total Body Surface Area (cm^2)	FSA Fraction SA Exposed (unitless)	RDA Relative Dermal Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals													
Acenaphthylene	0.13	0.2	18,000	0.22	0.10	195	1	1.00E-06	70	365	7.9E-08	NA	
Benzo(g,h,i)perylene	0.25	0.2	18,000	0.22	0.10	195	1	1.00E-06	70	365	1.5E-07	NA	
Phenanthrene	2.3	0.2	18,000	0.22	0.10	195	1	1.00E-06	70	365	1.4E-06	NA	
Pesticides/PCBs													
Aroclor-1242	2.4	0.2	18,000	0.22	0.06	195	1	1.00E-06	70	365	8.7E-07	NA	
Aroclor-1254	8.3	0.2	18,000	0.22	0.06	195	1	1.00E-06	70	365	3.0E-06	2.00E-05	1.5E-01
Aroclor-1260	11	0.2	18,000	0.22	0.06	195	1	1.00E-06	70	365	4.0E-06	NA	
Inorganic Chemicals													
Arsenic	18	0.2	18,000	0.22	0.032	195	1	1.00E-06	70	365	3.5E-06	3.00E-04	1.2E-02
Beryllium	1.4	0.2	18,000	0.22	0.25	195	1	1.00E-06	70	365	2.2E-06	5.00E-03	4.3E-04
Iron	31,604	0.2	18,000	0.22	0.01	195	1	1.00E-06	70	365	1.9E-03	3.00E-01	6.4E-03
Manganese	3,447	0.2	18,000	0.22	0.20	195	1	1.00E-06	70	365	4.2E-03	2.30E-02	1.8E-01
												Sum =	3.5E-01

Table H-15

Sector: Middle Buildings
Scenario: Future Outdoor Construction
Pathway: Ingestion of Subsurface Soil
Receptor: Construction Worker (>18 years)

CARCINOGENIC
CDI = Cs x IgR x IgRF x ROA x EF x ED x CF x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	Cs Subsurface Soil Concentration (mg/kg)	IgR Soil Ingestion Rate (mg/d)	IgRF Fraction Ingested Attributable to Site (unitless)	ROA Relative Oral Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals												
Acenaphthylene	0.13	125	1.0	1.0	195	1	1.00E-06	70	25,550	1.8E-09	NA	
Benzo(g,h,i)perylene	0.25	125	1.0	1.0	195	1	1.00E-06	70	25,550	3.4E-09	NA	
Phenanthrene	2.3	125	1.0	1.0	195	1	1.00E-06	70	25,550	3.1E-08	NA	
Pesticides/PCBs												
Aroclor-1242	2.4	125	1.0	1.0	195	1	1.00E-06	70	25,550	3.3E-08	2.00E+00	7E-08
Aroclor-1254	8.3	125	1.0	1.0	195	1	1.00E-06	70	25,550	1.1E-07	2.00E+00	2E-07
Aroclor-1260	11	125	1.0	1.0	195	1	1.00E-06	70	25,550	1.5E-07	2.00E+00	3E-07
Inorganic Chemicals												
Arsenic	18	125	1.0	1.0	195	1	1.00E-06	70	25,550	2.5E-07	1.50E+00	4E-07
Beryllium	1.4	125	0.04	1.0	195	1	1.00E-06	70	25,550	7.8E-10	4.30E+00	3E-09
Iron	31,604	125	0.04	1.0	195	1	1.00E-06	70	25,550	1.7E-05	NA	
Manganese	3,447	125	0.05	1.0	195	1	1.00E-06	70	25,550	2.3E-06	NA	
											Sum =	1E-06

Table H-16

Sector: Middle Buildings
Scenario: Future Outdoor Construction
Pathway: Ingestion of Subsurface Soil
Receptor: Construction Worker (>18 years)

NONCARCINOGENIC
CDI = Cs x IgR x IgRF x ROA x EF x ED x CF x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	Cs Subsurface Soil Concentration (mg/kg)	IgR Sediment Ingestion Rate (mg/d)	IgRF Fraction Ingested Attributable to Site (unitless)	ROA Relative Oral Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals												
Acenaphthylene	0.13	125	1.0	1.0	195	1	1.00E-06	70	365	1.2E-07	NA	
Benzo(g,h,i)perylene	0.25	125	1.0	1.0	195	1	1.00E-06	70	365	2.4E-07	NA	
Phenanthrene	2.3	125	1.0	1.0	195	1	1.00E-06	70	365	2.2E-06	NA	
Pesticides/PCBs												
Aroclor-1242	2.4	125	1.0	1.0	195	1	1.00E-06	70	365	2.3E-06	NA	
Aroclor-1254	8.3	125	1.0	1.0	195	1	1.00E-06	70	365	7.9E-06	2.00E-05	4.0E-01
Aroclor-1260	11	125	1.0	1.0	195	1	1.00E-06	70	365	1.0E-05	NA	
Inorganic Chemicals												
Arsenic	18	125	1.0	1.0	195	1	1.00E-06	70	365	1.7E-05	3.00E-04	5.8E-02
Beryllium	1.4	125	0.04	1.0	195	1	1.00E-06	70	365	5.5E-08	5.00E-03	1.1E-05
Iron	31,604	125	0.04	1.0	195	1	1.00E-06	70	365	1.2E-03	3.00E-01	4.0E-03
Manganese	3,447	125	0.05	1.0	195	1	1.00E-06	70	365	1.6E-04	2.30E-02	7.1E-03
											Sum =	4.7E-01

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Table H-17

Sector: Middle Buildings
Scenario: Future Outdoor Construction
Pathway: Inhalation of Dust and Vapor[a]
Receptor: Construction Worker (>18 years)

CARCINOGENIC

$CDI = ((Cvs + Cvg) \times IhR) + (Cs \times Cp \times CF1 \times IhR \times LDF)) \times RIA \times EF \times ED \times 1/BW \times 1/ATc$

$Risk = CDI \times CSF$

Chemical	Cvs Vapor Concentration (mg/m^3)	Cvg Vapor Concentration (mg/m^3)	Cs Subsurface Soil Concentration (mg/kg)	Cp Particulate Concentration (mg/m^3)	IhR Inhalation Rate (m^3/day)	LDF Lung Deposition Fraction (unitless)	RIA Relative Inhalation Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Volatile Organics															
Vinyl Chloride	NA	1.0E-06	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	2.3E-09	3.00E-01	7E-10
1,1-Dichloroethene	NA	1.2E-07	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	2.7E-10	1.75E-01	5E-11
1,2-Dichloroethene (total)	NA	5.5E-07	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	1.2E-09	NA	
1,2-Dichloroethane	NA	3.6E-07	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	7.9E-10	9.10E-02	7E-11
1,1,1-Trichloroethane	NA	3.8E-06	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	8.4E-09	NA	
Trichloroethene	NA	2.1E-04	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	4.6E-07	6.00E-03	3E-09
Benzene	NA	1.9E-05	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	4.1E-08	2.90E-02	1E-09
Tetrachloroethene	NA	1.4E-06	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	3.0E-09	2.03E-03	6E-12
Chlorobenzene	NA	1.7E-05	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	3.8E-08	NA	
Semivolatile Organics															
1,3-Dichlorobenzene	NA	2.4E-05	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	5.2E-08	NA	
1,4-Dichlorobenzene	NA	1.6E-05	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	3.6E-08	NA	
1,2-Dichlorobenzene	NA	4.2E-05	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	9.1E-08	NA	
1,2,4-Trichlorobenzene	NA	1.5E-03	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	3.4E-06	NA	
2,4-Dichlorophenol	NA	NA	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	NA	NA	
Acenaphthylene	5.3E-07	NA	0.13	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	1.2E-09	NA	
Benzo(g,h,i)perylene	1.5E-10	NA	0.25	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	1.4E-10	NA	
Phenanthrene	5.6E-05	NA	2.3	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	1.2E-07	NA	
PCBs/Pesticides															
Aroclor-1242	7.7E-05	1.6E-07	2.4	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	1.7E-07	2.00E+00	3E-07
Aroclor-1248	NA	8.6E-04	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	1.9E-06	2.00E+00	4E-06
Aroclor-1254	6.1E-04	1.3E-03	8.3	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	4.2E-06	2.00E+00	8E-06
Aroclor-1260	4.0E-04	1.4E-07	11	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	8.8E-07	2.00E+00	2E-06
Inorganic Chemicals [b]															
Arsenic	NA	NA	18	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	9.9E-09	1.50E+01	1E-07
Beryllium	NA	NA	1.4	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	7.8E-10	8.40E+00	7E-09
Lead	NA	NA	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	NA	NA	
Iron	NA	NA	31,604	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	1.7E-05	NA	
Manganese	NA	NA	3,447	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	1.9E-06	NA	
Mercury	NA	NA	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	NA	NA	
														Sum =	1E-05

Notes:

a - Table H-17a summarizes the risks using EPA Region III's computed groundwater-specific vapor concentration (Cvg) and PM-10 concentration (Cp) of 0.082 mg/m^3. See Appendix I for EPA's PM-10 computation.

b - Although several metals were defined as COPCs for the Alluvial Central and Southern groundwater, they were not included in this assessment because they are not volatile.

Table H-17a

Sector: Middle Buildings
Scenario: Future Outdoor Construction
Pathway: Inhalation of Dust and Vapor[a]
Receptor: Construction Worker (>18 years)

CARCINOGENIC

$CDI = ((Cvs + Cvg) \times IhR) + (Cs \times Cp \times CF1 \times IhR \times LDF) \times RIA \times EF \times ED \times 1/BW \times 1/ATc$

Risk = CDI x CSF

Chemical	Cvs Vapor Concentration (mg/m^3)	Cvg Vapor Concentration (mg/m^3)	Cs Subsurface Soil Concentration (mg/kg)	Cp Particulate Concentration (mg/m^3)	IhR Inhalation Rate (m^3/day)	LDF Lung Deposition Fraction (unitless)	RIA Relative Inhalation Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Volatile Organics															
Vinyl Chloride	NA	4.8E-07	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	1.0E-09	3.00E-01	3E-10
1,1-Dichloroethene	NA	5.6E-08	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	1.2E-10	1.75E-01	2E-11
1,2-Dichloroethene (total)	NA	2.5E-07	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	5.5E-10	NA	
1,2-Dichloroethane	NA	1.7E-07	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	3.6E-10	9.10E-02	3E-11
1,1,1-Trichloroethane	NA	1.8E-06	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	3.8E-09	NA	
Trichloroethene	NA	9.6E-05	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	2.1E-07	6.00E-03	1E-09
Benzene	NA	8.5E-06	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	1.9E-08	2.90E-02	5E-10
Tetrachloroethene	NA	6.2E-07	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	1.4E-09	2.03E-03	3E-12
Chlorobenzene	NA	8.0E-06	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	1.7E-08	NA	
Semivolatile Organics															
1,3-Dichlorobenzene	NA	1.1E-05	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	2.4E-08	NA	
1,4-Dichlorobenzene	NA	7.5E-06	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	1.6E-08	NA	
1,2-Dichlorobenzene	NA	1.9E-05	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	4.2E-08	NA	
1,2,4-Trichlorobenzene	NA	7.0E-04	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	1.5E-06	NA	
2,4-Dichlorophenol	NA	NA	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	NA	NA	
Acenaphthylene	1.9E-06	NA	0.13	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	4.1E-09	NA	
Benzo(g,h,i)perylene	2.8E-10	NA	0.25	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	6.2E-12	NA	
Phenanthrene	1.1E-05	NA	2.3	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	2.4E-08	NA	
PCBs/Pesticides															
Aroclor-1242	1.5E-05	7.1E-08	2.4	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	3.3E-08	2.00E+00	7E-08
Aroclor-1248	NA	3.9E-04	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	8.6E-07	2.00E+00	2E-06
Aroclor-1254	3.4E-05	6.0E-04	8.3	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	1.4E-06	2.00E+00	3E-06
Aroclor-1260	1.7E-05	6.4E-08	11	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	3.7E-08	2.00E+00	7E-08
Inorganic Chemicals [b]															
Arsenic	NA	NA	18	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	4.1E-10	1.50E+01	6E-09
Beryllium	NA	NA	1.4	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	3.2E-11	8.40E+00	3E-10
Lead	NA	NA	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	NA	NA	
Iron	NA	NA	31,604	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	7.1E-07	NA	
Manganese	NA	NA	3,447	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	7.7E-08	NA	
Mercury	NA	NA	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	25,550	NA	NA	
Notes:														Sum =	5E-06

a - Table H-17a summarizes the risks using EPA's groundwater-and soil-specific vapor concentration (Cvg and Cvs) and PM-10 concentration (Cp) of 0.082 mg/m^3. See Appendix I for EPA's PM-10 computation.

b - Although several metals were defined as COPCs for the Alluvial Central and Southern groundwater, they were not included in this assessment because they are not volatile.

Table H-18

Sector: Middle Buildings
Scenario: Future Outdoor Construction
Pathway: Inhalation of Dust and Vapor[a]
Receptor: Construction Worker (>18 years)

NONCARCINOGENIC
CDI =((Cvs + Cvg) x IhR) + (Cs x Cp x CF1 x IhR x LDF))x RIA x EF x ED x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	Cvs Vapor Concentration (mg/m^3)	Cvg Vapor Concentration (mg/m^3)	Cs Subsurface Soil Concentration (mg/kg)	Cp[a] Particulate Concentration (mg/m^3)	IhR Inhalation Rate (m^3/day)	LDF Lung Deposition Fraction (unitless)	RIA Relative Inhalation Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Volatile Organics															
Vinyl Chloride	NA	1.0E-06	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	1.6E-07	NA	
1,1-Dichloroethene	NA	1.2E-07	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	1.9E-08	NA	
1,2-Dichloroethene (total)	NA	5.5E-07	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	8.4E-08	NA	
1,2-Dichloroethane	NA	3.6E-07	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	5.5E-08	2.86E-03	1.9E-05
1,1,1-Trichloroethane	NA	3.8E-06	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	5.9E-07	2.86E-01	2.0E-06
Trichloroethene	NA	2.1E-04	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	3.2E-05	NA	
Benzene	NA	1.9E-05	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	2.8E-06	1.71E-03	1.7E-03
Tetrachloroethene	NA	1.4E-06	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	2.1E-07	NA	
Chlorobenzene	NA	1.7E-05	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	2.7E-06	5.71E-03	4.7E-04
Semivolatile Organics															
1,3-Dichlorobenzene	NA	2.4E-05	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	3.6E-06	NA	
1,4-Dichlorobenzene	NA	1.6E-05	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	2.5E-06	2.29E-01	1.1E-05
1,2-Dichlorobenzene	NA	4.2E-05	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	6.4E-06	4.00E-02	1.6E-04
1,2,4-Trichlorobenzene	NA	1.5E-03	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	2.4E-04	5.71E-02	4.1E-03
2,4-Dichlorophenol	NA	NA	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	NA	NA	
Acenaphthylene	5.3E-07	NA	0.13	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	8.6E-08	NA	
Benzo(g,h,i)perylene	1.5E-10	NA	0.25	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	9.6E-09	NA	
Phenanthrene	5.6E-05	NA	2.3	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	8.6E-06	NA	
PCBs/Pesticides															
Aroclor-1242	7.7E-05	1.6E-07	2.4	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	1.2E-05	NA	
Aroclor-1248	NA	8.6E-04	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	1.3E-04	NA	
Aroclor-1254	6.1E-04	1.3E-03	8.3	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	3.0E-04	NA	
Aroclor-1260	4.0E-04	1.4E-07	11	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	6.1E-05	NA	
Inorganic Chemicals[b]															
Arsenic	NA	NA	18	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	6.9E-07	NA	
Beryllium	NA	NA	1.4	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	5.5E-08	NA	
Iron	NA	NA	31604	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	1.2E-03	NA	
Manganese	NA	NA	3447	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	1.3E-04	1.43E-05	9.2E+00
Mercury	NA	NA	NA	2	20.0	0.125	1.0	195	1	1.00E-06	70	365	NA	8.60E-05	
														Sum =	9.2E+00

Notes:
a - Table H-18a summarize the hazards using EPA Region III's computed groundwater-specific vapor concentration (Cvg) and PM-10 concentration (Cp) of 0.082 mg/m^3. See Appendix I for EPA's PM-10 computation.
b - Although several metals were defined as COPCs for the Alluvial Central and Southern groundwater, they were not included in this assessment because they are not volatile.

G:\CL[illegible]\FINALCALC\FINAL[illegible]\Middle Buildings

Table H-18a

Sector: Middle Buildings
Scenario: Future Outdoor Construction
Pathway: Inhalation of Dust and Vapor[a]
Receptor: Construction Worker (>18 years)

NONCARCINOGENIC

CDI =((Cvs + Cvg) x InR) + (Cs x Cp x CF1 x InR x LDF))x RIA x EF x ED x 1/BW x 1/ATnc

HQ = CDI/RfD

Chemical	Cvs Vapor Concentration (mg/m^3)	Cvg Vapor Concentration (mg/m^3)	Cs Subsurface Soil Concentration (mg/kg)	Cp Particulate Concentration (mg/m^3)	InR Inhalation Rate (m^3/day)	LDF Lung Deposition Fraction (unitless)	RIA Relative Inhalation Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Volatile Organics															
Vinyl Chloride	NA	4.8E-07	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	7.3E-08	NA	
1,1-Dichloroethene	NA	5.6E-08	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	8.5E-09	NA	
1,2-Dichloroethene (total)	NA	2.5E-07	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	3.9E-08	NA	
1,2-Dichloroethane	NA	1.7E-07	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	2.5E-08	2.86E-03	8.9E-06
1,1,1-Trichloroethane	NA	1.8E-06	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	2.7E-07	2.86E-01	9.3E-07
Trichloroethene	NA	9.6E-05	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	1.5E-05	NA	
Benzene	NA	8.5E-06	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	1.3E-06	1.71E-03	7.6E-04
Tetrachloroethene	NA	6.2E-07	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	9.5E-08	NA	
Chlorobenzene	NA	8.0E-06	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	1.2E-06	5.71E-03	2.1E-04
Semivolatile Organics															
1,3-Dichlorobenzene	NA	1.1E-05	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	1.7E-06	NA	
1,4-Dichlorobenzene	NA	7.5E-06	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	1.1E-06	2.29E-01	5.0E-06
1,2-Dichlorobenzene	NA	1.9E-05	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	2.9E-06	4.00E-02	7.3E-05
1,2,4-Trichlorobenzene	NA	7.0E-04	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	1.1E-04	5.71E-02	1.9E-03
2,4-Dichlorophenol	NA	NA	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	NA	NA	
Acenaphthylene	1.9E-06	NA	0.13	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	2.9E-07	NA	
Benzo(g,h,i)perylene	2.8E-10	NA	0.25	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	4.3E-10	NA	
Phenanthrene	1.1E-05	NA	2.3	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	1.7E-06	NA	
PCBs/Pesticides															
Aroclor-1242	1.5E-05	7.1E-08	2.4	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	2.3E-06	NA	
Aroclor-1248	NA	3.9E-04	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	6.0E-05	NA	
Aroclor-1254	3.4E-05	6.0E-04	8.3	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	9.7E-05	NA	
Aroclor-1260	1.7E-05	6.4E-08	11	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	2.6E-06	NA	
Inorganic Chemicals[b]															
Arsenic	NA	NA	18	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	2.8E-08	NA	
Beryllium	NA	NA	1.4	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	2.2E-09	NA	
Iron	NA	NA	31604	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	4.9E-05	NA	
Manganese	NA	NA	3447	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	5.4E-06	1.43E-05	3.8E-01
Mercury	NA	NA	NA	0.082	20.0	0.125	1.0	195	1	1.00E-06	70	365	NA	8.60E-05	
														Sum =	3.8E-01

Notes:

a - Table H-18a summarizes the risks using EPA's groundwater-and soil-specific vapor concentration (Cvg and Cvs) and PM-10 concentration (Cp) of 0.082 mg/m^3. See Appendix I for EPA's PM-10 computation.

b - Although several metals were defined as COPCs for the Alluvial Central and Southern groundwater, they were not included in this assessment because they are not volatile.

Table H-19

Sector: Moat
Scenario: Maintenance
Pathway: Dermal Contact with Surface Soil
Receptor: Maintenance Worker (>18 years)

CARCINOGENIC

CDI = Cs x DAF x SA x FSA x RDA x EF x ED x CF x 1/BW x 1/ATc

Risk = CDI x CSF

Chemical	Cs Surface Soil Concentration (mg/kg)	DAF Dermal Adherence Factor (mg/cm^2-d)	SA Total Body Surface Area (cm^2)	FSA Fraction SA Exposed (unitless)	RDA Relative Dermal Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals													
Acenaphthylene	0.0	0.2	18,000	0.14	0.10	7	25	1.00E-06	70	25,550	3.9E-11	NA	
Benzo(g,h,i)perylene	0.16	0.2	18,000	0.14	0.10	7	25	1.00E-06	70	25,550	7.9E-10	NA	
Phenanthrene	0.25	0.2	18,000	0.14	0.10	7	25	1.00E-06	70	25,550	1.2E-09	NA	
Benzo(a)pyrene	0.32	0.2	18,000	0.14	0.10	7	25	1.00E-06	70	25,550	1.6E-09	7.30E+00	1E-08
Pesticides/PCBs													
Aroclor-1248	120	0.2	18,000	0.14	0.06	7	25	1.00E-06	70	25,550	3.6E-07	2.00E+00	7E-07
Aroclor-1254	81	0.2	18,000	0.14	0.06	7	25	1.00E-06	70	25,550	2.4E-07	2.00E+00	5E-07
Aroclor-1260	58	0.2	18,000	0.14	0.06	7	25	1.00E-06	70	25,550	1.7E-07	2.00E+00	3E-07
Dieldrin	0.055	0.2	18,000	0.14	0.06	7	25	1.00E-06	70	25,550	1.6E-10	1.60E+01	3E-09
Inorganics													
Aluminum	8,040	0.2	18,000	0.14	0.01	7	25	1.00E-06	70	25,550	4.0E-06	NA	
Arsenic	102	0.2	18,000	0.14	0.032	7	25	1.00E-06	70	25,550	1.6E-07	1.50E+00	2E-07
Beryllium	1.4	0.2	18,000	0.14	0.25	7	25	1.00E-06	70	25,550	1.7E-08	4.30E+00	7E-08
Cadmium	4.0	0.2	18,000	0.14	0.01	7	25	1.00E-06	70	25,550	2.0E-09	NA	
Iron	33,900	0.2	18,000	0.14	0.01	7	25	1.00E-06	70	25,550	1.7E-05	NA	
Manganese	799	0.2	18,000	0.14	0.2	7	25	1.00E-06	70	25,550	7.9E-06	NA	
Mercury	1.2	0.2	18,000	0.14	0.01	7	25	1.00E-06	70	25,550	5.9E-10	NA	
Thallium	1.4	0.2	18,000	0.14	0.01	7	25	1.00E-06	70	25,550	6.9E-10	NA	
												Sum =	2E-06

Table H-20

Sector: Most
Scenario: Maintenance
Pathway: Dermal Contact with Surface Soil
Receptor: Maintenance Worker (>18 years)

NONCARCINOGENIC
CDI = Cs x DAF x SA x FSA x RDA x EF x ED x CF x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	Cs Surface Soil Concentration (mg/kg)	DAF Dermal Adherance Factor (mg/cm^2-d)	SA Total Body Surface Area (cm^2)	FSA Fraction SA Exposed (unitless)	RDA Relative Dermal Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals													
Acenaphthylene	0.0080	0.2	18,000	0.14	0.10	7	25	1.00E-06	70	9,125	1.1E-10	NA	
Benzo(g,h,i)perylene	0.16	0.2	18,000	0.14	0.10	7	25	1.00E-06	70	9,125	2.2E-09	NA	
Phenanthrene	0.25	0.2	18,000	0.14	0.10	7	25	1.00E-06	70	9,125	3.5E-09	NA	
Benzo(a)pyrene	0.32	0.2	18,000	0.14	0.10	7	25	1.00E-06	70	9,125	4.4E-09	NA	
Pesticides/PCBs													
Aroclor-1248	120	0.2	18,000	0.14	0.06	7	25	1.00E-06	70	9,125	9.9E-07	NA	
Aroclor-1254	81	0.2	18,000	0.14	0.06	7	25	1.00E-06	70	9,125	6.7E-07	2.00E-05	3.4E-02
Aroclor-1260	58	0.2	18,000	0.14	0.06	7	25	1.00E-06	70	9,125	4.8E-07	NA	
Dieldrin	0.055	0.2	18,000	0.14	0.06	7	25	1.00E-06	70	9,125	4.6E-10	5.00E-05	9.1E-06
Inorganics													
Aluminum	8,040	0.2	18,000	0.14	0.01	7	25	1.00E-06	70	9,125	1.1E-05	1.00E+00	1.1E-05
Arsenic	102	0.2	18,000	0.14	0.032	7	25	1.00E-06	70	9,125	4.5E-07	3.00E-04	1.5E-03
Beryllium	1.4	0.2	18,000	0.14	0.25	7	25	1.00E-06	70	9,125	4.8E-08	5.00E-03	9.7E-06
Cadmium	4.0	0.2	18,000	0.14	0.01	7	25	1.00E-06	70	9,125	5.5E-09	5.00E-04	1.1E-05
Iron	33,900	0.2	18,000	0.14	0.01	7	25	1.00E-06	70	9,125	4.7E-05	3.00E-01	1.6E-04
Manganese	799	0.2	18,000	0.14	0.2	7	25	1.00E-06	70	9,125	2.2E-05	2.30E-02	9.6E-04
Mercury	1.2	0.2	18,000	0.14	0.01	7	25	1.00E-06	70	9,125	1.7E-09	1.00E-04	1.7E-05
Thallium	1.4	0.2	18,000	0.14	0.01	7	25	1.00E-06	70	9,125	1.9E-09	8.00E-05	2.4E-05
												Sum =	3.6E-02

Table H-21

Sector: Moat
Scenario: Maintenance
Pathway: Ingestion of Surface Soil
Receptor: Maintenance Worker (>18 years)

CARCINOGENIC
CDI = Cs x IgR x IgRF x ROA x EF x ED x CF x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	Cs Surface Soil Concentration (mg/kg)	IgR Soil Ingestion Rate (mg/d)	IgRF Fraction Ingested Attributable to Site (unitless)	ROA Relative Oral Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals												
Acenaphthylene	0.0	100	1	1.0	7	25	1.00E-06	70	25,550	7.8E-11	NA	
Benzo(g,h,i)perylene	0.16	100	1	1.0	7	25	1.00E-06	70	25,550	1.6E-09	NA	
Phenanthrene	0.25	100	1	1.0	7	25	1.00E-06	70	25,550	2.4E-09	NA	
Benzo(a)pyrene	0.32	100	1	1.0	7	25	1.00E-06	70	25,550	3.1E-09	7.30E+00	2E-08
Pesticides/PCBs												
Aroclor-1248	120	100	1	1.0	7	25	1.00E-06	70	25,550	1.2E-06	2.00E+00	2E-06
Aroclor-1254	81	100	1	1.0	7	25	1.00E-06	70	25,550	7.9E-07	2.00E+00	2E-06
Aroclor-1260	58	100	1	1.0	7	25	1.00E-06	70	25,550	5.7E-07	2.00E+00	1E-06
Dieldrin	0.055	100	1	1.0	7	25	1.00E-06	70	25,550	5.4E-10	1.60E+01	9E-09
Inorganics												
Aluminum	8,040	100	1	1.0	7	25	1.00E-06	70	25,550	7.9E-05	NA	
Arsenic	102	100	1	1.0	7	25	1.00E-06	70	25,550	1.0E-06	1.50E+00	1E-06
Beryllium	1.4	100	1	1.0	7	25	1.00E-06	70	25,550	1.4E-08	4.30E+00	6E-08
Cadmium	4.0	100	1	1.0	7	25	1.00E-06	70	25,550	3.9E-08	NA	
Iron	33,900	100	1	1.0	7	25	1.00E-06	70	25,550	3.3E-04	NA	
Manganese	799	100	1	1.0	7	25	1.00E-06	70	25,550	7.8E-06	NA	
Mercury	1.2	100	1	1.0	7	25	1.00E-06	70	25,550	1.2E-08	NA	
Thallium	1.4	100	1	1.0	7	25	1.00E-06	70	25,550	1.4E-08	NA	
											Sum =	7E-06

Table H-22

Sector: Mont
Scenario: Maintenance
Pathway: Ingestion of Surface Soil
Receptor: Maintenance Worker (>18 years)

NONCARCINOGENIC
CDI = Cs x IgR x IgRF x ROA x EF x ED x CF x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	Cs Surface Soil Concentration (mg/kg)	IgR Soil Ingestion Rate (mg/d)	IgRF Fraction Ingested Attributable to Site (unitless)	ROA Relative Oral Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals												
Acenaphthylene	0.0080	100	1	1.0	7	25	1.00E-06	70	9,125	2.2E-10	NA	
Benzo(g,h,i)perylene	0.16	100	1	1.0	7	25	1.00E-06	70	9,125	4.4E-09	NA	
Phenanthrene	0.25	100	1	1.0	7	25	1.00E-06	70	9,125	6.8E-09	NA	
Benzo(a)pyrene	0.32	100	1	1.0	7	25	1.00E-06	70	9,125	8.8E-09	NA	
Pesticides/PCBs												
Aroclor-1248	120	100	1	1.0	7	25	1.00E-06	70	9,125	3.3E-06	NA	
Aroclor-1254	81	100	1	1.0	7	25	1.00E-06	70	9,125	2.2E-06	2.00E-05	1.1E-01
Aroclor-1260	58	100	1	1.0	7	25	1.00E-06	70	9,125	1.6E-06	NA	
Dieldrin	0.055	100	1	1.0	7	25	1.00E-06	70	9,125	1.5E-09	5.00E-05	3.0E-05
Inorganics												
Aluminum	8,040	100	1	1.0	7	25	1.00E-06	70	9,125	2.2E-04	1.00E+00	2.2E-04
Arsenic	102	100	1	1.0	7	25	1.00E-06	70	9,125	2.8E-06	3.00E-04	9.3E-03
Beryllium	1.4	100	1	1.0	7	25	1.00E-06	70	9,125	3.8E-08	5.00E-03	7.7E-06
Cadmium	4.0	100	1	1.0	7	25	1.00E-06	70	9,125	1.1E-07	5.00E-04	2.2E-04
Iron	33,900	100	1	1.0	7	25	1.00E-06	70	9,125	9.3E-04	3.00E-01	3.1E-03
Manganese	799	100	1	1.0	7	25	1.00E-06	70	9,125	2.2E-05	2.30E-02	9.5E-04
Mercury	1.2	100	1	1.0	7	25	1.00E-06	70	9,125	3.3E-08	1.00E-04	3.3E-04
Thallium	1.4	100	1	1.0	7	25	1.00E-06	70	9,125	3.8E-08	8.00E-05	4.8E-04
											Sum =	1.3E-01

Table H-23

Sector: Most
Scenario: Maintenance
Pathway: Inhalation of Soil Particulates
Receptor: Maintenance Worker (>18 years)

CARCINOGENIC
CDI = Cs x (1/PEF) x InR x LDF x RIA x EF x ED x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	Cs Surface Soil Concentration (mg/kg)	PEF Particulate Emission Factor (m^3/kg)	InR Inhalation Rate (m^3/day)	LDF Lung Deposition Fraction (unitless)	RIA Relative Inhalation Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals												
Acenaphthylene	0.0080	1.30E+09	20	0.125	1.0	7	25	70	25,550	1.5E-15	NA	
Benzo(g,h,i)perylene	0.16	1.30E+09	20	0.125	1.0	7	25	70	25,550	3.0E-14	NA	
Phenanthrene	0.25	1.30E+09	20	0.125	1.0	7	25	70	25,550	4.7E-14	NA	
Benzo(a)pyrene	0.32	1.30E+09	20	0.125	1.0	7	25	70	25,550	6.0E-14	6.10E+00	4E-13
Pesticides/PCBs												
Aroclor-1248	120	1.30E+09	20	0.125	1.0	7	25	70	25,550	2.3E-11	2.00E+00	5E-11
Aroclor-1254	81	1.30E+09	20	0.125	1.0	7	25	70	25,550	1.5E-11	2.00E+00	3E-11
Aroclor-1260	58	1.30E+09	20	0.125	1.0	7	25	70	25,550	1.1E-11	2.00E+00	2E-11
Dieldrin	0.055	1.30E+09	20	0.125	1.0	7	25	70	25,550	1.0E-14	1.61E+01	2E-13
Inorganics												
Aluminum	8,040	1.30E+09	20	0.125	1.0	7	25	70	25,550	1.5E-09	NA	
Arsenic	102	1.30E+09	20	0.125	1.0	7	25	70	25,550	1.9E-11	1.50E+01	3E-10
Beryllium	1.4	1.30E+09	20	0.125	1.0	7	25	70	25,550	2.6E-13	8.40E+00	2E-12
Cadmium	4.0	1.30E+09	20	0.125	1.0	7	25	70	25,550	7.5E-13	6.30E+00	5E-12
Iron	33,900	1.30E+09	20	0.125	1.0	7	25	70	25,550	6.4E-09	NA	
Manganese	799	1.30E+09	20	0.125	1.0	7	25	70	25,550	1.5E-10	NA	
Mercury	1.2	1.30E+09	20	0.125	1.0	7	25	70	25,550	2.3E-13	NA	
Thallium	1.4	1.30E+09	20	0.125	1.0	7	25	70	25,550	2.6E-13	NA	
											Sum =	4E-10

Table H-24

Sector: Mont
Scenario: Maintenance
Pathway: Inhalation of Soil Particulates
Receptor: Maintenance Worker (>18 years)

NONCARCINOGENIC

$CDI = Cs \times (1/PEF) \times IhR \times LDF \times RIA \times EF \times ED \times 1/BW \times 1/ATc$

$HQ = CDI/RfD$

Chemical	Cs Surface Soil Concentration (mg/kg)	PEF Particulate Emission Factor (m^3/kg)	IhR Inhalation Rate (m^3/day)	LDF Lung Deposition Fraction (unitless)	RIA Relative Inhalation Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals												
Acenaphthylene	0.0080	1.30E+09	20	0.125	1.0	7	25	70	9,125	4.2E-15	NA	
Benzo(g,h,i)perylene	0.16	1.30E+09	20	0.125	1.0	7	25	70	9,125	8.4E-14	NA	
Phenanthrene	0.25	1.30E+09	20	0.125	1.0	7	25	70	9,125	1.3E-13	NA	
Benzo(a)pyrene	0.32	1.30E+09	20	0.125	1.0	7	25	70	9,125	1.7E-13	NA	
Pesticides/PCBs												
Aroclor-1248	120	1.30E+09	20	0.125	1.0	7	25	70	9,125	6.3E-11	NA	
Aroclor-1254	81	1.30E+09	20	0.125	1.0	7	25	70	9,125	4.3E-11	NA	
Aroclor-1260	58	1.30E+09	20	0.125	1.0	7	25	70	9,125	3.1E-11	NA	
Dieldrin	0.055	1.30E+09	20	0.125	1.0	7	25	70	9,125	2.9E-14	NA	
Inorganics												
Aluminum	8,040	1.30E+09	20	0.125	1.0	7	25	70	9,125	4.2E-09	NA	
Arsenic	102	1.30E+09	20	0.125	1.0	7	25	70	9,125	5.4E-11	NA	
Beryllium	1.4	1.30E+09	20	0.125	1.0	7	25	70	9,125	7.4E-13	NA	
Cadmium	4.0	1.30E+09	20	0.125	1.0	7	25	70	9,125	2.1E-12	5.70E-05	3.7E-08
Iron	33,900	1.30E+09	20	0.125	1.0	7	25	70	9,125	1.8E-08	NA	
Manganese	799	1.30E+09	20	0.125	1.0	7	25	70	9,125	4.2E-10	1.43E-05	2.9E-05
Mercury	1.2	1.30E+09	20	0.125	1.0	7	25	70	9,125	6.3E-13	8.60E-05	7.4E-09
Thallium	1.4	1.30E+09	20	0.125	1.0	7	25	70	9,125	7.4E-13	NA	
											Sum =	2.9E-05

Table H-25

Sector: Moat
Scenario: Trespasser
Pathway: Dermal Contact with Surface Soil
Receptor: Child (7 to 12 years)

CARCINOGENIC

CDI = Cs x DAF x SA x FSA x RDA x EF x ED x CF x 1/BW x 1/ATc

Risk = CDI x CSF

Chemical	Cs Surface Soil Concentration (mg/kg)	DAF Dermal Adherence Factor (mg/cm^2-d)	SA Total Body Surface Area (cm^2)	FSA Fraction SA Exposed (unitless)	RDA Relative Dermal Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals													
Acenaphthylene	0.0080	0.2	10,500	0.22	0.10	4	6	1.00E-06	33	25,550	1.1E-11	NA	
Benzo(g,h,i)perylene	0.16	0.2	10,500	0.22	0.10	4	6	1.00E-06	33	25,550	2.1E-10	NA	
Phenanthrene	0.25	0.2	10,500	0.22	0.10	4	6	1.00E-06	33	25,550	3.3E-10	NA	
Benzo(a)pyrene	0.32	0.2	10,500	0.22	0.10	4	6	1.00E-06	33	25,550	4.2E-10	7.30E+00	3E-09
Pesticides/PCBs													
Aroclor-1248	120	0.2	10,500	0.22	0.06	4	6	1.00E-06	33	25,550	9.5E-08	2.00E+00	2E-07
Aroclor-1254	81	0.2	10,500	0.22	0.06	4	6	1.00E-06	33	25,550	6.4E-08	2.00E+00	1E-07
Aroclor-1260	58	0.2	10,500	0.22	0.06	4	6	1.00E-06	33	25,550	4.6E-08	2.00E+00	9E-08
Dieldrin	0.055	0.2	10,500	0.22	0.06	4	6	1.00E-06	33	25,550	4.3E-11	1.60E+01	7E-10
Inorganics													
Aluminum	8,040	0.2	10,500	0.22	0.01	4	6	1.00E-06	33	25,550	1.1E-06	NA	
Arsenic	102	0.2	10,500	0.22	0.032	4	6	1.00E-06	33	25,550	4.3E-08	1.50E+00	6E-08
Beryllium	1.4	0.2	10,500	0.22	0.25	4	6	1.00E-06	33	25,550	4.6E-09	4.30E+00	2E-08
Cadmium	4.0	0.2	10,500	0.22	0.01	4	6	1.00E-06	33	25,550	5.3E-10	NA	
Iron	33,900	0.2	10,500	0.22	0.01	4	6	1.00E-06	33	25,550	4.5E-06	NA	
Manganese	799	0.2	10,500	0.22	0.2	4	6	1.00E-06	33	25,550	2.1E-06	NA	
Mercury	1.2	0.2	10,500	0.22	0.01	4	6	1.00E-06	33	25,550	1.6E-10	NA	
Thallium	1.4	0.2	10,500	0.22	0.01	4	6	1.00E-06	33	25,550	1.8E-10	NA	
												Sum =	5E-07

Table H-26

Sector: Moss
Scenario: Trespasser
Pathway: Dermal Contact with Surface Soil
Receptor: Child (7 to 12 years)

NONCARCINOGENIC
CDI = Cs x DAF x SA x FSA x RDA x EF x ED x CF x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	Cs Surface Soil Concentration (mg/kg)	DAF Dermal Adherence Factor (mg/cm^2-d)	SA Total Body Surface Area (cm^2)	FSA Fraction SA Exposed (unitless)	RDA Relative Dermal Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals													
Acenaphthylene	0.0080	0.2	10,500	0.22	0.10	4	6	1.00E-06	33	2,190	1.2E-10	NA	
Benzo(g,h,i)perylene	0.16	0.2	10,500	0.22	0.10	4	6	1.00E-06	33	2,190	2.5E-09	NA	
Phenanthrene	0.25	0.2	10,500	0.22	0.10	4	6	1.00E-06	33	2,190	3.8E-09	NA	
Benzo(a)pyrene	0.32	0.2	10,500	0.22	0.10	4	6	1.00E-06	33	2,190	4.9E-09	NA	
Pesticides/PCBs													
Aroclor-1248	120	0.2	10,500	0.22	0.06	4	6	1.00E-06	33	2,190	1.1E-06	NA	
Aroclor-1254	81	0.2	10,500	0.22	0.06	4	6	1.00E-06	33	2,190	7.5E-07	2.00E-05	3.7E-02
Aroclor-1260	58	0.2	10,500	0.22	0.06	4	6	1.00E-06	33	2,190	5.3E-07	NA	
Dieldrin	0.055	0.2	10,500	0.22	0.06	4	6	1.00E-06	33	2,190	5.1E-10	5.00E-05	1.0E-05
Inorganics													
Aluminum	8,040	0.2	10,500	0.22	0.01	4	6	1.00E-06	33	2,190	1.2E-05	1.00E+00	1.2E-05
Arsenic	102	0.2	10,500	0.22	0.032	4	6	1.00E-06	33	2,190	5.0E-07	3.00E-04	1.7E-03
Beryllium	1.4	0.2	10,500	0.22	0.25	4	6	1.00E-06	33	2,190	5.4E-08	5.00E-03	1.1E-05
Cadmium	4.0	0.2	10,500	0.22	0.01	4	6	1.00E-06	33	2,190	6.1E-09	5.00E-04	1.2E-05
Iron	33,900	0.2	10,500	0.22	0.01	4	6	1.00E-06	33	2,190	5.2E-05	3.00E-01	1.7E-04
Manganese	799	0.2	10,500	0.22	0.2	4	6	1.00E-06	33	2,190	2.5E-05	2.30E-02	1.1E-03
Mercury	1.2	0.2	10,500	0.22	0.01	4	6	1.00E-06	33	2,190	1.8E-09	1.00E-04	1.8E-05
Thallium	1.4	0.2	10,500	0.22	0.01	4	6	1.00E-06	33	2,190	2.1E-09	8.00E-05	2.7E-05
												Sum =	4.0E-02

Table H-27

Sector: Moat
Scenario: Trespasser
Pathway: Dermal Contact with Surface Soil
Receptor: Adolescent (13 to 18 years)

CARCINOGENIC

CDI = Cs x DAF x SA x FSA x RDA x EF x ED x CF x 1/BW x 1/ATc

Risk = CDI x CSF

Chemical	Cs Surface Soil Concentration (mg/kg)	DAF Dermal Adherence Factor (mg/cm^2-d)	SA Total Body Surface Area (cm^2)	FSA Fraction SA Exposed (unitless)	RDA Relative Dermal Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals													
Acenaphthylene	0.0080	0.2	16,000	0.23	0.10	4	6	1.00E-06	56	25,550	9.9E-12	NA	
Benzo(g,h,i)perylene	0.16	0.2	16,000	0.23	0.10	4	6	1.00E-06	56	25,550	2.0E-10	NA	
Phenanthrene	0.25	0.2	16,000	0.23	0.10	4	6	1.00E-06	56	25,550	3.1E-10	NA	
Benzo(a)pyrene	0.32	0.2	16,000	0.23	0.10	4	6	1.00E-06	56	25,550	4.0E-10	7.30E+00	3E-09
Pesticides/PCBs													
Aroclor-1248	120	0.2	16,000	0.23	0.06	4	6	1.00E-06	56	25,550	8.9E-08	2.00E+00	2E-07
Aroclor-1254	81	0.2	16,000	0.23	0.06	4	6	1.00E-06	56	25,550	6.0E-08	2.00E+00	1E-07
Aroclor-1260	58	0.2	16,000	0.23	0.06	4	6	1.00E-06	56	25,550	4.3E-08	2.00E+00	9E-08
Dieldrin	0.055	0.2	16,000	0.23	0.06	4	6	1.00E-06	56	25,550	4.1E-11	1.60E+01	7E-10
Inorganics													
Aluminum	8,040	0.2	16,000	0.23	0.01	4	6	1.00E-06	56	25,550	9.9E-07	NA	
Arsenic	102	0.2	16,000	0.23	0.032	4	6	1.00E-06	56	25,550	4.0E-08	1.50E+00	6E-08
Beryllium	1.4	0.2	16,000	0.23	0.25	4	6	1.00E-06	56	25,550	4.3E-09	4.30E+00	2E-08
Cadmium	4.0	0.2	16,000	0.23	0.01	4	6	1.00E-06	56	25,550	4.9E-10	NA	
Iron	33,900	0.2	16,000	0.23	0.01	4	6	1.00E-06	56	25,550	4.2E-06	NA	
Manganese	799	0.2	16,000	0.23	0.2	4	6	1.00E-06	56	25,550	2.0E-06	NA	
Mercury	1.2	0.2	16,000	0.23	0.01	4	6	1.00E-06	56	25,550	1.5E-10	NA	
Thallium	1.4	0.2	16,000	0.23	0.01	4	6	1.00E-06	56	25,550	1.7E-10	NA	
												Sum =	5E-07

