APPENDIX H

TOXICOLOGICAL PROFILES

Screening Level Ecological Risk Assessment Protocol

August 1999

APPENDIX H

TOXICOLOGICAL PROFILES

APPENDIX H

TOXICOLOGICAL PROFILES

ACETONE

1.0 SUMMARY

Acetone is a highly volatile organic compound. Volatilization and biodegradation are the major fate processes affecting acetone released to soil, surface water, and sediment. Routes of exposure for wildlife include ingestion, inhalation, and dermal uptake. Acetone is not bioconcentrated by aquatic organisms, and is not bioaccumulated by mammals and birds. Therefore, it does not bioaccumulate in aquatic or terrestrial food chains.

The following is a profile of the fate of acetone in soil, surface water and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER AND SEDIMENT

Volatilization and leaching are the two primary transport properties affecting the fate of acetone in soils (HSDB 1997). Volatilization is more significant than leaching. The extent of leaching depends on soil characteristics. Evidence also suggests that acetone rapidly degrades in soil (HSDB 1997).

Volatilization and biodegradation are the major fate processes affecting the fate of acetone in surface water. The volatilization half-life for acetone from a model river is approximately 18 hours when estimated using 1-meter depth, a current of 1 m/second, and wind velocity of 3 m/second (Thomas 1982). In addition, acetone does not partition well to sediments because it is highly soluble in water. Dispersion of acetone from the water column to sediment and suspended solids in water is likely to be insignificant, due to the complete miscibility of acetone in water.

Biodegredation is the most significant degradation process of acetone in water (Rathbun et al. 1982). Studies on wastewater have shown that aquatic microbial communities quickly acclimate to acetone, and rapidly biodegrade it (Urano and Kato 1986a,b). When tested in seawater, acetone was biodegraded much slower than when tested in freshwater (Takemoto et al. 1981).

Photolysis as a degradation process for acetone in water is insignificant. Studies have shown that photodecomposition was not observed when acetone contaminated distilled or natural water was exposed to sunlight for 2-3 days (Rathbun et al. 1982).

3.0 FATE IN ECOLOGICAL RECEPTORS

For most aquatic systems, acetone will exist in water rather than sediment, due to acetone's high water solubility and low sediment adsorption coefficient. Bioaccumulation does not occur in aquatic organisms as suggested by the low log K_{ow} value for acetone (Rathbun et al. 1982). Adult haddock tested under static conditions at 7.9°C showed a bioconcentration factor of 1 for acetone (Rustung et al. 1931). Biomagnification along the aquatic food chain is also considered insignificant for acetone as suggested by the low K_{ow} value.

Acetone is a highly volatile compound and may be inhaled in large quantities. Acetone is very water soluble, so it is quickly absorbed following inhalation into the blood stream and dispersed throughout the body. A large portion of acetone is excreted primarily unchanged through the lungs and urine, with only a small portion reduced and excreted as carbon dioxide (Encyclopedia of Occupational Health and Safety 1983). Because acetone is quickly eliminated, wildlife receptors will not accumulate it in tissues.

No information was available on the fate of acetone after exposure by birds or plants.

4.0 REFERENCES

ATSDR. 1994. *Toxicological Profile for Acetone*. Agency for Toxic Substances and Disease Registry, Atlanta, GA.

Encyclopedia of Occupational Health and Safety. 1983. p 38. As cited in HSDB 1997.

HSDB. 1997. Hazardous Substances Data Bank.

Rathbun R, Stephens D, Schultz D,Tai D. 1982. "Fate of Acetone in Water." Chemosphere 11:1097-1114.

Rustung E, Frithjof K, Foyen A. 1931. "The Uptake and Distribution of Acetone in the Coldblooded Organism." Biochem Z 242:366-376.

Takemoto S, Kuge Y, Nakamoto M. 1981. "The Measurement of BOD in Sea Water." Suishitsu Okaku Kenkyu 4:80-90. As cited in ATSDR 1994.

Thomas R. 1982. "Volatilization from Water." In: Lyman W, Reehl W, Rosenblatt D, eds. *Handbook of Chemical Property Estimation Methods.* McGraw-Hill Book Company, New York. pp 15-1 to 15-34.

Urano K, Kato Z. 1986a. "Evaluation of Biodegradation Rates of Priority Organic Compounds." J Haz Matr 13:147-159.

Urano K, Kato Z. 1986b. "A Method to Classify Biodegradabilities of Organic Compounds." J Haz Matr 13:135-145.

ACRYLONITRILE

1.0 SUMMARY

Acrylonitrile is a highly water soluble volatile organic compound. Volatilization and biodegradation are the major fate processes affecting acrylonitrile released to surface