Table H-28

Sector: Most
Scenario: Trespasser
Pathway: Dermal Contact with Surface Soil
Receptor: Adolescent (13 to 18 years)

NONCARCINOGENIC
CDI = Cs x DAF x SA x FSA x RDA x EF x ED x CF x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	Cs Surface Soil Concentration (mg/kg)	DAF Dermal Adherence Factor (mg/cm^2-d)	SA Total Body Surface Area (cm^2)	FSA Fraction SA Exposed (unitless)	RDA Relative Dermal Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals													
Acenaphthylene	0.0080	0.2	16,000	0.23	0.10	4	6	1.00E-06	56	2,190	1.2E-10	NA	
Benzo(g,h,i)perylene	0.16	0.2	16,000	0.23	0.10	4	6	1.00E-06	56	2,190	2.3E-09	NA	
Phenanthrene	0.25	0.2	16,000	0.23	0.10	4	6	1.00E-06	56	2,190	3.6E-09	NA	
Benzo(a)pyrene	0.32	0.2	16,000	0.23	0.10	4	6	1.00E-06	56	2,190	4.6E-09	NA	
Pesticides/PCBs													
Aroclor-1248	120	0.2	16,000	0.23	0.06	4	6	1.00E-06	56	2,190	1.0E-06	NA	
Aroclor-1254	81	0.2	16,000	0.23	0.06	4	6	1.00E-06	56	2,190	7.0E-07	2.00E-05	3.5E-02
Aroclor-1260	58	0.2	16,000	0.23	0.06	4	6	1.00E-06	56	2,190	5.0E-07	NA	
Dieldrin	0.055	0.2	16,000	0.23	0.06	4	6	1.00E-06	56	2,190	4.8E-10	5.00E-05	9.5E-06
Inorganics													
Aluminum	8,040	0.2	16,000	0.23	0.01	4	6	1.00E-06	56	2,190	1.2E-05	1.00E+00	1.2E-05
Arsenic	102	0.2	16,000	0.23	0.032	4	6	1.00E-06	56	2,190	4.7E-07	3.00E-04	1.6E-03
Beryllium	1.4	0.2	16,000	0.23	0.25	4	6	1.00E-06	56	2,190	5.0E-08	5.00E-03	1.0E-05
Cadmium	4.0	0.2	16,000	0.23	0.01	4	6	1.00E-06	56	2,190	5.8E-09	5.00E-04	1.2E-05
Iron	33,900	0.2	16,000	0.23	0.01	4	6	1.00E-06	56	2,190	4.9E-05	3.00E-01	1.6E-04
Manganese	799	0.2	16,000	0.23	0.2	4	6	1.00E-06	56	2,190	2.3E-05	2.30E-02	1.0E-03
Mercury	1.2	0.2	16,000	0.23	0.01	4	6	1.00E-06	56	2,190	1.7E-09	1.00E-04	1.7E-05
Thallium	1.4	0.2	16,000	0.23	0.01	4	6	1.00E-06	56	2,190	2.0E-09	8.00E-05	2.5E-05
												Sum =	3.8E-02

Table H-29

Sector: Moss
Scenario: Trespasser
Pathway: Ingestion of Surface Soil
Receptor: Child (7 to 12 years)

CARCINOGENIC
CDI = Cs x IgR x IgRF x ROA x EF x ED x CF x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	Cs Surface Soil Concentration (mg/kg)	IgR Surface Soil Ingestion Rate (mg/d)	IgRF Fraction Ingested Attributable to Site (unitless)	ROA Relative Oral Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals												
Acenaphthylene	0.0080	200	0.5	1.0	4	6	1.00E-06	33	25,550	2.3E-11	NA	
Benzo(g,h,i)perylene	0.16	200	0.5	1.0	4	6	1.00E-06	33	25,550	4.6E-10	NA	
Phenanthrene	0.25	200	0.5	1.0	4	6	1.00E-06	33	25,550	7.1E-10	NA	
Benzo(a)pyrene	0.32	200	0.5	1.0	4	6	1.00E-06	33	25,550	9.1E-10	7.30E+00	7E-09
Pesticides/PCBs												
Aroclor-1248	120	200	0.5	1.0	4	6	1.00E-06	33	25,550	3.4E-07	2.00E+00	7E-07
Aroclor-1254	81	200	0.5	1.0	4	6	1.00E-06	33	25,550	2.3E-07	2.00E+00	5E-07
Aroclor-1260	58	200	0.5	1.0	4	6	1.00E-06	33	25,550	1.7E-07	2.00E+00	3E-07
Dieldrin	0.055	200	0.5	1.0	4	6	1.00E-06	33	25,550	1.6E-10	1.60E+01	3E-09
Inorganics												
Aluminum	8,040	200	0.5	1.0	4	6	1.00E-06	33	25,550	2.3E-05	NA	
Arsenic	102	200	0.5	1.0	4	6	1.00E-06	33	25,550	2.9E-07	1.50E+00	4E-07
Beryllium	1.4	200	0.5	1.0	4	6	1.00E-06	33	25,550	4.0E-09	4.30E+00	2E-08
Cadmium	4.0	200	0.5	1.0	4	6	1.00E-06	33	25,550	1.1E-08	NA	
Iron	33,900	200	0.5	1.0	4	6	1.00E-06	33	25,550	9.6E-05	NA	
Manganese	799	200	1.0	0.05	4	6	1.00E-06	33	25,550	2.3E-07	NA	
Mercury	1.2	200	1.0	0.05	4	6	1.00E-06	33	25,550	3.4E-10	NA	
Thallium	1.4	200	0.5	1.0	4	6	1.00E-06	33	25,550	4.0E-09	NA	
											Sum =	2E-06

Table H-30

Sector: Most
Scenario: Trespasser
Pathway: Ingestion of Surface Soil
Receptor: Child (7 to 12 years)

NONCARCINOGENIC
CDI = Cs x IgR x IgRF x ROA x EF x ED x CF x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	Cs Surface Soil Concentration (mg/kg)	IgR Surface Soil Ingestion Rate (mg/d)	IgRF Fraction Ingested Attributable to Site (unitless)	ROA Relative Oral Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals												
Acenaphthylene	0.0080	200	0.5	1.0	4	6	1.00E-06	33	2,190	2.7E-10	NA	
Benzo(g,h,i)perylene	0.16	200	0.5	1.0	4	6	1.00E-06	33	2,190	5.3E-09	NA	
Phenanthrene	0.25	200	0.5	1.0	4	6	1.00E-06	33	2,190	8.3E-09	NA	
Benzo(a)pyrene	0.32	200	0.5	1.0	4	6	1.00E-06	33	2,190	1.1E-08	NA	
Pesticides/PCBs												
Aroclor-1248	120	200	0.5	1.0	4	6	1.00E-06	33	2,190	4.0E-06	NA	
Aroclor-1254	81	200	0.5	1.0	4	6	1.00E-06	33	2,190	2.7E-06	2.00E-05	1.3E-01
Aroclor-1260	58	200	0.5	1.0	4	6	1.00E-06	33	2,190	1.9E-06	NA	
Dieldrin	0.055	200	0.5	1.0	4	6	1.00E-06	33	2,190	1.8E-09	5.00E-05	3.7E-05
Inorganics												
Aluminum	8,040	200	0.5	1.0	4	6	1.00E-06	33	2,190	2.7E-04	1.00E+00	2.7E-04
Arsenic	102	200	0.5	1.0	4	6	1.00E-06	33	2,190	3.4E-06	3.00E-04	1.1E-02
Beryllium	1.4	200	0.5	1.0	4	6	1.00E-06	33	2,190	4.6E-08	5.00E-03	9.3E-06
Cadmium	4.0	200	0.5	1.0	4	6	1.00E-06	33	2,190	1.3E-07	5.00E-04	2.7E-04
Iron	33,900	200	0.5	1.0	4	6	1.00E-06	33	2,190	1.1E-03	3.00E-01	3.8E-03
Manganese	799	200	1.0	0.05	4	6	1.00E-06	33	2,190	2.7E-06	2.30E-02	1.2E-04
Mercury	1.2	200	1.0	0.05	4	6	1.00E-06	33	2,190	4.0E-09	1.00E-04	4.0E-05
Thallium	1.4	200	0.5	1.0	4	6	1.00E-06	33	2,190	4.6E-08	8.00E-05	5.8E-04
											Sum =	1.5E-01

Table H-31

Sector: Moat
Scenario: Trespasser
Pathway: Ingestion of Surface Soil
Receptor: Adolescent (13 to 18 years)

CARCINOGENIC
CDI = Cs x IgR x IgRF x ROA x EF x ED x CF x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	Cs Surface Soil Concentration (mg/kg)	IgR Soil Ingestion Rate (mg/d)	IgRF Fraction Ingested Attributable to Site (unitless)	ROA Relative Oral Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals												
Acenaphthylene	0.0080	100	0.5	1.0	4	6	1.00E-06	56	25,550	6.7E-12	NA	
Benzo(g,h,i)perylene	0.16	100	0.5	1.0	4	6	1.00E-06	56	25,550	1.3E-10	NA	
Phenanthrene	0.25	100	0.5	1.0	4	6	1.00E-06	56	25,550	2.1E-10	NA	
Benzo(a)pyrene	0.32	100	0.5	1.0	4	6	1.00E-06	56	25,550	2.7E-10	7.30E+00	2E-09
Pesticides/PCBs												
Aroclor-1248	120	100	0.5	1.0	4	6	1.00E-06	56	25,550	1.0E-07	2.00E+00	2E-07
Aroclor-1254	81	100	0.5	1.0	4	6	1.00E-06	56	25,550	6.8E-08	2.00E+00	1E-07
Aroclor-1260	58	100	0.5	1.0	4	6	1.00E-06	56	25,550	4.9E-08	2.00E+00	1E-07
Dieldrin	0.055	100	0.5	1.0	4	6	1.00E-06	56	25,550	4.6E-11	1.60E+01	7E-10
Inorganics												
Aluminum	8,040	100	0.5	1.0	4	6	1.00E-06	56	25,550	6.7E-06	NA	
Arsenic	102	100	0.5	1.0	4	6	1.00E-06	56	25,550	8.6E-08	1.50E+00	1E-07
Beryllium	1.4	100	0.5	1.0	4	6	1.00E-06	56	25,550	1.2E-09	4.30E+00	5E-09
Cadmium	4.0	100	0.5	1.0	4	6	1.00E-06	56	25,550	3.4E-09	NA	
Iron	33,900	100	0.5	1.0	4	6	1.00E-06	56	25,550	2.8E-05	NA	
Manganese	799	100	0.5	1.0	4	6	1.00E-06	56	25,550	6.7E-07	NA	
Mercury	1.2	100	0.5	1.0	4	6	1.00E-06	56	25,550	1.0E-09	NA	
Thallium	1.4	100	0.5	1.0	4	6	1.00E-06	56	25,550	1.2E-09	NA	
											Sum =	6E-07

Table H-32

Sector: Mont
Scenario: Trespasser
Pathway: Ingestion of Surface Soil
Receptor: Adolescent (13 to 18 years)

NONCARCINOGENIC
CDI = Cs x IgR x IgRF x ROA x EF x ED x CF x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	Cs Surface Soil Concentration (mg/kg)	IgR Sediment Ingestion Rate (mg/d)	IgRF Fraction Ingested Attributable to Site (unitless)	ROA Relative Oral Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals												
Acenaphthylene	0.0080	100	0.5	1.0	4	6	1.00E-06	56	2,190	7.8E-11	NA	
Benzo(g,h,i)perylene	0.16	100	0.5	1.0	4	6	1.00E-06	56	2,190	1.6E-09	NA	
Phenanthrene	0.25	100	0.5	1.0	4	6	1.00E-06	56	2,190	2.4E-09	NA	
Benzo(a)pyrene	0.32	100	0.5	1.0	4	6	1.00E-06	56	2,190	3.1E-09	NA	
Pesticides/PCBs												
Aroclor-1248	120	100	0.5	1.0	4	6	1.00E-06	56	2,190	1.2E-06	NA	
Aroclor-1254	81	100	0.5	1.0	4	6	1.00E-06	56	2,190	7.9E-07	2.00E-05	4.0E-02
Aroclor-1260	58	100	0.5	1.0	4	6	1.00E-06	56	2,190	5.7E-07	NA	
Dieldrin	0.055	100	0.5	1.0	4	6	1.00E-06	56	2,190	5.4E-10	5.00E-05	1.1E-05
Inorganics												
Aluminum	8,040	100	0.5	1.0	4	6	1.00E-06	56	2,190	7.9E-05	1.00E+00	7.9E-05
Arsenic	102	100	0.5	1.0	4	6	1.00E-06	56	2,190	1.0E-06	3.00E-04	3.3E-03
Beryllium	1.4	100	0.5	1.0	4	6	1.00E-06	56	2,190	1.4E-08	5.00E-03	2.7E-06
Cadmium	4.0	100	0.5	1.0	4	6	1.00E-06	56	2,190	3.9E-08	5.00E-04	7.8E-05
Iron	33,900	100	0.5	1.0	4	6	1.00E-06	56	2,190	3.3E-04	3.00E-01	1.1E-03
Manganese	799	100	0.5	1.0	4	6	1.00E-06	56	2,190	7.8E-06	2.30E-02	3.4E-04
Mercury	1.2	100	0.5	1.0	4	6	1.00E-06	56	2,190	1.2E-08	1.00E-04	1.2E-04
Thallium	1.4	100	0.5	1.0	4	6	1.00E-06	56	2,190	1.4E-08	8.00E-05	1.7E-04
											Sum =	4.5E-02

Table H-33

Sector: Moat
Scenario: Storm Sewer Maintenance/Construction
Pathway: Dermal Contact with Subsurface Soil
Receptor: Construction Worker (>18 years)

CARCINOGENIC

CDI = Cs x DAF x SA x FSA x RDA x EF x ED x CF x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	Cs Subsurface Soil Concentration (mg/kg)	DAF Dermal Adherence Factor (mg/cm^2-d)	SA Total Body Surface Area (cm^2)	FSA Fraction SA Exposed (unitless)	RDA Relative Dermal Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals													
Benzo(g,h,i)perylene	14	0.2	18,000	0.14	0.10	0.40	25	1.00E-06	70	25,550	3.9E-09	NA	
Phenanthrene	22	0.2	18,000	0.14	0.10	0.40	25	1.00E-06	70	25,550	6.2E-09	NA	
Benzo(a)anthracene	23	0.2	18,000	0.14	0.10	0.40	25	1.00E-06	70	25,550	6.5E-09	7.30E-01	5E-09
Benzo(a)pyrene	19	0.2	18,000	0.14	0.10	0.40	25	1.00E-06	70	25,550	5.4E-09	7.30E+00	4E-08
Benzo(b)fluoranthene	17	0.2	18,000	0.14	0.10	0.40	25	1.00E-06	70	25,550	4.8E-09	7.30E-01	3E-09
Indeno(1,2,3-cd)pyrene	13	0.2	18,000	0.14	0.10	0.40	25	1.00E-06	70	25,550	3.7E-09	NA	
Pesticides/PCBs													
Aroclor-1260	840	0.2	18,000	0.14	0.06	0.40	25	1.00E-06	70	25,550	1.4E-07	2.00E+00	3E-07
Inorganics													
Arsenic	49	0.2	18,000	0.14	0.032	0.40	25	1.00E-06	70	25,550	4.5E-09	1.50E+00	7E-09
Beryllium	3.1	0.2	18,000	0.14	0.25	0.40	25	1.00E-06	70	25,550	2.2E-09	4.30E+00	9E-09
Lead	451	0.2	18,000	0.14	0.02	0.40	25	1.00E-06	70	25,550	2.5E-08	NA	
												Sum =	3E-07

Table H-34

Sector: Mont
Scenario: Storm Sewer Maintenance/Construction
Pathway: Dermal Contact with Subsurface Soil
Receptor: Construction Worker (>18 years)

NONCARCINOGENIC

CDI = Cs x DAF x SA x FSA x RDA x EF x ED x CF x 1/BW x 1/ATnc

HQ = CDI/RfD

Chemical	Cs Subsurface Soil Concentration (mg/kg)	DAF Dermal Adherence Factor (mg/cm^2-d)	SA Total Body Surface Area (cm^2)	FSA Fraction SA Exposed (unitless)	RDA Relative Dermal Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals													
Benzo(g,h,i)perylene	14	0.2	18,000	0.14	0.10	0.40	25	1.00E-06	70	9,125	1.1E-08	NA	
Phenanthrene	22	0.2	18,000	0.14	0.10	0.40	25	1.00E-06	70	9,125	1.7E-08	NA	
Benzo(a)anthracene	23	0.2	18,000	0.14	0.10	0.40	25	1.00E-06	70	9,125	1.8E-08	NA	
Benzo(a)pyrene	19	0.2	18,000	0.14	0.10	0.40	25	1.00E-06	70	9,125	1.5E-08	NA	
Benzo(b)fluoranthene	17	0.2	18,000	0.14	0.10	0.40	25	1.00E-06	70	9,125	1.3E-08	NA	
Indeno(1,2,3-cd)pyrene	13	0.2	18,000	0.14	0.10	0.40	25	1.00E-06	70	9,125	1.0E-08	NA	
Pesticides/PCBs													
Aroclor-1260	840	0.2	18,000	0.14	0.06	0.40	25	1.00E-06	70	9,125	4.0E-07	NA	
Inorganics													
Arsenic	49	0.2	18,000	0.14	0.032	0.40	25	1.00E-06	70	9,125	1.2E-08	3.00E-04	4.2E-05
Beryllium	3.1	0.2	18,000	0.14	0.25	0.40	25	1.00E-06	70	9,125	6.1E-09	5.00E-03	1.2E-06
Lead	451	0.2	18,000	0.14	0.02	0.40	25	1.00E-06	70	9,125	7.1E-08	NA	
												Sum =	4.3E-05

Table H-35

Sector: Most
Scenario: Storm Sewer Maintenance/Construction
Pathway: Ingestion of Subsurface Soil
Receptor: Construction Worker (>18 years)

CARCINOGENIC

CDI = Cs x IgR x IgRF x ROA x EF x ED x CF x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	Cs Subsurface Soil Concentration (mg/kg)	IgR Soil Ingestion Rate (mg/d)	IgRF Fraction Ingested Attributable to Site (unitless)	ROA Relative Oral Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals												
Benzo(g,h,i)perylene	14	101	1	1.0	0.40	25	1.00E-06	70	25,550	7.9E-09	NA	
Phenanthrene	22	101	1	1.0	0.40	25	1.00E-06	70	25,550	1.2E-08	NA	
Benzo(a)anthracene	23	101	1	1.0	0.40	25	1.00E-06	70	25,550	1.3E-08	7.30E-01	9E-09
Benzo(a)pyrene	19	101	1	1.0	0.40	25	1.00E-06	70	25,550	1.1E-08	7.30E+00	8E-08
Benzo(b)fluoranthene	17	101	1	1.0	0.40	25	1.00E-06	70	25,550	9.6E-09	7.30E-01	7E-09
Indeno(1,2,3-cd)pyrene	13	101	1	1.0	0.40	25	1.00E-06	70	25,550	7.3E-09	NA	
Pesticides/PCBs												
Aroclor-1260	840	101	1	1.0	0.40	25	1.00E-06	70	25,550	4.7E-07	2.00E+00	9E-07
Inorganics												
Arsenic	49	101	1	1.0	0.40	25	1.00E-06	70	25,550	2.8E-08	1.50E+00	4E-08
Beryllium	3.1	101	1	1.0	0.40	25	1.00E-06	70	25,550	1.7E-09	4.30E+00	7E-09
Lead	451	101	1	1.0	0.40	25	1.00E-06	70	25,550	2.5E-07	NA	
											Sum =	1E-06

Table H-36

Sector: Moat
Scenario: Storm Sewer Maintenance/Construction
Pathway: Ingestion of Subsurface Soil
Receptor: Construction Worker (>18 years)

NONCARCINOGENIC
CDI = Cs x IgR x IgRF x ROA x EF x ED x CF x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	Cs Subsurface Soil Concentration (mg/kg)	IgR Soil Ingestion Rate (mg/d)	IgRF Fraction Ingested Attributable to Site (unitless)	ROA Relative Oral Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals												
Benzo(g,h,i)perylene	14	101	1	1.0	0.40	25	1.00E-06	70	9,125	2.2E-08	NA	
Phenanthrene	22	101	1	1.0	0.40	25	1.00E-06	70	9,125	3.5E-08	NA	
Benzo(a)anthracene	23	101	1	1.0	0.40	25	1.00E-06	70	9,125	3.6E-08	NA	
Benzo(a)pyrene	19	101	1	1.0	0.40	25	1.00E-06	70	9,125	3.0E-08	NA	
Benzo(b)fluoranthene	17	101	1	1.0	0.40	25	1.00E-06	70	9,125	2.7E-08	NA	
Indeno(1,2,3-cd)pyrene	13	101	1	1.0	0.40	25	1.00E-06	70	9,125	2.0E-08	NA	
Pesticides/PCBs												
Aroclor-1260	840	101	1	1.0	0.40	25	1.00E-06	70	9,125	1.3E-06	NA	
Inorganics												
Arsenic	49	101	1	1.0	0.40	25	1.00E-06	70	9,125	7.8E-08	NA	
Beryllium	3.1	101	1	1.0	0.40	25	1.00E-06	70	9,125	4.9E-09	5.00E-03	9.7E-07
Lead	451	101	1	1.0	0.40	25	1.00E-06	70	9,125	7.1E-07	NA	
											Sum =	9.7E-07

Table H-37

Sector: Moat
Scenario: Storm Sewer Maintenance/Construction
Pathway: Inhalation of Soil Particulates
Receptor: Construction Worker (>18 years)

CARCINOGENIC

CDI = Cs x Cp x IhR x LDF x RIA x EF x ED x CF1 x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	Cs Subsurface Soil Concentration (mg/kg)	Cp [a] Particulate Concentration (mg/m^3)	IhR Inhalation Rate (m^3/day)	LDF Lung Deposition Fraction (unitless)	RIA Relative Inhalation Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals													
Benzo(g,h,i)perylene	14	6.92E-02	20	0.125	1.0	0.40	25	1.00E-06	70	25,550	1.4E-11	NA	
Phenanthrene	22	6.92E-02	20	0.125	1.0	0.40	25	1.00E-06	70	25,550	2.1E-11	NA	
Benzo(a)anthracene	23	6.92E-02	20	0.125	1.0	0.40	25	1.00E-06	70	25,550	2.2E-11	NA	
Benzo(a)pyrene	19	6.92E-02	20	0.125	1.0	0.40	25	1.00E-06	70	25,550	1.8E-11	6.10E+00	1E-10
Benzo(b)fluoranthene	17	6.92E-02	20	0.125	1.0	0.40	25	1.00E-06	70	25,550	1.6E-11	6.10E-01	1E-11
Indeno(1,2,3-cd)pyrene	13	6.92E-02	20	0.125	1.0	0.40	25	1.00E-06	70	25,550	1.3E-11	NA	
Pesticides/PCBs													
Aroclor-1260	840	6.92E-02	20	0.125	1.0	0.40	25	1.00E-06	70	25,550	8.1E-10	2.00E+00	2E-09
Inorganics													
Arsenic	49	6.92E-02	20	0.125	1.0	0.40	25	1.00E-06	70	25,550	4.8E-11	1.50E+01	7E-10
Beryllium	3.1	6.92E-02	20	0.125	1.0	0.40	25	1.00E-06	70	25,550	3.0E-12	8.40E+00	2E-11
Lead	451	6.92E-02	20	0.125	1.0	0.40	25	1.00E-06	70	25,550	4.4E-10	NA	
												Sum =	2E-09

Notes:
a - Table H-37a summarize the risks associated with this pathway using EPA Region III's computed a PM-10 concentration of 0.0016 ug/m^3. See Appendix I for EPA's PM-10 computation.

Table H-37a

Sector: Moat
Scenario: Storm Sewer Maintenance/Construction
Pathway: Inhalation of Soil Particulates
Receptor: Construction Worker (>18 years)

CARCINOGENIC

CDI = Cs x Cp x IhR x LDF x RIA x EF x ED x CF1 x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	Cs Subsurface Soil Concentration (mg/kg)	Cp [a] Particulate Concentration (mg/m^3)	IhR Inhalation Rate (m^3/day)	LDF Lung Deposition Fraction (unitless)	RIA elative Inhalatio Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals													
Benzo(g,h,i)perylene	14	1.70E+00	20	0.125	1.0	0.40	25	1.00E-06	70	25,550	3.3E-10	NA	
Phenanthrene	22	1.70E+00	20	0.125	1.0	0.40	25	1.00E-06	70	25,550	5.2E-10	NA	
Benzo(a)anthracene	23	1.70E+00	20	0.125	1.0	0.40	25	1.00E-06	70	25,550	5.5E-10	NA	
Benzo(a)pyrene	19	1.70E+00	20	0.125	1.0	0.40	25	1.00E-06	70	25,550	4.5E-10	6.10E+00	3E-09
Benzo(b)fluoranthene	17	1.70E+00	20	0.125	1.0	0.40	25	1.00E-06	70	25,550	4.0E-10	6.10E-01	2E-10
Indeno(1,2,3-cd)pyrene	13	1.70E+00	20	0.125	1.0	0.40	25	1.00E-06	70	25,550	3.1E-10	NA	
Pesticides/PCBs													
Aroclor-1260	840	1.70E+00	20	0.125	1.0	0.40	25	1.00E-06	70	25,550	2.0E-08	2.00E+00	4E-08
Inorganics													
Arsenic	49	1.70E+00	20	0.125	1.0	0.40	25	1.00E-06	70	25,550	1.2E-09	1.50E+01	2E-08
Beryllium	3.1	1.70E+00	20	0.125	1.0	0.40	25	1.00E-06	70	25,550	7.3E-11	8.40E+00	6E-10
Lead	451	1.70E+00	20	0.125	1.0	0.40	25	1.00E-06	70	25,550	1.1E-08	NA	
												Sum =	6E-08

Notes:
a - Table H-37a summarize the risks associated with this pathway using EPA Region III's computed a PM-10 concentration of 1.7 mg/m^3. See Appendix 1 for EPA's PM-10 computation.

Table H-38

Sector: Moat
Scenario: Storm Sewer Maintenance/Construction
Pathway: Inhalation of Soil Particulates
Receptor: Construction Worker (>18 years)

NONCARCINOGENIC

CDI = Cs x Cp x IhR x LDF x RIA x EF x ED x CF1 x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	Cs Subsurface Soil Concentration (mg/kg)	Cp[a] Particulate Concentration (mg/m^3)	IhR Inhalation Rate (m^3/day)	LDF Lung Deposition Fraction (unitless)	RIA Relative Inhalation Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals													
Benzo(g,h,i)perylene	14	6.92E-02	20	0.125	1.0	0.40	25	1.00E-06	70	9,125	3.8E-11	NA	
Phenanthrene	22	6.92E-02	20	0.125	1.0	0.40	25	1.00E-06	70	9,125	6.0E-11	NA	
Benzo(a)anthracene	23	6.92E-02	20	0.125	1.0	0.40	25	1.00E-06	70	9,125	6.2E-11	NA	
Benzo(a)pyrene	19	6.92E-02	20	0.125	1.0	0.40	25	1.00E-06	70	9,125	5.1E-11	NA	
Benzo(b)fluoranthene	17	6.92E-02	20	0.125	1.0	0.40	25	1.00E-06	70	9,125	4.6E-11	NA	
Indeno(1,2,3-cd)pyrene	13	6.92E-02	20	0.125	1.0	0.40	25	1.00E-06	70	9,125	3.5E-11	NA	
Pesticides/PCBs													
Aroclor-1260	840	6.92E-02	20	0.125	1.0	0.40	25	1.00E-06	70	9,125	2.3E-09	NA	
Inorganics													
Arsenic	49	6.92E-02	20	0.125	1.0	0.40	25	1.00E-06	70	9,125	1.3E-10	NA	
Beryllium	3.1	6.92E-02	20	0.125	1.0	0.40	25	1.00E-06	70	9,125	8.3E-12	NA	
Lead	451	6.92E-02	20	0.125	1.0	0.40	25	1.00E-06	70	9,125	1.2E-09	NA	
Notes:												Sum =	0.0E+00

a - Table H-38a summarize the hazards associated with this pathway using EPA Region III's computed a PM-10 concentration of 0.0016 ug/m^3. See Appendix I for EPA's PM-10 computation.

Table H-38a

Sector: Moat
Scenario: Storm Sewer Maintenance/Construction
Pathway: Inhalation of Soil Particulates
Receptor: Construction Worker (>18 years)

NONCARCINOGENIC

CDI = Cs x Cp x IhR x LDF x RIA x EF x ED x CF1 x 1/BW x 1/ATnc

HQ = CDI/RfD

Chemical	Cs Subsurface Soil Concentration (mg/kg)	Cp [a] Particulate Concentration (mg/m^3)	IhR Inhalation Rate (m^3/day)	LDF Lung Deposition Fraction (unitless)	RIA elative Inhalatio Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals													
Benzo(g,h,i)perylene	14	1.70E+00	20	0.125	1.0	0.40	25	1.00E-06	70	9,125	9.3E-10	NA	
Phenanthrene	22	1.70E+00	20	0.125	1.0	0.40	25	1.00E-06	70	9,125	1.5E-09	NA	
Benzo(a)anthracene	23	1.70E+00	20	0.125	1.0	0.40	25	1.00E-06	70	9,125	1.5E-09	NA	
Benzo(a)pyrene	19	1.70E+00	20	0.125	1.0	0.40	25	1.00E-06	70	9,125	1.3E-09	NA	
Benzo(b)fluoranthene	17	1.70E+00	20	0.125	1.0	0.40	25	1.00E-06	70	9,125	1.1E-09	NA	
Indeno(1,2,3-cd)pyrene	13	1.70E+00	20	0.125	1.0	0.40	25	1.00E-06	70	9,125	8.6E-10	NA	
Pesticides/PCBs													
Aroclor-1260	840	1.70E+00	20	0.125	1.0	0.40	25	1.00E-06	70	9,125	5.6E-08	NA	
Inorganics													
Arsenic	49	1.70E+00	20	0.125	1.0	0.40	25	1.00E-06	70	9,125	3.3E-09	NA	
Beryllium	3.1	1.70E+00	20	0.125	1.0	0.40	25	1.00E-06	70	9,125	2.0E-10	NA	
Lead	451	1.70E+00	20	0.125	1.0	0.40	25	1.00E-06	70	9,125	3.0E-08	NA	
												Sum =	0.0E+00

Notes:

a - Table H-38a summarize the hazards associated with this pathway using EPA Region III's computed a PM-10 concentration of 1.7 mg/m^3. See Appendix I for EPA's PM-10 computation.

Table H-39

Sector: Railroad ROW
Scenario: Maintenance
Pathway: Inhalation of Surface Soil Particulates
Receptor: Maintenance Worker (>18 years)

CARCINOGENIC

CDI = Cs x (1/PEF) x IhR x LDF x RIA x EF x ED x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	Cs Surface Soil Concentration (mg/kg)	PEF Particulate Emission Factor (m^3/kg)	IhR Inhalation Rate (m^3/day)	LDF Lung Deposition Fraction (unitless)	RIA Relative Inhalation Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals												
Acenaphthylene	0.059	1.30E+09	20	0.125	1.0	5	25	70	25,550	NA	NA	
Benzo(g,h,i)perylene	3.0	1.30E+09	20	0.125	1.0	5	25	70	25,550	NA	NA	
Benzo(a)anthracene	5.1	1.30E+09	20	0.125	1.0	5	25	70	25,550	NA	NA	
Benzo(a)pyrene	4.8	1.30E+09	20	0.125	1.0	5	25	70	25,550	6.5E-13	6.10E+00	4E-12
Benzo(b)fluoranthene	8.0	1.30E+09	20	0.125	1.0	5	25	70	25,550	1.1E-12	6.10E-01	7E-13
Dibenz(a,h)anthracene	0.2	1.30E+09	20	0.125	1.0	5	25	70	25,550	2.2E-14	6.10E+00	1E-13
Indeno(1,2,3-cd)pyrene	3.3	1.30E+09	20	0.125	1.0	5	25	70	25,550	NA	NA	
Phenanthrene	4.5	1.30E+09	20	0.125	1.0	5	25	70	25,550	NA	NA	
Pesticides/PCBs												
Aroclor-1248	210	1.30E+09	20	0.125	1.0	5	25	70	25,550	2.8E-11	2.00E+00	6E-11
Aroclor-1254	270	1.30E+09	20	0.125	1.0	5	25	70	25,550	3.6E-11	2.00E+00	7E-11
Aroclor-1260	170	1.30E+09	20	0.125	1.0	5	25	70	25,550	2.3E-11	2.00E+00	5E-11
Dieldrin	0.44	1.30E+09	20	0.125	1.0	5	25	70	25,550	5.9E-14	1.61E+01	1E-12
Heptachlor Epoxide	0.22	1.30E+09	20	0.125	1.0	5	25	70	25,550	2.9E-14	9.10E+00	3E-13
Endrin aldehyde	3.2	1.30E+09	20	0.125	1.0	5	25	70	25,550	NA	NA	
Dioxins/Furans												
Total Equivalent 2,3,7,8-TCDD	0.0016	1.30E+09	20	0.125	1.0	5	25	70	25,550	2.1E-16	1.10E+05	2E-11
Inorganic Chemicals												
Aluminum	11,000	1.30E+09	20	0.125	1.0	5	25	70	25,550	NA	NA	
Antimony	29.8	1.30E+09	20	0.125	1.0	5	25	70	25,550	NA	NA	
Arsenic	48	1.30E+09	20	0.125	1.0	5	25	70	25,550	6.5E-12	1.50E+01	1E-10
Beryllium	1.6	1.30E+09	20	0.125	1.0	5	25	70	25,550	2.2E-13	8.40E+00	2E-12
Cadmium	26	1.30E+09	20	0.125	1.0	5	25	70	25,550	3.5E-12	6.30E+00	2E-11
Chromium	81	1.30E+09	20	0.125	1.0	5	25	70	25,550	1.1E-11	4.20E+01	5E-10
Copper	972	1.30E+09	20	0.125	1.0	5	25	70	25,550	NA	NA	
Iron	59,800	1.30E+09	20	0.125	1.0	5	25	70	25,550	NA	NA	
Lead	624	1.30E+09	20	0.125	1.0	5	25	70	25,550	NA	NA	
Manganese	1,215	1.30E+09	20	0.125	1.0	5	25	70	25,550	NA	NA	
Mercury	0.95	1.30E+09	20	0.125	1.0	5	25	70	25,550	NA	NA	
Thallium	3.6	1.30E+09	20	0.125	1.0	5	25	70	25,550	NA	NA	
Zinc	15,600	1.30E+09	20	0.125	1.0	5	25	70	25,550	NA	NA	
											Sum =	8E-10

Table H-40

Sector: Railroad ROW
Scenario: Maintenance
Pathway: Inhalation of Surface Soil Particulates
Receptor: Maintenance Worker (>18 years)

NONCARCINOGENIC
CDI = Cs x (1/PEF) x InR x LDF x RIAx EF x ED x 1/BW x 1/ATc
HQ = CDI/RfD

Chemical	Cs Surface Soil Concentration (mg/kg)	PEF Particulate Emission Factor (m^3/kg)	InR Inhalation Rate (m^3/day)	LDF Lung Deposition Fraction (unitless)	RIA Relative Inhalation Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals												
Acenaphthylene	0.059	1.30E+09	20	0.125	1.0	5	25	70	9,125	NA	NA	
Benzo(g,h,i)perylene	3.0	1.30E+09	20	0.125	1.0	5	25	70	9,125	NA	NA	
Benzo(a)anthracene	5.1	1.30E+09	20	0.125	1.0	5	25	70	9,125	NA	NA	
Benzo(a)pyrene	4.8	1.30E+09	20	0.125	1.0	5	25	70	9,125	NA	NA	
Benzo(b)fluoranthene	8.0	1.30E+09	20	0.125	1.0	5	25	70	9,125	NA	NA	
Dibenz(a,h)anthracene	0.2	1.30E+09	20	0.125	1.0	5	25	70	9,125	NA	NA	
Indeno(1,2,3-cd)pyrene	3.3	1.30E+09	20	0.125	1.0	5	25	70	9,125	NA	NA	
Phenanthrene	4.5	1.30E+09	20	0.125	1.0	5	25	70	9,125	NA	NA	
Pesticides/PCBs												
Aroclor-1248	210	1.30E+09	20	0.125	1.0	5	25	70	9,125	NA	NA	
Aroclor-1254	270	1.30E+09	20	0.125	1.0	5	25	70	9,125	NA	NA	
Aroclor-1260	170	1.30E+09	20	0.125	1.0	5	25	70	9,125	NA	NA	
Dieldrin	0.44	1.30E+09	20	0.125	1.0	5	25	70	9,125	NA	NA	
Heptachlor Epoxide	0.22	1.30E+09	20	0.125	1.0	5	25	70	9,125	8.1E-14	1.30E-05	6.2E-09
Endrin aldehyde	3.2	1.30E+09	20	0.125	1.0	5	25	70	9,125	NA	NA	
Dioxins/Furans												
Total Equivalent 2,3,7,8-TCDD	0.0016	1.30E+09	20	0.125	1.0	5	25	70	9,125	NA	NA	
Inorganic Chemicals												
Aluminum	11,000	1.30E+09	20	0.125	1.0	5	25	70	9,125	4.1E-09	1.00E+00	4.1E-09
Antimony	29.8	1.30E+09	20	0.125	1.0	5	25	70	9,125	NA	NA	
Arsenic	48	1.30E+09	20	0.125	1.0	5	25	70	9,125	NA	NA	
Beryllium	1.6	1.30E+09	20	0.125	1.0	5	25	70	9,125	NA	NA	
Cadmium	26	1.30E+09	20	0.125	1.0	5	25	70	9,125	9.7E-12	5.70E-05	1.7E-07
Chromium	81	1.30E+09	20	0.125	1.0	5	25	70	9,125	3.0E-11	5.70E-07	5.3E-05
Copper	972	1.30E+09	20	0.125	1.0	5	25	70	9,125	NA	NA	
Iron	59,800	1.30E+09	20	0.125	1.0	5	25	70	9,125	NA	NA	
Lead	624	1.30E+09	20	0.125	1.0	5	25	70	9,125	NA	NA	
Manganese	1,215	1.30E+09	20	0.125	1.0	5	25	70	9,125	4.6E-10	1.43E-05	3.2E-05
Mercury	0.95	1.30E+09	20	0.125	1.0	5	25	70	9,125	3.6E-13	8.60E-05	4.2E-09
Thallium	3.6	1.30E+09	20	0.125	1.0	5	25	70	9,125	NA	NA	
Zinc	15,600	1.30E+09	20	0.125	1.0	5	25	70	9,125	NA	NA	
											Sum =	8.6E-05

Table H-41

Sector: Railroad ROW
Scenario: Maintenance
Pathway: Dermal Contact with Surface Soil
Receptor: Maintenance Worker (>18 years)

CARCINOGENIC

CDI = Cs x DAF x SA x FSA x RDA x EF x ED x CF x 1/BW x 1/ATc

Risk = CDI x CSF

Chemical	Cs Surface Soil Concentration (mg/kg)	DAF Dermal Adherence Factor (mg/cm^2-d)	SA Total Body Surface Area (cm^2)	SAF Fraction SA Exposed (unitless)	RDA Relative Dermal Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slop Factor mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals													
Acenaphthylene	0.059	0.2	18,000	0.14	0.10	5	25	1.00E-06	70	25550	2.08E-10	NA	
Benzo(g,h,i)perylene	3.0	0.2	18,000	0.14	0.10	5	25	1.00E-06	70	25550	1.04E-08	NA	
Benzo(a)anthracene	5.1	0.2	18,000	0.14	0.10	5	25	1.00E-06	70	25550	1.78E-08	7.30E-01	1E-08
Benzo(a)pyrene	4.8	0.2	18,000	0.14	0.10	5	25	1.00E-06	70	25550	1.69E-08	7.30E+00	1E-07
Benzo(b)fluoranthene	8.0	0.2	18,000	0.14	0.10	5	25	1.00E-06	70	25550	2.82E-08	7.30E-01	2E-08
Dibenz(a,h)anthracene	0.2	0.2	18,000	0.14	0.10	5	25	1.00E-06	70	25550	5.64E-10	7.30E+00	4E-09
Indeno(1,2,3-cd)pyrene	3.3	0.2	18,000	0.14	0.10	5	25	1.00E-06	70	25550	1.16E-08	NA	
Phenanthrene	4.5	0.2	18,000	0.14	0.10	5	25	1.00E-06	70	25550	1.59E-08	NA	
Pesticides/PCBs													
Aroclor-1248	210	0.2	18,000	0.14	0.06	5	25	1.00E-06	70	25550	4.44E-07	2.00E+00	9E-07
Aroclor-1254	270	0.2	18,000	0.14	0.06	5	25	1.00E-06	70	25550	5.71E-07	2.00E+00	1E-06
Aroclor-1260	170	0.2	18,000	0.14	0.06	5	25	1.00E-06	70	25550	3.59E-07	2.00E+00	7E-07
Dieldrin	0.44	0.2	18,000	0.14	0.10	5	25	1.00E-06	70	25550	1.55E-09	1.60E+01	2E-08
Heptachlor Epoxide	0.22	0.2	18,000	0.14	0.10	5	25	1.00E-06	70	25550	7.57E-10	9.10E+00	7E-09
Endrin aldehyde	3.2	0.2	18,000	0.14	0.10	5	25	1.00E-06	70	25550	1.13E-08	NA	
Dioxins/Furans													
Total Equivalent 2,3,7,8-TCDD	0.0016	0.2	18,000	0.14	0.03	5	25	1.00E-06	70	25550	1.65E-12	1.50E+05	2E-07
Inorganic Chemicals													
Aluminum	11,000	0.2	18,000	0.14	0.01	5	25	1.00E-06	70	25550	3.87E-06	NA	
Antimony	29.8	0.2	18,000	0.14	0.01	5	25	1.00E-06	70	25550	1.05E-08	NA	
Arsenic	48	0.2	18,000	0.14	0.032	5	25	1.00E-06	70	25550	5.42E-08	1.50E+00	8E-08
Beryllium	1.6	0.2	18,000	0.14	0.25	5	25	1.00E-06	70	25550	1.41E-08	4.30E+00	6E-08
Cadmium	26	0.2	18,000	0.14	0.01	5	25	1.00E-06	70	25550	9.12E-09	NA	
Chromium	81	0.2	18,000	0.14	0.01	5	25	1.00E-06	70	25550	2.85E-08	NA	
Copper	972	0.2	18,000	0.14	0.01	5	25	1.00E-06	70	25550	3.42E-07	NA	
Iron	59,800	0.2	18,000	0.14	0.01	5	25	1.00E-06	70	25550	2.11E-05	NA	
Lead	624	0.2	18,000	0.14	0.02	5	25	1.00E-06	70	25550	4.40E-07	NA	
Manganese	1,215	0.2	18,000	0.14	0.20	5	25	1.00E-06	70	25550	8.56E-06	NA	
Mercury	0.95	0.20	18000.00	0.14	0.01	5.00	25.00	0.00	70.00	25550.00	0.00	NA	
Thallium	3.6	0.2	18,000	0.14	0.01	5	25	1.00E-06	70	25550	1.27E-09	NA	
Zinc	15,600	0.2	18,000	0.14	0.01	5	25	1.00E-06	70	25550	5.50E-06	NA	
												Sum =	3E-06

Table H-42

Sector: Railroad ROW
Scenario: Maintenance
Pathway: Dermal Contact with Surface Soil
Receptor: Maintenance Worker (>18 years)

NONCARCINOGENIC
CDI = Cs x DAF x SA x FSA x RDA x EF x ED x CF x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	Cs Surface Soil Concentration (mg/kg)	DAF Dermal Adherence Factor (mg/cm^2-d)	SA Total Body Surface Area (cm^2)	SAF Fraction SA Exposed (unitless)	RDA Relative Dermal Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals													
Acenaphthylene	0.059	0.2	18,000	0.14	0.10	5	25	1.00E-06	70	9,125	5.82E-10	NA	
Benzo(g,h,i)perylene	3.0	0.2	18,000	0.14	0.10	5	25	1.00E-06	70	9,125	2.91E-08	NA	
Benzo(a)anthracene	5.1	0.2	18,000	0.14	0.10	5	25	1.00E-06	70	9,125	4.98E-08	NA	
Benzo(a)pyrene	4.8	0.2	18,000	0.14	0.10	5	25	1.00E-06	70	9,125	4.73E-08	NA	
Benzo(b)fluoranthene	8.0	0.2	18,000	0.14	0.10	5	25	1.00E-06	70	9,125	7.89E-08	NA	
Dibenz(a,h)anthracene	0.2	0.2	18,000	0.14	0.10	5	25	1.00E-06	70	9,125	1.58E-09	NA	
Indeno(1,2,3-cd)pyrene	3.3	0.2	18,000	0.14	0.10	5	25	1.00E-06	70	9,125	3.25E-08	NA	
Phenanthrene	4.5	0.2	18,000	0.14	0.10	5	25	1.00E-06	70	9,125	4.44E-08	NA	
Pesticides/PCBs													
Aroclor-1248	210	0.2	18,000	0.14	0.06	5	25	1.00E-06	70	9,125	1.24E-06	NA	
Aroclor-1254	270	0.2	18,000	0.14	0.06	5	25	1.00E-06	70	9,125	1.60E-06	2.00E-05	8.0E-02
Aroclor-1260	170	0.2	18,000	0.14	0.06	5	25	1.00E-06	70	9,125	1.01E-06	NA	
Dieldrin	0.44	0.2	18,000	0.14	0.10	5	25	1.00E-06	70	9,125	4.34E-09	5.00E-05	8.7E-05
Heptachlor Epoxide	0.22	0.2	18,000	0.14	0.10	5	25	1.00E-06	70	9,125	2.12E-09	1.30E-05	1.6E-04
Endrin aldehyde	3.2	0.2	18,000	0.14	0.10	5	25	1.00E-06	70	9,125	3.16E-08	3.00E-04	1.1E-04
Dioxins/Furans													
Total Equivalent 2,3,7,8-TCDD	0.0016	0.2	18,000	0.14	0.03	5	25	1.00E-06	70	9,125	4.62E-12	NA	
Inorganic Chemicals													
Aluminum	11,000	0.2	18,000	0.14	0.01	5	25	1.00E-06	70	9,125	1.08E-05	1.00E+00	1.1E-05
Antimony	29.8	0.2	18,000	0.14	0.01	5	25	1.00E-06	70	9,125	2.94E-08	4.00E-04	7.3E-05
Arsenic	48	0.2	18,000	0.14	0.032	5	25	1.00E-06	70	9,125	1.52E-07	3.00E-04	5.1E-04
Beryllium	1.6	0.2	18,000	0.14	0.25	5	25	1.00E-06	70	9,125	3.95E-08	5.00E-03	7.9E-06
Cadmium	26	0.2	18,000	0.14	0.01	5	25	1.00E-06	70	9,125	2.55E-08	5.00E-04	5.1E-05
Chromium	81	0.2	18,000	0.14	0.01	5	25	1.00E-06	70	9,125	7.97E-08	5.00E-03	1.6E-05
Copper	972	0.2	18,000	0.14	0.01	5	25	1.00E-06	70	9,125	9.59E-07	4.00E-02	2.4E-05
Iron	59,800	0.2	18,000	0.14	0.01	5	25	1.00E-06	70	9,125	5.90E-05	3.00E-01	2.0E-04
Lead	624	0.2	18,000	0.14	0.02	5	25	1.00E-06	70	9,125	1.23E-06	NA	
Manganese	1,215	0.2	18,000	0.14	0.20	5	25	1.00E-06	70	9,125	2.40E-05	2.30E-02	1.0E-03
Mercury	0.95	0.2	18,000	0.14	0.01	5	25	1.00E-06	70	9,125	9.37E-10	1.00E-04	9.4E-06
Thallium	3.6	0.2	18,000	0.14	0.01	5	25	1.00E-06	70	9,125	3.55E-09	8.00E-05	4.4E-05
Zinc	15,600	0.2	18,000	0.14	0.01	5	25	1.00E-06	70	9,125	1.54E-05	3.00E-01	5.1E-05
												Sum =	8.2E-02

Table H-43

Sector: Railroad ROW
Scenario: Maintenance
Pathway: Incidental Ingestion of Surface Soil
Receptor: Maintenance Worker (>18 years)

CARCINOGENIC

CDI = Cs x IgR x IgRF x ROA x EF x ED x CF x 1/BW x 1/ATc

Risk = CDI x CSF

Chemical	Cs Surface Soil Concentration (mg/kg)	IgR Soil Ingestion Rate (mg/d)	IgRF Fraction Ingested Attributable to Site (unitless)	ROA Relative Oral Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals												
Acenaphthylene	0.059	100	1	1.0	5	25	1.00E-06	70	25550	4.12E-10	NA	
Benzo(g,h,i)perylene	3.0	100	1	1.0	5	25	1.00E-06	70	25550	2.06E-08	NA	
Benzo(a)anthracene	5.1	100	1	1.0	5	25	1.00E-06	70	25550	3.53E-08	7.30E-01	3E-08
Benzo(a)pyrene	4.8	100	1	1.0	5	25	1.00E-06	70	25550	3.35E-08	7.30E+00	2E-07
Benzo(b)fluoranthene	8.0	100	1	1.0	5	25	1.00E-06	70	25550	5.59E-08	7.30E-01	4E-08
Dibenz(a,h)anthracene	0.2	100	1	1.0	5	25	1.00E-06	70	25550	1.12E-09	7.30E+00	8E-09
Indeno(1,2,3-cd)pyrene	3.3	100	1	1.0	5	25	1.00E-06	70	25550	2.31E-08	NA	
Phenanthrene	4.5	100	1	1.0	5	25	1.00E-06	70	25550	3.15E-08	NA	
Pesticides/PCBs												
Aroclor-1248	210	100	1	1.0	5	25	1.00E-06	70	25550	1.47E-06	2.00E+00	3E-06
Aroclor-1254	270	100	1	1.0	5	25	1.00E-06	70	25550	1.89E-06	2.00E+00	4E-06
Aroclor-1260	170	100	1	1.0	5	25	1.00E-06	70	25550	1.19E-06	2.00E+00	2E-06
Dieldrin	0.44	100	1	1.0	5	25	1.00E-06	70	25550	3.08E-09	1.60E+01	5E-08
Heptachlor Epoxide	0.22	100	1	1.0	5	25	1.00E-06	70	25550	1.50E-09	9.10E+00	1E-08
Endrin aldehyde	3.2	100	1	1.0	5	25	1.00E-06	70	25550	2.24E-08	NA	
Dioxins/Furans												
Total Equivalent 2,3,7,8-TCDD	0.0016	100	1	1.0	5	25	1.00E-06	70	25550	1.09E-11	1.50E+05	2E-06
Inorganic Chemicals												
Aluminum	11,000	100	1	1.0	5	25	1.00E-06	70	25550	7.69E-05	NA	
Antimony	29.8	100	1	1.0	5	25	1.00E-06	70	25550	2.08E-07	NA	
Arsenic	48	100	1	1.0	5	25	1.00E-06	70	25550	3.36E-07	1.50E+00	5E-07
Beryllium	1.6	100	1	1.0	5	25	1.00E-06	70	25550	1.12E-08	4.30E+00	5E-08
Cadmium	26	100	1	1.0	5	25	1.00E-06	70	25550	1.81E-07	NA	
Chromium	81	100	1	1.0	5	25	1.00E-06	70	25550	5.65E-07	NA	
Copper	972	100	1	1.0	5	25	1.00E-06	70	25550	6.79E-06	NA	
Iron	59,800	100	1	1.0	5	25	1.00E-06	70	25550	4.18E-04	NA	
Lead	624	100	1	1.0	5	25	1.00E-06	70	25550	4.36E-06	NA	
Manganese	1,215	100	1	1.0	5	25	1.00E-06	70	25550	8.49E-06	NA	
Mercury	0.95	100	1	1.0	5	25	1.00E-06	70	25550	6.64E-09	NA	
Thallium	3.6	100	1	1.0	5	25	1.00E-06	70	25550	2.52E-08	NA	
Zinc	15,600	100	1	1.0	5	25	1.00E-06	70	25550	1.09E-04	NA	
											Sum =	1E-05

Table H-44

Sector: Railroad ROW
Scenario: Maintenance
Pathway: Incidental Ingestion of Surface Soil
Receptor: Maintenance Worker (>18 years)

NONCARCINOGENIC
CDI = Cs x IgR x IgRF x ROA x EF x ED x CF x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	Cs Surface Soil Concentration (mg/kg)	IgR Soil Ingestion Rate (mg/d)	IgRF Fraction Ingested Attributable to Site (unitless)	ROA Relative Oral Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals												
Acenaphthylene	0.059	100	1	1.0	5	25	1.00E-06	70	9,125	1.15E-09	NA	
Benzo(g,h,i)perylene	3.0	100	1	1.0	5	25	1.00E-06	70	9,125	5.77E-08	NA	
Benzo(a)anthracene	5.1	100	1	1.0	5	25	1.00E-06	70	9,125	9.88E-08	NA	
Benzo(a)pyrene	4.8	100	1	1.0	5	25	1.00E-06	70	9,125	9.39E-08	NA	
Benzo(b)fluoranthene	8.0	100	1	1.0	5	25	1.00E-06	70	9,125	1.57E-07	NA	
Dibenz(a,h)anthracene	0.2	100	1	1.0	5	25	1.00E-06	70	9,125	3.13E-09	NA	
Indeno(1,2,3-cd)pyrene	3.3	100	1	1.0	5	25	1.00E-06	70	9,125	6.46E-08	NA	
Phenanthrene	4.5	100	1	1.0	5	25	1.00E-06	70	9,125	8.81E-08	NA	
Pesticides/PCBs												
Aroclor-1248	210	100	1	1.0	5	25	1.00E-06	70	9,125	4.11E-06	NA	
Aroclor-1254	270	100	1	1.0	5	25	1.00E-06	70	9,125	5.28E-06	2.00E-05	2.6E-01
Aroclor-1260	170	100	1	1.0	5	25	1.00E-06	70	9,125	3.33E-06	NA	
Dieldrin	0.44	100	1	1.0	5	25	1.00E-06	70	9,125	8.61E-09	5.00E-05	1.7E-04
Heptachlor Epoxide	0.22	100	1	1.0	5	25	1.00E-06	70	9,125	4.21E-09	1.30E-05	3.2E-04
Endrin aldehyde	3.2	100	1	1.0	5	25	1.00E-06	70	9,125	6.26E-08	3.00E-04	2.1E-04
Dioxins/Furans												
Total Equivalent 2,3,7,8-TCDD	0.0016	100	1	1.0	5	25	1.00E-06	70	9,125	3.06E-11	NA	
Inorganic Chemicals												
Aluminum	11,000	100	1	1.0	5	25	1.00E-06	70	9,125	2.15E-04	1.00E+00	2.2E-04
Antimony	29.8	100	1	1.0	5	25	1.00E-06	70	9,125	5.83E-07	4.00E-04	1.5E-03
Arsenic	48	100	1	1.0	5	25	1.00E-06	70	9,125	9.41E-07	3.00E-04	3.1E-03
Beryllium	1.6	100	1	1.0	5	25	1.00E-06	70	9,125	3.13E-08	5.00E-03	6.3E-06
Cadmium	26	100	1	1.0	5	25	1.00E-06	70	9,125	5.07E-07	5.00E-04	1.0E-03
Chromium	81	100	1	1.0	5	25	1.00E-06	70	9,125	1.58E-06	5.00E-03	3.2E-04
Copper	972	100	1	1.0	5	25	1.00E-06	70	9,125	1.90E-05	4.00E-02	4.8E-04
Iron	59,800	100	1	1.0	5	25	1.00E-06	70	9,125	1.17E-03	3.00E-01	3.9E-03
Lead	624	100	1	1.0	5	25	1.00E-06	70	9,125	1.22E-05	NA	
Manganese	1,215	100	1	1.0	5	25	1.00E-06	70	9,125	2.38E-05	2.30E-02	1.0E-03
Mercury	0.95	100	1	1.0	5	25	1.00E-06	70	9,125	1.86E-08	1.00E-04	1.9E-04
Thallium	3.6	100	1	1.0	5	25	1.00E-06	70	9,125	7.05E-08	8.00E-05	8.8E-04
Zinc	15,600	100	1	1.0	5	25	1.00E-06	70	9,125	3.05E-04	3.00E-01	1.0E-03
											Sum =	2.8E-01

Table H-45

Sector: Railroad ROW
Scenario: Trespasser
Pathway: Incidental Ingestion of Surface Soil
Receptor: Child (7 to 12 years)

CARCINOGENIC

CDI = Cs x IgR x IgRF x ROA x EF x ED x CF x 1/BW x 1/ATc

Risk = CDI x CSF

Chemical	Cs Surface Soil Concentration (mg/kg)	IgR Soil Ingestion Rate (mg/d)	IgRF Fraction Ingested Attributable to Site (unitless)	ROA Relative Oral Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals												
Acenaphthylene	0.059	200	0.5	1.0	78	6	1.00E-06	33	25550	3.27E-09	NA	
Benzo(g,h,i)perylene	3.0	200	0.5	1.0	78	6	1.00E-06	33	25550	1.64E-07	NA	
Benzo(a)anthracene	5.1	200	0.5	1.0	78	6	1.00E-06	33	25550	2.80E-07	7.30E-01	2E-07
Benzo(a)pyrene	4.8	200	0.5	1.0	78	6	1.00E-06	33	25550	2.66E-07	7.30E+00	2E-06
Benzo(b)fluoranthene	8.0	200	0.5	1.0	78	6	1.00E-06	33	25550	4.44E-07	7.30E-01	3E-07
Dibenz(a,h)anthracene	0.2	200	0.5	1.0	78	6	1.00E-06	33	25550	8.88E-09	7.30E+00	6E-08
Indeno(1,2,3-cd)pyrene	3.3	200	0.5	1.0	78	6	1.00E-06	33	25550	1.83E-07	NA	
Phenanthrene	4.5	200	0.5	1.0	78	6	1.00E-06	33	25550	2.50E-07	NA	
Pesticides/PCBs												
Aroclor-1248	210	200	0.5	1.0	78	6	1.00E-06	33	25550	1.17E-05	2.00E+00	2E-05
Aroclor-1254	270	200	0.5	1.0	78	6	1.00E-06	33	25550	1.50E-05	2.00E+00	3E-05
Aroclor-1260	170	200	0.5	1.0	78	6	1.00E-06	33	25550	9.44E-06	2.00E+00	2E-05
Dieldrin	0.44	200	0.5	1.0	78	6	1.00E-06	33	25550	2.44E-08	1.60E+01	4E-07
Heptachlor Epoxide	0.22	200	0.5	1.0	78	6	1.00E-06	33	25550	1.19E-08	9.10E+00	1E-07
Endrin aldehyde	3.2	200	0.5	1.0	78	6	1.00E-06	33	25550	1.78E-07	NA	
Dioxins/Furans												
Total Equivalent 2,3,7,8-TCDD	0.0016	200	0.5	1.0	78	6	1.00E-06	33	25550	8.67E-11	1.50E+05	1E-05
Inorganic Chemicals												
Aluminum	11,000	200	0.5	1.0	78	6	1.00E-06	33	25550	6.11E-04	NA	
Antimony	29.8	200	0.5	1.0	78	6	1.00E-06	33	25550	1.65E-06	NA	
Arsenic	48	200	0.5	1.0	78	6	1.00E-06	33	25550	2.67E-06	1.50E+00	4E-06
Beryllium	1.6	200	0.5	1.0	78	6	1.00E-06	33	25550	8.88E-08	4.30E+00	4E-07
Cadmium	26	200	0.5	1.0	78	6	1.00E-06	33	25550	1.44E-06	NA	
Chromium	81	200	0.5	1.0	78	6	1.00E-06	33	25550	4.48E-06	NA	
Copper	972	200	0.5	1.0	78	6	1.00E-06	33	25550	5.40E-05	NA	
Iron	59,800	200	0.5	1.0	78	6	1.00E-06	33	25550	3.32E-03	NA	
Lead	624	200	0.5	1.0	78	6	1.00E-06	33	25550	3.46E-05	NA	
Manganese	1,215	200	0.5	1.0	78	6	1.00E-06	33	25550	6.74E-05	NA	
Mercury	0.95	200	0.5	1.0	78	6	1.00E-06	33	25550	5.27E-08	NA	
Thallium	3.6	200	0.5	1.0	78	6	1.00E-06	33	25550	2.00E-07	NA	
Zinc	15,600	200	0.5	1.0	78	6	1.00E-06	33	25550	8.66E-04	NA	
											Sum =	9E-05

Table H-46

Sector: Railroad ROW
Scenario: Trespasser
Pathway: Incidental Ingestion of Surface Soil
Receptor: Child (7 to 12 years)

NONCARCINOGENIC
CDI = Cs x IgR x IgRF x ROA x EF x ED x CF x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	Cs Surface Soil Concentration (mg/kg)	IgR Soil Ingestion Rate (mg/d)	IgRF Fraction Ingested Attributable to Site (unitless)	ROA Relative Oral Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals												
Acenaphthylene	0.059	200	0.5	1.0	78	6	1.00E-06	33	2,190	3.62E-08	NA	
Benzo(g,h,i)perylene	3.0	200	0.5	1.0	78	6	1.00E-06	33	2,190	1.91E-06	NA	
Benzo(a)anthracene	5.1	200	0.5	1.0	78	6	1.00E-06	33	2,190	3.27E-06	NA	
Benzo(a)pyrene	4.8	200	0.5	1.0	78	6	1.00E-06	33	2,190	3.11E-06	NA	
Benzo(b)fluoranthene	8.0	200	0.5	1.0	78	6	1.00E-06	33	2,190	5.18E-06	NA	
Dibenz(a,h)anthracene	0.2	200	0.5	1.0	78	6	1.00E-06	33	2,190	1.04E-07	NA	
Indeno(1,2,3-cd)pyrene	3.3	200	0.5	1.0	78	6	1.00E-06	33	2,190	2.14E-06	NA	
Phenanthrene	4.5	200	0.5	1.0	78	6	1.00E-06	33	2,190	2.91E-06	NA	
Pesticides/PCBs												
Aroclor-1248	210	200	0.5	1.0	78	6	1.00E-06	33	2,190	1.36E-04	NA	
Aroclor-1254	270	200	0.5	1.0	78	6	1.00E-06	33	2,190	1.75E-04	2.00E-05	8.7E+00
Aroclor-1260	170	200	0.5	1.0	78	6	1.00E-06	33	2,190	1.10E-04	NA	
Dieldrin	0.44	200	0.5	1.0	78	6	1.00E-06	33	2,190	2.85E-07	5.00E-05	5.7E-03
Heptachlor Epoxide	0.22	200	0.5	1.0	78	6	1.00E-06	33	2,190	1.39E-07	1.30E-05	1.1E-02
Endrin aldehyde	3.2	200	0.5	1.0	78	6	1.00E-06	33	2,190	2.07E-06	3.00E-04	6.9E-03
Dioxins/Furans												
Total Equivalent 2,3,7,8-TCDD	0.0016	200	0.5	1.0	78	6	1.00E-06	33	2,190	1.01E-09	NA	
Inorganic Chemicals												
Aluminum	11,000	200	0.5	1.0	78	6	1.00E-06	33	2,190	7.12E-03	1.00E+00	7.1E-03
Antimony	29.8	200	0.5	1.0	78	6	1.00E-06	33	2,190	1.93E-05	4.00E-04	4.8E-02
Arsenic	48	200	0.5	1.0	78	6	1.00E-06	33	2,190	3.11E-05	3.00E-04	1.0E-01
Beryllium	1.6	200	0.5	1.0	78	6	1.00E-06	33	2,190	1.04E-06	5.00E-03	2.1E-04
Cadmium	26	200	0.5	1.0	78	6	1.00E-06	33	2,190	1.68E-05	5.00E-04	3.4E-02
Chromium	81	200	0.5	1.0	78	6	1.00E-06	33	2,190	5.23E-05	5.00E-03	1.0E-02
Copper	972	200	0.5	1.0	78	6	1.00E-06	33	2,190	6.29E-04	4.00E-02	1.6E-02
Iron	59,800	200	0.5	1.0	78	6	1.00E-06	33	2,190	3.87E-02	3.00E-01	1.3E-01
Lead	624	200	0.5	1.0	78	6	1.00E-06	33	2,190	4.04E-04	NA	
Manganese	1,215	200	0.5	1.0	78	6	1.00E-06	33	2,190	7.87E-04	2.30E-02	3.4E-02
Mercury	0.95	200	0.5	1.0	78	6	1.00E-06	33	2,190	6.15E-07	1.00E-04	6.2E-03
Thallium	3.6	200	0.5	1.0	78	6	1.00E-06	33	2,190	2.33E-06	8.00E-05	2.9E-02
Zinc	15,600	200	0.5	1.0	78	6	1.00E-06	33	2,190	1.01E-02	3.00E-01	3.4E-02
											Sum =	9.2E+00

Table H-47

Sector: Railroad ROW
Scenario: Trespasser
Pathway: Incidental Ingestion of Surface Soil
Receptor: Adolescent (13 to 18 years)

CARCINOGENIC

CDI = Cs x IgR x IgRF x ROA x EF x ED x CF x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	Cs Surface Soil Concentration (mg/kg)	IgR Soil Ingestion Rate (mg/d)	IgRF Fraction Ingested Attributable to Site (unitless)	ROA Relative Oral Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals												
Acenaphthylene	0.059	100	0.5	1.0	78	6	1.00E-06	56	25550	9.65E-10	NA	
Benzo(g,h,i)perylene	3.0	100	0.5	1.0	78	6	1.00E-06	56	25550	4.82E-08	NA	
Benzo(a)anthracene	5.1	100	0.5	1.0	78	6	1.00E-06	56	25550	8.26E-08	7.30E-01	6E-08
Benzo(a)pyrene	4.8	100	0.5	1.0	78	6	1.00E-06	56	25550	7.85E-08	7.30E+00	6E-07
Benzo(b)fluoranthene	8.0	100	0.5	1.0	78	6	1.00E-06	56	25550	1.31E-07	7.30E-01	1E-07
Dibenz(a,h)anthracene	0.2	100	0.5	1.0	78	6	1.00E-06	56	25550	2.62E-09	7.30E+00	2E-08
Indeno(1,2,3-cd)pyrene	3.3	100	0.5	1.0	78	6	1.00E-06	56	25550	5.40E-08	NA	
Phenanthrene	4.5	100	0.5	1.0	78	6	1.00E-06	56	25550	7.36E-08	NA	
Pesticides/PCBs												
Aroclor-1248	210	100	0.5	1.0	78	6	1.00E-06	56	25550	3.43E-06	2.00E+00	7E-06
Aroclor-1254	270	100	0.5	1.0	78	6	1.00E-06	56	25550	4.42E-06	2.00E+00	9E-06
Aroclor-1260	170	100	0.5	1.0	78	6	1.00E-06	56	25550	2.78E-06	2.00E+00	6E-06
Dieldrin	0.44	100	0.5	1.0	78	6	1.00E-06	56	25550	7.20E-09	1.60E+01	1E-07
Heptachlor Epoxide	0.22	100	0.5	1.0	78	6	1.00E-06	56	25550	3.52E-09	9.10E+00	3E-08
Endrin aldehyde	3.2	100	0.5	1.0	78	6	1.00E-06	56	25550	5.23E-08	NA	
Dioxins/Furans												
Total Equivalent 2,3,7,8-TCDD	0.0016	100	0.5	1.0	78	6	1.00E-06	56	25550	2.55E-11	1.50E+05	4E-06
Inorganic Chemicals												
Aluminum	11,000	100	0.5	1.0	78	6	1.00E-06	56	25550	1.80E-04	NA	
Antimony	29.8	100	0.5	1.0	78	6	1.00E-06	56	25550	4.87E-07	NA	
Arsenic	48	100	0.5	1.0	78	6	1.00E-06	56	25550	7.87E-07	1.50E+00	1E-06
Beryllium	1.6	100	0.5	1.0	78	6	1.00E-06	56	25550	2.62E-08	4.30E+00	1E-07
Cadmium	26	100	0.5	1.0	78	6	1.00E-06	56	25550	4.24E-07	NA	
Chromium	81	100	0.5	1.0	78	6	1.00E-06	56	25550	1.32E-06	NA	
Copper	972	100	0.5	1.0	78	6	1.00E-06	56	25550	1.59E-05	NA	
Iron	59,800	100	0.5	1.0	78	6	1.00E-06	56	25550	9.78E-04	NA	
Lead	624	100	0.5	1.0	78	6	1.00E-06	56	25550	1.02E-05	NA	
Manganese	1,215	100	0.5	1.0	78	6	1.00E-06	56	25550	1.99E-05	NA	
Mercury	0.95	100	0.5	1.0	78	6	1.00E-06	56	25550	1.55E-08	NA	
Thallium	3.6	100	0.5	1.0	78	6	1.00E-06	56	25550	5.89E-08	NA	
Zinc	15,600	100	0.5	1.0	78	6	1.00E-06	56	25550	2.55E-04	NA	
											Sum =	3E-05

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Table H-48

Sector: Railroad ROW
Scenario: Trespasser
Pathway: Incidental Ingestion of Surface Soil
Receptor: Adolescent (13 to 18 years)

NONCARCINOGENIC
CDI = Cs x IgR x IgRF x ROA x EF x ED x CF x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	Cs Surface soil Concentration (mg/kg)	IgR Soil Ingestion Rate (mg/d)	IgRF Fraction Ingested Attributable to Site (unitless)	ROA Relative Oral Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals												
Acenaphthylene	0.059	100	0.5	1.0	78	6	1.00E-06	56	2,190	1.13E-08	NA	
Benzo(g,h,i)perylene	3.0	100	0.5	1.0	78	6	1.00E-06	56	2,190	5.63E-07	NA	
Benzo(a)anthracene	5.1	100	0.5	1.0	78	6	1.00E-06	56	2,190	9.64E-07	NA	
Benzo(a)pyrene	4.8	100	0.5	1.0	78	6	1.00E-06	56	2,190	9.16E-07	NA	
Benzo(b)fluoranthene	8.0	100	0.5	1.0	78	6	1.00E-06	56	2,190	1.53E-06	NA	
Dibenz(a,h)anthracene	0.2	100	0.5	1.0	78	6	1.00E-06	56	2,190	3.05E-08	NA	
Indeno(1,2,3-cd)pyrene	3.3	100	0.5	1.0	78	6	1.00E-06	56	2,190	6.30E-07	NA	
Phenanthrene	4.5	100	0.5	1.0	78	6	1.00E-06	56	2,190	8.59E-07	NA	
Pesticides/PCBs												
Aroclor-1248	210	100	0.5	1.0	78	6	1.00E-06	56	2,190	4.01E-05	NA	
Aroclor-1254	270	100	0.5	1.0	78	6	1.00E-06	56	2,190	5.15E-05	2.00E-05	2.6E+00
Aroclor-1260	170	100	0.5	1.0	78	6	1.00E-06	56	2,190	3.24E-05	NA	
Dieldrin	0.44	100	0.5	1.0	78	6	1.00E-06	56	2,190	8.40E-08	5.00E-05	1.7E-03
Heptachlor Epoxide	0.22	100	0.5	1.0	78	6	1.00E-06	56	2,190	4.10E-08	1.30E-05	3.2E-03
Endrin aldehyde	3.2	100	0.5	1.0	78	6	1.00E-06	56	2,190	6.11E-07	3.00E-04	2.0E-03
Dioxins/Furans												
Total Equivalent 2,3,7,8-TCDD	0.0016	100	0.5	1.0	78	6	1.00E-06	56	2,190	2.98E-10	NA	
Inorganic Chemicals												
Aluminum	11,000	100	0.5	1.0	78	6	1.00E-06	56	2,190	2.10E-03	1.00E+00	2.1E-03
Antimony	29.8	100	0.5	1.0	78	6	1.00E-06	56	2,190	5.69E-06	4.00E-04	1.4E-02
Arsenic	48	100	0.5	1.0	78	6	1.00E-06	56	2,190	9.18E-06	3.00E-04	3.1E-02
Beryllium	1.6	100	0.5	1.0	78	6	1.00E-06	56	2,190	3.05E-07	5.00E-03	6.1E-05
Cadmium	26	100	0.5	1.0	78	6	1.00E-06	56	2,190	4.94E-06	5.00E-04	9.9E-03
Chromium	81	100	0.5	1.0	78	6	1.00E-06	56	2,190	1.54E-05	5.00E-03	3.1E-03
Copper	972	100	0.5	1.0	78	6	1.00E-06	56	2,190	1.85E-04	4.00E-02	4.6E-03
Iron	59,800	100	0.5	1.0	78	6	1.00E-06	56	2,190	1.14E-02	3.00E-01	3.8E-02
Lead	624	100	0.5	1.0	78	6	1.00E-06	56	2,190	1.19E-04	NA	
Manganese	1,215	100	0.5	1.0	78	6	1.00E-06	56	2,190	2.32E-04	2.30E-02	1.0E-02
Mercury	0.95	100	0.5	1.0	78	6	1.00E-06	56	2,190	1.81E-07	1.00E-04	1.8E-03
Thallium	3.6	100	0.5	1.0	78	6	1.00E-06	56	2,190	6.87E-07	8.00E-05	8.6E-03
Zinc	15,600	100	0.5	1.0	78	6	1.00E-06	56	2,190	2.98E-03	3.00E-01	9.9E-03
											Sum =	2.7E+00

Table H-49

Sector: Railroad ROW
Scenario: Trespasser
Pathway: Dermal Contact with Surface Soil
Receptor: Child (7 to 12 years)

CARCINOGENIC
CDI = Cs x DAF x SA x FSA x RDA x EF x ED x CF x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	Cs Surface Soil Concentration (mg/kg)	DAF Dermal Adherence Factor (mg/cm^2-d)	SA Total Body Surface Area (cm^2)	SAF Fraction SA Exposed (unitless)	RDA Relative Dermal Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals													
Acenaphthylene	0.059	0.2	10,500	0.22	0.10	78	6	1.00E-06	33	25550	1.51E-09	NA	
Benzo(g,h,i)perylene	3.0	0.2	10,500	0.22	0.10	78	6	1.00E-06	33	25550	7.56E-08	NA	
Benzo(a)anthracene	5.1	0.2	10,500	0.22	0.10	78	6	1.00E-06	33	25550	1.30E-07	7.30E-01	9E-08
Benzo(a)pyrene	4.8	0.2	10,500	0.22	0.10	78	6	1.00E-06	33	25550	1.23E-07	7.30E+00	9E-07
Benzo(b)fluoranthene	8.0	0.2	10,500	0.22	0.10	78	6	1.00E-06	33	25550	2.05E-07	7.30E-01	1E-07
Dibenz(a,h)anthracene	0.2	0.2	10,500	0.22	0.10	78	6	1.00E-06	33	25550	4.10E-09	7.30E+00	3E-08
Indeno(1,2,3-cd)pyrene	3.3	0.2	10,500	0.22	0.10	78	6	1.00E-06	33	25550	8.46E-08	NA	
Phenanthrene	4.5	0.2	10,500	0.22	0.10	78	6	1.00E-06	33	25550	1.15E-07	NA	
Pesticides/PCBs													
Aroclor-1248	210	0.2	10,500	0.22	0.06	78	6	1.00E-06	33	25550	3.23E-06	2.00E+00	6E-06
Aroclor-1254	270	0.2	10,500	0.22	0.06	78	6	1.00E-06	33	25550	4.15E-06	2.00E+00	8E-06
Aroclor-1260	170	0.2	10,500	0.22	0.06	78	6	1.00E-06	33	25550	2.62E-06	2.00E+00	5E-06
Dieldrin	0.44	0.2	10,500	0.22	0.10	78	6	1.00E-06	33	25550	1.13E-08	1.60E+01	2E-07
Heptachlor Epoxide	0.22	0.2	10,500	0.22	0.10	78	6	1.00E-06	33	25550	5.51E-09	9.10E+00	5E-08
Endrin aldehyde	3.2	0.2	10,500	0.22	0.10	78	6	1.00E-06	33	25550	8.21E-08	NA	
Dioxins/Furans													2E-05
Total Equivalent 2,3,7,8-TCDD	0.0016	0.2	10,500	0.22	0.03	78	6	1.00E-06	33	25550	1.20E-11	1.50E+05	2E-06
Inorganic Chemicals													
Aluminum	11,000	0.2	10,500	0.22	0.01	78	6	1.00E-06	33	25550	2.82E-05	NA	
Antimony	29.8	0.2	10,500	0.22	0.01	78	6	1.00E-06	33	25550	7.64E-08	NA	
Arsenic	48	0.2	10,500	0.22	0.032	78	6	1.00E-06	33	25550	3.95E-07	1.50E+00	6E-07
Beryllium	1.6	0.2	10,500	0.22	0.25	78	6	1.00E-06	33	25550	1.03E-07	4.30E+00	4E-07
Cadmium	26	0.2	10,500	0.22	0.01	78	6	1.00E-06	33	25550	6.64E-08	NA	
Chromium	81	0.2	10,500	0.22	0.01	78	6	1.00E-06	33	25550	2.07E-07	NA	
Copper	972	0.2	10,500	0.22	0.01	78	6	1.00E-06	33	25550	2.49E-06	NA	
Iron	59,800	0.2	10,500	0.22	0.01	78	6	1.00E-06	33	25550	1.53E-04	NA	
Lead	624	0.2	10,500	0.22	0.02	78	6	1.00E-06	33	25550	3.20E-06	NA	
Manganese	1,215	0.2	10,500	0.22	0.20	78	6	1.00E-06	33	25550	6.23E-05	NA	
Mercury	0.95	0.2	10,500	0.22	0.20	78	6	1.00E-06	33	25550	4.87E-08	NA	
Thallium	3.6	0.2	10,500	0.22	0.01	78	6	1.00E-06	33	25550	9.23E-09	NA	
Zinc	15,600	0.2	10,500	0.22	0.01	78	6	1.00E-06	33	25550	4.00E-05	NA	
												Sum =	5E-05

Table H-50

Sector: Railroad ROW
Scenario: Trespasser
Pathway: Dermal Contact with Surface Soil
Receptor: Child (7 to 12 years)

NONCARCINOGENIC
CDI = Cs x DAF x SA x FSA x RDA x EF x ED x CF x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	Cs Surface Soil Concentration (mg/kg)	DAF Dermal Adherence Factor (mg/cm^2-d)	SA Total Body Surface Area (cm^2)	SAF Fraction SA Exposed (unitless)	RDA Relative Dermal Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals													
Acenaphthylene	0.059	0.2	10,500	0.22	0.10	78	6	1.00E-06	33	2,190	1.77E-08	NA	
Benzo(g,h,i)perylene	3.0	0.2	10,500	0.22	0.10	78	6	1.00E-06	33	2,190	8.83E-07	NA	
Benzo(a)anthracene	5.1	0.2	10,500	0.22	0.10	78	6	1.00E-06	33	2,190	1.51E-06	NA	
Benzo(a)pyrene	4.8	0.2	10,500	0.22	0.10	78	6	1.00E-06	33	2,190	1.44E-06	NA	
Benzo(b)fluoranthene	8.0	0.2	10,500	0.22	0.10	78	6	1.00E-06	33	2,190	2.39E-06	NA	
Dibenz(a,h)anthracene	0.2	0.2	10,500	0.22	0.10	78	6	1.00E-06	33	2,190	4.79E-08	NA	
Indeno(1,2,3-cd)pyrene	3.3	0.2	10,500	0.22	0.10	78	6	1.00E-06	33	2,190	9.87E-07	NA	
Phenanthrene	4.5	0.2	10,500	0.22	0.10	78	6	1.00E-06	33	2,190	1.35E-06	NA	
Pesticides/PCBs													
Aroclor-1248	210	0.2	10,500	0.22	0.06	78	6	1.00E-06	33	2,190	3.77E-05	NA	
Aroclor-1254	270	0.2	10,500	0.22	0.06	78	6	1.00E-06	33	2,190	4.85E-05	2.00E-05	2.4E+00
Aroclor-1260	170	0.2	10,500	0.22	0.06	78	6	1.00E-06	33	2,190	3.05E-05	NA	
Dieldrin	0.44	0.2	10,500	0.22	0.10	78	6	1.00E-06	33	2,190	1.32E-07	5.00E-05	2.6E-03
Heptachlor Epoxide	0.22	0.2	10,500	0.22	0.10	78	6	1.00E-06	33	2,190	6.43E-08	1.30E-05	4.9E-03
Endrin aldehyde	3.2	0.2	10,500	0.22	0.10	78	6	1.00E-06	33	2,190	9.57E-07	3.00E-04	3.2E-03
Dioxins/Furans													
Total Equivalent 2,3,7,8-TCDD	0.0016	0.2	10,500	0.22	0.03	78	6	1.00E-06	33	2,190	1.40E-10	NA	
Inorganic Chemicals													
Aluminum	11,000	0.2	10,500	0.22	0.01	78	6	1.00E-06	33	2,190	3.29E-04	1.00E+00	3.3E-04
Antimony	29.8	0.2	10,500	0.22	0.01	78	6	1.00E-06	33	2,190	8.92E-07	4.00E-04	2.2E-03
Arsenic	48	0.2	10,500	0.22	0.032	78	6	1.00E-06	33	2,190	4.60E-06	3.00E-04	1.5E-02
Beryllium	1.6	0.2	10,500	0.22	0.25	78	6	1.00E-06	33	2,190	1.20E-06	5.00E-03	2.4E-04
Cadmium	26	0.2	10,500	0.22	0.01	78	6	1.00E-06	33	2,190	7.75E-07	5.00E-04	1.5E-03
Chromium	81	0.2	10,500	0.22	0.01	78	6	1.00E-06	33	2,190	2.42E-06	5.00E-03	4.8E-04
Copper	972	0.2	10,500	0.22	0.01	78	6	1.00E-06	33	2,190	2.91E-05	4.00E-02	7.3E-04
Iron	59,800	0.2	10,500	0.22	0.01	78	6	1.00E-06	33	2,190	1.79E-03	3.00E-01	6.0E-03
Lead	624	0.2	10,500	0.22	0.02	78	6	1.00E-06	33	2,190	3.73E-05	NA	
Manganese	1,215	0.2	10,500	0.22	0.20	78	6	1.00E-06	33	2,190	7.27E-04	2.30E-02	3.2E-02
Mercury	0.95	0.2	10,500	0.22	0.01	78	6	1.00E-06	33	2,190	2.84E-08	1.00E-04	2.8E-04
Thallium	3.6	0.2	10,500	0.22	0.01	78	6	1.00E-06	33	2,190	1.08E-07	8.00E-05	1.3E-03
Zinc	15,600	0.2	10,500	0.22	0.01	78	6	1.00E-06	33	2,190	4.67E-04	3.00E-01	1.6E-03
												Sum =	2.5E+00

Table H-51

Sector: Railroad ROW
Scenario: Trespasser
Pathway: Dermal Contact with Surface Soil
Receptor: Adolescent (13 to 18 years)

CARCINOGENIC

CDI = Cs x DAF x SA x FSA x RDA x EF x ED x CF x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	Cs Surface Soil Concentration (mg/kg)	DAF Dermal Adherence Factor (mg/cm^2-d)	SA Total Body Surface Area (cm^2)	SAF Fraction SA Exposed (unitless)	RDA Relative Dermal Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATc Averaging Time Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	CSF Cancer Slope Factor (mg/kg-d)^-1	Risk
Semivolatile Organic Chemicals													
Acenaphthylene	0.059	0.2	16,000	0.23	0.10	78	6	1.00E-06	56	25550	1.42E-09	NA	
Benzo(g,h,i)perylene	3.0	0.2	16,000	0.23	0.10	78	6	1.00E-06	56	25550	7.10E-08	NA	
Benzo(a)anthracene	5.1	0.2	16,000	0.23	0.10	78	6	1.00E-06	56	25550	1.22E-07	7.30E-01	9E-08
Benzo(a)pyrene	4.8	0.2	16,000	0.23	0.10	78	6	1.00E-06	56	25550	1.16E-07	7.30E+00	8E-07
Benzo(b)fluoranthene	8.0	0.2	16,000	0.23	0.10	78	6	1.00E-06	56	25550	1.93E-07	7.30E-01	1E-07
Dibenz(a,h)anthracene	0.2	0.2	16,000	0.23	0.10	78	6	1.00E-06	56	25550	3.85E-09	7.30E+00	3E-08
Indeno(1,2,3-cd)pyrene	3.3	0.2	16,000	0.23	0.10	78	6	1.00E-06	56	25550	7.94E-08	NA	
Phenanthrene	4.5	0.2	16,000	0.23	0.10	78	6	1.00E-06	56	25550	1.08E-07	NA	
Pesticides/PCBs													
Aroclor-1248	210	0.2	16,000	0.23	0.06	78	6	1.00E-06	56	25550	3.03E-06	2.00E+00	6E-06
Aroclor-1254	270	0.2	16,000	0.23	0.06	78	6	1.00E-06	56	25550	3.90E-06	2.00E+00	8E-06
Aroclor-1260	170	0.2	16,000	0.23	0.06	78	6	1.00E-06	56	25550	2.46E-06	2.00E+00	5E-06
Dieldrin	0.44	0.2	16,000	0.23	0.10	78	6	1.00E-06	56	25550	1.06E-08	1.60E+01	2E-07
Heptachlor Epoxide	0.22	0.2	16,000	0.23	0.10	78	6	1.00E-06	56	25550	5.18E-09	9.10E+00	5E-08
Endrin aldehyde	3.2	0.2	16,000	0.23	0.10	78	6	1.00E-06	56	25550	7.70E-08	NA	
Dioxins/Furans													
Total Equivalent 2,3,7,8-TCDD	0.0016	0.2	16,000	0.23	0.03	78	6	1.00E-06	56	25550	1.13E-11	1.50E+05	2E-06
Inorganic Chemicals													
Aluminum	11,000	0.2	16,000	0.23	0.01	78	6	1.00E-06	56	25550	2.65E-05	NA	
Antimony	29.8	0.2	16,000	0.23	0.01	78	6	1.00E-06	56	25550	7.17E-08	NA	
Arsenic	48	0.2	16,000	0.23	0.032	78	6	1.00E-06	56	25550	3.71E-07	1.50E+00	6E-07
Beryllium	1.6	0.2	16,000	0.23	0.25	78	6	1.00E-06	56	25550	9.63E-08	4.30E+00	4E-07
Cadmium	26	0.2	16,000	0.23	0.01	78	6	1.00E-06	56	25550	6.24E-08	NA	
Chromium	81	0.2	16,000	0.23	0.01	78	6	1.00E-06	56	25550	1.95E-07	NA	
Copper	972	0.2	16,000	0.23	0.01	78	6	1.00E-06	56	25550	2.34E-06	NA	
Iron	59,800	0.2	16,000	0.23	0.01	78	6	1.00E-06	56	25550	1.44E-04	NA	
Lead	624	0.2	16,000	0.23	0.02	78	6	1.00E-06	56	25550	3.00E-06	NA	
Manganese	1,215	0.2	16,000	0.23	0.20	78	6	1.00E-06	56	25550	5.85E-05	NA	
Mercury	0.95	0.2	16,000	0.23	0.01	78	6	1.00E-06	56	25550	2.29E-09	NA	
Thallium	3.6	0.2	16,000	0.23	0.01	78	6	1.00E-06	56	25550	8.67E-09	NA	
Zinc	15,600	0.2	16,000	0.23	0.01	78	6	1.00E-06	56	25550	3.76E-05	NA	
												Sum =	2E-05

Table H-52

Sector: Railroad ROW
Scenario: Trespasser
Pathway: Dermal Contact with Surface Soil
Receptor: Adolescent (13 to 18 years)

NONCARCINOGENIC
CDI = Cs x DAF x SA x FSA x RDA x EF x ED x CF x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	Cs Surface Soil Concentration (mg/kg)	DAF Dermal Adherence Factor (mg/cm^2-d)	SA Total Body Surface Area (cm^2)	SAF Fraction SA Exposed (unitless)	RDA Relative Dermal Absorption (unitless)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF Conversion Factor (kg/mg)	BW Body Weight (kg)	ATnc Averaging Time Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organic Chemicals													
Acenaphthylene	0.059	0.2	16,000	0.23	0.10	78	6	1.00E-06	56	2,190	1.66E-08	NA	
Benzo(g,h,i)perylene	3.0	0.2	16,000	0.23	0.10	78	6	1.00E-06	56	2,190	8.29E-07	NA	
Benzo(a)anthracene	5.1	0.2	16,000	0.23	0.10	78	6	1.00E-06	56	2,190	1.42E-06	NA	
Benzo(a)pyrene	4.8	0.2	16,000	0.23	0.10	78	6	1.00E-06	56	2,190	1.35E-06	NA	
Benzo(b)fluoranthene	8.0	0.2	16,000	0.23	0.10	78	6	1.00E-06	56	2,190	2.25E-06	NA	
Dibenz(a,h)anthracene	0.2	0.2	16,000	0.23	0.10	78	6	1.00E-06	56	2,190	4.49E-08	NA	
Indeno(1,2,3-cd)pyrene	3.3	0.2	16,000	0.23	0.10	78	6	1.00E-06	56	2,190	9.27E-07	NA	
Phenanthrene	4.5	0.2	16,000	0.23	0.10	78	6	1.00E-06	56	2,190	1.26E-06	NA	
Pesticides/PCBs													
Aroclor-1248	210	0.2	16,000	0.23	0.06	78	6	1.00E-06	56	2,190	3.54E-05	NA	
Aroclor-1254	270	0.2	16,000	0.23	0.06	78	6	1.00E-06	56	2,190	4.55E-05	2.00E-05	2.3E+00
Aroclor-1260	170	0.2	16,000	0.23	0.06	78	6	1.00E-06	56	2,190	2.86E-05	NA	
Dieldrin	0.44	0.2	16,000	0.23	0.10	78	6	1.00E-06	56	2,190	1.24E-07	5.00E-05	2.5E-03
Heptachlor Epoxide	0.22	0.2	16,000	0.23	0.10	78	6	1.00E-06	56	2,190	6.04E-08	1.30E-05	4.6E-03
Endrin aldehyde	3.2	0.2	16,000	0.23	0.10	78	6	1.00E-06	56	2,190	8.99E-07	3.00E-04	3.0E-03
Dioxins/Furans													
Total Equivalent 2,3,7,8-TCDD	0.0016	0.2	16,000	0.23	0.03	78	6	1.00E-06	56	2,190	1.32E-10	NA	
Inorganic Chemicals													
Aluminum	11,000	0.2	16,000	0.23	0.01	78	6	1.00E-06	56	2,190	3.09E-04	1.00E+00	3.1E-04
Antimony	29.8	0.2	16,000	0.23	0.01	78	6	1.00E-06	56	2,190	8.37E-07	4.00E-04	2.1E-03
Arsenic	48	0.2	16,000	0.23	0.032	78	6	1.00E-06	56	2,190	4.32E-06	3.00E-04	1.4E-02
Beryllium	1.6	0.2	16,000	0.23	0.25	78	6	1.00E-06	56	2,190	1.12E-06	5.00E-03	2.2E-04
Cadmium	26	0.2	16,000	0.23	0.01	78	6	1.00E-06	56	2,190	7.27E-07	5.00E-04	1.5E-03
Chromium	81	0.2	16,000	0.23	0.01	78	6	1.00E-06	56	2,190	2.27E-06	5.00E-03	4.5E-04
Copper	972	0.2	16,000	0.23	0.01	78	6	1.00E-06	56	2,190	2.73E-05	4.00E-02	6.8E-04
Iron	59,800	0.2	16,000	0.23	0.01	78	6	1.00E-06	56	2,190	1.68E-03	3.00E-01	5.6E-03
Lead	624	0.2	16,000	0.23	0.02	78	6	1.00E-06	56	2,190	3.51E-05	NA	
Manganese	1,215	0.2	16,000	0.23	0.20	78	6	1.00E-06	56	2,190	6.82E-04	2.30E-02	3.0E-02
Mercury	0.95	0.2	16,000	0.23	0.01	78	6	1.00E-06	56	2,190	2.67E-08	1.00E-04	2.7E-04
Thallium	3.6	0.2	16,000	0.23	0.01	78	6	1.00E-06	56	2,190	1.01E-07	8.00E-05	1.3E-03
Zinc	15,600	0.2	16,000	0.23	0.01	78	6	1.00E-06	56	2,190	4.38E-04	3.00E-01	1.5E-03
												Sum =	2.3E+00

Table H-53

Sector: Railroad ROW
Scenario: Trespasser
Pathway: Dermal Contact with Pooled Storm Water Runoff
Receptor: Child (Ages 7 to 12)

CARCINOGENIC

CDI = Cw x PC x SA x FSA x t x EF x ED x CF1 x 1/BW x 1/ATc

Risk = CDI x CSF

Chemical	C_w Runoff Water Concentration (mg/L)	PC Permeability Coeficient (cm/hr)	SA Total Body Surface Area (cm2)	FSA Fraction SA Exposed (unitless)	t Time Spent in Puddle (hr/d)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (L/cm3)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	CSF Cancer Slope Factor $(mg/kg\text{-}day)^{-1}$	Risk
Volatile Organic Chemicals :													
Trichloroethene	0.092	1.60E-02	10500	0.22	2.6	4	6	0.001	33	25550	2.52E-07	1.10E-02	3E-09
Pesticides/PCBs													
Aroclor-1248	0.0028	7.10E-01	10500	0.22	2.6	4	6	0.001	33	25550	3.40E-07	2.00E+00	7E-07
Aroclor-1260	0.0082	7.10E-01	10500	0.22	2.6	4	6	0.001	33	25550	9.95E-07	2.00E+00	2E-06
												Sum	3E-06

Table H-54

Sector: Railroad ROW
Scenario: Trespasser
Pathway: Dermal Contact with Pooled Storm Water Runoff
Receptor: Child (Ages 7 to 12)

NON-CARCINOGENIC
CDI = Cw x PC x SA x FSA x t x EF x ED x CF1 x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	C_w Runoff Water Concentration (mg/L)	PC Permeability Coeficient (cm/hr)	SA Total Body Surface Area (cm2)	FSA Fraction SA Exposed (unitless)	t Time Spent in Puddle (hr/d)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (L/cm3)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Volatile Organic Chemicals :													
Trichloroethene	0.092	1.60E-02	10500	0.22	2.6	4	6	0.001	33	9125	7.05E-07	6.00E-03	1.2E-04
Pesticides/PCBs													
Aroclor-1248	0.0028	7.10E-01	10500	0.22	2.6	4	6	0.001	33	9125	9.52E-07	NA	
Aroclor-1260	0.0082	7.10E-01	10500	0.22	2.6	4	6	0.001	33	9125	2.79E-06	NA	
												Sum =	1.2E-04

Table H-55

Sector: Railroad ROW
Scenario: Trespasser
Pathway: Dermal Contact with Pooled Storm Water Runoff
Receptor: Child (Ages 12 to 18)

CARCINOGENIC

CDI = Cw x PC x SA x FSA x t x EF x ED x CF1 x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	C_w Runoff Water Concentration (mg/L)	PC Permeability Coeficient (cm/hr)	SA Total Body Surface Area (cm2)	FSA Fraction SA Exposed (unitless)	t Time Spent in Puddle (hr/d)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (L/cm3)	BW Body Weight (kg)	ATc raging Ti Carcinoge (days)	CDI Chronic Daily Intake (mg/kg-day)	CSF Cancer Slope Factor $(mg/kg\text{-}day)^{-1}$	Risk
Volatile Organic Chemicals :													
Trichloroethene	0.092	1.60E-02	16000	0.23	2.6	4	6	0.001	56	25550	2.36E-07	1.10E-02	3E-09
Pesticides/PCBs													
Aroclor-1248	0.0028	7.10E-01	16000	0.23	2.6	4	6	0.001	56	25550	3.19E-07	2.00E+00	6E-07
Aroclor-1260	0.0082	7.10E-01	16000	0.23	2.6	4	6	0.001	56	25550	9.34E-07	2.00E+00	2E-06
												Sum	3E-06

Table H-56

Sector: Railroad ROW
Scenario: Trespasser
Pathway: Dermal Contact with Pooled Storm Water Runoff
Receptor: Child (Ages 12 to 18)

NON-CARCINOGENIC
CDI = Cw x PC x SA x FSA x t x EF x ED x CF1 x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	C_w Runoff Water Concentration (mg/L)	PC Permeability Coeficient (cm/hr)	SA Total Body Surface Area (cm2)	FSA Fraction SA Exposed (unitless)	t Time Spent in Puddle (hr/d)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (L/cm3)	BW Body Weight (kg)	ATnc Averaging Ti Noncarcinog (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Volatile Organic Chemicals :													
Trichloroethene	0.092	1.60E-02	16000	0.23	2.6	4	6	0.001	56	9125	6.61E-07	6.00E-03	1.1E-04
Pesticides/PCBs													
Aroclor-1248	0.0028	7.10E-01	16000	0.23	2.6	4	6	0.001	56	9125	8.93E-07	NA	
Aroclor-1260	0.0082	7.10E-01	16000	0.23	2.6	4	6	0.001	56	9125	2.62E-06	NA	
												Sum =	1.1E-04

Table H-57

Alluvial Southern Groundwater
Scenario: Future Worker
Pathway: Inhalation of Groundwater Contaminants while Showering
Receptor: Hypothetical Worker (>18 years)

CARCINOGENIC
CDI =Ca x IhR x t x EF x ED x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	Ca Shower Air Concentration (mg/m3)	IhR Inhalation Rate (m3/min)	t Time Spent in Shower (min/day)	RIA Relative Inhalation Absorption (unitless)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	CSF Cancer Slope Factor $(mg/kg\text{-}day)^{-1}$	Risk
Volatile Organics :											
Vinyl Chloride	0.35	0.01	12	1.00	250	25	70	25550	1.45E-04	3.00E-01	4E-05
1,2-Dichloroethene (total)	76	0.01	12	1.00	250	25	70	25550	3.20E-02	NA	
1,1-Dichloroethene	0.046	0.01	12	1.00	250	25	70	25550	1.94E-05	1.75E-01	3E-06
Chlorobenzene	5.3	0.01	12	1.00	250	25	70	25550	2.23E-03	NA	
Trichloroethene	3.9	0.01	12	1.00	250	25	70	25550	1.65E-03	6.00E-03	1E-05
										Sum =	6E-05

Table H-58

Alluvial Southern Groundwater
Scenario: Future Worker
Pathway: Inhalation of Groundwater Contaminants while Showering
Receptor: Hypothetical Worker (>18 years)

NON-CARCINOGENIC
CDI =Ca x InR x t x EF x ED x 1/BW x 1/ATc
HQ = CDI/RfD

Chemical	Ca Shower Air Concentration (mg/m3)	InR Inhalation Rate (m3/min)	t Time Spent in Shower (min/day)	RIA Relative Inhalation Absorption (unitless)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	RfD Reference Dose (mg/kg-day)	HQ Hazard Quotient
Volatile Organics :											
Vinyl Chloride	0.35	0.01	12	1.00	250	25	70	9125	4.07E-04	NA	
1,2-Dichloroethene (total)	76	0.01	12	1.00	250	25	70	9125	8.95E-02	NA	
1,1-Dichloroethene	0.046	0.01	12	1.00	250	25	70	9125	5.42E-05	NA	
Chlorobenzene	5.3	0.01	12	1.00	250	25	70	9125	6.24E-03	5.71E-03	1.1E+00
Trichloroethene	3.9	0.01	12	1.00	250	25	70	9125	4.61E-03	NA	
										Sum =	1.1E+00

Table H-59

Alluvial Southern Groundwater
Scenario: Future Worker
Pathway: Dermal Contact with Groundwater Contaminants while Showering
Receptor: Hypothetical Worker (>18 years)

CARCINOGENIC
CDI = Cw x PC x SA x FSA x t x EF x ED x CF1 x CF2 x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	C_w Groundwater Concentration (mg/L)	PC Permeability Coefficient (cm/hr)	SA Total Body Surface Area (cm^2)	FSA Fraction SA Exposed (unitless)	t Time Spent in Shower (min/d)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (L/cm3)	CF2 Conversion Factor (hr/min)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	CSF Cancer Slope Factor $(mg/kg\text{-}day)^{-1}$	Risk
Volatile Organics :														
Vinyl Chloride	0.0064	7.30E-03	18000	1	12	250	25	0.001	0.017	70	25550	5.87E-07	1.90E+00	1E-06
1,2-Dichloroethene (total)	1.5	1.02E-02	18000	1	12	250	25	0.001	0.017	70	25550	1.92E-04	NA	
1,1-Dichloroethene	0.00094	1.60E-02	18000	1	12	250	25	0.001	0.017	70	25550	1.90E-07	6.00E-01	1E-07
Chlorobenzene	0.11	4.10E-02	18000	1	12	250	25	0.001	0.017	70	25550	5.85E-05	NA	
Trichloroethene	0.088	1.60E-02	18000	1	12	250	25	0.001	0.017	70	25550	1.76E-05	1.10E-02	2E-07
PCBs (ug/l):														
Aroclor-1254	0.014	7.10E-01	18000	1	12	250	25	0.001	0.017	70	25550	1.25E-04	2.00E+00	3E-04
Semivolatile Organics :														
1,3-Dichlorobenzene	0.56	8.70E-02	18000	1	12	250	25	0.001	0.017	70	25550	6.13E-04	NA	
1,4-Dichlorobenzene	0.45	6.20E-02	18000	1	12	250	25	0.001	0.017	70	25550	3.51E-04	2.40E-02	8E-06
1,2-Dichlorobenzene	0.15	6.10E-02	18000	1	12	250	25	0.001	0.017	70	25550	1.17E-04	NA	
1,2,4-Trichlorobenzene	0.11	1.00E-01	18000	1	12	250	25	0.001	0.017	70	25550	1.38E-04	NA	
2,4-Dichlorophenol	0.011	2.30E-02	18000	1	12	250	25	0.001	0.017	70	25550	3.11E-06	NA	
Inorganics														
Aluminum	98	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	1.23E-03	NA	
Arsenic	0.39	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	4.96E-06	1.50E+00	7E-06
Barium	0.90	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	1.13E-05	NA	
Beryllium	0.0069	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	8.68E-08	4.30E+00	4E-07
Cadmium	0.0046	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	5.76E-08	NA	
Chromium	0.48	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	6.06E-06	NA	
Copper	0.39	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	4.84E-06	NA	
Iron	308	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	3.87E-03	NA	
Lead	0.25	4.00E-06	18000	1	12	250	25	0.001	0.017	70	25550	1.24E-08	NA	
Manganese	7.5	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	9.37E-05	NA	
Mercury	0.0015	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	1.89E-08	NA	
Nickel	0.72	9.00E-06	18000	1	12	250	25	0.001	0.017	70	25550	8.12E-08	NA	
Vanadium	0.18	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	2.20E-06	NA	
Zinc	1.8	6.00E-04	18000	1	12	250	25	0.001	0.017	70	25550	1.35E-05	NA	
Dioxins														
Total Equivalent 2,3,7,8-TCDD	4.73E-09	1.40E+00	18000	1	12	250	25	0.001	0.017	70	25550	8.33E-11	1.50E+05	1E-05
													Sum	3E-04

Table H-60

Alluvial Southern Groundwater
Scenario: Future Worker
Pathway: Dermal Contact with Groundwater Contaminants while Showering
Receptor: Hypothetical Worker (>18 years)

NON-CARCINOGENIC
CDI = Cw x PC x SA x FSA x t x EF x ED x CF1 x CF2 x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	C_w Groundwater Concentration (mg/L)	PC Permeability Coefficient (cm/hr)	SA Total Body Surface Area (cm2)	FSA Fraction SA Exposed (unitless)	t Time Spent in Shower (min/d)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (L/cm3)	CF2 Conversion Factor (hr/min)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Volatile Organics :														
Vinyl Chloride	0.0064	7.30E-03	18000	1	12	250	25	0.001	0.017	70	9125	1.64E-06	NA	
1,2-Dichloroethene (total)	1.5	1.02E-02	18000	1	12	250	25	0.001	0.017	70	9125	5.38E-04	9.00E-03	6.0E-02
1,1-Dichloroethene	0.00094	1.60E-02	18000	1	12	250	25	0.001	0.017	70	9125	5.32E-07	9.00E-03	5.9E-05
Chlorobenzene	0.11	4.10E-02	18000	1	12	250	25	0.001	0.017	70	9125	1.64E-04	2.00E-02	8.2E-03
Trichloroethene	0.088	1.60E-02	18000	1	12	250	25	0.001	0.017	70	9125	4.93E-05	6.00E-03	8.2E-03
PCBs (ug/l):														
Aroclor-1254	0.014	7.10E-01	18000	1	12	250	25	0.001	0.017	70	9125	3.50E-04	2.00E-05	1.8E+01
Semivolatile Organics :														
1,3-Dichlorobenzene	0.56	8.70E-02	18000	1	12	250	25	0.001	0.017	70	9125	1.72E-03	8.90E-02	1.9E-02
1,4-Dichlorobenzene	0.45	6.20E-02	18000	1	12	250	25	0.001	0.017	70	9125	9.83E-04	NA	
1,2-Dichlorobenzene	0.15	6.10E-02	18000	1	12	250	25	0.001	0.017	70	9125	3.27E-04	9.00E-02	3.6E-03
1,2,4-Trichlorobenzene	0.11	1.00E-01	18000	1	12	250	25	0.001	0.017	70	9125	3.87E-04	1.00E-02	3.9E-02
2,4-Dichlorophenol	0.011	2.30E-02	18000	1	12	250	25	0.001	0.017	70	9125	8.72E-06	3.00E-03	2.9E-03
Inorganics														
Aluminum	98	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	3.45E-03	1.00E+00	3.4E-03
Arsenic	0.39	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	1.39E-05	3.00E-04	4.6E-02
Barium	0.90	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	3.17E-05	7.00E-02	4.5E-04
Beryllium	0.0069	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	2.43E-07	5.00E-03	4.9E-05
Cadmium	0.0046	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	1.61E-07	5.00E-04	3.2E-04
Chromium	0.48	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	1.70E-05	5.00E-03	3.4E-03
Copper	0.39	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	1.36E-05	4.00E-02	3.4E-04
Iron	308	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	1.08E-02	3.00E-01	3.6E-02
Lead	0.25	4.00E-06	18000	1	12	250	25	0.001	0.017	70	9125	3.47E-08	NA	
Mercury	0.0015	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	5.28E-08	1.00E-04	5.3E-04
Manganese	7.5	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	2.62E-04	2.30E-02	1.1E-02
Nickel	0.72	9.00E-06	18000	1	12	250	25	0.001	0.017	70	9125	2.27E-07	2.00E-02	1.1E-05
Vanadium	0.18	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	6.16E-06	7.00E-03	8.8E-04
Zinc	1.8	6.00E-04	18000	1	12	250	25	0.001	0.017	70	9125	3.78E-05	3.00E-01	1.3E-04
Dioxins														
Total Equivalent 2,3,7,8-TCDD	4.73E-09	1.40E+00	18000	1	12	250	25	0.001	0.017	70	9125	2.33E-10	NA	
													Sum =	1.8E+01

Table H-61

Alluvial Southern Groundwater
Scenario: Future Worker
Pathway: Ingestion of Groundwater
Receptor: Hypothetical Worker (>18 years)

CARCINOGENIC
CDI = Cw x IR x EF x ED x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	C_w Groundwater Concentration (mg/L)	IR Ingestion Rate (l/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	BW Body Weight (Kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	CSF Cancer Slope Factor $(mg/kg\text{-}day)^{-1}$	Risk
Volatile Organics :									
Vinyl Chloride	0.015	1	250	25	70	25550	5.24E-05	1.90E+00	1E-04
1,2-Dichloroethene (total)	3.3	1	250	25	70	25550	1.15E-02	NA	
1,1-Dichloroethene	0.0020	1	250	25	70	25550	6.99E-06	6.00E-01	4E-06
Chlorobenzene	0.23	1	250	25	70	25550	8.04E-04	NA	
Trichloroethene	0.17	1	250	25	70	25550	5.94E-04	1.10E-02	7E-06
PCBs (ug/l):									
Aroclor-1254	0.014	1	250	25	70	25550	4.89E-05	2.00E+00	2E-05
Semivolatile Organics :									
1,3-Dichlorobenzene	0.56	1	250	25	70	25550	1.96E-03	NA	
1,4-Dichlorobenzene	0.45	1	250	25	70	25550	1.57E-03	2.40E-02	4E-05
1,2-Dichlorobenzene	0.15	1	250	25	70	25550	5.31E-04	NA	
1,2,4-Trichlorobenzene	0.11	1	250	25	70	25550	3.84E-04	NA	
2,4-Dichlorophenol	0.011	1	250	25	70	25550	3.76E-05	NA	
Inorganics									
Aluminum	98	1	250	25	70	25550	3.42E-01	NA	
Arsenic	0.39	1	250	25	70	25550	1.38E-03	1.50E+00	2E-03
Barium	0.90	1	250	25	70	25550	3.14E-03	NA	
Beryllium	0.0069	1	250	25	70	25550	2.41E-05	4.30E+00	1E-04
Cadmium	0.0046	1	250	25	70	25550	1.60E-05	NA	
Chromium	0.48	1	250	25	70	25550	1.68E-03	NA	
Copper	0.39	1	250	25	70	25550	1.35E-03	NA	
Iron	308	1	250	25	70	25550	1.08E+00	NA	
Lead	0.25	1	250	25	70	25550	8.60E-04	NA	
Manganese	7.5	1	250	25	70	25550	2.60E-02	NA	
Mercury	0.0015	1	250	25	70	25550	5.24E-06	NA	
Nickel	0.72	1	250	25	70	25550	2.51E-03	NA	
Vanadium	0.18	1	250	25	70	25550	6.12E-04	NA	
Zinc	1.8	1	250	25	70	25550	6.26E-03	NA	
Dioxins									
Total Equivalent 2,3,7,8-TCDD	4.73E-09	1	250	25	70	25550	1.65E-11	1.50E+05	2E-06
								Sum =	2E-03

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Table H-62

Alluvial Southern Groundwater
Scenario: Future Worker
Pathway: Ingestion of Groundwater
Receptor: Hypothetical Worker (>18 years)

NON-CARCINOGENIC
CDI = Cw x IR x EF x ED x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	C_w Groundwater Concentration (mg/L)	IR Ingestion Rate (l/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	BW Body Weight (Kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Volatile Organics :									
Vinyl Chloride	0.015	1	250	25	70	9125	1.47E-04	NA	
1,2-Dichloroethene (total)	3.3	1	250	25	70	9125	3.23E-02	9.00E-03	3.6E+00
1,1-Dichloroethene	0.0020	1	250	25	70	9125	1.96E-05	9.00E-03	2.2E-03
Chlorobenzene	0.23	1	250	25	70	9125	2.25E-03	2.00E-02	1.1E-01
Trichloroethene	0.17	1	250	25	70	9125	1.66E-03	6.00E-03	2.8E-01
PCBs (ug/l):									
Aroclor-1254	0.014	1	250	25	70	9125	1.37E-04	2.00E-05	6.8E+00
Semivolatile Organics :									
1,3-Dichlorobenzene	0.56	1	250	25	70	9125	5.48E-03	8.90E-02	6.2E-02
1,4-Dichlorobenzene	0.45	1	250	25	70	9125	4.40E-03	NA	
1,2-Dichlorobenzene	0.15	1	250	25	70	9125	1.49E-03	9.00E-02	1.7E-02
1,2,4-Trichlorobenzene	0.11	1	250	25	70	9125	1.08E-03	1.00E-02	1.1E-01
2,4-Dichlorophenol	0.011	1	250	25	70	9125	1.05E-04	3.00E-03	3.5E-02
Inorganics									
Aluminum	98	1	250	25	70	9125	9.58E-01	1.00E+00	9.6E-01
Arsenic	0.39	1	250	25	70	9125	3.86E-03	3.00E-04	1.3E+01
Barium	0.90	1	250	25	70	9125	8.80E-03	7.00E-02	1.3E-01
Beryllium	0.0069	1	250	25	70	9125	6.75E-05	5.00E-03	1.4E-02
Cadmium	0.0046	1	250	25	70	9125	4.48E-05	5.00E-04	9.0E-02
Chromium	0.48	1	250	25	70	9125	4.72E-03	5.00E-03	9.4E-01
Copper	0.39	1	250	25	70	9125	3.77E-03	4.00E-02	9.4E-02
Iron	308	1	250	25	70	9125	3.01E+00	3.00E-01	1.0E+01
Lead	0.25	1	250	25	70	9125	2.41E-03	NA	
Mercury	0.0015	1	250	25	70	9125	1.47E-05	1.00E-04	1.5E-01
Manganese	7.5	1	250	25	70	9125	7.29E-02	2.30E-02	3.2E+00
Nickel	0.72	1	250	25	70	9125	7.02E-03	2.00E-02	3.5E-01
Vanadium	0.18	1	250	25	70	9125	1.71E-03	7.00E-03	2.4E-01
Zinc	1.8	1	250	25	70	9125	1.75E-02	3.00E-01	5.8E-02
Dioxins									
Total Equivalent 2,3,7,8-TCDD	4.73E-09	1	250	25	70	9125	4.63E-11	NA	
								Sum =	4.0E+01

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Table H-63

Alluvial Southern Groundwater
Scenario: Future Resident
Pathway: Inhalation of Groundwater Contaminants while Showering
Receptor: Hypothetical Resident (>18 years)

CARCINOGENIC
CDI =Ca x InR x t x EF x ED x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	Ca Shower Air Concentration (mg/m3)	InR Inhalation Rate (m3/min)	t Time Spent in Shower (min/day)	RIA Relative Inhalation Absorption (unitless)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	CSF Cancer Slope Factor $(mg/kg\text{-}day)^{-1}$	Risk
Volatile Organics :											
Vinyl Chloride	0.35	0.01	12	1.00	350	30	70	25550	2.44E-04	3.00E-01	7E-05
1,2-Dichloroethene (total)	76	0.01	12	1.00	350	30	70	25550	5.37E-02	NA	
1,1-Dichloroethane	0.046	0.01	12	1.00	350	30	70	25550	3.25E-05	1.75E-01	6E-06
Chlorobenzene	5.3	0.01	12	1.00	350	30	70	25550	3.74E-03	NA	
Trichloroethene	3.9	0.01	12	1.00	350	30	70	25550	2.77E-03	6.00E-03	2E-05
										Sum =	1E-04

Table H-64

Alluvial Southern Groundwater
Scenario: Future Resident
Pathway: Inhalation of Groundwater Contaminants while Showering
Receptor: Hypothetical Resident (>18 years)

NON-CARCINOGENIC
CDI =Ca x InR x t x EF x ED x 1/BW x 1/ATc
HQ = CDI/RfD

Chemical	Ca Shower Air Concentration (mg/m3)	InR Inhalation Rate (m3/min)	t Time Spent in Shower (min/day)	RIA Relative Inhalation Absorption (unitless)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	RfD Reference Dose (mg/kg-day)	HQ Hazard Quotient
Volatile Organics :											
Vinyl Chloride	0.35	0.01	12	1.00	350	30	70	10950	5.69E-04	NA	
1,2-Dichloroethene (total)	76	0.01	12	1.00	350	30	70	10950	1.25E-01	NA	
1,1-Dichloroethene	0.046	0.01	12	1.00	350	30	70	10950	7.59E-05	NA	
Chlorobenzene	5.3	0.01	12	1.00	350	30	70	10950	8.73E-03	5.71E-03	1.5E+00
Trichloroethene	3.9	0.01	12	1.00	350	30	70	10950	6.45E-03	NA	
										Sum =	1.5E+00

AR304322

Table H-65

Alluvial Southern Groundwater
Scenario: Future Resident
Pathway: Dermal Contact with Groundwater Contaminants while Showering
Receptor: Hypothetical Resident (>18 years)

CARCINOGENIC

CDI = Cw x PC x SA x FSA x t x EF x ED x CF1 x CF2 x 1/BW x 1/ATc

Risk = CDI x CSF

Chemical	C_w Groundwater Concentration (mg/L)	PC Permeability Coefficient (cm/hr)	SA Total Body Surface Area (cm2)	FSA Fraction SA Exposed (unitless)	t Time Spent in Shower (min/d)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (L/cm3)	CF2 Conversion Factor (hr/min)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	CSF Cancer Slope Factor (mg/kg-day)$^{-1}$	Risk
Volatile Organics :														
Vinyl Chloride	0.0064	7.30E-03	18000	1	12	350	30	0.001	0.017	70	25550	9.86E-07	1.90E+00	2E-06
1,2-Dichloroethene (total)	1.50	1.02E-02	18000	1	12	350	30	0.001	0.017	70	25550	2.23E-04	NA	
1,1-Dichloroethene	0.00094	1.60E-02	18000	1	12	350	30	0.001	0.017	70	25550	3.19E-07	6.00E-01	2E-07
Chlorobenzene	0.11	4.10E-02	18000	1	12	350	30	0.001	0.017	70	25550	9.83E-05	NA	
Trichloroethene	0.09	1.60E-02	18000	1	12	350	30	0.001	0.017	70	25550	2.96E-05	1.10E-02	3E-07
PCBs (ug/l):														
Aroclor-1254	0.014	7.10E-01	18000	1	12	350	30	0.001	0.017	70	25550	2.10E-04	2.00E+00	1E-04
Semivolatile Organics :														
1,3-Dichlorobenzene	0.56	8.70E-02	18000	1	12	350	30	0.001	0.017	70	25550	1.83E-03	NA	
1,4-Dichlorobenzene	0.45	6.20E-02	18000	1	12	350	30	0.001	0.017	70	25550	5.90E-04	2.40E-02	1E-05
1,2-Dichlorobenzene	0.15	6.10E-02	18000	1	12	350	30	0.001	0.017	70	25550	1.96E-04	NA	
1,2,4-Trichlorobenzene	0.11	1.80E-01	18000	1	12	350	30	0.001	0.017	70	25550	2.32E-04	NA	
2,4-Dichlorophenol	0.011	2.30E-02	18000	1	12	350	30	0.001	0.017	70	25550	5.23E-06	NA	
Inorganics														
Aluminum	98	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	2.07E-03	NA	
Arsenic	0.39	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	8.33E-06	1.50E+00	1E-05
Barium	0.90	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	1.90E-05	NA	
Beryllium	0.0069	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	1.46E-07	4.30E+00	6E-07
Cadmium	0.0046	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	9.67E-08	NA	
Chromium	0.48	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	1.02E-05	NA	
Copper	0.39	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	8.14E-06	NA	
Iron	308	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	6.51E-03	NA	
Lead	0.25	4.00E-06	18000	1	12	350	30	0.001	0.017	70	25550	2.09E-08	NA	
Manganese	7.5	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	1.57E-04	NA	
Mercury	0.0015	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	3.17E-08	NA	
Nickel	0.72	9.00E-06	18000	1	12	350	30	0.001	0.017	70	25550	1.36E-07	NA	
Vanadium	0.18	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	3.70E-06	NA	
Zinc	1.8	6.00E-06	18000	1	12	350	30	0.001	0.017	70	25550	2.27E-05	NA	
Dioxins														
Total Equivalent 2,3,7,8-TCDD	4.73E-09	1.40E+00	18000	1	12	350	30	0.001	0.017	70	25550	1.40E-10	1.50E+05	2E-05
													Sum	2E-04

Table H-66

Alluvial Southern Groundwater
Scenario: Future Resident
Pathway: Dermal Contact with Groundwater Contaminants while Showering
Receptor: Hypothetical Resident (>18 years)

NON-CARCINOGENIC
CDI = Cw x PC x SA x FSA x t x EF x ED x CF1 x CF2 x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	C_w Groundwater Concentration (mg/L)	PC Permeability Coefficient (cm/hr)	SA Total Body Surface Area (cm2)	FSA Fraction SA Exposed (unitless)	t Time Spent in Shower (min/d)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (L/cm3)	CF2 Conversion Factor (hr/min)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogens (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Volatile Organics :														
Vinyl Chloride	0.0064	7.30E-05	18000	1	12	350	30	0.001	0.017	70	10950	2.30E-06	NA	
1,2-Dichloroethane (total)	1.5	1.02E-02	18000	1	12	350	30	0.001	0.017	70	10950	7.53E-04	9.00E-03	8.4E-02
1,1-Dichloroethane	0.00084	1.60E-02	18000	1	12	350	30	0.001	0.017	70	10950	7.45E-07	9.00E-03	8.3E-05
Chloroform	0.11	4.10E-02	18000	1	12	350	30	0.001	0.017	70	10950	2.29E-04	2.00E-02	1.1E-02
Trichloroethene	0.068	1.60E-02	18000	1	12	350	30	0.001	0.017	70	10950	6.91E-05	6.00E-03	1.2E-02
PCBs (ug/l):														
Aroclor-1254	0.014	7.30E-01	18000	1	12	350	30	0.001	0.017	70	10950	4.90E-04	2.00E-05	2.5E+01
Semivolatile Organics :														
1,3-Dichlorobenzene	0.56	8.70E-02	18000	1	12	350	30	0.001	0.017	70	10950	2.40E-03	8.90E-02	2.7E-02
1,4-Dichlorobenzene	0.45	6.20E-02	18000	1	12	350	30	0.001	0.017	70	10950	1.36E-03	NA	
1,2-Dichlorobenzene	0.15	6.10E-02	18000	1	12	350	30	0.001	0.017	70	10950	4.57E-04	9.00E-02	5.1E-03
1,2,4-Trichlorobenzene	0.11	1.00E-01	18000	1	12	350	30	0.001	0.017	70	10950	5.42E-04	1.00E-02	5.4E-02
2,4-Dichlorophenol	0.011	2.30E-02	18000	1	12	350	30	0.001	0.017	70	10950	1.22E-05	3.00E-03	4.1E-03
Inorganics														
Aluminum	98	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	4.83E-03	1.00E+00	4.8E-03
Arsenic	0.39	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	1.94E-05	3.00E-04	6.5E-02
Barium	0.90	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	4.43E-05	7.00E-02	6.3E-04
Beryllium	0.0069	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	3.40E-07	5.00E-03	6.8E-05
Cadmium	0.0046	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	2.26E-07	5.00E-04	4.5E-04
Chromium	0.48	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	2.38E-05	5.00E-03	4.8E-03
Copper	0.39	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	1.90E-05	4.00E-02	4.7E-04
Iron	308	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	1.52E-02	3.00E-01	5.1E-02
Lead	0.25	4.00E-06	18000	1	12	350	30	0.001	0.017	70	10950	4.85E-08	NA	
Mercury	0.0015	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	7.40E-08	1.80E-04	7.4E-04
Manganese	7.5	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	3.67E-04	2.30E-02	1.6E-02
Nickel	0.72	9.00E-06	18000	1	12	350	30	0.001	0.017	70	10950	3.18E-07	2.00E-02	1.6E-05
Vanadium	0.18	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	8.63E-06	7.00E-03	1.2E-03
Zinc	1.8	6.00E-04	18000	1	12	350	30	0.001	0.017	70	10950	5.30E-05	3.00E-01	1.8E-04
Dioxins														
Total Equivalent 2,3,7,8-TCDD	4.73E-09	1.40E+00	18000	1	12	350	30	0.001	0.017	70	10950	3.27E-10	NA	
													Sum =	2.6E+01

CARCINOGENIC
CDI = Cs x DAF x SA x FSA x RDA x EF x ED x CF x 1/BW x 1/ATc

Table H-67

Alluvial Southern Groundwater
Scenario: Future Resident
Pathway: Ingestion of Groundwater
Receptor: Hypothetical Resident (>18 years)

CARCINOGENIC
CDI = Cw x IR x EF x ED x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	C_w Groundwater Concentration (mg/L)	IR Ingestion Rate (l/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	BW Body Weight (Kg)	ATc Averaging Time, Carcinogens (days)	CDI Chronic Daily Intake (mg/kg-day)	CSF Cancer Slope Factor $(mg/kg\text{-}day)^{-1}$	Risk
Volatile Organics :									
Vinyl Chloride	0.015	2	350	30	70	25550	1.76E-04	1.90E+00	3E-04
1,2-Dichloroethene (total)	3.3	2	350	30	70	25550	3.87E-02	NA	
1,1-Dichloroethane	0.0020	2	350	30	70	25550	2.35E-05	6.00E-01	1E-05
Chlorobenzene	0.23	2	350	30	70	25550	2.70E-03	NA	
Trichloroethene	0.17	2	350	30	70	25550	2.00E-03	1.10E-02	2E-05
PCBs (mg/l):									
Aroclor-1254	0.014	2	350	30	70	25550	1.64E-04	2.00E+00	8E-05
Semivolatile Organics :									
1,3-Dichlorobenzene	0.56	2	350	30	70	25550	6.58E-03	NA	
1,4-Dichlorobenzene	0.45	2	350	30	70	25550	5.28E-03	2.40E-02	1E-04
1,2-Dichlorobenzene	0.15	2	350	30	70	25550	1.78E-03	NA	
1,2,4-Trichlorobenzene	0.11	2	350	30	70	25550	1.29E-03	NA	
2,4-Dichlorophenol	0.011	2	350	30	70	25550	1.26E-04	NA	
Inorganics									
Aluminum	98	2	350	30	70	25550	1.15E+00	NA	
Arsenic	0.39	2	350	30	70	25550	4.63E-03	1.50E+00	7E-03
Barium	0.90	2	350	30	70	25550	1.06E-02	NA	
Beryllium	0.0069	2	350	30	70	25550	8.10E-05	4.30E+00	3E-04
Cadmium	0.0046	2	350	30	70	25550	5.37E-05	NA	
Chromium	0.48	2	350	30	70	25550	5.66E-03	NA	
Copper	0.39	2	350	30	70	25550	4.52E-03	NA	
Iron	308	2	350	30	70	25550	3.62E+00	NA	
Lead	0.25	2	350	30	70	25550	2.89E-03	NA	
Manganese	7.5	2	350	30	70	25550	8.75E-02	NA	
Mercury	0.0015	2	350	30	70	25550	1.76E-05	NA	
Nickel	0.72	2	350	30	70	25550	8.42E-03	NA	
Vanadium	0.18	2	350	30	70	25550	2.05E-03	NA	
Zinc	1.8	2	350	30	70	25550	2.10E-02	NA	
Dioxins									
Total Equivalent 2,3,7,8-TCDD	4.73E-09	2	350	30	70	25550	5.55E-11	1.50E+05	8E-06
								Sum =	8E-03

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Table H-68

Alluvial Southern Groundwater
Scenario: Future Resident
Pathway: Ingestion of Groundwater
Receptor: Hypothetical Resident (>18 years)

NON-CARCINOGENIC
CDI = Cw x IR x EF x ED x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	C_w Groundwater Concentration (mg/L)	IR Ingestion Rate (l/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	BW Body Weight (Kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Volatile Organics :									
Vinyl Chloride	0.015	2	350	30	70	10950	4.11E-04	NA	
1,2-Dichloroethene (total)	3.3	2	350	30	70	10950	9.04E-02	9.00E-03	1.0E+01
1,1-Dichloroethene	0.0020	2	350	30	70	10950	5.48E-05	9.00E-03	6.1E-03
Chlorobenzene	0.23	2	350	30	70	10950	6.30E-03	2.00E-02	3.2E-01
Trichloroethene	0.17	2	350	30	70	10950	1.53E-02	6.00E-03	2.6E+00
PCBs (ug/l):									
Aroclor-1254	0.014	2	350	30	70	10950	3.84E-04	2.00E-05	1.9E+01
Semivolatile Organics :									
1,3-Dichlorobenzene	0.56	2	350	30	70	10950	4.16E-03	8.90E-02	4.7E-02
1,4-Dichlorobenzene	0.45	2	350	30	70	10950	1.23E-02	NA	
1,2-Dichlorobenzene	0.15	2	350	30	70	10950	4.16E-03	9.00E-02	4.6E-02
1,2,4-Trichlorobenzene	0.11	2	350	30	70	10950	3.01E-03	1.00E-02	3.0E-01
2,4-Dichlorophenol	0.011	2	350	30	70	10950	2.95E-04	3.00E-03	9.8E-02
Inorganics									
Aluminum	98	2	350	30	70	10950	2.68E+00	1.00E+00	2.7E+00
Arsenic	0.39	2	350	30	70	10950	1.06E-02	3.00E-04	3.6E+01
Barium	0.90	2	350	30	70	10950	2.46E-02	7.00E-02	3.5E-01
Beryllium	0.0069	2	350	30	70	10950	1.89E-04	5.00E-03	3.8E-02
Cadmium	0.0046	2	350	30	70	10950	1.25E-04	5.00E-04	2.5E-01
Chromium	0.48	2	350	30	70	10950	1.32E-02	5.00E-03	2.6E+00
Copper	0.39	2	350	30	70	10950	1.05E-02	4.00E-02	2.6E-01
Iron	308	2	350	30	70	10950	8.44E+00	3.00E-01	2.8E+01
Lead	0.25	2	350	30	70	10950	6.74E-03	NA	
Mercury	0.0015	2	350	30	70	10950	4.11E-05	1.00E-04	4.1E-01
Manganese	7.5	2	350	30	70	10950	2.06E-01	2.30E-02	8.9E+00
Nickel	0.72	2	350	30	70	10950	1.96E-02	2.00E-02	9.8E-01
Vanadium	0.18	2	350	30	70	10950	4.79E-03	7.00E-03	6.8E-01
Zinc	1.8	2	350	30	70	10950	4.90E-02	3.00E-01	1.6E-01
Dioxins									
Total Equivalent 2,3,7,8-TCDD	4.73E-09	2	350	30	70	10950	1.30E-10	NA	
								Sum =	1.1E+02

Table H-69

Bedrock Groundwater
Scenario: Future Worker
Pathway: Dermal Contact with Groundwater Contaminants while Showering
Receptor: Hypothetical Worker (>18 years)

CARCINOGENIC
CDI = Cw x PC x SA x FSA x t x EF x ED x CF1 x CF2 x 1/BW x 1/ATc
Risk = CDI x CSF x DAF x SA x FSA x RDA x EF x ED x CF x 1/BW x 1/ATc

Chemical	C_w Groundwater Concentration (mg/L)	PC Permeability Coeficient (cm/hr)	SA Total Body Surface Area (cm2)	FSA Fraction SA Exposed (unitless)	t Time Spent in Shower (min/d)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (L/cm3)	CF2 Conversion Factor (hr/min)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	CSF Cancer Slope Factor (mg/kg-day)$^{-1}$	Risk
Semivolatile Organics :														
Phenanthrene	0.0019	2.30E-01	18000	1	12	250	25	0.001	0.017	70	25550	5.30E-06	NA	
Benzo(a)anthracene	0.0014	8.10E-01	18000	1	12	250	25	0.001	0.017	70	25550	1.43E-05	7.30E-01	1E-05
bis(2-Ethylhexyl) phthalate	0.0071	3.30E-02	18000	1	12	250	25	0.001	0.017	70	25550	2.99E-06	NA	
PCBs:														
Aroclor-1254	0.0033	7.10E-01	18000	1	12	250	25	0.001	0.017	70	25550	2.95E-05	2.00E+00	6E-05
Aroclor-1260	0.0048	7.10E-01	18000	1	12	250	25	0.001	0.017	70	25550	4.33E-05	2.00E+00	9E-05
Inorganics:														
Aluminum	7.7	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	9.47E-05	NA	
Arsenic	0.0097	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	1.22E-07	1.50E+00	2E-07
Barium	0.27	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	3.39E-06	NA	
Beryllium	0.00042	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	5.20E-09	4.30E+00	2E-08
Cadmium	0.0025	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	3.15E-08	NA	
Iron	14	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	1.80E-04	NA	
Manganese	0.028	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	3.53E-07	NA	
Dioxins														
Total Equivalent 2,3,7,8-TCDD	1.34E-09	1.40E+00	18000	1	12	250	25	0.001	0.017	70	25550	2.18E-11	1.30E+05	3E-06
													Sum =	2E-04

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Table H-70

Bedrock Groundwater
Scenario: Future Worker
Pathway: Dermal Contact with Groundwater Contaminants while Showering
Receptor: Hypothetical Worker (>18 years)

NON-CARCINOGENIC
CDI = Cw x PC x SA x FSA x t x EF x ED x CF1 x CF2 x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	C_w Groundwater Concentration (mg/L)	PC Permeability Coefficient (cm/hr)	SA Total Body Surface Area (cm^2)	FSA Fraction SA Exposed (unitless)	t Time Spent in Shower (min/d)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (L/cm3)	CF2 Conversion Factor (hr/min)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organics :														
Benzo(a)anthracene	0.0014	2.30E-01	18000	1	12	250	25	0.001	0.017	70	9125	1.13E-05	NA	
bis(2-Ethylhexyl) phthalate	0.0071	8.10E-01	18000	1	12	250	25	0.001	0.017	70	9125	2.02E-04	NA	
Phenanthrene	0.0019	3.36E-02	18000	1	12	250	25	0.001	0.017	70	9125	2.25E-06	NA	
PCBs:														
Aroclor-1254	0.0033	7.10E-01	18000	1	12	250	25	0.001	0.017	70	9125	8.25E-05	2.00E-05	4.1E+00
Aroclor-1260	0.0048	7.10E-01	18000	1	12	250	25	0.001	0.017	70	9125	1.21E-04	NA	
Inorganics:														
Aluminum	7.7	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	2.71E-04	1.00E+00	
Arsenic	0.0097	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	3.42E-07	3.00E-04	
Barium	0.27	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	9.39E-06	7.00E-02	
Beryllium	0.00042	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	1.46E-08	5.00E-03	
Cadmium	0.0025	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	8.81E-08	5.00E-04	
Iron	14	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	5.04E-04	3.00E-01	
Manganese	0.028	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	9.89E-07	2.30E-02	
Dioxins														
Total Equivalent 2,3,7,8-TCDD	1.34E-09	1.00E+00	18000	1	12	250	25	0.001	0.017	70	9125	6.12E-11	NA	
													Sums	4.1E+00

Table H-71

Sector: Bedrock Groundwater
Scenario: Future Worker
Pathway: Ingestion of Groundwater
Receptor: Hypothetical Worker (>18 years)

CARCINOGENIC
CDI = Cw x IR x EF x ED x 1/BW x 1/ATc
Risk = CDI x CSF

CARCINOGENIC
CDI = Cs x DAF x SA x RDA x EF x ED x CF x 1/BW x 1/ATc

Chemical	C_w Groundwater Concentration (mg/L)	IR Ingestion Rate (l/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	BW Body Weight (Kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	CSF Cancer Slope Factor $(mg/kg\text{-}day)^{-1}$	Risk
Semivolatile Organics :									
Phenanthrene	0.0019	1	250	25	70	25550	6.64E-06	NA	
Benzo(a)anthracene	0.0014	1	250	25	70	25550	4.89E-06	7.30E-01	4E-06
bis(2-Ethylhexyl) phthalate	0.0071	1	250	25	70	25550	2.47E-05	NA	
PCBs:									
Aroclor-1254	0.0033	1	250	25	70	25550	1.15E-05	2.00E+00	2E-05
Aroclor-1260	0.0048	1	250	25	70	25550	1.69E-05	2.00E+00	3E-05
Inorganics									
Aluminum		[illegible]	250	25	70	25550	2.69E-02	NA	
Arsenic	0.0097	1	250	25	70	25550	3.39E-05	1.50E+00	5E-05
Barium	0.27	1	250	25	70	25550	9.31E-04	NA	
Beryllium	0.00042	1	250	25	70	25550	1.47E-06	4.30E+00	6E-06
Cadmium	0.0025	1	250	25	70	25550	8.74E-06	NA	
Iron	14	1	250	25	70	25550	5.00E-02	NA	
Manganese	0.028	1	250	25	70	25550	9.81E-05	NA	
Dioxins									
Total Equivalent 2,3,7,8-TCDD	1.24E-09	1	250	25	70	25550	4.33E-12	1.50E+05	6E-07
								Sum =	1E-04

Table H-72

Bedrock Groundwater
Scenario: Future Worker
Pathway: Ingestion of Groundwater
Receptor: Hypothetical Worker (>18 years)

NON-CARCINOGENIC
CDI = Cw x IR x EF x ED x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	C_w Groundwater Concentration (mg/L)	IR Ingestion Rate (l/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	BW Body Weight (Kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organics :									
Benzo(a)anthracene	0.0014	1	250	25	70	9125	1.37E-05	NA	
bis(2-Ethylhexyl) phthalate	0.0071	1	250	25	70	9125	6.92E-05	2.00E-02	3.5E-03
Phenanthrene	0.0019	1	250	25	70	9125	1.86E-05	NA	
PCBs:									
Aroclor-1254	0.0033	1	250	25	70	9125	3.23E-05	2.00E-05	1.6E+00
Aroclor-1260	0.0038	1	250	25	70	9125	3.72E-05	NA	
Inorganics:									
Aluminum	7.7	1	250	25	70	9125	7.52E-02	1.00E+00	7.5E-02
Arsenic	0.0097	1	250	25	70	9125	9.49E-05	3.00E-04	3.2E-01
Barium	0.27	1	250	25	70	9125	2.61E-03	7.00E-02	3.7E-02
Beryllium	0.00042	1	250	25	70	9125	4.11E-06	5.00E-03	8.2E-04
Cadmium	0.0025	1	250	25	70	9125	2.45E-05	5.00E-04	4.9E-02
Iron	14	1	250	25	70	9125	1.40E-01	3.00E-01	4.7E-01
Manganese	0.133	1	250	25	70	9125	1.30E-03	2.30E-02	5.7E-02
Dioxins									
Total Equivalent 2,3,7,8-TCDD	3.6E-09	1	250	25	70	9125	3.52E-11	NA	
								Sum=	2.6E+00

Table H-73

Bedrock Groundwater
Scenario: Future Resident
Pathway: Dermal Contact with Groundwater Contaminants while Showering
Receptor: Hypothetical Resident (>18 years)

CARCINOGENIC
CDI = Cw x PC x SA x PSA x t x EF x ED x CF1 x CF2 x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	C_w Groundwater Concentration (mg/L)	PC Permeability Coefficient (cm/hr)	SA Total Body Surface Area (cm2)	FSA Fraction SA Exposed (unitless)	t Time Spent in Shower (min/d)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (L/cm3)	CF2 Conversion Factor (hr/min)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	CSF Cancer Slope Factor $(mg/kg-day)^{-1}$	Risk
Semivolatile Organics :														
Phenanthrene	0.0019	2.30E-01	18000	1	12	350	30	0.001	0.017	70	25550	9.24E-06	NA	
Benzo(a)anthracene	0.0014	8.10E-01	18000	1	12	350	30	0.001	0.017	70	25550	2.40E-05	7.30E-01	2E-05
bis(2-Ethylhexyl) phthalate	0.0071	3.30E-02	18000	1	12	350	30	0.001	0.017	70	25550	5.02E-06	NA	
PCBs:														
Aroclor-1254	0.0033	7.10E-01	18000	1	12	350	30	0.001	0.017	70	25550	4.95E-05	2.00E+00	1E-04
Aroclor-1260	0.0048	7.10E-01	18000	1	12	350	30	0.001	0.017	70	25550	7.27E-05	2.00E+00	1E-04
Inorganics:														
Aluminum	7.7	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	1.63E-04	NA	
Arsenic	0.0097	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	2.05E-07	1.50E+00	3E-07
Barium	0.27	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	5.63E-06	NA	
Beryllium	0.00042	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	8.89E-09	4.30E+00	4E-08
Cadmium	0.0025	1.80E-03	18000	1	12	350	30	0.001	0.017	70	25550	5.28E-08	NA	
Iron	14	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	3.02E-04	NA	
Manganese	0.028	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	5.93E-07	NA	
Dioxins														
Total Equivalent 2,3,7,8-TCDD	1.2E-09	1.40E+00	18000	1	12	350	30	0.001	0.017	70	25550	3.67E-11	1.50E+05	6E-06
													Sum =	3E-04

Table H-74

Bedrock Groundwater
Scenario: Future Resident
Pathway: Dermal Contact with Groundwater Contaminants while Showering
Receptor: Hypothetical Resident (>18 years)

NON-CARCINOGENIC
CDI = Cw x PC x SA x FSA x t x EF x ED x CF1 x CF2 x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	C_w Groundwater Concentration (mg/L)	PC Permeability Coefficient (cm/hr)	SA Total Body Surface Area (cm2)	FSA Fraction SA Exposed (unitless)	t Time Spent in Shower (min/d)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (L/cm3)	CF2 Conversion Factor (hr/min)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organics:														
Benzo(a)anthracene	0.0014	8.10E-01	18000	1	12	350	30	0.001	0.017	70	10950	5.59E-05	NA	
bis(2-Ethylhexyl) phthalate	0.0071	3.36E-02	18000	1	12	350	30	0.001	0.017	70	10950	1.17E-05	NA	
Phenanthrene	0.0019	2.30E-01	18000	1	12	350	30	0.001	0.017	70	10950	2.16E-05	NA	
PCBs:														
Aroclor-1254	0.0033	7.10E-01	18000	1	12	350	30	0.001	0.017	70	10950	1.16E-04	2.00E-05	5.8E+00
Aroclor-1260	0.0048	7.10E-01	18000	1	12	350	30	0.001	0.017	70	10950	1.70E-04	NA	
Inorganics:														
Aluminum	7.7	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	3.79E-04	1.00E+00	3.8E-04
Arsenic	0.0097	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	4.78E-07	3.00E-04	1.6E-03
Barium	0.27	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	1.31E-05	7.00E-02	1.9E-04
Beryllium	0.00042	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	2.07E-08	5.00E-03	4.1E-06
Cadmium	0.0025	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	1.23E-07	5.00E-04	2.5E-04
Iron	14	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	7.05E-04	3.00E-01	2.4E-03
Manganese	0.028	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	1.38E-06	2.30E-02	6.0E-05
Dioxins														
Total Equivalent 2,3,7,8-TCDD	1.24E-09	1.40E+00	18000	1	12	350	30	0.001	0.017	70	10950	8.56E-11	NA	
													Sum=	5.8E+00

Table H-75

Bedrock Groundwater
Scenario: Future Resident
Pathway: Ingestion of Groundwater
Receptor: Hypothetical Resident (>18 years)

CARCINOGENIC
CDI = Cw x IR x EF x ED x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	C_w Groundwater Concentration (mg/L)	IR Ingestion Rate (l/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	BW Body Weight (Kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	CSF Cancer Slope Factor $(mg/kg\text{-}day)^{-1}$	Risk
Semivolatile Organics :									
Phenanthrene	0.0019	2	350	30	70	25550	2.23E-05	NA	
Benzo(a)anthracene	0.0014	2	350	30	70	25550	1.64E-05	7.30E-01	1E-05
bis(2-Ethylhexyl) phthalate	0.0071	2	350	30	70	25550	8.31E-05	1.40E-02	1E-06
PCBs:									
Aroclor-1254	0.0033	2	350	30	70	25550	3.87E-05	2.00E+00	8E-05
Aroclor-1260	0.0038	2	350	30	70	25550	4.46E-05	2.00E+00	9E-05
Inorganics:									
Aluminum	7.7	2	350	30	70	25550	9.03E-02	NA	
Arsenic	0.0097	2	350	30	70	25550	1.14E-04	1.50E+00	2E-04
Barium	0.27	2	350	30	70	25550	3.13E-03	NA	
Beryllium	0.00042	2	350	30	70	25550	4.93E-06	4.30E+00	2E-05
Cadmium	0.0025	2	350	30	70	25550	2.94E-05	NA	
Iron	14	2	350	30	70	25550	1.68E-01	NA	
Manganese	0.133	2	350	30	70	25550	1.56E-03	NA	
Dioxins									
Total Equivalent 2,3,7,8-TCDD	3.6E-09	2	350	30	70	25550	4.23E-11	1.50E+05	6E-06
								Sum =	4E-04

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Table H-76

Bedrock Groundwater
Scenario: Future Resident
Pathway: Ingestion of Groundwater Contaminants while Showering
Receptor: Hypothetical Resident (>18 years)

NON-CARCINOGENIC
CDI = Cw x IR x EF x ED x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	C_w Groundwater Concentration (mg/L)	IR Ingestion Rate (l/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	BW Body Weight (Kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Semivolatile Organics :									
Benzo(a)anthracene	0.0014	2	350	30	70	10950	3.84E-05	NA	
bis(2-Ethylhexyl) phthalate	0.0091	2	350	30	70	10950	1.94E-04	NA	
Phenanthrene	0.0019	2	350	30	70	10950	5.21E-05	NA	
PCBs:									
Aroclor-1254	0.0033	2	350	30	70	10950	9.04E-05	2.00E-05	4.5E+00
Aroclor-1260	0.0048	2	350	30	70	10950	1.32E-04	NA	
Inorganics:									
Aluminum	7.7	2	350	30	70	10950	2.11E-01	1.00E+00	2.1E-01
Arsenic	0.0097	2	350	30	70	10950	2.66E-04	3.00E-04	8.9E-01
Barium	0.27	2	350	30	70	10950	7.30E-03	7.00E-02	1.0E-01
Beryllium	0.00042	2	350	30	70	10950	1.15E-05	5.00E-03	2.3E-03
Cadmium	0.0025	2	350	30	70	10950	6.85E-05	5.00E-04	1.4E-01
Iron	14	2	350	30	70	10950	3.92E-04	3.00E-01	1.3E+00
Manganese	0.028	2	350	30	70	10950	7.68E-04	2.30E-02	3.3E-02
Dioxins									
Total Equivalent 2,3,7,8-TCDD	1.24E-09	2	350	30	70	10950	3.40E-11	NA	
								Sum=	7.2E+00

Table H-77

Alluvial Central Groundwater
Scenario: Future Worker
Pathway: Inhalation of Groundwater Contaminants while Showering
Receptor: Hypothetical Worker (>18 years)

CARCINOGENIC
CDI =Ca x InR x t x EF x ED x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	Ca Shower Air Concentration (mg/m3)	InR Inhalation Rate (m3/min)	t Time Spent in Shower (min/day)	RIA Relative Inhalation Absorption (unitless)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	CSF Cancer Slope Factor $(mg/kg\text{-}day)^{-1}$	Risk
Volatile Organics:											
1,1-Dichloroethene	0.12	0.01	12	1.00	250	25	70	25550	4.84E-05	1.75E-01	8E-06
1,2-Dichloroethene (total)	0.21	0.01	12	1.00	250	25	70	25550	8.72E-05	NA	
1,2-Dichloroethane	0.14	0.01	12	1.00	250	25	70	25550	5.81E-05	9.10E-02	5E-06
1,1,1-Trichloroethane	1.5	0.01	12	1.00	250	25	70	25550	6.30E-04	NA	
Trichloroethene	93	0.01	12	1.00	250	25	70	25550	3.90E-02	6.00E-03	2E-04
Benzene	6.7	0.01	12	1.00	250	25	70	25550	2.81E-03	2.90E-02	8E-05
Tetrachloroethene	0.58	0.01	12	1.00	250	25	70	25550	2.42E-04	2.03E-03	5E-07
Chlorobenzene	7.2	0.01	12	1.00	250	25	70	25550	3.00E-03	NA	
										Sum =	3E-04

Table H-78

Alluvial Central Groundwater
Scenario: Future Worker
Pathway: Inhalation of Groundwater Contaminants while Showering
Receptor: Hypothetical Worker (>18 years)

NON-CARCINOGENIC
CDI =Ca x IhR x t x EF x ED x 1/BW x 1/ATc
HQ = CDI/RfD

Chemical	Ca Shower Air Concentration (mg/m3)	IhR Inhalation Rate (m3/min)	t Time Spent in Shower (min/day)	RIA Relative Inhalation Absorption (unitless)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	RfD Reference Dose (mg/kg-day)	HQ Hazard Quotient
Volatile Organics											
1,1-Dichloroethane	0.12	0.01	12	1.00	250	25	70	9125	1.36E-04	NA	
1,2-Dichloroethane (total)	0.21	0.01	12	1.00	250	25	70	9125	2.44E-04	NA	
1,2-Dichloroethene	0.14	0.01	12	1.00	250	25	70	9125	1.63E-04	2.86E-03	5.7E-02
1,1,1-Trichloroethane	1.5	0.01	12	1.00	250	25	70	9125	1.75E-03	2.86E-01	6.2E-03
Trichloroethene	99	0.01	12	1.00	250	25	70	9125	1.09E-01	NA	
Benzene	6.7	0.01	12	1.00	250	25	70	9125	7.84E-03	1.71E-03	4.6E+00
Tetrachloroethene	0.58	0.01	12	1.00	250	25	70	9125	6.78E-04	NA	
Chlorobenzene	7.2	0.01	12	1.00	250	25	70	9125	8.41E-03	5.71E-03	1.5E+00
										Sum =	6.1E+00

Table H-79

Alluvial Central Groundwater
Scenario: Future Worker
Pathway: Dermal Contact with Groundwater Contaminants while Showering
Receptor: Hypothetical Worker (>18 years)

CARCINOGENIC
CDI = Cw x PC x SA x FSA x t x EF x ED x CF1 x CF2 x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	C_w Groundwater Concentration (mg/L)	PC Permeability Coefficient (cm/hr)	SA Total Body Surface Area (cm2)	FSA Fraction SA Exposed (unitless)	t Time Spent in Shower (min/d)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (L/cm3)	CF2 Conversion Factor (hr/min)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	CSF Cancer Slope Factor $(mg/kg-day)^{-1}$	Risk
Volatile Organics:														
1,1-Dichloroethene	0.0004	1.60E-02	18000	1	12	250	25	0.001	0.017	70	25550	4.75E-07	6.00E-01	3E-07
1,2-Dichloroethene (total)	0.0041	1.02E-02	18000	1	12	250	25	0.001	0.017	70	25550	5.24E-07	NA	
1,2-Dichloroethane	0.0028	5.30E-03	18000	1	12	250	25	0.001	0.017	70	25550	1.89E-07	9.10E-02	2E-08
1,1,1-Trichloroethane	0.032	1.70E-02	18000	1	12	250	25	0.001	0.017	70	25550	6.04E-06	NA	
Trichloroethene	2.1	1.60E-02	18000	1	12	250	25	0.001	0.017	70	25550	4.18E-04	1.10E-02	5E-06
Benzene	0.13	2.10E-02	18000	1	12	250	25	0.001	0.017	70	25550	3.45E-05	2.90E-02	1E-06
Tetrachloroethene	0.013	4.80E-02	18000	1	12	250	25	0.001	0.017	70	25550	8.07E-06	5.20E-02	4E-07
Chlorobenzene	0.15	4.10E-02	18000	1	12	250	25	0.001	0.017	70	25550	7.89E-05	NA	
Semivolatile Organics:														
1,3-Dichlorobenzene	0.51	8.70E-02	18000	1	12	250	25	0.001	0.017	70	25550	5.58E-04	NA	
1,4-Dichlorobenzene	0.36	6.20E-02	18000	1	12	250	25	0.001	0.017	70	25550	2.81E-04	2.40E-02	7E-06
1,2-Dichlorobenzene	0.91	6.10E-02	18000	1	12	250	25	0.001	0.017	70	25550	6.98E-04	NA	
1,2,4-Trichlorobenzene	48	1.00E-01	18000	1	12	250	25	0.001	0.017	70	25550	6.04E-02	NA	
PCBs:														
Aroclor-1242	0.0057	7.10E-01	18000	1	12	250	25	0.001	0.017	70	25550	5.05E-05	2.00E+00	1E-04
Aroclor-1248	30	7.10E-01	18000	1	12	250	25	0.001	0.017	70	25550	2.68E-01	2.00E+00	4E-01
Aroclor-1254	47	7.10E-01	18000	1	12	250	25	0.001	0.017	70	25550	4.20E-01	2.00E+00	6E-01
Aroclor-1260	0.0056	7.10E-01	18000	1	12	250	25	0.001	0.017	70	25550	5.00E-05	2.00E+00	1E-04
Inorganics:														
Aluminum	140	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	1.77E-05	NA	
Arsenic	0.035	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	4.39E-07	1.50E+00	7E-07
Barium	0.98	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	1.23E-05	NA	
Beryllium	0.012	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	1.45E-07	4.30E+00	6E-07
Cadmium	0.043	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	5.40E-07	NA	
Chromium	0.25	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	3.15E-06	NA	
Copper	0.78	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	9.80E-06	NA	
Iron	477	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	6.00E-03	NA	
Lead	0.38	4.00E-06	18000	1	12	250	25	0.001	0.017	70	25550	1.42E-08	NA	
Manganese	50	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	6.35E-04	NA	
Mercury	0.0010	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	1.21E-08	NA	
Nickel	0.29	9.00E-06	18000	1	12	250	25	0.001	0.017	70	25550	3.28E-08	NA	
Silver	0.014	6.00E-04	18000	1	12	250	25	0.001	0.017	70	25550	1.08E-07	NA	
Vanadium	0.27	1.00E-03	18000	1	12	250	25	0.001	0.017	70	25550	3.34E-06	NA	
Zinc	18	6.00E-04	18000	1	12	250	25	0.001	0.017	70	25550	1.37E-04	NA	
Cyanide (ug/l):	0.006	6.00E-04	18000	1	12	250	25	0.001	0.017	70	25550	2.74E-07	NA	
Dioxins:														
Total Equivalent 2,3,7,8-TCDD	8.30E-09	1.40E+00	18000	1	12	250	25	0.001	0.017	70	25550	1.46E-10	1.30E+05	2E-05
													Sum =	1E+00

Table H-80

Alluvial Central Groundwater
Scenario: Future Worker
Pathway: Dermal Contact with Groundwater Contaminants while Showering
Receptor: Hypothetical Worker (>18 years)

NON-CARCINOGENIC
CDI = Cw x PC x SA x FSA x t x EF x ED x CF1 x CF2 x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	C_w Groundwater Concentration (mg/L)	PC Permeability Coefficient (cm/hr)	SA Total Body Surface Area (cm2)	FSA Fraction SA Exposed (unitless)	t Time Spent in Shower (min/d)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (L/cm3)	CF2 Conversion Factor (hr/min)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Volatile Organics:														
1,1-Dichloroethene	0.0050	1.60E-02	18000	1	12	250	25	0.001	0.017	70	9125	2.82E-06	9.00E-03	3.1E-04
1,2-Dichloroethene (total)	0.0080	1.00E-02	18000	1	12	250	25	0.001	0.017	70	9125	3.23E-06	9.00E-03	3.6E-04
1,2-Dichloroethane	0.0060	5.30E-03	18000	1	12	250	25	0.001	0.017	70	9125	1.12E-06	3.00E-02	3.7E-05
1,1,1-Trichloroethane	0.066	1.70E-02	18000	1	12	250	25	0.001	0.017	70	9125	3.95E-05	3.50E-02	1.1E-03
Trichloroethene	4.0	1.60E-02	18000	1	12	250	25	0.001	0.017	70	9125	2.27E-03	6.00E-03	3.8E-01
Benzene	0.29	2.10E-02	18000	1	12	250	25	0.001	0.017	70	9125	2.15E-04	3.00E-03	7.2E-02
Tetrachloroethene	0.005	4.80E-02	18000	1	12	250	25	0.001	0.017	70	9125	4.23E-05	1.00E-02	4.2E-03
Chlorobenzene	0.31	4.10E-02	18000	1	12	250	25	0.001	0.017	70	9125	4.48E-04	2.00E-02	2.2E-02
Semivolatile Organics :														
1,3-Dichlorobenzene	0.51	8.70E-02	18000	1	12	250	25	0.001	0.017	70	9125	1.56E-03	8.90E-02	1.8E-02
1,4-Dichlorobenzene	0.36	6.20E-02	18000	1	12	250	25	0.001	0.017	70	9125	7.86E-04	NA	
1,2-Dichlorobenzene	0.91	6.10E-02	18000	1	12	250	25	0.001	0.017	70	9125	1.96E-03	9.00E-02	2.2E-02
1,2,4-Trichlorobenzene	48	1.00E-01	18000	1	12	250	25	0.001	0.017	70	9125	1.69E-01	1.00E-02	1.7E+01
PCBs :														
Aroclor-1242	0.0057	7.10E-01	18000	1	12	250	25	0.001	0.017	70	9125	1.41E-04	NA	
Aroclor-1248	30	7.10E-01	18000	1	12	250	25	0.001	0.017	70	9125	7.50E-01	NA	
Aroclor-1254	47	7.10E-01	18000	1	12	250	25	0.001	0.017	70	9125	1.18E+00	2.00E-05	5.9E+04
Aroclor-1260	0.0056	7.10E-01	18000	1	12	250	25	0.001	0.017	70	9125	1.40E-04	NA	
Inorganics :														
Aluminum	140	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	4.94E-03	1.00E+00	4.9E-03
Arsenic	0.035	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	1.23E-06	3.00E-04	4.1E-03
Barium	0.98	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	3.45E-05	7.00E-02	4.9E-04
Beryllium	0.012	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	4.05E-07	5.00E-03	8.1E-05
Cadmium	0.043	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	1.53E-06	5.00E-04	3.1E-03
Chromium	0.25	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	8.81E-06	5.00E-03	1.8E-03
Copper	0.78	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	2.74E-05	4.00E-02	6.9E-04
Iron	477	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	1.68E-02	3.00E-01	5.6E-02
Lead	0.38	4.00E-06	18000	1	12	250	25	0.001	0.017	70	9125	3.97E-08	NA	
Manganese	50	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	1.76E-03	2.30E-02	7.7E-02
Mercury	0.0010	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	3.36E-08	1.00E-04	3.4E-04
Nickel	0.29	9.00E-06	18000	1	12	250	25	0.001	0.017	70	9125	9.19E-08	2.00E-02	4.6E-06
Silver	0.014	6.00E-04	18000	1	12	250	25	0.001	0.017	70	9125	2.96E-07	5.00E-03	5.9E-05
Vanadium	0.27	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	9.34E-06	7.00E-03	1.3E-03
Zinc	18	6.00E-04	18000	1	12	250	25	0.001	0.017	70	9125	3.85E-04	3.00E-01	1.3E-03
Cyanide (ug/l):	0.036	1.00E-03	18000	1	12	250	25	0.001	0.017	70	9125	1.28E-06	NA	
Dioxins :														
Total Equivalent 2,3,7,8-TCDD	8.3E-09	1.40E+00	18000	1	12	250	25	0.001	0.017	70	9125	4.09E-10	NA	
													Sum =	5.9E+04

Table H-81

Alluvial Central Groundwater
Scenario: Future Worker
Pathway: Ingestion of Groundwater
Receptor: Hypothetical Worker (>18 years)

CARCINOGENIC
CDI = Cw x IR x EF x ED x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	C_w Groundwater Concentration (mg/L)	IR Ingestion Rate (l/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	BW Body Weight (Kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	CSF Cancer Slope Factor $(mg/kg\text{-}day)^{-1}$	Risk
Volatile Organics:									
1,1-Dichloroethene	0.0050	1	250	25	70	25550	1.75E-05	6.00E-01	1E-05
1,2-Dichloroethene (total)	0.0090	1	250	25	70	25550	3.15E-05	NA	
1,2-Dichloroethane	0.0060	1	250	25	70	25550	2.10E-05	9.10E-02	2E-06
1,1,1-Trichloroethane	0.066	1	250	25	70	25550	2.30E-04	NA	
Trichloroethene	4.0	1	250	25	70	25550	1.41E-02	1.10E-02	2E-04
Benzene	0.29	1	250	25	70	25550	1.01E-03	2.90E-02	3E-05
Tetrachloroethene	0.025	1	250	25	70	25550	8.74E-05	5.20E-02	5E-06
Chlorobenzene	0.31	1	250	25	70	25550	1.08E-03	NA	
Semivolatile Organics:									
1,3-Dichlorobenzene	0.51	1	250	25	70	25550	1.78E-03	NA	
1,4-Dichlorobenzene	0.36	1	250	25	70	25550	1.26E-03	2.40E-02	3E-05
1,2-Dichlorobenzene	0.91	1	250	25	70	25550	3.18E-03	NA	
1,2,4-Trichlorobenzene	48	1	250	25	70	25550	1.68E-01	NA	
PCBs:									
Aroclor-1242	0.0057	1	250	25	70	25550	1.97E-05	2.00E+00	4E-05
Aroclor-1248	30	1	250	25	70	25550	1.05E-01	2.00E+00	2E-01
Aroclor-1254	47	1	250	25	70	25550	1.64E-01	2.00E+00	3E-01
Aroclor-1260	0.0056	1	250	25	70	25550	1.96E-05	2.00E+00	4E-05
Inorganics:									
Aluminum	140	1	250	25	70	25550	4.91E-01	NA	
Arsenic	0.035	1	250	25	70	25550	1.23E-04	1.50E+00	2E-04
Barium	0.98	1	250	25	70	25550	3.42E-03	NA	
Beryllium	0.012	1	250	25	70	25550	4.02E-05	4.30E+00	2E-04
Cadmium	0.043	1	250	25	70	25550	1.52E-04	NA	
Chromium	0.25	1	250	25	70	25550	8.74E-04	NA	
Copper	0.78	1	250	25	70	25550	2.72E-03	NA	
Iron	477	1	250	25	70	25550	1.67E+00	NA	
Lead	0.28	1	250	25	70	25550	9.81E-04	NA	
Manganese	50	1	250	25	70	25550	1.76E-01	NA	
Mercury	0.0010	1	250	25	70	25550	3.35E-06	NA	
Nickel	0.29	1	250	25	70	25550	1.01E-03	NA	
Silver	0.014	1	250	25	70	25550	4.90E-05	NA	
Vanadium	0.27	1	250	25	70	25550	9.27E-04	NA	
Zinc	18	1	250	25	70	25550	6.36E-02	NA	
Cyanide (ug/l):	0.036	1	250	25	70	25550	1.27E-04	NA	
Dioxins:									
Total Equivalent 2,3,7,8-TCDD	8.30E-09	1	250	25	70	25550	2.90E-11	1.50E+05	4E-06
								Sum =	5E-01

Table H-82

Alluvial Central Groundwater
Scenario: Future Worker
Pathway: Ingestion of Groundwater
Receptor: Hypothetical Worker (>18 years)

NONCARCINOGENIC
CDI = Cw x IR x EF x ED x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	C_w Groundwater Concentration (mg/L)	IR Ingestion Rate (l/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	BW Body Weight (Kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Volatile Organics:									
1,1-Dichloroethane	0.0050	1	250	25	70	9125	4.89E-05	9.00E-03	5.4E-03
1,2-Dichloroethane (total)	0.0080	1	250	25	70	9125	8.81E-05	9.00E-03	9.8E-03
1,2-Dichloroethane	0.0060	1	250	25	70	9125	5.87E-05	3.00E-02	2.0E-03
1,1,1-Trichloroethane	0.066	1	250	25	70	9125	6.45E-04	3.50E-02	1.8E-02
Trichloroethene	4.03	1	250	25	70	9125	3.94E-02	6.00E-03	6.6E+00
Benzene	0.29	1	250	25	70	9125	2.84E-03	3.00E-03	9.5E-01
Tetrachloroethene	0.025	1	250	25	70	9125	2.45E-04	1.00E-02	2.4E-02
Chlorobenzene	0.31	1	250	25	70	9125	3.03E-03	2.00E-02	1.5E-01
Semi-volatile Organics:									
1,3-Dichlorobenzene	0.51	1	250	25	70	9125	4.99E-03	8.90E-02	5.6E-02
1,4-Dichlorobenzene	0.36	1	250	25	70	9125	3.52E-03	NA	
1,2-Dichlorobenzene	0.91	1	250	25	70	9125	8.90E-03	9.00E-02	9.9E-02
1,2,4-Trichlorobenzene	48	1	250	25	70	9125	4.70E-01	1.00E-02	4.7E+01
PCBs:									
Aroclor-1242	0.0057	1	250	25	70	9125	5.53E-05	NA	
Aroclor-1248	30	1	250	25	70	9125	2.94E-01	NA	
Aroclor-1254	47	1	250	25	70	9125	4.60E-01	2.00E-05	2.3E+04
Aroclor-1260	0.0056	1	250	25	70	9125	5.48E-05	NA	
Inorganics:									
Aluminum	140	1	250	25	70	9125	1.37E+00	1.00E+00	1.4E+00
Arsenic	0.035	1	250	25	70	9125	3.41E-04	3.00E-04	1.1E+00
Barium	0.98	1	250	25	70	9125	9.59E-03	7.00E-02	1.4E-01
Beryllium	0.012	1	250	25	70	9125	1.13E-04	5.00E-03	2.3E-02
Cadmium	0.043	1	250	25	70	9125	4.25E-04	5.00E-04	8.5E-01
Chromium	0.25	1	250	25	70	9125	2.45E-03	5.00E-03	4.9E-01
Copper	0.78	1	250	25	70	9125	7.62E-03	4.00E-02	1.9E-01
Iron	477	1	250	25	70	9125	4.67E+00	3.00E-01	1.6E+01
Lead	0.28	1	250	25	70	9125	2.76E-03	NA	
Manganese	50	1	250	25	70	9125	4.94E-01	2.30E-02	2.1E+01
Mercury	0	1	250	25	70	9125	9.39E-06	1.00E-04	9.4E-02
Nickel	0.29	1	250	25	70	9125	2.84E-03	2.00E-02	1.4E-01
Silver	0.014	1	250	25	70	9125	1.37E-04	5.00E-03	2.7E-02
Vanadium	0.27	1	250	25	70	9125	2.60E-03	7.00E-03	3.7E-01
Zinc	18	1	250	25	70	9125	1.76E-01	3.00E-01	5.9E-01
Cyanide (ug/l):	0.036	1	250	25	70	9125	3.55E-04	NA	
Dioxins:									
Total Equivalent 2,3,7,8-TCDD	8.30E-09	1	250	25	70	9125	8.12E-11	NA	
								Sum =	2.3E+04

Table H-83

Alluvial Central Groundwater
Scenario: Future Resident
Pathway: Inhalation of Groundwater Contaminants while Showering
Receptor: Hypothetical Resident (>18 years)

CARCINOGENIC
CDI =Ca x IbR x t x EF x ED x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	Ca Shower Air Concentration (mg/m3)	IbR Inhalation Rate (m3/min)	t Time Spent in Shower (min/day)	RIA Relative Inhalation Absorption (unitless)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	CSF Cancer Slope Factor $(mg/kg-day)^{-1}$	Risk
Volatile Organics:											
1,1-Dichloroethene	0.12	0.01	12	1.00	350	30	70	25550	8.13E-05	1.75E-01	1E-05
1,2-Dichloroethene (total)	0.21	0.01	12	1.00	350	30	70	25550	1.46E-04	NA	
1,2-Dichloroethane	0.14	0.01	12	1.00	350	30	70	25550	9.76E-05	9.10E-02	9E-06
1,1,1-Trichloroethane	1.5	0.01	12	1.00	350	30	70	25550	1.07E-03	NA	
Trichloroethene	93	0.01	12	1.00	350	30	70	25550	6.56E-02	6.00E-03	4E-04
Benzene	6.7	0.01	12	1.00	350	30	70	25550	4.72E-03	2.90E-02	1E-04
Tetrachloroethene	0.58	0.01	12	1.00	350	30	70	25550	4.07E-04	2.03E-03	8E-07
Chlorobenzene	7.2	0.01	12	1.00	350	30	70	25550	5.04E-03	NA	
										Sum =	6E-04

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Table H-84

Alluvial Central Groundwater
Scenario: Future Resident
Pathway: Inhalation of Groundwater Contaminants while Showering
Receptor: Hypothetical Resident (>18 years)

NON-CARCINOGENIC
CDI = Ca x InR x t x EF x ED x 1/BW x 1/ATc
HQ = CDI/RfD

Chemical	Ca Shower Air Concentration (mg/m3)	InR Inhalation Rate (m3/min)	t Time Spent in Shower (min/day)	RIA Relative Inhalation Absorption (unitless)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogens (days)	CDI Chronic Daily Intake (mg/kg-day)	RfD Reference Dose (mg/kg-day)	HQ Hazard Quotient
Volatile Organics											
1,1-Dichloroethene	0.12	0.01	12	1.00	350	30	70	10950	1.90E-04	NA	
1,2-Dichloroethene (total)	0.21	0.01	12	1.00	350	30	70	10950	3.42E-04	NA	
1,2-Dichloroethane	0.14	0.01	12	1.00	350	30	70	10950	2.28E-04	2.86E-03	8.0E-02
1,1,1-Trichloroethane	1.5	0.01	12	1.00	350	30	70	10950	2.50E-03	2.86E-01	8.7E-03
Trichloroethene	.93	0.01	12	1.00	350	30	70	10950	1.53E-03	NA	
Benzene	6.7	0.01	12	1.00	350	30	70	10950	1.10E-02	1.71E-03	6.4E+00
Tetrachloroethene	0.58	0.01	12	1.00	350	30	70	10950	9.49E-04	NA	
Chlorobenzene	7.2	0.01	12	1.00	350	30	70	10950	1.18E-02	5.71E-03	2.1E+00
										Sum =	8.6E+00

AR:304342

Table H-85

Alluvial Central Groundwater
Scenario: Future Resident
Pathway: Dermal Contact with Groundwater Contaminants while Showering
Receptor: Hypothetical Resident (>18 years)

CARCINOGENIC
CDI = Cw x PC x SA x PSA x t x EF x ED x CF1 x CF2 x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	C_w Groundwater Concentration (mg/L)	PC Permeability Coefficient (cm/hr)	SA Total Body Surface Area (cm2)	PSA Fraction SA Exposed (unitless)	t Time Spent in Shower (min/d)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (L/cm3)	CF2 Conversion Factor (hr/min)	BW Body Weight (kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	CSF Cancer Slope Factor $(mg/kg\text{-}day)^{-1}$	Risk
Volatile Organics:														
1,1-Dichloroethane	0.0024	1.80E-02	18000	1	12	350	30	0.001	0.017	70	25550	7.99E-07	6.00E-01	5E-07
1,2-Dichloroethane (total)	0.0041	1.02E-02	18000	1	12	350	30	0.001	0.017	70	25550	8.80E-07	NA	
1,2-Dichloroethane	0.0028	5.30E-03	18000	1	12	350	30	0.001	0.017	70	25550	3.18E-07	9.10E-02	3E-08
1,1,1-Trichloroethane	0.032	1.70E-02	18000	1	12	350	30	0.001	0.017	70	25550	1.15E-05	NA	
Trichloroethene	2.1	1.80E-02	18000	1	12	350	30	0.001	0.017	70	25550	7.02E-04	1.10E-02	8E-06
Benzene	0.13	2.10E-02	18000	1	12	350	30	0.001	0.017	70	25550	5.79E-05	2.90E-02	2E-06
Tetrachloroethene	0.013	4.80E-02	18000	1	12	350	30	0.001	0.017	70	25550	1.36E-05	5.20E-02	7E-07
Chlorobenzene	0.15	4.10E-02	18000	1	12	350	30	0.001	0.017	70	25550	1.32E-04	NA	
Semivolatile Organics:														
1,3-Dichlorobenzene	0.51	8.70E-02	18000	1	12	350	30	0.001	0.017	70	25550	9.38E-04	NA	
1,4-Dichlorobenzene	0.36	6.20E-02	18000	1	12	350	30	0.001	0.017	70	25550	4.72E-04	2.40E-02	1E-05
1,2-Dichlorobenzene	0.91	6.10E-02	18000	1	12	350	30	0.001	0.017	70	25550	1.17E-03	NA	
1,2,4-Trichlorobenzene	48	1.00E-01	18000	1	12	350	30	0.001	0.017	70	25550	1.01E-01	NA	
PCBs:														
Aroclor-1242	0.0057	7.10E-01	18000	1	12	350	30	0.001	0.017	70	25550	8.48E-05	2.00E+00	2E-04
Aroclor-1248	30	7.10E-01	18000	1	12	350	30	0.001	0.017	70	25550	4.50E-01	2.00E+00	6E-01
Aroclor-1254	47	7.10E-01	18000	1	12	350	30	0.001	0.017	70	25550	7.05E-01	2.00E+00	8E-01
Aroclor-1260	0.0056	7.10E-01	18000	1	12	350	30	0.001	0.017	70	25550	8.40E-05	2.00E+00	2E-04
Inorganics:														
Aluminum	140	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	2.97E-03	NA	
Arsenic	0.035	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	7.38E-07	1.50E+00	1E-06
Barium	0.98	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	2.07E-05	NA	
Beryllium	0.012	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	2.43E-07	4.30E+00	1E-06
Cadmium	0.043	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	9.17E-07	NA	
Chromium	0.25	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	5.28E-06	NA	
Copper	0.78	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	1.65E-05	NA	
Iron	477	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	1.01E-02	NA	
Lead	0.28	4.00E-06	18000	1	12	350	30	0.001	0.017	70	25550	2.38E-08	NA	
Manganese	50	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	1.07E-03	NA	
Mercury	0	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	2.03E-08	NA	
Nickel	0.29	9.00E-06	18000	1	12	350	30	0.001	0.017	70	25550	5.52E-08	NA	
Silver	0.014	6.00E-04	18000	1	12	350	30	0.001	0.017	70	25550	1.78E-07	NA	
Vanadium	0.27	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	5.61E-06	NA	
Zinc	18	6.00E-04	18000	1	12	350	30	0.001	0.017	70	25550	2.31E-04	NA	
Cyanide (ng/l):	0.056	1.00E-03	18000	1	12	350	30	0.001	0.017	70	25550	7.67E-07	NA	
Dioxins:														
Total Equivalent 2,3,7,8-TCDD	8.30E-09	1.40E+00	18000	1	12	350	30	0.001	0.017	70	25550	2.46E-10	1.50E+05	4E-05
													Sum =	1E+00

Table H-86

Alluvial Central Groundwater
Scenario: Future Resident
Pathway: Dermal Contact with Groundwater Contaminants while Showering
Receptor: Hypothetical Resident (>18 years)

NON-CARCINOGENIC
CDI = Cw x PC x SA x FSA x t x EF x ED x CF1 x CF2 x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	C_w Groundwater Concentration (mg/L)	PC Permeability Coefficient (cm/hr)	SA Total Body Surface Area (cm2)	FSA Fraction SA Exposed (unitless)	t Time Spent in Shower (min/d)	EF Exposure Frequency (d/yr)	ED Exposure Duration (yrs)	CF1 Conversion Factor (L/cm3)	CF2 Conversion Factor (hr/min)	BW Body Weight (kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-d)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Volatile Organics:														
1,1-Dichloroethene	0.0050	1.60E-02	18000	1	12	350	30	0.001	0.017	70	10950	3.95E-06	9.00E-03	4.4E-04
1,2-Dichloroethene (total)	0.0060	1.02E-02	18000	1	12	350	30	0.001	0.017	70	10950	4.53E-06	9.00E-03	5.0E-04
1,2-Dichloroethane	0.0060	5.30E-03	18000	1	12	350	30	0.001	0.017	70	10950	1.57E-06	3.00E-02	5.2E-05
1,1,1-Trichloroethane	0.086	1.70E-02	18000	1	12	350	30	0.001	0.017	70	10950	5.52E-05	3.50E-02	1.6E-03
Trichloroethene	4.0	1.60E-02	18000	1	12	350	30	0.001	0.017	70	10950	3.18E-03	6.00E-03	5.3E-01
Benzene	0.20	2.10E-02	18000	1	12	350	30	0.001	0.017	70	10950	3.00E-04	3.00E-03	1.0E-01
Tetrachloroethene	0.025	4.80E-02	18000	1	12	350	30	0.001	0.017	70	10950	5.92E-05	1.00E-02	5.9E-03
Chlorobenzene	0.31	4.10E-02	18000	1	12	350	30	0.001	0.017	70	10950	6.27E-04	2.00E-02	3.1E-02
Semivolatile Organics :													NA	
1,3-Dichlorobenzene	0.51	8.70E-02	18000	1	12	350	30	0.001	0.017	70	10950	2.19E-03	8.90E-02	2.5E-02
1,4-Dichlorobenzene	0.36	6.20E-02	18000	1	12	350	30	0.001	0.017	70	10950	1.10E-03	NA	
1,2-Dichlorobenzene	0.91	6.10E-02	18000	1	12	350	30	0.001	0.017	70	10950	2.74E-03	9.00E-02	3.0E-02
1,2,4-Trichlorobenzene	48	1.00E-01	18000	1	12	350	30	0.001	0.017	70	10950	2.37E-01	1.00E-02	2.4E+01
PCBs :														
Aroclor-1242	0.0057	7.10E-01	18000	1	12	350	30	0.001	0.017	70	10950	1.98E-04	NA	
Aroclor-1248	30	7.10E-01	18000	1	12	350	30	0.001	0.017	70	10950	1.05E+00	NA	
Aroclor-1254	47	7.10E-01	18000	1	12	350	30	0.001	0.017	70	10950	1.65E+00	2.00E-05	8.2E+04
Aroclor-1260	0.0056	7.10E-01	18000	1	12	350	30	0.001	0.017	70	10950	1.96E-04	NA	
Inorganics :														
Aluminum	140	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	6.92E-03	1.00E+00	6.9E-03
Arsenic	0.035	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	1.72E-06	3.00E-04	5.7E-03
Barium	0.98	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	4.83E-05	7.00E-02	6.9E-04
Beryllium	0.012	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	5.67E-07	5.00E-03	1.1E-04
Cadmium	0.043	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	2.14E-06	5.00E-04	4.3E-03
Chromium	0.25	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	1.23E-05	5.00E-03	2.5E-03
Copper	0.78	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	3.84E-05	4.00E-02	9.6E-04
Iron	477	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	2.35E-02	3.00E-01	7.8E-02
Lead	0.38	4.00E-06	18000	1	12	350	30	0.001	0.017	70	10950	5.56E-08	NA	
Manganese	50	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	2.49E-03	2.30E-02	1.1E-01
Mercury	0.0010	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	4.73E-08	1.00E-04	4.7E-04
Nickel	0.29	9.00E-06	18000	1	12	350	30	0.001	0.017	70	10950	1.29E-07	2.00E-02	6.4E-06
Silver	0.014	6.00E-04	18000	1	12	350	30	0.001	0.017	70	10950	4.15E-07	5.00E-03	8.3E-05
Vanadium	0.27	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	1.31E-05	7.00E-03	1.9E-03
Zinc	18	6.00E-04	18000	1	12	350	30	0.001	0.017	70	10950	5.38E-04	3.00E-01	1.8E-03
Cyanide (ug/l):	0.036	1.00E-03	18000	1	12	350	30	0.001	0.017	70	10950	1.79E-06	NA	
Dioxins :														
Total Equivalent 2,3,7,8-TCDD	8.3E-09	1.40E+00	18000	1	12	350	30	0.001	0.017	70	10950	5.73E-10	NA	
													Sum =	8.2E+04

Table H-87

Alluvial Central Groundwater
Scenario: Future Resident
Pathway: Ingestion of Groundwater
Receptor: Hypothetical Resident (>18 years)

CARCINOGENIC
CDI = Cw x IR x EF x ED x 1/BW x 1/ATc
Risk = CDI x CSF

Chemical	C_w Groundwater Concentration (mg/L)	IR Ingestion Rate (l/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	BW Body Weight (Kg)	ATc Averaging Time, Carcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	CSF Cancer Slope Factor $(mg/kg\text{-}day)^{-1}$	Risk
Volatile Organics:									
1,1-Dichloroethene	0.0050	2	350	30	70	25550	5.87E-05	6.00E-01	4E-05
1,2-Dichloroethene (total)	0.0090	2	350	30	70	25550	1.06E-04	NA	
1,2-Dichloroethane	0.0060	2	350	30	70	25550	7.05E-05	9.10E-02	6E-06
1,1,1-Trichloroethane	0.066	2	350	30	70	25550	7.74E-04	NA	
Trichloroethene	4.0	2	350	30	70	25550	4.73E-02	1.10E-02	5E-04
Benzene	0.29	2	350	30	70	25550	3.41E-03	2.90E-02	1E-04
Tetrachloroethene	0.025	2	350	30	70	25550	2.94E-04	5.20E-02	2E-05
Chlorobenzene	0.31	2	350	30	70	25550	3.64E-03	NA	
Semivolatile Organics:									
1,3-Dichlorobenzene	0.51	2	350	30	70	25550	5.99E-03	NA	
1,4-Dichlorobenzene	0.36	2	350	30	70	25550	4.23E-03	2.40E-02	1E-04
1,2-Dichlorobenzene	0.91	2	350	30	70	25550	1.07E-02	NA	
1,2,4-Trichlorobenzene	48	2	350	30	70	25550	5.64E-01	NA	
PCBs:									
Aroclor-1242	0.0057	2	350	30	70	25550	6.63E-05	2.00E+00	1E-04
Aroclor-1248	30	2	350	30	70	25550	3.52E-01	2.00E+00	5E-01
Aroclor-1254	47	2	350	30	70	25550	5.52E-01	2.00E+00	7E-01
Aroclor-1260	0.0056	2	350	30	70	25550	6.58E-05	2.00E+00	1E-04
Inorganics:									
Aluminum	140	2	350	30	70	25550	1.63E+00	NA	
Arsenic	0.035	2	350	30	70	25550	4.10E-04	1.50E+00	6E-04
Barium	0.98	2	350	30	70	25550	1.15E-02	NA	
Beryllium	0.012	2	350	30	70	25550	1.35E-04	4.30E+00	6E-04
Cadmium	0.043	2	350	30	70	25550	5.10E-04	NA	
Chromium	0.25	2	350	30	70	25550	2.94E-03	NA	
Copper	0.78	2	350	30	70	25550	9.15E-03	NA	
Iron	477	2	350	30	70	25550	5.60E+00	NA	
Lead	0.28	2	350	30	70	25550	3.31E-03	NA	
Manganese	50	2	350	30	70	25550	5.95E-01	NA	
Mercury	0.0010	2	350	30	70	25550	1.13E-05	NA	
Nickel	0.29	2	350	30	70	25550	3.41E-03	NA	
Silver	0.014	2	350	30	70	25550	1.63E-04	NA	
Vanadium	0.27	2	350	30	70	25550	3.11E-03	NA	
Zinc	18	2	350	30	70	25550	2.14E-01	NA	
Cyanide (ug/l):	0.036	2	350	30	70	25550	4.26E-04	NA	
Dioxins:									
Total Equivalent 2,3,7,8-TCDD	8.30E-09	2	350	30	70	25550	9.75E-11	1.50E+05	1E-05
								Sum =	1E+00

Table H-88

Alluvial Central Groundwater
Scenario: Future Resident
Pathway: Ingestion of Groundwater
Receptor: Hypothetical Resident (>18 years)

NONCARCINOGENIC
CDI = Cw x IR x EF x ED x 1/BW x 1/ATnc
HQ = CDI/RfD

Chemical	C_w Groundwater Concentration (mg/L)	IR Ingestion Rate (l/day)	EF Exposure Frequency (days/year)	ED Exposure Duration (years)	BW Body Weight (Kg)	ATnc Averaging Time, Noncarcinogen (days)	CDI Chronic Daily Intake (mg/kg-day)	RfD Reference Dose (mg/kg-d)	HQ Hazard Quotient
Volatile Organics:									
1,1-Dichloroethene	0.0010	2	350	30	70	10950	1.37E-04	9.00E-03	1.5E-02
1,2-Dichloroethene (total)	0.0090	2	350	30	70	10950	2.47E-04	9.00E-03	2.7E-02
1,2-Dichloroethane	0.0060	2	350	30	70	10950	1.64E-04	3.00E-02	5.5E-03
1,1,1-Trichloroethane	0.066	2	350	30	70	10950	1.81E-03	3.50E-02	5.2E-02
Trichloroethene	4.03	2	350	30	70	10950	1.10E-01	6.00E-03	1.8E+01
Benzene	0.29	2	350	30	70	10950	7.95E-03	3.00E-03	2.6E+00
Tetrachloroethene	0.025	2	350	30	70	10950	6.85E-04	1.00E-02	6.8E-02
Chlorobenzene	0.31	2	350	30	70	10950	8.49E-03	2.00E-02	4.2E-01
Semivolatile Organics:									
1,3-Dichlorobenzene	0.51	2	350	30	70	10950	1.40E-02	8.90E-02	1.6E-01
1,4-Dichlorobenzene	0.36	2	350	30	70	10950	9.86E-03	NA	
1,2-Dichlorobenzene	0.91	2	350	30	70	10950	2.49E-02	9.00E-02	2.8E-01
1,2,4-Trichlorobenzene	48	2	350	30	70	10950	1.32E+00	1.00E-02	1.3E+02
PCBs:									
Aroclor-1242	0.0057	2	350	30	70	10950	1.55E-04	NA	
Aroclor-1248	30	2	350	30	70	10950	8.22E-01	NA	
Aroclor-1254	47	2	350	30	70	10950	1.29E+00	2.00E-05	6.4E+04
Aroclor-1260	0.0056	2	350	30	70	10950	1.53E-04	NA	
Inorganics:									
Aluminum	140	2	350	30	70	10950	3.85E+00	1.00E+00	3.8E+00
Arsenic	0.035	2	350	30	70	10950	9.56E-04	3.00E-04	3.2E+00
Barium	0.98	2	350	30	70	10950	2.68E-02	7.00E-02	3.8E-01
Beryllium	0.012	2	350	30	70	10950	3.15E-04	5.00E-03	6.3E-02
Cadmium	0.043	2	350	30	70	10950	1.18E-03	5.00E-04	2.4E+00
Chromium	0.25	2	350	30	70	10950	6.85E-03	5.00E-03	1.4E+00
Copper	0.78	2	350	30	70	10950	2.13E-02	4.00E-02	5.3E-01
Iron	477	2	350	30	70	10950	1.31E+01	3.00E-01	4.4E+01
Lead	0.28	2	350	30	70	10950	7.73E-03	NA	
Manganese	50	2	350	30	70	10950	1.38E+00	2.30E-02	6.0E+01
Mercury	0.0010	2	350	30	70	10950	2.63E-05	1.00E-04	2.6E-01
Nickel	0.29	2	350	30	70	10950	7.95E-03	2.00E-02	4.0E-01
Silver	0.014	2	350	30	70	10950	3.84E-04	5.00E-03	7.7E-02
Vanadium	0.27	2	350	30	70	10950	7.27E-03	7.00E-03	1.0E+00
Zinc	18	2	350	30	70	10950	4.96E-01	3.00E-01	1.7E+00
Cyanide (ug/l):	0.036	2	350	30	70	10950	9.95E-04	NA	
Dioxins:									
Total Equivalent 2,3,7,8-TCDD	8.30E-09	2	350	30	70	10950	2.27E-10	NA	
								Sum =	6.8E+04

Appendix I

EPA Comments on the Baseline Risk Assessment

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Pennsylvania Dept. of Env. Protection
230 Chestnut Street
Meadville, PA 16335-3481

RE: Finalization of Human Health Risk Assessment at the Former Sharon Transformer Plant Site

Dear Mr. Tordella:

On behalf of CBS Corporation (CBS) (herein after all references to Westinghouse have been changed to CBS), ChemRisk has reviewed the United States Environmental Protection Agency (EPA) Region III comments on our November 26, 1997 revised Human Health Risk Assessment Report received by CBS on February 12, 1998. In response to these comments, this letter transmits revised text, tables, and an additional appendix for incorporation into the Risk Assessment Report, including new risk summary tables that demonstrate the consistency of results computed by ChemRisk and EPA.

ChemRisk had previously computed risks for 19 exposure scenarios as reported in November, 1997. The EPA review comments do not modify any of the risks from these 19 exposure scenarios, to the extent that they would, under National Contingency Plan (NCP) requirements, be included (or excluded) in the assessment of alternatives by the Feasibility Study (FS).

EPA computed risks for an additional exposure scenario (unrestricted worker access to the Moat) that results in human health risks outside the NCP target range. This additional exposure scenario also does not represent the baseline condition. Unrestricted access to the Moat is precluded by existing institutional controls installed by CBS several years ago as mandated by the consent agreement between CBS and the Pennsylvania Department of Environmental Protection (PADEP) that also governs the performance of the RI/FS. Nonetheless, to prevent further delay in remediation, it is our understanding that CBS will address mitigation of unacceptable risk due to the hypothetical unrestricted worker access to the Moat in the FS.

ChemRisk is hereby providing responses to the February 10, 1998 comments by PADEP and EPA. To aid in the insertion of revised pages into the final risk assessment, all attached text and tables are included in the order they appear in the risk assessment document. In addition, ChemRisk has included revisions to Sections 1.0 and 6.0 to clarify any changes made in the risk assessment report. In those instances where a response is not provided, CBS accepts the comment and has included the comment in the new Appendix I, which will contain both the full text of the February 10, 1998 comments by EPA and PADEP and this letter in response to those comments.

Response to EPA Comments

Comment #2: See response to Comment #3e.

Comment #3e: Bullet #2: In accordance with the RI Work Plan, sediment samples were only scheduled to be analyzed for site-specific constituents of interest as defined by the Phase IB Field Sampling Plan. The lab data reports, however, included the TCL compounds. The EPA validator then reviewed and validated all TCL data in the sediment samples. Although many of these data results were not intended for use in the CBS risk assessment, EPA's inclusion of these results does not substantially change the risk results. In fact, using the RI/FS TCL data results in a lower risk compared to using the TCL data collected by PADEP.
Bullet #4: The revised TCB/DCB river sediment data are from the CBS data set, which did undergo EPA validation. The previous version had used the PADEP TCB/DCB data.

Comment #3f: As reported previously, supporting documentation for some lead values could not be located. These samples were analyzed in 1992 and the laboratory has since come under new ownership.

Comment #3h: Table C-9, CBS agrees that the original detection limits should be restored, as noted by EPA. A revised Table C-9 is included.
In addition, for the 56th bullet, information on non-verification of results was added to the third paragraph on page 5-13 in the final risk assessment and not the fourth paragraph, as indicated by EPA. A revised page 5-13 is attached.

Comment #3i: The information on non-verification of results in Table C-10 was added to the third paragraph on page 5-13 in the final risk assessment and not the fourth paragraph, as indicated by EPA. A revised page 5-13 is attached.
In addition, for sample R-1 in Table C-10 (19th bullet), the EPA validator incorrectly transcribed the 2,3,7,8-TCDD concentration from the edited lab sheet to the validator's summary sheet. Thus, the correct TEQ for R-1 should be 0.0007, which when averaged with the duplicate sample results in a TEQ for the RA of 0.0004 ng/l. A revised Table C-10 is attached.

Comment #7a,b: Total Petroleum Hydrocarbons (TPH) were deleted from Tables C-2 and C-3, as noted by EPA. Revised Tables C-2 and C-3 are included.

Comment #10: Information supporting the appropriateness of the lognormal assumption was added to the second paragraph on page 3-6 in the final risk assessment and not the third paragraph as noted by EPA. A revised page 3-6 is included.

Comment #11a: See Comment #3e.

Comment #11g: 1,2-DCE was not on the PADEP approved Phase IB parameter list for this well. The concentration of 3300 ug/l was reported when the lab was asked to provide full TCL data in 1994 (2 years later). Other than the data summary sheet, provided in 1994, no additional supporting data for this result is available.

Comment #11h: The exposure point concentrations for cadmium, lead in Tables 3-9 and D-9 have been updated consistent with EPA comments. Revised tables are attached.

Comment #11i: The exposure point concentrations for manganese and Aroclor 1260 in Tables 3-10 and D-10 have been updated consistent with EPA comments. Revised tables are attached.

Comment #25: The "Source" column in Table 3-22 was updated to read "5 days/year for 25 years". A revised Table 3-22 is included.

Comment #38: Tables 4-7 and 4-8 have been noted to refer the reader to Appendix I. Revised tables are attached.

Comment #43: The second paragraph on page G-110 has been deleted. A revised page is attached.

Comment #48: Tables H-37, H-38, H-53, H-54, H-55, H-56, H-72, and H-75 have been updated in accordance with EPA comments. The revised tables are attached.

Comment # 49: Utility worker inhalation cancer risk and hazard has been updated in Tables 5-1, H-37 and H-38. In addition, all risk results reported in EPA's comments have been updated in Section 5. Corrected tables as well as a revised Section 5 are attached.

Comment #50: See comment #49.

Comment #55: A rationale for the use of unfiltered groundwater samples has been added to Section 2.4.5. Revised pages 2-16 and 2-17 are attached.

Comment #61: The HHRA results for Bedrock groundwater indicate both incremental carcinogenic risk and hazard index values above the NCP target range. Therefore, even though the data utilized in the HHRA for bedrock groundwater are not thought to be representative of aquifer conditions, bedrock groundwater will be addressed in the Feasibility Study. As a minimum, additional sampling of the bedrock groundwater will be undertaken in an effort to confirm conditions in bedrock aquifer and accomplish resolution of this issue consistent with the provisions of the NCP.

Comment #65,67: Comment #65 was not addressed as it was deleted by PADEP. The concentration of the components of LNAPL shown in Table E-1b were calculated by dividing the maximum Aroclor concentration in LNAPL as shown in the RI Table 4-6 and dividing by an approximate LNAPL density of 0.91 mg/L. Table E-1b has been revised to provide additional information regarding the calculation of Ci and Co. The units of ug/L on Table E-3 have been changed to mg/L. The revised table E-3 is also attached.

Comment #87: This sentence was changed in the fourth paragraph on page 5-7. The revised page is attached.

Comment #88: This sentence was deleted from the second paragraph on page 5-14. The revised page is attached.

New Comments on Added/Deleted Material

Bullet #1: The particle emission rate, Cp, calculated in Table 3-15 was used in Tables H-17 and H-18. The value of Cp in Table 3-15 is 2.177 mg/m^3. This value is rounded to 2 mg/m^3 in Tables H-17 and H-18. However, this value is less than that calculated by EPA for the same scenario (0.0822 mg/m^3). Table 3-15 has been revised to reflect the rounding process and to introduce EPA's computed PM-10 concentration. In addition, Tables H-17a and H-18a have been added, which calculated the hypothetical risks using EPA's PM-10 concentration. Revised Table 3-15 and Tables H-17a and H-18a are attached.

Bullet #2: See response for bullet #1, above.

Bullet #3: The last sentence in the first paragraph on page 3-14 was revised to read "PCBs north of the site have been reported to range from non-detect to 0.240 ppm; PCBs at and downstream of Clark sewer outfall, Wishart outfall, and the Franklin outfall have been reported to range from non-detect to 25 ppm, 0.82 to 13 ppm, and non-detect to 4.1 ppm, respectively." Although EPA indicates that this correction should be made to page 3-14, second paragraph, in the final version of the risk assessment, this change was made to the first paragraph on page 3-14. The revised page is attached.

Bullet #5: This sentence was deleted from the third paragraph on page 5-13 of the final risk assessment. The revised page is attached.

Review of the Air Modeling Portions of the Human Health Risk Assessment for the CBS Site'

Comment #1: EPA is correct in pointing out an error in the footnoted equation on Table E-1b in comment 1(b). However, the term ρi was not used in a spreadsheet equation. The correct term (Pv) was used to calculate the value of Co in the spreadsheet. A revised Table E-1b is attached.

EPA suggests that the term D/Lt has units of cm^2/m-sec. In fact, using the units information provided in Table E-3, it can be concluded that the term D/Lt has units of cm/sec. Thus, the emission rates calculated by ChemRisk are not in error by a factor of 1×10^{-2} as suggested by EPA.

Finally, EPA is correct that a factor of 0.168 is incorrectly applied to the indoor construction worker air calculations. The error arises from the use of an incorrect ceiling height in the spreadsheet calculation. ChemRisk should have used a ceiling height of 18.3 m; however, a value of 3.08 m was used. Therefore, the air concentrations used to compute the inhalation risks to the hypothetical indoor construction worker are approximately six times greater than they should be. Table E-3 has been revised and is attached. In addition, Tables H-11 and H-12 have been revised to use the corrected air concentrations from the revised Table E-3.

Comment #2: As pointed out in the above response, the units of D/Lt are correctly reported in the risk assessment as cm/sec. Therefore, the emission rates calculated by ChemRisk are not in error by a factor of 1×10^{-2} as reported by EPA. Rather they are reported correctly in Table E-4. It should be noted, however, that although Table E-4 reported correctly computed air concentrations, they were not correctly reported in risk tables H-17 and H-18. These tables have been revised to correct this error. In addition, EPA has calculated alternative air concentrations for this scenario. The EPA computed values have been added to Table E-4 and are used to compute alternative risk values in Tables H-17a and H-18a.

Comment # 8 Table 3-19 was revised to include a corrected derivation of the factor EF and to include EPA's PM-10 concentration estimate. Tables H-37 and H-38 have also been revised to reflect the corrected EF value. In addition, risk tables H-37a and H-38a have been added to reflect the risks associated with EPA's computed PM-10 values.

Summary of Comparative Risks

In addition to addressing specific points raised by EPA, CBS and ChemRisk believe that it is beneficial to present summary risks tabulated by ChemRisk together with EPA's to ascertain the ultimate effect of each set of results. The attached table shows cumulative carcinogenic and noncarcinogenic risk estimates as compiled by CBS and EPA. This table replaces Table 5-1 of the Risk Assessment Report. Below is a summary analysis of the comparative results:

- Both EPA and ChemRisk conclude that vapor inhalation in the Middle Building exceeds NCP target cancer and noncancer risk benchmarks. CBS is working with PADEP and EPA on implementing a pilot remediation program to decontaminate impacted building surfaces that are thought to be a source of indoor air contamination in the Middle Building, as many of the constituents in air were not measured in underlying groundwater. Full-scale interior decontamination will proceed after evaluation of the Pilot Study results.

- Both EPA and ChemRisk conclude that the dust and vapor inhalation exposure pathway for the hypothetical Indoor and Outdoor Construction Worker results in noncancer risk beyond the NCP target range.

- EPA concludes that hypothetical unrestricted worker access to the Moat would result in excess (unacceptable) carcinogenic and noncarcinogenic risk. EPA and ChemRisk's analyses of restricted access exposures (including a worker and child and adult trespasser), which are in keeping with both baseline, present, and likely future use of this land, did not result in unacceptable risks for these receptor populations. Current access restrictions are mandated as a requirement of the RI/FS consent agreement.

- Both EPA and ChemRisk calculations indicate that the railroad right-of-way surface concentrations of site chemicals of potential concern (COPC) exceed the target cancer and noncancer risk ranges for the hypothetical child and adolescent trespassers.

- Groundwater was estimated by EPA and ChemRisk to pose a hypothetical health risk beyond the NCP target range, assuming consumption, showering, and inhalation of vapors occurred on a long-term basis.

Given CBS's decision to include evaluation of the worker unrestricted access scenario for the Moat in the FS, it appears that PADEP, EPA, and CBS are now in complete concurrence with the results of the Human Health Risk Assessment as they effect the selection of site media for remedial alternatives evaluation in the FS.

Included as attachments to this letter are revisions to the Table of Contents, Section 1.0 (Introduction), Section 6.0 (Summary and Conclusions) and new Appendix I (EPA review comments dated 2/3/98 and this response letter prepared by ChemRisk on behalf of CBS). Also included are the tables and text modifications referred to herein. It is our belief that incorporation of these inserts into the November 1997 Risk Assessment Report will fulfill EPA requirements for the baseline risk assessment.

Sincerely,

Mark C. Maritato
Senior Health Scientist

cc: Vic Janosik
Jennifer Hubbard
Gordon Taylor
Pat O'Hara

Pennsylvania Department of Environmental Protection

230 Chestnut Street
Meadville, PA 16335-3481
February 10, 1998

Northwest Regional Office

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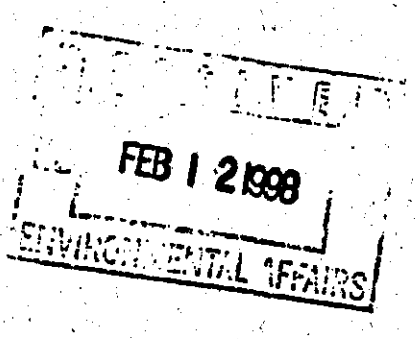

Mr. Gordon Taylor
Westinghouse Electric Corporation
1525 Westinghouse Building
Pittsburgh, PA 15222

RE: Westinghouse Electric (Sharon Plant) Site
November 26, 1997 Revised Baseline Human Health Risk Assessment

Dear Mr. Taylor:

Enclosed please find the comments prepared by the US EPA Toxicologist, Jennifer Hubbard, concerning the November 26, 1997 Revised Human Health Risk Assessment Report ("Risk Assessment") for the Sharon Transformer NPL Site. Also, enclosed are comments on the air modeling portions of the Risk Assessment (see comment Nos. 65 and 66 of J. Hubbards review), which were prepared by Patricia Flores, EPA Air Modeler.

Please revise the Risk Assessment to address these comments and submit the revised Risk Assessment within thirty (30) days of receipt of this letter.

As usual, if you have any questions, please give me a call.

Sincerely,

Charles L. Tordella
Project Manager
Environmental Cleanup

CLT:jb

Enclosure

cc: Mr. Janosik
Ms. Stainbrook

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION III
841 Chestnut Building
Philadelphia, Pennsylvania 19107

SUBJECT: Review of Westinghouse Sharon Revised Draft Risk Assessment

DATE: 2/3/98

FROM: Jennifer Hubbard, Toxicologist
Technical Support Section (3HS41) JRH

Received 2/4/98
by V. Janosik, RPM

TO: Vic Janosik, RPM
Western PA Remedial Section (3HS22)

The above document has been reviewed. Portions of this document were also referred to other EPA personnel; for example, the air modeling portions were reviewed by an air modeling expert.

Comments that were satisfactorily addressed are not discussed below; they are considered to be resolved. Comment numbers, below, refer to comments as numbered in my original 7/16/97 review. "Westinghouse's response" refers to the 11/26/97 letter to Charles Tordella of PADEP, which included responses to most of the risk assessment comments.

The discussion of previous comments is followed by a discussion of any new issues that arose as a result of additions to or deletions from the previous draft report.

DATA QUALITY

2. Westinghouse stated, in their comment response, that "PADEP data would be used except in cases where data obtained and validated by Westinghouse were available for the same compound." Westinghouse data were added, but not for all chemicals. For example, PADEP benzo[a]pyrene data were used even though validated Westinghouse data for benzo[a]pyrene were available.

   Also, Westinghouse used only data from locations SD-11A, SD-11B, SD-12A, SD-13A, SD-14A, SD-14AD, SD-15A, and SD-16A. Additional validated data from other river sediment locations are available.

   The source of the new trichlorobenzene and dichlorobenzene for these river sediment locations is also not clear; EPA did not appear to have these data.

   The consequences of the resulting discrepancies are discussed in Comments 3e, 7e, and 11a, as well as the discussion of the river sediment risks.

Celebrating 25 Years of Environmental Progress

3. Appendix C:

Some discrepancies between the reported data set and the EPA CRL evaluation occurred. Discrepancies that were not corrected include:

c) Table C-4, 12th bullet: Phenols in TB-7,S-2 should be rejected, R. This should include all phenolic compounds, not just phenol itself. However, this does not change the COPCs.

Also on this table, antimony in TB-8,S-3 and TB-8,S-4 should be J rather than R. However, this does not affect the COPC selection.

e) Table C-6, 1st bullet: The values shown on this table were from the unvalidated PADEP data set. For validated Westinghouse data, see the CRL data reviews.

Westinghouse data were used for some chemicals, but not for 1,1,2,2-PCA, 1,1,2-TCA, 1,2-DCA, 1,2-DCE, 1,2-dichloropropane, 2-hexanone, 4-methyl-2-pentanone, acetone, benzene, bromodichloromethane, bromoform, bromomethane, carbon disulfide, carbon tetrachloride, chloroethane, chloroform, chloromethane, cis-1,3-dichloropropene, dibromochloromethane, ethylbenzene, methylene chloride, styrene, PCE, trans-1,3-dichloropropene, vinyl acetate, vinyl chlroide, semivolatiles, and pesticides.

The result for Aroclor 1260 in SD-12A should be 4000 J ug/kg.

The source of the new trichlorobenzene and dichlorobenzene results for these river sediment locations is also not clear; EPA did not appear to have these data.

Also, Westinghouse used only data from locations SD-11A, SD-11B, SD-12A, SD-13A, SD-14A, SD-14AD, SD-15A, and SD-16A. Additional validated data from other river sediment locations are available.

f) Table C-7, 2nd bullet: The lead values for SW-5, 7/29; SW-7; and SW-9, 7/29 could not be confirmed; supporting data were not submitted in the comment response package.

g) Table C-8: Westinghouse's response refers to changes in samples M-15 and M-17. These actually refer to Table C-9, not C-8, and are discussed under item 3h,

directly below.

h) Table C-9, 1st bullet: The original comment was partly in error and is withdrawn in part, as follows. Benzene in M-5 and MW-16B should not be flagged B. Westinghouse correctly did not apply the B to M-5, but the B on MW-16B should be removed.

Table C-9, 15th bullet: The reported detection limits for 2-butanone in M-14 should be 10 ug/l; this change was omitted.

Table C-9, 18th bullet: The reported detection limits for trichloroethene in S-12R should be 20 ug/l; this change was omitted.

Table C-9, 24th bullet: Westinghouse made the changes to M-1, but omitted the following changes to M-15: Pesticide/PCB results should not be NA, but detection limits should be reported as follows:

500 ug/l for aldrin, the BHCs, alpha-chlordane, endosulfan I, gamma-chlordane, heptachlor, heptachlor epoxide;
1000 ug/l for the DDX compounds, dieldrin, endosulfan II, endosulfan sulfate, endrin, endrin aldehyde, endrin ketone;
5000 ug/l for methoxychlor;
10000 ug/l for Aroclor 1016, Aroclor 1232;
20000 ug/l for Aroclor 1221;
50000 ug/l for toxaphene.

Table C-9, 29th bullet: Westinghouse's response to comment 3g, above, refers to changes in samples M-15 and M-17. These actually refer to Table C-9, not C-8, and are discussed herein. The changes appear to be appropriate except the reference to PCBs in sample M-17. EPA's copy of the Form I data sheet for this sample shows detection limits of 50, 500, and 5000 ug/l rather than 500, 5000, and 50000 ug/l. (These detection limits were reflected in EPA's original comment, here at the 29th bullet of comment 3h.) Westinghouse is requested to restore the original detection limits or provide the background information for raising the detection limits.

Table C-9, 56th bullet: Except for manganese and cyanide, inorganic results and detection limits for M-15 were not verified because of difficulty obtaining supporting data. This information should be added to page 5-13, 4th paragraph.

i) Table C-10, 1st bullet: The reported results and

detection limits for certain VOCs in M-9, M-11B, S-1B, S-2B, S-8B, N-2B, N-3B, N-6B, and N-7B could not be verified because supporting data were not found. This information should be added to page S-13, 4th paragraph.

Table C-10, 13th bullet: August 1994 inorganic results are available for cyanide (10 ug/l) in S-1B, but were omitted.
Table C-10, 19th bullet: Westinghouse is correct that M-9 TCDD TEQs should be 0.0012 ng/l. However, the R-1 concentration was derived by averaging the results for R-1 (0.007 ng/l) and R-1D (0.0001 ng/l), and therefore should be 0.00355 ng/l, as in the original comment. Also, the result for S-8B was obscured on the table, but should be 0.00057 ng/l, as in the original comment.

COPC SCREENING

6. One relevant phrase was missed. Delete Section 2.4.5, 4th-5th line, the phrase, "and also to available federal and state MCLs."

7. Appendix C:

a) Table C-2: The 1,1,1-trichloroethane RBC should be 160000 ug/kg. However, this does not affect the final COPC selection. The 2-hexanone RBC on this table should be 310000, not 310, ug/kg. However, this does not affect the final COPC selection. It is still not clear why Total Petroleum Hydrocarbons (TPH) were selected as a COPC or what criteria could be used to evaluate TPH risk.

b) Table C-3: The 1,1,1-trichloroethane RBC should be 160000 ug/kg. However, this does not affect the final COPC selection. The 2-hexanone RBC on this table should be 310000, not 310, ug/kg. However, this does not affect the final COPC selection. It is still not clear why Total Petroleum Hydrocarbons (TPH) were selected as a COPC or what criteria could be used to evaluate TPH risk. The cyanide RBC should be 160 mg/kg, not 1600000 ug/l. However, this does not affect the final COPC selection.

c) Table C-4: The 1,1,1-trichloroethane RBC should be 4100000 ug/kg. However, this does not affect the final COPC selection. The 2-hexanone RBC on this table should be 8200000, not 8200, ug/kg. However, this does not affect the final COPC selection.

d) Table C-5: The 1,1,1-trichloroethane RBC should be

4100000 ug/kg. However, this does not affect the final COPC selection. The 2-hexanone RBC on this table should be 8200000, not 8200, ug/kg. However, this does not affect the final COPC selection.

e) Table C-6: For the Westinghouse vs. PADEP data set issue, see Comment 2, above. However, as stated in original EPA comment 49, the cancer risks using Westinghouse data would be about the same as those using the PADEP data, and HIs from the Westinghouse data would be lower than those from the PADEP data.

f) Table C-8: The RBC for 1,1,1-trichloroethane should be 54 ug/l. However, this does not affect the final COPC selection. The PCB RBC should be 0.03 ug/l; the source of 0.0087 ug/l is not clear. However, this does not change the final COPC selection. The cyanide RBC should be 73 ug/l. However, this does not affect the final COPC selection.

g) Table C-9: The RBC for 1,1,1-trichloroethane should be 54 ug/l. However, this does not change the final COPC selection. The PCB RBC should be 0.03 ug/l; the source of 0.0087 ug/l is not clear. However, this does not change the final COPC selection. The cyanide RBC should be 73 ug/l. However, this does not change the final COPC selection.

h) Table C-10: The RBC for 1,1,1-trichloroethane should be 54 ug/l. However, this does not change the final COPC selection. The methylmercury RBC should be 0.00037 mg/l. However, this does not change the final COPC selection. The cyanide RBC should be 73 ug/l. However, this does not change the final COPC selection.

8. The basis for the PADEP surface water criteria was added. For completeness, add to Section 2.4.4, "For a discussion of river surface water and RBCs, see Section 2.2.1."

EPCs

10. This report used an assumption of lognormality for all data sets. This assumption should have been supported by distributional tests but was not. EPA found that this assumption was acceptable for the Westinghouse data sets by testing with the Wilk-Shapiro test. The report should include information supporting the appropriateness of the lognormal assumption for this site, such as on page 3-6, after the 3rd paragraph. EPA's evaluation is repeated below, if sample wording is needed:

"Some of the data sets could be considered normal. However,

when using the Wilk-Shapiro test at alpha = 0.05, most data sets were confirmed to be lognormal. For data sets where normality had a higher W than lognormality, the lognormal assumption still passed the W test at alpha = 0.05 (except in one case: arsenic in bedrock groundwater). For data sets where neither the normal nor lognormal values passed at alpha = 0.05, the lognormal W was typically higher (i.e., was a better fit) than the normal W. Therefore, the lognormal assumption is acceptable for the data sets. This assumption is also expected to make little difference in the overall risks, based on a brief assessment of the use of normal upper confidence limits (UCLs) for southern groundwater data (risks were the same as or within an order of magnitude of the risks based on lognormal UCLs)."

11. a) Table 3-2 and Table D-2:

See also Comment 2. Under the current circumstances, Westinghouse used mostly the PADEP data, but Westinghouse data for PCBs and lead. EPA verified the EPCs that would be derived using the PADEP data, although different EPCs could be derived using the available Westinghouse data (however, see Comment 48 for the ultimate outcome of the two data sets).

EPA did not obtain the same EPC for Aroclor 1260 and lead, however, that appear on Tables D-2 and 3-2, using Westinghouse data. This is apparently due to Westinghouse's use of only samples SD-11A through SD16A. If the full complement of downstream samples were used, the EPC for Aroclor 1260 would be 25000 ug/kg, and for lead would be 1500 mg/kg. (Using the unvalidated PADEP data, the EPC for Aroclor 1260 would be 4700 ug/kg. Obviously, there is considerable variation.) The table should be footnoted, or this information should be added to the river assessment discussion. (This lead result also affects page 5-8, 3rd paragraph.)

g) Tables 3-8 and D-8: EPA could not find the supporting data or information for the reported detection of 1,2-DCE at 3300 E ug/l in S-10. (Furthermore, the E indicates that the concentration is an estimate because it exceeded the range determined by the standards; it should have been diluted and reanalyzed.) Therefore, EPA used the next highest concentration, 560 ug/l, in its original confirmation check. With the result of 3300, the risks are five-fold higher for this chemical.

h) Tables 3-9 and D-9:

The discrepancies in the aluminum and zinc EPCs is due

to the inclusion or exclusion of sample M-15, whose results could not be verified. (Westinghouse included the data.) Still, the EPCs, while noticeably different, are not vastly different (less than a factor of 2). The cadmium EPC should be 0.0674 mg/l; R data was incorrectly included in Westinghouse's calculation (see Comment 12). The lead EPC should be 0.692 mg/l; a transcription error on Table D-9 led to this being erroneously listed as 0.0692 mg/l (the sample in question is N-1). As noted previously, the benzene result in MW-16B should not be B, but this does not appear to affect the EPC. As noted previously, the TCE detection limit should be 20 ug/l in S-12R, but this does not appear to significantly affect the EPC.

i) Tables 3-10 and D-10:

The EPC for Aroclor 1260 should be 3.6 ug/l; Tables 3-10 and D-10 used the wrong n (should be 12 instead of 11). The EPC for manganese should be 0.133 mg/l. Tables 3-10 and D-10 apparently used the wrong value for S-8B, which should be 0.0066 K rather than 0.0066 B (and therefore should not be halved for the UCL calculation). Since the lognormal UCL exceeds the maximum positive concentration, the maximum of 0.133 mg/l should be used. See Comment 3i, above: the TCDD EPC should be 0.0035 ng/l.

12. EPA requested that the report clarify how B and R data were handled. This explanation was added. However, the stated approach to R data is inappropriate. Because R data are rejected, they are not suited for any data use, either qualitatively or quantitatively, to confirm or rule out a chemical's presence. They should be removed from the data set, not treated as detection limits as on page 3-6.

This also affects Tables 3-7, D-7, 3-9, D-9, 3-10, D-10.

EXPOSURE ASSESSMENT

20. The indoor worker skin fraction exposed was made consistent with the 22% value everywhere except on page 3-46, 1st paragraph, where it should also be changed to 0.22.

25. In changing Table 3-22 from 7 to 5 days/yr, the risk assessor neglected the rational in the "Source" column, which reads "1/mo, 7 months." The terms should be reconciled.

TOXICITY ASSESSMENT

34. Table 4-2: Nickel is not a carcinogen via the oral route.

Westinghouse did not add chemicals to these tables if the chemicals were only found above RBCs in Westinghouse river data. This is sufficient if the existence of these data is acknowledged elsewhere, as it is in Sections 2.2.1 and 5. See also Comments 2 and 48 for discussion of river risks.

35. Table 4-3:

b), c), e), f) See Comment 34, above, for conclusions relevant to benzo[k]fluoranthene, heptachlor, aldrin, and chloroform.

h) The inhalation CSF for TCDD is 1.5E5 per mg/kg/day.

i) Bis(2-ethylhexyl)phthalate has a provisional inhalation CSF of 1.4E-2 per mg/kg/day.

36. Table 4-5:

j), k) See Comment 34, above, for conclusions relevant to heptachlor and aldrin.

o) 1,1,1-trichloroethane has a provisional oral RfD of 2E-2 mg/kg/day. This has existed since August 1996.

37. Table 4-6:

a) There is a provisional inhalation RfD of 1.4E-3 mg/kg/day for 1,2-dichloroethane.

c) Westinghouse's response stated that the mercury RfC is 3E-4 and not 8.6E-5. Indeed, this is the RfC, in units of mg/m^3. However, the 8.6E-5 mg/kg/day value is the inhalation RfD derived from the RfC. Westinghouse did use the 8.6E-5 mg/kg/day RfD appropriately on Table 4-6, so no corrections are required.

e) The chromium inhalation RfD of 5.7E-7 mg/kg/day was added to the table. The chromium inhalation value has been further updated, with 1E-7 mg/kg/day (10 times less than the provisional subchronic RfD) now being recommended by NCEA. This would mainly impact only Tables H-40 and 5-1 for the railroad ROW maintenance worker inhalation pathway. However, the new RfD would not cause the HI to exceed 1.

38. Tables 4-7 and 4-8:

Again, these tables attempt to combine two different concepts into one factor. The result, in the dermal column, is a mixture of dermal ABS and oral-to-dermal adjustment factors. For example, factors such as 0.032 for arsenic and

0.03 for VOCs are dermal absorption factors for soil (used to adjust the intake), while 0.27 for aluminum is an oral-to-dermal adjustment factor (used to adjust the RfD).

The difficulty in trying to cover both bases in one factor for two different exposure routes (inhalation, which is calculated separately, is not addressed in this comment) is seen by the example of arsenic. Arsenic has a dermal absorption factor of 0.032 from soil, and therefore the dermal dose incorporates this factor. However, the toxicity factor, which is orally based, incorporates an approximate oral absorption of probably 0.95. RfDs would be adjusted by multiplying the RfD by 0.95; CSFs would be adjusted by dividing the CSF by 0.95. The relative absorption factors would then be 1 for oral exposure (0.95/0.95), but would involve both 0.032 and 0.95 for dermal exposure, with multiplication and division as appropriate for non-cancer or cancer routes. (Technically, as small an adjustment as 0.95 is not recommended and should be rounded to 1, but the example is used for illustration.) For dermal exposures from water, the RfD and CSF would still be adjusted for oral-to-dermal exposure, but instead of using a dermal absorption factor, the Kp and its related values are used to estimate the dermal intake for that route.

Therefore, the values shown in Table 4-8 may not be appropriate, depending upon their use in Appendix H. It is recommended that absorption factors and oral-to-dermal adjustment factors be separated, to avoid this confusion. Attachment 1 to this memo specifically outlines this for the Westinghouse site. The impact upon risks in Appendix H is discussed below in Comment 48.

The suggested values for 1,2-dichloroethane, 1,1-dichloroethene, and 1,2-dichloroethene were added to the table, but the old values were not removed. The TCDD factor does not appear to be appropriate, as noted before. Groundwater COPCs were not included. Also, the carcinogenic PAHs should not be evaluated by the oral-to-dermal method, since they are believed to act at the point of contact for dermal carcinogenicity. These issues are also rectified in Attachment 1.

39. Table 4-9:

e), g), j), m) See Comment 34, above, for conclusions relevant to benzo[k]fluoranthene, heptachlor, aldrin, and chloroform.

h) The dibenz[a,h]anthracene Kp should be 2.7 cm/hr; in spite of the response, this was not added to the table.

k) Westinghouse did not add chemicals to these tables if the chemicals were only found above RBCs in residential soil, as with chlordane. This is sufficient if the existence of these data is acknowledged elsewhere, as it is in Section 2.

43. Appendix G, chemicals not updated include:

Page G-2, the inhalation CSF for 1,1-DCE is 1.75E-1 per mg/kg/day based on the IRIS unit risk. (The inhalation CSF that appears on IRIS is incorrect, according to the IRIS toxicologist and a comparison with the unit risk.) It appears that the correct CSF of 1.75E-1 was used in all the calculations.

Page G-7, the provisional inhalation RfD for 1,2-dichlorobenzene is now 9E-3 mg/kg/day. (This also affects Table 4-6.)

Page G-25, the provisional oral RfD for 1,3-dichlorobenzene is now 3E-2 mg/kg/day and the provisional inhalation RfD is now 2E-3 mg/kg/day. (This also affects Tables 4-5 and 4-6.)

Page G-29, the provisional oral RfD for 1,4-dichlorobenzene is now 3E-2 mg/kg/day. (This also affects Table 4-5.)

Page G-30, the provisional inhalation RfD for aluminum is 1E-3 mg/kg/day. (This also affects Table 4-6.)

Page G-69, the provisional inhalation RfD for chromium is now 1E-7 mg/kg/day. (This also affects Table 4-6.)

Page G-112: Delete the second paragraph, which was superseded by the new shaded section.

Page G-120, the provisional inhalation CSF for benzo[a]pyrene is 3.1 per mg/kg/day. (Also affects Table 4-3 for the carcinogenic PAHs).

Page G-124, the provisional inhalation RfD for PCE is now 1.4E-1 mg/kg/day. (This also affects Table 4-6.)

Page G-149, the provisional oral RfD for 1,1,1-TCA is now 2E-2 mg/kg/day.

Page G-157, the oral RfD for methylmercury is 1E-4 mg/kg/day. It appears that the correct RfD was used in the calculations.

RISK CHARACTERIZATION

47. EPA's showering evaluation using the Foster and Chrostowski model showed that the McKone and Bogen inhalation risk estimates were very near those of the Foster and Chrostowski model for cancer risk (less than an order of magnitude below the Foster and Chrostowski estimates) but were often two orders of magnitude below the Foster and Chrostowski-based hazard indices. The major difference appears to be that the risk assessment did not evaluate inhalation risks for di- and tri- chlorinated benzenes, which are semivolatile but may evaporate, especially when heated. In any case, both shower models result in risks above the NCP risk range.

Attachment 2 contains a comparison of results using the two shower models.

48. Calculations in Appendix H were to be adjusted in response to comments. Discrepancies and their consequences are listed below. This time, each specific instance is listed separately:

Tables H-1 and H-2: If the full Westinghouse data set were used for Aroclor 1260 and lead, their respective EPCs would be 8 and 2 times higher. However, the overall "PADEP" cancer risk and HI would not change. (Using all validated Westinghouse data, the cancer risk would be 8E-6 and the HI would be 6E-3.)

Table H-3: As stated in original comment 38, the dermal cancer risks for PAHs should not be quantitated based on the oral CSFs, since these compounds appear to act locally. As per Attachment 1, the beryllium ABS should be 0.01 and the dermal CSF should be 430, for a beryllium cancer risk of 2E-6. If the full Westinghouse data set were used for Aroclor 1260 and lead, their respective EPCs would be 8 and 2 times higher. With this and the other adjustments, the "PADEP" cancer risk of 2E-6 would be approximately 3E-6 to 4E-6. (Using all validated Westinghouse data, the cancer risk would be 2E-6.)

Table H-4: If the full Westinghouse data set were used for Aroclor 1260 and lead, their respective EPCs would be 8 and 2 times higher. However, the overall "PADEP" HI would not change for that reason. As per Attachment 1, the ABS for beryllium and manganese should be 0.01, with RfDs adjusted so that the dermal RfDs would be as follows: aluminum, 2.7E-1; antimony, 4E-5; beryllium, 5E-5; cadmium, 2.5E-5; chromium, 5E-5; vanadium, 1E-4; zinc, 7.5E-2. The HI would then be 0.5. (Using all validated Westinghouse data, the HI would be 0.004.)

Table H-6: The tetrachloroethene RfD should be 1.4E-1, for an HQ of 5E-3. However, this does not change the overall HI

of 1.7.

Table H-7: As per Attachment 1, the beryllium ABS should be 0.01 and the dermal CSF should be 430, for a beryllium cancer risk of 4E-7. The total cancer risk would then be 6E-7.

Table H-8: As per Attachment 1, the ABS for beryllium and manganese should be 0.01, with RfDs adjusted so that the dermal RfDs would be as follows: beryllium, 5E-5. The total HI would then be 6E-2.

Tables H-11 and H-12: According to the EPA air modeler, the Cvs for Aroclor 1242 and the Cvg for the other Aroclors were incorrect. (Furthermore, the Cvg values that are shown on this table do not match the tables Appendix E, so it is not clear where they came from.) When these changes are made, the total cancer risk is 4E-7 rather than 5E-7. However, no impact is seen on the HI of 7.7, which was due solely to manganese.

Table H-13: As per Attachment 1, the beryllium ABS should be 0.01 and the dermal CSF should be 430, for a beryllium cancer risk of 5E-7. The total cancer risk would then be 8E-7.

Table H-14: As per Attachment 1, the ABS for beryllium and manganese should be 0.01, with RfDs adjusted so that the dermal RfDs would be as follows: beryllium, 5E-5. The total HI would then be 0.2.

Tables H-15 and H-16: The IgRF values do not appear to have been adjusted to the recommended value of 1 for beryllium, iron, and manganese. While the beryllium cancer risk should therefore be 8E-8, the overall cancer risk of 1E-6 does not change. The total HI should therefore be 0.7.

Tables H-17 and H-18: While the Cvg numbers do not match those in Appendix E, they do match the Tscreen numbers generated by the EPA air modeler and are believed to be correct. However, the value for 1,2-DCE may be biased high since it was driven by a high "E"-flagged result in groundwter. The Cvg for TCDD should be 2.3E-15 (an incorrect concentration for this appears in Table E-29, and the whole compound was crossed out on Tables H-17 and H-18). The Cvg and Cvs values appear to be based on hourly rates; annual averages (which are usually preferred for long-term risk assessment) would be lower. The Cvs values appeared to be in error, based on the EPA air modeler's review. If the trend of using hourly rates is continued, the Cvs for acenaphthylene should be 2.3E-5, for benzo[g,h,i]perylene should be 3.5E-9, for phenanthrene should be 1.4E-4, for

Aroclor 1242 could be up to 3.6E-4, for Aroclor 1254 should be 4.2E-4, and for Aroclor 1260 should be 2E-4. The inhalation CSF for TCDD should be 1.5E5. However, none of these numbers significantly change the total cancer risk of 5E-6. The RfDs should be as follows: 1,2-DCA, 1.4E-3; PCE, 1.4E-1; 1,3-DCB, 2E-3; 1,2-DCB, 9E-3. However, these factors do not significantly change the total HI of approximately 9.2.

Table H-19: As stated in original comment 38, the dermal cancer risks for PAHs should not be quantitated based on the oral CSFs, since these compounds appear to act locally. As per Attachment 1, the ABS values for dieldrin, beryllium, and manganese should be 0.1, 0.01, and 0.01, respectively. The beryllium dermal CSF should be 430. However, none of these changes significantly impact the total cancer risk of 2E-6.

Table H-20: As per Attachment 1, the ABS values for dieldrin, beryllium, and manganese should be 0.1, 0.01, and 0.01, respectively, with RfDs adjusted so that the dermal RfDs would be as follows: aluminum, 2.7E-1; beryllium, 5E-5; cadmium, 2.5E-5; mercury, 1.5E-5. However, the total HI of 0.04 would not be significantly impacted.

Table H-23: The benzo[a]pyrene inhalation CSF should be 3.1, but that does not significantly change the total cancer risk of 4E-10.

Tables H-25 and H-27: As per Attachment 1, the ABS values for dieldrin, beryllium and manganese should be 0.1, 0.01, and 0.01, respectively. The beryllium dermal CSF should be 430. However, the overall cancer risk of 5E-7 does not change significantly.

Tables H-26 and H-28: As per Attachment 1, the ABS values for dieldrin, beryllium, and manganese should be 0.1, 0.01, and 0.01, respectively, with RfDs adjusted so that the dermal RfDs would be as follows: aluminum, 2.7E-1; beryllium, 5E-5; cadmium, 2.5E-5; mercury, 1.5E-5. However, the total HI of 0.04 would not be significantly impacted.

Tables H-29 and H-30: The IgRF for manganese and mercury does not match that of the other metals. It appears that these numbers should have been applied to the ROA column instead, so the IgRF should be 0.5 and the ROA 1.0. However, the total cancer risk of 2E-6 does not change, nor does the total HI of 0.15 change significantly.

Table H-33: As stated in original comment 38, the dermal cancer risks for PAHs should not be quantitated based on the oral CSFs, since these compounds appear to act locally. As

per Attachment 1, the ABS value for beryllium should be 0.01. The beryllium dermal CSF should be 430. However, none of these changes significantly impact the total cancer risk of 3E-7.

Table H-34: As per Attachment 1, the ABS for beryllium should be 0.01, with the RfD adjusted so that the dermal RfD would be as follows: beryllium, 5E-5. The total HI would then be 5E-5, not a great change from 4E-5.

Table H-36: The arsenic RfD should not have been crossed out; the HQ should be 3E-4, and the total HI should be 3E-4.

Tables H-37 and H-38: The equation at the top of this table does not match the factors that are shown on the table (for example, PEF vs. Cp). EPA believes the Cp should also be 1.3 (hourly) or 0.1 (annual) (see EPA air modeler's review). The PAH CSFs should also be updated. Because of the uncertainties, the cancer risk and HI could not be verified. (Also affected by Table 3-19.)

Table H-39: The PAH CSFs should be updated. The TCDD CSF should be 1.5E5. However, these changes do not greatly affect the total cancer risk of 8E-10.

Table H-40: The dieldrin RfD is oral. New provisional RfDs for aluminum and chromium inhalation are 1E-3 and 1E-7, respectively. Using these new factors, the cancer risk (due to chromium) would be 3E-4.

Table H-41: As stated in original comment 38, the dermal cancer risks for PAHs should not be quantitated based on the oral CSFs, since these compounds appear to act locally. As per Attachment 1, the ABS values for beryllium and manganese should be 0.01. The beryllium dermal CSF should be 430. However, none of these changes significantly impact the total cancer risk of 3E-6.

Table H-42: As per Attachment 1, the ABS values for beryllium and manganese should be 0.01, with RfDs adjusted so that the dermal RfDs would be as follows: aluminum, 2.7E-1; antimony, 4E-5; beryllium, 5E-5; cadmium, 2.5E-5; chromium, 5E-5; copper, 2E-2; mercury, 1.5E-5; zinc, 7.5E-2. However, the total HI of 0.08 would not be significantly impacted.

Table H-49: As stated in original comment 38, the dermal cancer risks for PAHs should not be quantitated based on the oral CSFs, since these compounds appear to act locally. There is a stray risk of "2E-05" just above the TCDD risk that does not appear to belong. As per Attachment 1, the ABS values for beryllium, manganese and mercury should be

0.01. The beryllium dermal CSF should be 430. The total cancer risk should therefore be 2E-5 rather than 5E-5.

Table H-50: As per Attachment 1, the ABS values for beryllium, mercury and manganese should be 0.01, with RfDs adjusted so that the dermal RfDs would be as follows: aluminum, 2.7E-1; antimony, 4E-5; beryllium, 5E-5; cadmium, 2.5E-5; chromium, 5E-5; copper, 2E-2; mercury, 1.5E-5; zinc, 7.5E-2. However, the total HI of 2.5 would not be significantly impacted.

Table H-51: As stated in original comment 38, the dermal cancer risks for PAHs should not be quantitated based on the oral CSFs, since these compounds appear to act locally. As per Attachment 1, the ABS values for beryllium, manganese and mercury should be 0.01. The beryllium dermal CSF should be 430. However, the total cancer risk of 2E-5 would not be significantly impacted.

Table H-52: As per Attachment 1, the ABS values for beryllium, mercury and manganese should be 0.01, with RfDs adjusted so that the dermal RfDs would be as follows: aluminum, 2.7E-1; antimony, 4E-5; beryllium, 5E-5; cadmium, 2.5E-5; chromium, 5E-5; copper, 2E-2; mercury, 1.5E-5; zinc, 7.5E-2. However, the total HI of 2.3 would not be significantly impacted.

Tables H-53 and H-55: The factor of 0.017 hr/min is inappropriate on this table because here, t is given in hours rather than minutes. Therefore, CF2 is unnecessary and risks are 60 times too low. Non-steady-state risks would total 2E-5; steady-state risks would total 2E-6, using the estimated PC for TCE. (Steady-state risks, using the measured PC for TCE, would be 3E-6.)

Table H-54: The factor of 0.017 hr/min is inappropriate on this table because here, t is given in hours rather than minutes. Therefore, CF2 is unnecessary and HIs are 60 times too low. Non-steady-state HIs would total 7E-4; steady-state HIs would total 1E-4, using the estimated PC for TCE. (Steady-state HIs, using the measured PC for TCE, would be 5E-4.)

Table H-56: The factor of 0.017 hr/min is inappropriate on this table because here, t is given in hours rather than minutes. Therefore, CF2 is unnecessary and HIs are 60 times too low. Non-steady-state HIs would total 6E-4; steady-state HIs would total 1E-4, using the estimated PC for TCE. (Steady-state HIs, using the measured PC for TCE, would be 5E-4.)

Table H-57: Using the Foster and Chrostowski model and

including all organic COPCs, this cancer risk would be 9E-5.

Table H-58: Using the Foster and Chrostowski model and including all organic COPCs, this HI would be 9.5.

Table H-59: Because the shower model used affects the concentrations available for dermal risk, the McKone and Bogen vs. Foster and Chrostowski risks are likely to differ slightly. Also, this table uses the steady-state model, and the non-steady-state model is also available. The beryllium dermal CSF should be 430. However, the total McKone, steady-state cancer risk of 3E-4 would not be significantly impacted. (Using the Foster and Chrostowski and non-steady-state assumptions, the risk would be 5E-3.)

Table H-60: The RfDs for 1,3-DCB and 1,4-DCB are now 3E-2. As per Attachment 1, the RfDs should be adjusted so that the dermal RfDs would be as follows: aluminum, 2.7E-1; beryllium, 5E-5; cadmium, 2.5E-5; chromium, 5E-5; copper, 2E-2; mercury, 1.5E-5; nickel, 2E-4; vanadium, 1E-4; zinc, 7.5E-2. The total McKone, steady-state HI should therefore be 19 rather than 18. (Using the Foster and Chrostowski and non-steady-state assumptions, the HI would be 310.)

Table H-61: The TCDD concentration does not match the EPC on Table D-8. It should be 1.4E-8 mg/l, with a cancer risk of 7E-6. The Aroclor 1254 risk also seems to be miscalculated; it should be 9E-5 rather than 2E-5. However, the total risk of 2E-3 is not significantly affected.

Table H-62: The TCDD concentration does not match the EPC on Table D-8. It should be 1.4E-8 mg/l. The RfDs for 1,3-DCB and 1,4-DCB are now 3E-2, with HIs of 0.2. However, these issues do not greatly impact the total HI of approximately 40.

Table H-63: Using the Foster and Chrostowski model and including all organic COPCs, this cancer risk would also be 1E-4.

Table H-64: Using the Foster and Chrostowski model and including all organic COPCs, this HI would be 13.

Table H-65: Because the shower model used affects the concentrations available for dermal risk, the McKone and Bogen vs. Foster and Chrostowski risks are likely to differ slightly. Also, this table uses the steady-state model, and the non-steady-state model is also available. The beryllium dermal CSF should be 430, with a McKone, steady-state cancer risk of 5E-5. The TCDD concentration does not match the EPC on Table D-8. It should be 1.4E-8 mg/l, with a McKone, steady-state cancer risk of 6E-5. Therefore, the total

McKone, steady-state cancer risk should be 3E-4. (Using the Foster and Chrostowski and non-steady-state assumptions, the risk would be 7E-3.)

Table H-66: The RfDs for 1,3-DCB and 1,4-DCB are now 3E-2. As per Attachment 1, the RfDs should be adjusted so that the dermal RfDs would be as follows: aluminum, 2.7E-1; beryllium, 5E-5; cadmium, 2.5E-5; chromium, 5E-5; copper, 2E-2; mercury, 1.5E-5; nickel, 2E-4; vanadium, 1E-4; zinc, 7.5E-2. The TCDD concentration does not match the EPC on Table D-8. It should be 1.4E-8 mg/l. The total McKone, steady-state HI should therefore be 26 rather than 25. (Using the Foster and Chrostowski and non-steady-state assumptions, the HI would be 440.)

Table H-67; The TCDD concentration does not match the EPC on Table D-8. It should be 1.4E-8 mg/l, with a cancer risk of 2E-5. The Aroclor 1254 risk also seems to be miscalculated; it should be 3E-4 rather than 8E-5. However, the total risk of 8E-3 is not significantly affected.

Table H-68: The TCDD concentration does not match the EPC on Table D-8. It should be 1.4E-8 mg/l. The RfDs for 1,3-DCB and 1,4-DCB are now 3E-2, with HIs of 0.5 and 0.4, respectively. The TCE HI also appears to be miscalculated; it should be 0.8 rather than 2.6. However, these issues do not greatly impact the total HI of approximately 110.

Table H-69: The EPCs for Aroclor 1260, manganese, and TCDD should be 0.0036, 0.133, and 3.6E-9 mg/l, respectively. Because the shower model used affects the concentrations available for dermal risk, the McKone and Bogen vs. Foster and Chrostowski risks are likely to differ slightly. Also, this table uses the steady-state model, and the non-steady-state model is also available. The beryllium dermal CSF should be 430, with a McKone, steady-state cancer risk of 2E-6. As stated in original comment 38, the dermal cancer risks for PAHs should not be quantitated based on the oral CSFs, since these compounds appear to act locally. Bis(2-ethylhexyl)phthalate's adjusted dermal CSF would be 2.6E-2. However, the total McKone, steady-state cancer risk would not deviate from 2E-4. (Using the Foster and Chrostowski and non-steady-state assumptions, the risk would be 2E-3.)

Table H-70: The EPCs for Aroclor 1260, manganese, and TCDD should be 0.0036, 0.133, and 3.6E-9 mg/l, respectively. Bis(2-ethylhexyl)phthalate's adjusted dermal RfD should be 1.1E-2. The adjusted dermal RfDs for metals should be as follows: aluminum, 2.7E-1; cadmium, 2.5E-5; beryllium, 5E-5. The metals HIs are missing from this table. However, the total McKone, steady-state HI would not deviate significantly from 4.1. (Using the Foster and Chrostowski

and non-steady-state assumptions, the HI would be 75.)

Table H-71: The EPCs for Aroclor 1260, manganese, and TCDD should be 0.0036, 0.133, and 3.6E-9 mg/l, respectively. The bis(2-ethylhexyl)phthalate CSF of 1.4E-2 is missing from this table. However, the total cancer risk would not be significantly impacted.

Table H-72: This table should be labeled, "Ingestion" rather than "Dermal Contact . . . while Showering." The EPCs for Aroclor 1260, manganese, and TCDD should be 0.0036, 0.133, and 3.6E-9 mg/l, respectively. The bis(2-ethylhexyl)phthalate RfD of 2E-2 is missing from this table. The metals HIs are missing from this table. Therefore, the total HI should be 2.6 rather than 1.6.

Table H-73: The EPCs for Aroclor 1260, manganese, and TCDD should be 0.0036, 0.133, and 3.6E-9 mg/l, respectively. Because the shower model used affects the concentrations available for dermal risk, the McKone and Bogen vs. Foster and Chrostowski risks are likely to differ slightly. Also, this table uses the steady-state model, and the non-steady-state model is also available. The beryllium dermal CSF should be 430, with a McKone, steady-state cancer risk of 3E-6. As stated in original comment 38, the dermal cancer risks for PAHs should not be quantitated based on the oral CSFs, since these compounds appear to act locally. Bis(2-ethylhexyl)phthalate's adjusted dermal CSF would be 2.6E-2. Therefore, the total McKone, steady-state cancer risk would be 5E-4 rather than 3E-4. (Using the Foster and Chrostowski and non-steady-state assumptions, the risk would be 3E-3.)

Table H-74: The EPCs for Aroclor 1260, manganese, and TCDD should be 0.0036, 0.133, and 3.6E-9 mg/l, respectively. Bis(2-ethylhexyl)phthalate's adjusted dermal RfD would be 1.1E-2. The adjusted dermal RfDs for metals should be as follows: aluminum, 2.7E-1; cadmium, 2.5E-5; beryllium, 5E-5. However, the total McKone, steady-state HI would not deviate significantly from 5.8. (Using the Foster and Chrostowski and non-steady-state assumptions, the HI would be 110.)

Table H-75: This table should be labeled "Bedrock" rather than "North." The EPCs for Aroclor 1260, manganese, and TCDD should be 0.0036, 0.133, and 3.6E-9 mg/l, respectively. The bis(2-ethylhexyl)phthalate CSF of 1.4E-2 is missing from this table. However, the total cancer risk would not be significantly impacted.

Table H-76: The EPCs for Aroclor 1260, manganese, and TCDD should be 0.0036, 0.133, and 3.6E-9 mg/l, respectively. The bis(2-ethylhexyl)phthalate RfD of 2E-2 is missing from this table. However, the HI of 7.2 is not significantly

impacted.

Table H-77: Using the Foster and Chrostowski model and including all organic COPCs, this cancer risk would also be 3E-4.

Table H-78: The provisional RfDs for 1,2-DCA and PCE are 1.4E-3 and 0.14, respectively. However, the total McKone HI would not be greatly affected. Using the Foster and Chrostowski model and including all organic COPCs, this HI would be 380.

Table H-79: Because the shower model used affects the concentrations available for dermal risk, the McKone and Bogen vs. Foster and Chrostowski risks are likely to differ slightly. Also, this table uses the steady-state model, and the non-steady-state model is also available. The beryllium dermal CSF should be 430, with a McKone, steady-state cancer risk of 6E-5. The metals EPCs include results from M-15, which could not be verified. However, the McKone, steady-state cancer risk would not change. (Using the Foster and Chrostowski and non-steady-state assumptions, the risk would also approach 1.)

Table H-80: 1,1,1-TCA's dermal RfD should be 2E-2. The RfDs for 1,3-DCB and 1,4-DCB are now 3E-2. As per Attachment 1, the adjusted dermal RfDs for metals should be as follows: aluminum, 2.7E-1; cadmium, 2.5E-5; beryllium, 5E-5; chromium, 5E-5; copper, 2E-4; mercury, 1E-5; nickel, 2E-4; vanadium, 1E-4; zinc, 7.5E-2; cyanide, 2E-2. However, the total McKone, steady-state HI would not deviate significantly from 5.9E4. (Using the Foster and Chrostowski and non-steady-state assumptions, the HI would be 1E6.)

Table H-81: The metals EPCs include results from M-15, which could not be verified. However, the cancer risk of 0.5 would not change.

Table H-82: The metals EPCs include results from M-15, which could not be verified. 1,1,1-TCA's RfD is 2E-2. The RfDs for 1,3-DCB and 1,4-DCB are now 3E-2. Cyanide's RfD is 2E-2. However, the HI of 2.3E4 would not change.

Table H-83: Using the Foster and Chrostowski model and including all organic COPCs, this cancer risk would be 5E-4.

Table H-84: The provisional RfDs for 1,2-DCA and PCE are 1.4E-3 and 0.14, respectively. However, the total McKone HI would not be greatly affected. Using the Foster and Chrostowski model and including all organic COPCs, this HI would be 520.

Table H-85: Because the shower model used affects the concentrations available for dermal risk, the McKone and Bogen vs. Foster and Chrostowski risks are likely to differ slightly. Also, this table uses the steady-state model, and the non-steady-state model is also available. The beryllium dermal CSF should be 430, with a McKone, steady-state cancer risk of 8E-5. The metals EPCs include results from M-15, which could not be verified. However, the McKone, steady-state cancer risk would not change. (Using the Foster and Chrostowski and non-steady-state assumptions, the risk would also approach 1.)

Table H-86: 1,1,1-TCA's dermal RfD should be 2E-2. The RfDs for 1,3-DCB and 1,4-DCB are now 3E-2. As per Attachment 1, the adjusted dermal RfDs for metals should be as follows: aluminum, 2.7E-1; cadmium, 2.5E-5; beryllium, 5E-5; chromium, 5E-5; copper, 2E-4; mercury, 1E-5; nickel, 2E-4; vanadium, 1E-4; zinc, 7.5E-2; cyanide, 2E-2. However, the total McKone, steady-state HI would not deviate significantly from 8.2E4. (Using the Foster and Chrostowski and non-steady-state assumptions, the HI would be 1.5E6.)

Table H-87: The metals EPCs include results from M-15, which could not be verified. However, the cancer risk approaching 1 would not change.

Table H-88: The metals EPCs include results from M-15, which could not be verified. 1,1,1-TCA's RfD is 2E-2. The RfDs for 1,3-DCB and 1,4-DCB are now 3E-2. Cyanide's RfD is 2E-2. However, the HI of 6.5E4 would not change.

49. Table 5-1 was to be adjusted in accordance with comments. Some adjustments were made. The availability of alternate risk methodology (more than one shower model, more than one dermal model) and additional data sets (Westinghouse and PADEP) leads to multiple estimates for certain pathways. Also, Table 5-1 does not include scenarios for which EPA risk estimates are available. Therefore, Attachment 2 includes a comprehensive risk table that may be consulted for risk management decisions. Attachment 2 should be updated as the remaining outstanding issues are addressed (risk estimates that are highlighted in Attachment 2 represent EPA's estimates of what the risks will be when the risk assessment is updated in accordance with this review).

Below are comments that refer only to the scenarios presented on Table 5-1.

Railroad ROW: Maintenance worker inhalation HI should be 3E-4, due to chromium. Child trespasser dermal soil cancer risk should be 2E-5. Child trespasser dermal stormwater (steady-state) cancer risk should be 3E-6. Child trespasser

dermal stormwater (steady-state) HI should be 1E-4. Adolescent trespasser dermal stormwater (steady-state) cancer risk should be 2E-6. Adolescent trespasser dermal stormwater (steady-state) HI should be 5E-4. The child and adolescent HIs should not be added (HIs for different lifetime segments should not be summed).

Southern alluvial groundwater: Resident dermal (incorporating reported McKone & Bogen model) cancer risk should be 3E-4. Resident dermal (incorporating reported McKone & Bogen model) HI should be 26. Worker dermal (incorporating reported McKone & Bogen model) HI should be 19.

Bedrock groundwater: Resident dermal (incorporating reported McKone & Bogen model without semivolatiles) cancer risk should be 5E-4. Worker ingestion HI should be 2.6.

River sector: Child wader dermal sediment cancer risk should be 3E-6 to 4E-6. Child wader dermal sediment HI should be 0.5. The total child wader HI should therefore be 1.3, but the drivers, iron and chromium, are not additive so the HI does not truly exceed 1.

North/middle/south sectors: Employee vapor inhalation HI should be 1.7, in accordance with Appendix H. Indoor construction worker dermal soil cancer risk should be 6E-7. Indoor construction worker dermal soil HI should be 0.06. Indoor construction worker inhalation cancer risk should be 4E-7. Outdoor construction worker dermal soil cancer risk should be 8E-7. Outdoor construction worker ingestion soil HI should be 0.7. Outdoor construction worker dermal soil HI should be 0.2.

Moat: The child and adolescent HIs should not be added (HIs for different lifetime segments should not be summed). Utility worker ingestion soil HI should be 3E-4. Utility worker dermal soil HI should be 5E-5. Utility worker inhalation cancer risk and HI could not be verified.

50. Section 5 risks and hazards were to be adjusted in accordance with necessary adjustments to calculations and reported risks. The remaining adjustments to Sections 5.1 and 5.2 are still needed:

Page 5-2, last paragraph: Child wader cancer risk is 7E-6 to 8E-6.

Page 5-3, 2nd paragraph: Outdoor worker cancer risk is 1E-5. Utility worker air risk could not be verified. To the end of the paragraph, add, "For further EPA evaluation of the moat, see Section 5.3."

Page 5-3, 3rd paragraph: Trespasser stormwater risk should be 5E-6.

Page 5-4, 1st paragraph: Several VOCs of both the chlorinated ethene/ethane and BTEX families were found both in groundwater and in indoor air. Because of the concentration and toxicity of 1,2-DCA, this VOC happened to drive the indoor air risks. While the risk is uncertain due to a small number of samples, the discussion on page 5-4 does not adequately reflect the site conditions. There is no way of knowing at this point whether the air chemicals are or are not derived from the groundwater and, in fact, these preliminary data for groundwater and air seem more similar than dissimilar. See Attachment 3. (Therefore, page 6-2, 1st paragraph, last sentence, should also be deleted.)

Page 5-4, 2nd paragraph: There is not enough evidence at this time to support either conclusion 2) or 3) in the last sentence. These should be stricken or replaced with, "The source of the VOCs in the building air is currently unknown."

Page 5-5, 2nd full paragraph: Delete the discussion of the indoor employee. The HI does exceed 1 and this is discussed at the end of this page.

Page 5-5, 3rd full paragraph: To this paragraph, add, "The manganese may also be naturally occurring, since manganese is a common element in soils. This can be determined by comparing the on-site data to the background data." This background comparison should be performed, after which a stronger statement than the one used herein may be substituted.

Page 5-5, 4th full paragraph: Utility worker air risks could not be verified.

GENERAL GROUNDWATER AND HYDROGEOLOGY

55. The report should include a rationale for the use of unfiltered groundwater samples. The response did not address this comment. The point of the comment was not to challenge the taking of both filtered and unfiltered samples. The risk assessment should discuss (probably in Section 2.2.7 or 2.4.5) why the unfiltered metals data were considered to represent groundwater quality better than the filtered data.

61. Appendix B was not changed. Therefore, the following items are still outstanding: In dismissing the bedrock aquifer, the report fails to address the detections of PAHs, dioxins,

and furans in these wells.

Natural attenuation is briefly mentioned. However, if natural attenuation is being seriously proposed, considerable supporting information would have to be gathered (consult the hydrogeologist for guidance).

AIR MODELING

65, 67. No formal response to the 5/16/97 memo by Denis Lohman was discussed in the risk assessment response to comments. However, this comment and comment 67, which deal with the air modeling and Appendices E and F, have been reviewed by Patricia Flores, EPA air modeler. Consult her memo for an evaluation of the air modeling that supports the risk assessment.

However, the following items were still noted:

The LNAPL results on Table E-1b could not be found in the RI. Please supply the original data. Also, Table E-1b lists the concentrations as, e.g., 79170 mg/l, while Table E-3 lists the same concentrations as, e.g., 79170 ug/l. As previously noted, the concentration of TCDD on Table E-2a should be corrected.

UNCERTAINTY ANALYSIS

72. This report contains no central tendency, Monte Carlo, or other non-RME analysis, contrary to EPA expectations for multiple-descriptor risk assessments. The report also does not discuss whether sensitive subpopulations exist.

The comment response provided a rationale for this, which should be added to the uncertainty section of the risk assessment. Furthermore, the use of more than one set of data, more than one showering model, and more than one dermal model in the estimation and verification of risks does provide some multiple descriptors.

MISCELLANEOUS

77. One statement about EPA approval remains: page 3-1, where PADEP's name was accidentally crossed out instead of EPA's.

85. Table 4-4: The pentachlorinated dioxin should be 2,3,7,8-pentachlorodibenzo-p-dioxin. The point of this comment was that the "penta" TEF should be applied to 2,3,4,7,8-PeCDD as well as 1,2,3,7,8-PeCDD; in other words, any pentachlorinated dioxin with chlorines in the 2,3,7,8 positions. Only the 1,2,3,7,8- congener is indicated on the table.

86. The sentence in question is now page 5-7, 3rd paragraph, last sentence. Once again, this statement should be deleted. If this pathway were truly not a "practical" one for "developing sound risk management practices," then EPA would not have recommended its evaluation. EPA bases its risk assessment recommendations on information that will provide practical information for risk managers. Practical information includes the ability to evaluate the no-action alternative, select cleanup goals if necessary, evaluate a range of alternatives, and meet the requirements of the NCP.

The response claimed that the evaluation was "unwarranted due to the obvious outcome." Yet, what is "obvious" to environmental professionals may not be obvious to all stakeholders. At Superfund sites, there is an obligation to demonstrate the risks and calculations on which decisions may be based, not just to assume they are "obvious." That is the function of the Administrative Record.

With respect to the rest of that paragraph, it should be replaced with the following or similar wording: "The Agencies requested that risks to hypothetical residential receptors be quantified in this HHRA. As discussed above, this potential use is considered highly unlikely but could not be completely ruled out. Groundwater has been used locally for industrial purposes. Accompanying the assessment of the hypothetical resident is an assessment of potential exposure to workers who might drink and shower in the water. However, future use of groundwater, if any, would most probably be for production rather than human-use purposes. In the absence of existing restrictions to the contrary, potential receptors to groundwater were evaluated, though expectation of such usage in the foreseeable future is very low."

87. Page 5-8, 1st paragraph: Change "consistent with the practical future use of the property, not hypothetical extremes" to "reasonably anticipated current and future uses of the property. Due consideration must also be given to the no-action alternative, i.e., unrestricted use in the absence of remediation."

88. Page 5-14, 3rd paragraph: Delete the last sentence. The issue of whether additional cancer risk is or is not "compelling" is a matter of personal judgment. The numbers are presented, from which various stakeholders may make their own judgments about what they find personally "compelling." EPA does not object to the contextualization of risk (Westinghouse's rationale in the response), but this particular statement goes beyond risk contextualization.

89. Page 6-2, last paragraph: EPA's objection arises not because

the "FS will contain an evaluation of risk reduction associated with remedial options" or because the report identifies "the CERCLA process and subsequent steps of action at the Sharon site." Both of these are perfectly valid goals. However, the risk assessment wording is that "because risk is a function of exposure to an environmental toxicant, it follows that restricting or eliminating exposure is often the most prudent risk reduction measure . . ." (emph. added). This section concludes with the statement that the FS will include a "characterization of possible control measures than [sic] can be used to eliminate uncontrolled exposures to impacted media." Because risk is also a function of toxicity and environmental concentrations, not just a function of exposure, risk can also be reduced in ways different from or in addition to limiting exposure. The risk assessment should acknowledge this, rather than restricting future evaluation to exposure controls.

NEW COMMENTS ON ADDED/DELETED MATERIAL

- The appendices contain emissions calculations for soil excavation and truck hauling, but these emissions were apparently not used in the quantitative risk estimates.

- Page 3-11, 3rd paragraph: The report does not contain any reference for the 2 mg/m^3 emission rate.

- Page 3-14, 2nd paragraph: Report all sediment concentrations within the same paragraph in the same units (ppb or ppm) to facilitate comparison.

- Section 5.3: After first sentence, add, "As previously discussed, EPA examined potential residential soil risks." In the last sentence, change 1650 to 1670 (per changes due to some of Westinghouse's responses). To the end of the paragraph, add: "According to EPA, if surface water were quantitatively evaluated using the Westinghouse data, the only risks that might exceed the NCP risk range would be for swimming exposure to the outfall sample SWCLI, because of the reported PCBs (Aroclor 1248 at 2.8 ug/l, Aroclor 1260 at 4.5 ug/l). However, the likelihood of swimming at this location, and the current water quality at this location, are unknown. PCBs were not detected in water just downstream of this location." Also add, "EPA looked at the full set of Westinghouse data as well as the PADEP data for river sediment. Using these data, the child wader would have a cancer risk of approximately 9E-6 and an HI of approximately 0.01. This cancer risk is nearly the same as, and the HI is much lower than, those risks derived using PADEP data."

- Page 5-13, 4th paragraph: Delete the second and third sentences. The PADEP sediment data were not validated by national functional guidelines. These data only underwent lab QA/QC.

- Table C-11: The RBC for chloroethane should be 2.2; for freon 113 should be 3100; for 1,3,5-TMB should be 0.62; for 1,2,4-TMB should be 0.62; for 1,2-DCB should be 3.3; for PCB vapors should be 0.016. The only COPC selection that would change would be freon 113, which would no longer be a COPC.

Attachment 2 is provided as a convenient compilation of the risks that have been generated at various times and by various methods during the RI/risk assessment process. The attachment may be updated due to changes resulting from this review (numbers subject to change are highlighted). This attachment is recommended as a tool for risk managers during generation and review of the FS.

If you have any questions concerning these comments, please contact me at 215-566-3328.

3 Attachments

cc: Barbara Okorn Root (3HW41)
Eric Johnson (3HW41)

ATTACHMENT 1: SUBSTITUTE FOR TABLES 4-7 AND 4-8

CHEMICAL	DERMAL ABS FACTOR (soil)	ORAL-TO-DERMAL ADJUSTMENT FACTOR
ALUMINUM	0.01 a	0.27 b
ANTIMONY	0.01 a	0.1 c
ARSENIC	0.032 a	0.95 d
BARIUM	0.01 a	1 e
BERYLLIUM	0.01 a	0.01 c
CADMIUM	0.01 a	0.05 water RfD f 0.025 food RfD f
CHROMIUM	0.01 a	0.01 g
COPPER	0.01 a	0.6 d
IRON	0.01 a	1 h
LEAD	NA i	NA i
MANGANESE	0.01 a	1 h
MERCURY	0.01 a	0.15 e
NICKEL	0.01 a	0.1 d
SILVER	0.01 a	NA j
THALLIUM	0.01 a	1 b
VANADIUM	0.01 a	0.02 b
ZINC	0.01	0.25 d
BENZENE	0.0005 a	1 f
CHLOROBENZENE	0.03 a	1 h
1,2-DICHLOROETHANE	0.03 a	1 h
1,1-DICHLOROETHENE	0.0005 a	1 h
1,2-DICHLOROETHENE	0.0005 a	1 e
TETRACHLOROETHENE	0.03 a	1 e
TRICHLOROETHENE	0.03 a	1 h
VINYL CHLORIDE	0.0005 a	1 e
1,1,1-TRICHLOROETHANE	0.0005 a	1 h

CHEMICAL	DERMAL ABS FACTOR (soil)	ORAL-TO-DERMAL ADJUSTMENT FACTOR
ACENAPHTHYLENE	0.1 a	0.7 g
BENZ[A]ANTHRACENE	0.1 a	NA k
BENZO[A]PYRENE	0.1 a	NA k
BENZO[B]FLUORANTHENE	0.1 a	NA k
BENZO[G,H,I]PERYLENE	0.1 a	NA k
DIBENZ[A,H]ANTHRACENE	0.1 a	NA k
BIS(2-ETHYLHEXYL)PHTHALATE	0.1 a	0.55 l
1,2-DICHLOROBENZENE	0.1 a	1 h
1,3-DICHLOROBENZENE	0.1 a	1 h
1,4-DICHLOROBENZENE	0.1 a	1 h
INDENO[1,2,3-C,D]PYRENE	0.1 a	NA k
PHENANTHRENE	0.1 a	1 h
1,2,4-TRICHLOROBENZENE	0.1 a	1 h
2,4-DICHLOROPHENOL	0.1 a	1 h
CHLORDANE	0.1 a	0.8 m
AROCLOR 1242	0.06 a	1 f
AROCLOR 1248	0.06 a	1 f
AROCLOR 1254	0.06 a	1 f
AROCLOR 1260	0.06 a	1 f
DIELDRIN	0.1 a	1 h
ENDRIN ALDEHYDE	0.1 a	1 h
TCDD TEQs	0.03 a	1 h
HEPTACHLOR EPOXIDE	0.1 a	1 h
CYANIDE	1 h	NA j

*Used to derive dermal RfDs and CSFs for dermal exposure to soil and water, as per RAGS Appendix A.

a EPA, 1996a b ATSDR, 1992 c NCEA, 1994
d NCEA, 1992 e NCEA, 1993 f IRIS, 1997
g ATSDR, 1993
h Complete (100%) absorption is assumed as a default

i Not applicable since lead is evaluated using the IEUBK model
j NCEA, 1991
k These PAHs appear to act locally, so quantitative cancer risks based on the oral CSF are not recommended.
l ATSDR, 1991
m ATSDR, 1994

ATTACHMENT 2: SUMMARY OF RISK ESTIMATES

This summary brings together the reported risks (qualitative, quantitative, and semi-quantitative) that have been reported in various documents for the Westinghouse RI. Within this attachment, risks are red-lined if they do not appear as indicated here in the current draft of the risk assessment, but are anticipated to equal these values when corrected in accordance with this memo.

RIVER

WATER: In river water, using Westinghouse's target list, arsenic was the only compound that exceeded the Region III tap water RBC. All three sample stations were reported to have similar concentrations (4.2 to 4.4 ug/l), all of which were below the MCL of 50 ug/l. In addition, EPA reports that lead, chloroform, heptachlor, and dieldrin exceed the Region III tap water RBC in at least one surface water sample collected upstream of the Westinghouse facility, and that TCE, chloroform, heptachlor, heptachlor epoxide, and aldrin exceed the Region III tap water RBC in at least one surface water sample collected downstream of the Westinghouse facility. (Source: risk assessment; EPA review of risk assessment.)

According to EPA, if surface water were quantitatively evaluated using the Westinghouse data, the only risks that might exceed the NCP risk range would be for swimming exposure to the outfall sample SWCLI, because of the reported PCBs (Aroclor 1248 at 2.8 ug/l, Aroclor 1260 at 4.5 ug/l). However, the likelihood of swimming at this location, and the current water quality at this location, are unknown. PCBs were not detected in water just downstream of this location. (Source: EPA review of risk assessment.)

SEDIMENT:

CHILD WADER

CANCER RISK	CANCER RISK	HI	HI
PADEP/W'HOUSE (source: risk assessment)	W'HOUSE (source: EPA review of risk assessment)	PADEP/W'HOUSE (source: risk assessment)	W'HOUSE (source: EPA review of risk assessment)
7E-6 to 8E-6	9E-6	1.3 (but truly <1 because Fe and Cr not additive	0.01

FISH TISSUE: Given the available data, it was not possible to obtain fish tissue whose concentrations could be definitively linked to the Westinghouse site. (Source: risk assessment.) Because of the reported concentrations of PCBs in sediment (up to 0.24 ppm north of the site, up to 25 ppm downstream of the site), and the tendency of PCBs to bioconcentrate in fish tissue, there is a potential concern for fish consumption. Although the river is reportedly under a fish consumption advisory (source: risk assessment), compliance with the advisory is unknown. People have been observed fishing in the river downstream of the site, and fishing line has been found along the river banks. (Source: EPA site visit)

NORTH/MIDDLE/SOUTH SECTOR, INCLUDING BUILDING

RECEPTOR	CANCER RISK	HI
FUTURE EMPLOYEE (air inside bldg.)	2E-4	1.7
INDOOR CONSTRUCTION WORKER (contact with soil and soil emissions)	2E-6	8
OUTDOOR CONSTRUCTION WORKER (contact with soil and soil emissions)	1E-5	10

(Source: risk assessment)

For future employees, direct contact with dust inside building, the estimated cancer risks range from 2E-5 to 2E-2, depending upon the type of activity and area of the building visited. Lead in dust is also a potential concern. Approximately 25% of the building wipe samples exceeded a 1E-4 cancer risk level for RME exposure to potential workers. For the maintenance worker, almost half the wipe samples exceed a 1E-4 cancer risk level for RME exposure. There is also an open pit in the floor that could pose a physical hazard. (Source: EPA Middle Sector Building assessment.)

MOAT

RECEPTOR	CANCER RISK	HI
MAINTENANCE WORKER (source: risk assessment)	9E-6	0.2
CHILD TRESPASSER (source: risk assessment)	2E-6	0.2
ADOLESCENT TRESPASSER (source: risk assessment)	1E-6	0.08
UTILITY WORKER (source: risk assessment)	1E-6*	3E-4*

RECEPTOR	CANCER RISK	HI
WORKER: UNRESTRICTED ACCESS (source: EPA review of risk assessment)	2E-4	3.5
CONSTRUCTION WORKER (source: EPA review of risk assessment)	2E-5	0.5

*currently unable to verify inhalation risks; see comments on air modeling

RAILROAD ROW

SOIL

RECEPTOR	CANCER RISK	HI
MAINTENANCE WORKER	1E-5	0.4
CHILD TRESPASSER	1E-4	12
ADOLESCENT TRESPASSER	5E-5	5

(source: risk assessment)

POOLED STORM WATER

RECEPTOR	CANCER RISK, STEADY-STATE (source: risk assessment)	CANCER RISK, NON-STEADY-STATE (source: EPA review of risk assessment)	HI, STEADY-STATE (source: risk assessment)	HI, NON-STEADY-STATE (source: EPA review of risk assessment)
CHILD TRESPASSER	3E-6	2E-5	1E-4	5E-4
ADOLESCENT TRESPASSER	2E-6	2E-5	1E-4	6E-4

SOUTH ALLUVIAL GROUNDWATER

RECEPTOR	CANCER RISK, MCKONE, STEADY-STATE (source: risk assessment)	CANCER RISK, FOSTER, NON-STEADY-STATE (source: EPA review of risk assessment)	HI, MCKONE, STEADY-STATE (source: risk assessment)	HI, FOSTER, NON-STEADY-STATE (source: EPA review of risk assessment)
WORKER	3E-3	7E-3	60	360
ADULT RESIDENT	8E-3	2E-2	140	560
CHILD RESIDENT	--	9E-3	--	1670

CENTRAL ALLUVIAL GROUNDWATER

RECEPTOR	CANCER RISK, MCKONE, STEADY-STATE (source: risk assessment)	CANCER RISK, FOSTER, NON-STEADY-STATE (source: EPA review of risk assessment)	HI, MCKONE, STEADY-STATE (source: risk assessment)	HI, FOSTER, NON-STEADY-STATE (source: EPA review of risk assessment)
WORKER	appr. 1	appr. 1	8.2E4	1E6
ADULT RESIDENT	appr. 1	appr. 1	1.5E5	1.6E6
CHILD RESIDENT	--	appr. 1	--	5E6

BEDROCK GROUNDWATER

RECEPTOR	CANCER RISK, MCKONE, STEADY-STATE (source: risk assessment)	CANCER RISK, FOSTER, NON-STEADY-STATE (source: EPA review of risk assessment)	HI, MCKONE, STEADY-STATE (source: risk assessment)	HI, FOSTER, NON-STEADY-STATE (source: EPA review of risk assessment)
WORKER	3E-4	2E-3	7	78
ADULT RESIDENT	9E-4	3E-3	13	117

RECEPTOR	CANCER RISK, MCKONE, STEADY-STATE (source: risk assessment)	CANCER RISK, FOSTER, NON-STEADY-STATE (source: EPA review of risk assessment)	HI, MCKONE, STEADY-STATE (source: risk assessment)	HI, FOSTER, NON-STEADY-STATE (source: EPA review of risk assessment)
CHILD RESIDENT	--	2E-3	--	350

RESIDENTIAL SOIL

Most residential yards are associated with Hazard Indices and cancer risks at or below the target risks specified in the NCP. The exceptions were yards S12, S2, S14, S15-16, and S20. The risk drivers for these five yards were arsenic, beryllium, and PAHs. While these chemicals may be naturally occurring, or related to anthropogenic sources other than the site, this could not be confirmed with the available background data set. PAHs are associated with the combustion of organic material and are frequently found in the environment; they did not exhibit a consistent pattern in the residential neighborhood. Three of these five samples, S14, S20, and S02, were reportedly taken near garages.

The pattern of contamination found at the Westinghouse site was not observed in residential soils, particularly the high levels of PCBs. Low levels of PCBs and dioxins/furans were found in neighborhood soils, but these were far lower than on-site levels and did not contribute significantly to estimated residential risks.

Lead results in some yards may be of concern for children and pregnant women. No consistent pattern of lead contamination was observed. Overall risks due to lead are uncertain because only 1-2 samples were obtained from each yard and the amount of total lead exposure is unknown.

(Source: EPA review of risk assessment)

ATTACHMENT 3: COMPARISON OF VOCs IN MULTIPLE MEDIA

CHEMICAL	MIDDLE SECTOR SUBSOIL	ALLUVIAL SOUTH	ALLUVIAL CENTRAL	BUILDING AIR
PCE	X		X	X
TCE	X	X	X	X
12DCE		X	X	
11DCE		X	X	X
VINYL CHLORIDE		X		
111TCA	X		X	
12DCA			X	X
CHLOROETHANE		X		
BENZENE	X		X	X
TOLUENE	X			X
ETHYLBENZENE	X			X
XYLENES	X			X
2-BUTANONE	X			
2-HEXANONE	X			
CARBON DISULFIDE	X			
CHLOROBENZENE	X	X	X	
ACETONE			X	
DICHLORODIFLUOROMETHANE				X
TRICHLOROFLUOROMETHANE				X
FREON 113				X

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION III
841 Chestnut Building
Philadelphia, Pennsylvania 19107

SUBJECT: Review of the Air Modeling Portions of the Human Health Risk Assessment for the Former Westinghouse Transformer Plant Superfund Site

FEB 0 3 1998

FROM: Patricia I. Flores-Brown, Air/Superfund Coordinator
Technical Assessment Section (3AP22)

TO: Vic Janosik, Remedial Project Manager
Western PA Remedial Section (3HS22)

In response to a recent request from Jennifer Hubbard, Toxicologist, I have reviewed the air emissions estimation and modeling analysis submitted for the Former Westinghouse Transformer Plant. I reviewed the document for adequacy of the surmised exposure scenarios and for mathematical integrity. The air modeling portions were found in Chapters 2 and 3 as well as Appendixes E and F.

The document was very difficult to review if not impossible to decipher for a few reasons. First of all, the document lacked clarity in many places. The units on many of the inputs to the air modeling equations were not consistent; they frequently changed from page to page. Because of the lack of care in keeping track of the units, simple, unnecessary arithmetical errors were pervasive. Finally, most of the comments made by Mr. Dennis Lohman, Meteorologist, in a May 16, 1997 memorandum to you were not followed.

After deciphering most of what was contained in the document, I calculated ambient air concentrations for the 'air' scenarios in the document requested by Ms. Hubbard. My calculations are limited in their accuracy by the accuracy of the input data supplied by ChemRisk in the document.

The attached document contains my review and calculations. If you have any questions or concerns about its contents, please call me at x2193.

cc: J. Hubbard (3HS41)
M. Morris (3AP22)

This was telefaxed to Chuck Tordella of PADEP on 2-9-98. Vic Janosik

Review of the Air Modeling Portions of the Human Health Risk Assessment for the Westinghouse Superfund Site

The air modeling portions of the human health risk assessment were reviewed for adequacy of surmised exposure scenarios and for mathematical integrity. The air modeling portions were found in Chapters 2 and 3 as well as Appendixes E and H.

Ambient air concentrations for the outdoor scenarios were calculated by EPA using the EPA screening air dispersion model TSCREEN instead of a box model preferred by ChemRisk. Guidance for using the TSCREEN model to calculate ambient air concentrations for future land-use scenarios for area sources can be found starting on page 56 of the *Air/Superfund National Technical Guidance Study Series- Guideline for Predictive Baseline Emissions Estimation for Superfund Sites* - EPA-451/R-96-001.

1. Estimated Indoor Construction Worker Air Exposure Concentrations via Flux from LNAPL

a) The equations used in this analysis were similar to the equations found in *Air/Superfund National Technical Guidance Study Series - Assessing Potential Indoor Air Impacts for Superfund Sites* - EPA-451/R-92-002.

b) The inputs into this model are correct. However, in Table E-1b, equation (h) used to calculate C_e is incorrect. The equation listed is $C_e = X_i \times \rho_i \times MW \times 1/RT$. The correct equation should be $C_e = X_i \times Pv \times MW \times 1/RT$. The equations used to calculate C_e should be evaluated.

c) Using the equations and the values for the input parameters supplied by ChemRisk, the correct calculated indoor air concentrations should be the following:

Aroclor-1248 = 7.62E-8 mg/m^3
Aroclor-1254 = 8.45E-9 mg/m^3
Aroclor-1260 = 6.04E-9 mg/m^3

The discrepancies between these values and the values listed in table E-3 stem from the following:

D/Lt has units of cm^2/m-sec.
C_e has units of mg/m^3
$F = (D/Lt) \times C_e$ which has units of mg-cm^2/m^3-m-sec. If these units are multiplied by (m/100 cm), the units become (1E-2) mg-cm/m^3-sec.

For Aroclor-1248 the reported value of 1.64E-04 in Table E-1a should have units of mg-cm^2/m^3-m-sec. The reported value was never multiplied by (m/100 cm), thus the value for Aroclor-1248 is too high by two orders of magnitude.

Additionally, when the calculation of the concentration of vapor-phase chemical in indoor air was performed (Table E-3), the calculation is in error of a factor of approximately 0.168. There is no clue as to where the source of this error originates, but it is consistent for all three listed PCBs.

2. Estimated Outdoor Air Exposure Concentrations via Flux from Groundwater

a) Equations used in this scenario to calculate the flux from groundwater were the same for the indoor air calculations. The flux rates for the compounds found in Table E-4 are incorrect due to the following:

D/Lt has units of cm^2/m-sec.
C_w has units of μg/l
$F = (D/Lt) \times Cw \times H$ which has units of μg-cm^3/m-sec-l. If these units are multiplied by (m/100 cm), the units become (1E-2) μg-cm/sec-l.

For vinyl chloride the reported value of 5.74E-6 μg-cm/l-sec in Table E-2b and Table E-4 should be 5.74E-8 μg-cm/l-s or 5.74E-10 mg/m^3-s. The reported values for all of the compounds were never multiplied by (m/100 cm), thus the values are all too high by two orders of magnitude.

b) Ambient air concentrations calculated by ChemRisk were done by using a simple box model. EPA prefers that EPA's screening model TSCREEN be used to estimate hourly concentrations rather than the box model. The hourly ambient air concentrations listed below were derived using TSCREEN rather than the box model. The annual average ambient air concentrations were derived by multiplying the hourly concentrations by an adjustment factor of 0.08. The emitting area was assumed to be the same at 17,400 m2.

c) Using the equations and the values for the input parameters supplied by ChemRisk, the calculated the ambient air concentrations should be the following:

Contaminant		Hourly Conc.	Annual Conc.
vinyl chloride	=	5.945E-8 mg/m^3	4.756E-9 mg/m^3
1,1-dichloroethene	=	6.974E-9 mg/m^3	5.579E-10 mg/m^3
1,2-dichloroethene (total)	=	3.161E-8 mg/m^3	2.529E-9 mg/m^3
1,2-dichloroethane	=	2.075E-8 mg/m^3	1.660E-9 mg/m^3
1,1,1-trichloroethane	=	2.189E-7 mg/m^3	1.751E-8 mg/m^3
trichloroethene	=	1.195E-5 mg/m^3	9.560E-7 mg/m^3

benzene	=	1.063E-6 mg/m³	8.504E-8 mg/m³
tetrachloroethene	=	7.774E-8 mg/m³	6.219E-9 mg/m³
chlorobenzene	=	1.000E-6 mg/m³	8.000E-8 mg/m³
1,3-dichlorobenzene	=	1.366E-6 mg/m³	1.093E-7 mg/m³
1,4-dichlorobenzene	=	9.374E-7 mg/m³	7.499E-8 mg/m³
1,2-dichlorobenzene	=	2.395E-6 mg/m³	1.916E-7 mg/m³
1,2,4-trichlorobenzene	=	8.803E-5 mg/m³	7.042E-6 mg/m³
Aroclor-1242	=	8.917E-9 mg/m³	7.134E-10 mg/m³
Aroclor-1248	=	4.910E-5 mg/m³	3.928E-6 mg/m³
Aroclor-1254	=	7.545E-5 mg/m³	6.036E-6 mg/m³
Aroclor-1260	=	8.002E-9 mg/m³	6.402E-10 mg/m³
total 2378-TCDD Equivalent	=	2.274E-15 mg/m³	1.819E-16 mg/m³

(based on initial groundwater concentration of TCDD of 8E-6 ug/l)

3. Estimated Indoor Air Exposure concentrations via Flux from Subsurface Soil

a) The indoor air concentrations found in Table F-9 (Cv, mg/m³) are valid for all compounds except Aroclor-1242. The flux rate calculated for Aroclor-1242 is too low, most likely by no more than a factor of two. Table F-6, which is a print-out for the result of Aroclor-1242 using the BAM model, shows that the input value of Cto, the mass of solute per soil volume in mg/cm³, to be 0.227000E-02 mg/cm³. If the soil concentration of Aroclor-1242 listed in Table F-9 is correct, the value of Cto in Table F-6 should be:

(2.41 mg/kg-soil) x (1 kg-soil/1000 g) x (1.68 g/cm³ [soil density]) = 4.05E-3 mg/cm³.

b) The units on Js in Table F-9 should be mg/sec-cm² not mg/sec-cm²/mg/kg since the initial soil concentration was used to calculate Cto in the BAM model. Js should also be referred to as F.

c) The units reported for F on Table F-9 should be mg/cm²-sec not mg/m²-sec.

4. Estimated Outdoor Air Exposure Concentrations via Flux from Subsurface Soil

a) The BAM model was also used in this scenario to calculate the flux rate of the contaminants from subsurface soil, and the flux rates on Table F-10 are the same as for the indoor air scenario on Table F-9. The calculated flux rates for Aroclor-1242, again, are probably no more than a factor of two too low. Ambient air concentrations calculated by ChemRisk were done by using a box model. EPA prefers that EPA's screening model TSCREEN be used to estimate maximum hourly concentrations rather than a box model. The hourly ambient air concentrations below were derived using TSCREEN rather than the box model. The emitting area was assumed to be the same at 17,400 m2.

Contaminant		Hourly Conc.	Annual Conc.
Acenaphthylene	=	2.336E-5 mg/m³	1.869E-6 mg/m³
Benzo(g,h,i)perylene	=	3.545E-9 mg/m³	2.836E-10 mg/m³
Phenanthrene	=	1.395E-4 mg/m³	1.116E-5 mg/m³
Aroclor-1242	=	1.819E-4 mg/m³	1.455E-5 mg/m³
(this value is less than a factor of two low)			
Aroclor-1254	=	4.197E-4 mg/m³	3.358E-5 mg/m³
Aroclor-1260	=	2.078E-4 mg/m³	1.662E-5 mg/m³

b) The units on F in Table F-10 should be mg/sec-cm² not mg/sec-cm²/mg/kg since the initial soil concentration was used to calculate Cto in the BAM model.

c) The calculation of Cvs performed using the box model was multiplied by the soil concentration. This is incorrect due to the fact that the initial soil concentration was used to calculate Cto in the BAM model.

5. PM_{10} Emissions from Soil Excavation

The 5/16/97 comments by Denis M. Lohman, Meteorologist were not taken into account and the analysis was not revised. If the "top down approach" that ChemRisk used is assumed to be valid, the following comments made by Mr. Lohman still apply:

a) The volume of soil was calculated incorrectly. The soil volume should be 14,035 m³ not 11,325 m³ as reported by ChemRisk.

b) ChemRisk used an assumed wind speed of 2 m/s in place of the EPA recommended default of 4.4 m/s in the equation.

c) The calculated emission rate should be 5.434E-4 g/s instead of the 1.57E-4 g/s specified by ChemRisk in Table 3-15.

d) The EPA air dispersion model TSCREEN should be used instead of a box model.

e) Using the above corrections, PM_{10} concentrations from soil excavation of the slab were calculated in units of $\mu g/m^3$.

Contaminant		Hourly Conc.	Annual Conc.
PM_{10}	=	215.7 $\mu g/m^3$	17.26 $\mu g/m^3$

6. **PM10 Emissions from Truck Hauling Traffic**

Again, the 5/16/97 comments by Denis M. Lohman, Meteorologist were not taken into account.

a) ChemRisk used a silt surface loading value of 8 g/m^3 instead of the EPA default value of 5g/m^3 found in *Air/Superfund National Technical Guidance Study Series - Estimation of Air Impacts from Area Sources of Particulate Matter at Superfund Sites* - EPA-451/R-93-004, the guidance that ChemRisk claims to have used.

b) EPA calculates the PM_{10} emissions per vehicle kilometer traveled to be 1.692E2 g/VKT. Assuming that each truck travels 0.6 km round-trip, 101.5 g of PM_{10} are emitted for each round-trip. Assuming that 8 trucks a day are used, the PM_{10} emission rate is 2.819E-2 g/s.

c) The method that ChemRisk used to calculate an ambient air concentration for PM_{10} due to truck traffic is illogical as alluded to by Mr. Lohman. There is no apparent reason why the concentration behind the truck should be inversely proportional to the width or the height of the truck. The best way to calculate an ambient air concentration is outlined in the guidance document mentioned in (a). The traffic scenario in the guidance document assumes back and forth traffic in a confined area on-site. The emissions should be modeled as an area source where the truck trafficked area is assumed to be 2500 m^2.

Using TSCREEN:

Contaminant		Hourly Conc.	Annual Conc.
PM_{10}	=	811.2 μg/m^3	64.9 μg/m^3

d) The ambient annual air concentrations for Outdoor Construction Due to PM_{10} is thus 17.3 μg/m^3 (excavation) + 64.9 μg/m^3 (truck traffic) = 82.2 μg/m^3. This value should be listed in table 3-15.

7. **PM_{10} Emissions from the Construction Utility Worker - Trench Excavation**

a) The correct equation for this scenario was used. However, an average windspeed of 2 m/s was used instead of the EPA default value of 4.4 m/s. PM_{10} emissions from excavation, therefore, should be 52.15 g/day instead of the reported 18.7 g/day. The average PM_{10} emissions should be 2.414E-3 g/s instead of 8.66E-4 g/s.

b) Again, the TSCREEN model should be used instead of a box model. Trench excavation is supposed to occur for six hours out of an eight hour day and last only two days out of the year.

Using TSCREEN:

Contaminant		Hourly Conc.	Annual Conc.
PM_{10}	=	1703 $\mu g/m^3$	136.2 $\mu g/m^3$

8. **PM_{10} Emissions from the Construction Utility Worker - Active Soil Pile Near Trench**

a) The correct equation and the correct inputs to the equation were used. However, ChemRisk made a computational error and the emission rate they obtained was two orders of magnitude too low. ChemRisk calculated an emission rate of 1.44E-2 g/m^2-day, while the true emission rate should be 1.42 g/m^2-day.

b) ChemRisk's scenario assumed that the pile would be active for 8 hours during the workday. At the end of the workday, the soil pile is returned to the trench. However, in the equation that ChemRisk used to estimate particulate emissions, the pile was assumed to be active for 24 hours. An activity level for the pile of 8 hours is much more reasonable. EPA therefore calculates the emission rate to be 5.331E-3 g/s.

c) Again, EPA prefers use of the TSCREEN model. The TSCREEN model has a module to be used explicitly for active soil piles under the menu choice 'Particulate Matter Release Type - Fugitive Windblown Dust Emissions.' A soil pile diameter of 20 m was used in the model.

Using TSCREEN:

Contaminant		Hourly Conc.	Annual Conc.
PM_{10}	=	0.1903 $\mu g/m^3$	1.522E-2 $\mu g/m^3$

d) The ambient annual air concentrations for the Moat Maintenance/Utility Worker Trench Excavation due to PM_{10} is thus
1703 $\mu g/m^3$ (excavation) + 0.1903 $\mu g/m^3$ (soil pile) = 1703.2 $\mu g/m^3$. This value should be listed in Table 3-19.

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION III
841 Chestnut Building
Philadelphia, Pennsylvania 19107

March 11, 1998

SUBJECT: Review of the Response to Comments on the Air Modeling Portions of the Human Health Risk Assessment for the Former Westinghouse Transformer Plant Superfund Site

FROM: Patricia I. Flores-Brown, Air/Superfund Coordinator
Technical Assessment Section (3AP22)

TO: Vic Janosik, Remedial Project Manager
Western PA Remedial Section (3HS22)

In response to your recent request I have reviewed ChemRisk's response to EPA's comments on the air emissions estimation and modeling analysis submitted for the Former Westinghouse Transformer Plant. Below is my reply.

Comment #1

ChemRisk agreed that there was an error in the footnoted equation on Table E-1b. However, they did use the correct term (Pv) to calculate the value of Co. Therefore the values listed in Table E-1b were correct.

Upon reevaluation of the analysis and additional information submitted by ChemRisk, EPA agrees with ChemRisk that the term D/Lt has units of cm/sec. Therefore the emission rates calculated by ChemRisk are not in error by a factor of 1E-2. However, ChemRisk did admit that they used an incorrect ceiling height in the calculations. In response, ChemRisk resubmitted Table E-3 to EPA with the corrections. EPA considers the corrections to be acceptable. Therefore the correct calculated indoor air concentrations are the following:

Aroclor-1248 = 7.62E-6 mg/m^3
Aroclor-1254 = 8.45E-7 mg/m^3
Aroclor-1260 = 6.04E-7 mg/m^3

Comment #2

As stated in Comment #1, EPA agrees with ChemRisk that the term D/Lt has units of cm/sec. Therefore, EPA agrees that the emission rates calculated by ChemRisk and presented in Table E-4 are correct. However, EPA still prefers the use of the screening model TSCREEN to estimate hourly concentrations rather than the use of a box model. ChemRisk did not acknowledge EPA's

model preference and made no changes to Table E-4. Correcting the error made by assuming the wrong units and by using the EPA model TSCREEN, the calculated ambient air concentrations are listed below. These concentrations should be the values listed in Table E-4.

Contaminant		Hourly Conc.	Annual Conc.
vinyl chloride	=	5.945E-6 mg/m^3	4.756E-7 mg/m^3
1,1-dichloroethene	=	6.974E-7 mg/m^3	5.579E-8 mg/m^3
1,2-dichloroethene (total)	=	3.161E-6 mg/m^3	2.529E-7 mg/m^3
1,2-dichloroethane	=	2.075E-6 mg/m^3	1.660E-7 mg/m^3
1,1,1-trichloroethane	=	2.189E-5 mg/m^3	1.751E-6 mg/m^3
trichloroethene	=	1.195E-1 mg/m^3	9.560E-5 mg/m^3
benzene	=	1.063E-3 mg/m^3	8.504E-6 mg/m^3
tetrachloroethene	=	7.774E-6 mg/m^3	6.219E-7 mg/m^3
chlorobenzene	=	1.000E-4 mg/m^3	8.000E-6 mg/m^3
1,3-dichlorobenzene	=	1.366E-4 mg/m^3	1.093E-5 mg/m^3
1,4-dichlorobenzene	=	9.374E-5 mg/m^3	7.499E-6 mg/m^3
1,2-dichlorobenzene	=	2.395E-4 mg/m^3	1.916E-5 mg/m^3
1,2,4-trichlorobenzene	=	8.803E-3 mg/m^3	7.042E-4 mg/m^3
Aroclor-1242	=	8.917E-7 mg/m^3	7.134E-8 mg/m^3
Aroclor-1248	=	4.910E-3 mg/m^3	3.928E-4 mg/m^3
Aroclor-1254	=	7.545E-3 mg/m^3	6.036E-4 mg/m^3
Aroclor-1260	=	8.002E-7 mg/m^3	6.402E-8 mg/m^3
total 2378-TCDD Equivalent	=	2.274E-13 mg/m^3	1.819E-14 mg/m^3

(based on initial groundwater concentration of TCDD of 8E-6 ug/l)

Comments # 3-7

ChemRisk did not respond to Comments #3-7 and therefore accepts the comments. The tables that correspond to Comments #3-7 should be revised.

Comment #8

ChemRisk states that EPA's guidance document *Estimation of Air Impacts from Area Sources of Particulate Matter Emissions at Superfund Sites*, EPA-451/R-93-004, contains an error. Specifically, ChemRisk states that the units on equation 3-19 should be in kg/day-hectare or "(0.01) g/day-m^2". ChemRisk is correct; the guidance document does contains an error. However, ChemRisk claims that a factor of 0.01 (1000g/kg x hectare/10,000 m^2) is required to convert between the applied units. The correct factor to apply to equation 3-19 is a factor of 0.1 (1000g/kg x hectare/10,000 m^2). ChemRisk calculated an emission rate of 1.44E-2 g/m^2-day. The true emission rate should be 0.142 g/m^2-day.

The other comments made by EPA still apply. The correct concentrations for PM_{10} for this scenario are:
Hourly concentration = 0.02 ug/m^3
Annual concentration = 0.0016 ug/m^3

Table 3-19 should be revised.

If you have any questions or concerns, please contact me at x-2193.

cc: J. Hubbard (3HS41)
M. Morris (3AP22)

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION III
841 Chestnut Building
Philadelphia, Pennsylvania 19107

SUBJECT: Review of Health Risk Assessment **DATE:** 5/16/97
Westinghouse - Sharon

FROM: Denis M. Lohman, Meteorologist
Technical Assessment Section (3AT22)

TO: Vic Janosik, RMP
Western PA Remedial Section (3HW22)

Received 5-16-9
by V. Janosik, RI

As requested per the suggestion of Jennifer Hubbard, I have conducted a limited review of the Baseline Human Health Risk Assessment for the Former Westinghouse Transformer Plant in Sharon, Pennsylvania. My review is primarily limited to the future construction worker scenarios in the North, Middle and South Sectors.

Since I am not familiar with the Farmer emission flux model I did not make an in-depth evaluation of the indoor employee and indoor construction worker scenarios. I did determine that the procedure used to estimate potential exposures for those scenarios was logical. I would, however, prefer to characterize the exposures calculated as average rather than worst case since they assume a homogeneous distribution within the building.

The following comments address the Outdoor Construction Worker scenarios of section 3.3.2.3:

PM-10 Emissions from Excavation

The PM-10 emissions from soil excavation were estimated using the an equation found in the EPA's *Air/Superfund National Technical Guidance Study Series - Models for Estimating Air Emissions from Superfund Remedial Actions* (EPA, 1993b). The equation estimates the grams of particulate matter (PM-10) emitted from transfer operations as a function of the mass of soil handled, the wind speed and the percent soil moisture. The technique is not exact and ChemRisk chose to take, what I would call, a "top down approach" in which the total mass of soil to be excavated was calculated and an average emission rate was calculated. My preference would be to characterize an excavation "event" and estimate potential emissions from a "bottom up" or short term perspective. Neither approach is intrinsically right or wrong but the top down approach should not be characterized as a worst case.

Moreover, the calculation should be done correctly. I multiplied the 0.76 m by

243 m by 76 m and obtained a volume of soil of 14,035 m3. The value of 11,325 m3 reported by ChemRisk is wrong as is the subsequently calculated emission rate. In addition ChemRisk, without providing an explanation, used an assumed wind speed of 2 m/s in place of the EPA recommended default of 4.4 m/s. If the default wind speed is used with the correct soil volume, the emission rate calculated is 5.43x10-4 g/s instead of the 1.57x10-4 g/s specified by ChemRisk.

The mass balance box model used by ChemRisk is a simplified way of making order of magnitude concentration estimates. Because the model assumes uniform distribution within the "box" it cannot be used to determine a "worst case air concentration" as stated on page 3-31. Using the height of the worker and the lateral distance traveled by the worker is a convenient way to make the equation dimensionally correct but these are not logical inputs. The validity of the estimates depends upon some very subjective assumptions. If the worker were to sit down and, effectively, halve his/her height the concentration would double! Similarly, the lateral extent of the "box" should more properly be determined by the particular transfer operation rather than the mobility of the worker. And finally, the wind speed assumption of 2 m/s is used without explanation.

Since the emissions of PM-10 can be calculated and the characteristics of a transfer operation can be estimated it would be preferable to use EPA's screening model TSCREEN to estimate maximum hourly concentrations of PM-10.

PM-10 Emissions From Hauler Truck Traffic

The correct equation for estimating emissions from traffic on paved roads was used. However, the EPA default value for silt surface loading is 5 g/m3. ChemRisk used a silt surface loading value of 8 g/m3. This apparent mistake leads to an overestimate of emissions.

ChemRisk more than makes up for the emissions overestimate by incorrectly calculating the concentration. Instead of using the mass of vehicular kilometer traveled (g/VKT) the mass per 0.3 kilometers is used. The arithmetically correct estimate of concentration should be 62,640 µg/m3 instead of the 32,467 µg/m3 reported.

The equation used however is dimensionally correct but logically curious. There is no apparent reason why the concentration behind the truck should be inversely proportional to the width or the height of the truck. Also, if the one way haul distance should be 3 meters instead of 3 hundred meters the concentration would increase by two orders of magnitude!

Groundwater To Outdoor Air Vapor Transport

ChemRisk again uses a box model to calculate air concentrations of COPC. In this application the dimensions of the box are more logical but continue to be subjective. For comparison purposes I used ChemRisk's BAM estimates of methylene chloride emission rates in the TSCREEN model. The maximum concentration was estimated to be 5.713x10-3 µg/m3 of methylene chloride compared to the box model estimate of 3.880x10-3 µg/m3. Equivalent differences would be obtained for the other COPC.

Conclusion

As a result of a combination of carelessness, unnecessary simplification, and unwarranted assumptions ChemRisk has, in my opinion, underestimated the maximum risk due to air exposure by a factor of two or three. The exposure estimates derived by ChemRisk are more appropriately characteristic of average exposures according to the respective scenarios.

If you have additional questions or require additional analyses please contact me at (215) 566-2192.

cc: M. Morris (3AT22)
J. Hubbard (3HW41)
P. Flores-Brown (3AT22)

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION III
841 Chestnut Building
Philadelphia, Pennsylvania 19107

SUBJECT: Review of Westinghouse BLRA Revisions

FROM: Jennifer Hubbard, Toxicologist
Technical Support Section (3HS41) JR Hubbard

TO: Vic Janosik, RPM
Western PA Remedial Section (3HS22)

DATE: 3/26/98

Baseline Risk Assessment (Human Health) comments received 3/26/98 by V. Janosik, RPM

I have reviewed the CBS/Westinghouse comment response and BLRA revisions submitted 3/20/98. This memo contains a discussion of the remaining issues at the Westinghouse Sharon site. The memo is divided into three major sections:

a discussion of manganese hazard in soil;
a list of recommendations for the revised BLRA report; and
a record of BLRA pages and tables that were not explicitly changed, but for which Appendix I should be consulted whenever the BLRA is used as a source.

Comment numbers refer to the EPA toxicologist's previous comments on the baseline risk assessment, as discussed in the 2/3/98 memo to Vic Janosik. EPA Air/Superfund contact Patricia Flores-Brown was also consulted for issues related to the air modeling.

MANGANESE IN SOIL

During the review of the draft BLRA, it was uncertain whether manganese would be associated with an unacceptable HI (>1) for the indoor and outdoor construction workers. According to current EPA estimates, the total outdoor construction worker HI would be 1.3, or approximately 1. However, the indoor construction worker, by both Westinghouse and EPA estimates, would have an HI of approximately 8, due to inhalation of manganese. The manganese HI is driven primarily by the assumption that workers could inhale up to 5 mg/m^3 of particulates (the OSHA standard).

Before determining a course of action for manganese, project managers may wish to consider the following questions:

How well does the OSHA standard match up with expected exposure at the site?
How do the soil manganese levels compare to local background concentrations?
Are the manganese concentrations uniform, or is the HI driven by a "hot spot?"

Telefaxed to Chuck Tordella of PADEP 3/26/98

The answers to these questions may be useful in developing the FS, since they involve the issues of future use of the site, pattern of contamination, site attribution, and whether health-and-safety or institutional controls might be appropriate.

REMAINING RECOMMENDATIONS, BASED ON TOXICOLOGIST'S COMMENTS

PADEP and EPA have agreed that even where CBS acknowledges that numbers should have been changed or updated, these changes would not necessarily be made to the body of the report. Certain sections, considered to be the most significantly affected by the changes, were revised and resubmitted.

The remaining changes will not be made for two reasons: 1) the total risks, on which the remedy is based, would not change significantly; and 2) the correct information will be available in Appendix I of the report. While this will enable the project to move forward into the FS stage, it may make consulting specific tables in the BLRA a difficult task where minor errors persist. Therefore, to ensure that a consistent set of numbers is used to characterize the site in the FS, ROD, and other communications, it is recommended that the EPA estimates be used wherever CBS has acknowledged that EPA's corrections were appropriate.

The next section of this memo lists outstanding issues for which revisions are still recommended. These issues are either the most important for risk assessment and characterization, and/or they involve sections of the report that are already being revised for other reasons. (These comments are numbered beginning with an R, for revision, to distinguish them from earlier comment numbers.)

R-1 Section 2.4.5 was revised, but the revision requested by comment 6 was not made; the reference to MCLs could be deleted.

R-2 Page 3-6 was revised, but the 1st paragraph should also have been modified in accordance with comment 12, to reflect the appropriate rejection of R data.

R-3 Page 5-7, 3rd paragraph, even though resubmitted, still contained the last sentence, which was recommended by EPA for deletion (comment 86).

R-4 On Tables 3-9 and D-9, a transcription error and the use of R data affected the EPCs for cadmium and lead, which should change significantly, according to comments 11h and 12.

R-5 The EPCs on Tables 3-10 and D-10 for manganese and Aroclor 1260 should be changed in accordance with comments 11i and 12.

R-6 Tables 4-7 and 4-8 were not changed. Because of the confusion with dermal factors, as described in comment 38, it may be preferable to simply remove these tables from the report and refer the reader to Appendix I.

R-7 Table C-9 was revised, but the RBCs were not corrected and updated as recommended in comment 7g.

R-8 Table C-10 should be reprinted because the current formatting makes the dioxin results

illegible (comment 3i). In preparing this table for resubmission, the other changes can be made: the cyanide non-detect can be reported (comment 3i) and the RBCs can be corrected or updated (comment 7h).

R-9 Table H-72 was partially corrected. The EPCs and RfDs could be corrected or updated based on comment 48.

R-10 Table H-75 should be reprinted because the title is incorrect and misleading (it should be "bedrock" rather than "north sector," based on comment 48). In preparing this table for resubmission, the other changes can be made: the EPCs and CSF could be corrected or updated (based on comment 48).

R-11 Page 5-4, 1st and 2nd paragraphs, and page 6-2, 2nd paragraph, last sentence, still contain inadequately supported conclusions about 1,2-DCA, even though both Sections 5 and 6 were resubmitted. Revisions in accordance with comment 50 are recommended.

R-12 Tables H-17a and H-18a used EPA factors for all but the Cvs. According to the EPA air modeler, these Cvs values should be as follows: acenaphthylene (hourly 2.3E-5 mg/m^3, annual 1.9E-6 mg/m^3); benzo[g,h,i]perylene (hourly 3.5E-9 mg/m^3, annual 2.8E-10 mg/m^3); phenanthrene (hourly 1.4E-4 mg/m^3; annual 1.1E-5 mg/m^3); Aroclor 1242 (1.8E-4 mg/m^3, annual 1.4E-5 mg/m^3); Aroclor 1254 (hourly 4.2E-4 mg/m^3; annual 3.4E-5 mg/m^3); Aroclor 1260 (hourly 2.1 mg/m^3, annual 1.7E-5 mg/m^3).

R-13 Tables 37a, 38a, and 3-19 (footnote) used the incorrect EPA Cp value. The 0.0016 value was for the pile only; the total hourly Cp is still approximately 1.7 mg/m^3 and the annual 0.14 mg/m^3. This would change the cancer risk on Table 37a by 5 to 6 orders of magnitude.

R-14 The response letter indicates that comment 61 would not be addressed. However, on our conference call, CBS agreed to address the groundwater characterization issue to the extent necessary. Basically, this involves a discussion of the bedrock groundwater detections of dioxins, furans, and PAHs as a supplement to the information that appears in Appendix B.

R-15 Table 5-1 should either be corrected to show what the CBS risks would be if all the corrections were made, or EPA estimates should be relied on where corrections were not made.

REPORT SECTIONS AFFECTED BY MINOR CHANGES

The following report sections are not expected to change. Whenever a reader consults the BLRA, the BLRA pages on this list should not be used as references until the reader has checked Appendix I for the appropriate adjustments.

BLRA Sections that should only be used in conjunction with Appendix I:

Section 2.4.4 (should refer reader to the river discussion; based on comment 8)
Page 3-1 (should indicate PADEP rather than EPA; based on comment 77)
Page 3-37, 3rd paragraph (should indicate 0.22 skin fraction for indoor worker; based on

comment 20)
Page 5-3, 2nd paragraph (should refer reader to Section 5.3 for EPA moat evaluation; based on comment 50)
Page 5-5, indoor construction worker discussion (should have included more information on manganese; based on comment 50): see also manganese recommendations at the beginning of this memo
Section 5.3 (should include information on other pathways such as surface water, sediment, and local soil; based on bullet 4)
Page 6-2 (should indicate a wider range of potential further actions; based on comment 89)
The uncertainty section (could have included information about multiple risk descriptors; this information appears on Table 5-1 instead; based on comment 72)
Pages G-2, G-7, G-25, G-29, G-37, G-68, G-118, G-122, G-147, G-155 (newer RfDs and CSFs were available; based on comment 43)

Table 3-7 (R data should not have been used, although the effect on EPCs was negligible; based on comment 12)
Table 4-2 (the carcinogenic status of nickel should have been corrected; based on commment 34)
Table 4-3 (the inhalation CSFs for TCDD and DEHP should have been corrected or updated; based on comments 35h and 35i)
Table 4-4 (other pentachlorinated CDDs should be included; based on comment 85)
Table 4-5 (some RfDs should have been corrected or updated; based on comments 36o and 43)
Table 4-6 (some RfDs should have been corrected or updated; based on comments 37a, 37e, and 43)
Table 4-9 (Kp for dibenz[a,h]anthracene should have been corrected; based on comment 39h)
Table C-4 (R data should have been rejected and RBCs should have been corrected or updated; based on comments 3c and 7c)
Table C-5 (RBCs should have been corrected or updated; based on comment 7d)
Table C-8 (RBCs should have been corrected or updated; based on comment 7f)
Table C-11 (RBCs should have been corrected or updated and one chemical would no longer be a COPC; based on bullet 6)
Table D-7 (R data should have been rejected, causing slight changes to EPCs; based on comment 12)
Table E-4 (TCDD concentration; see air modeler's comments and toxicologist's comments on alluvial southern and central TCDD EPCs)
Tables H-11 and H-12 (Aroclor 1242 Cvs should have been corrected; based on comment 48)

Based on the comments, corresponding changes should also have been made to Appendix H. While total risks on these tables would generally not change significantly, individual factors or chemical results may need to be corrected or updated. The following tables are affected by this issue (based on comments 37e, 48, and 49):

Table 5-1		
Table H-3	Table H-4	Table H-6
Table H-7	Table H-8	Table H-13
Table H-14	Table H-15	Table H-16
Table H-19	Table H-20	Table H-23
Table H-25	Table H-26	Table H-27
Table H-28	Table H-29	Table H-30
Table H-33	Table H-34	Table H-36
Table H-39	Table H-40	Table H-41

Table H-42	Table H-49	Table H-50
Table H-51	Table H-52	Table H-60
Table H-61	Table H-62	Table H-65
Table H-66	Table H-67	Table H-68
Table H-69	Table H-70	Table H-71
Table H-73	Table H-74	Table H-76
Table H-78	Table H-79	Table H-80
Table H-82	Table H-84	Table H-85
Table H-86	Table H-88	

The adolescent trespasser's dermal-contact-with-storm-water Westinghouse estimate of cancer risk should be 3E-6 rather than 2E-6 on page 5-3 and Table 5-1, to conform with new Table H-55.

If you have any questions concerning these comments, please contact me at x3328.

cc: Eric Johnson (3HS41)
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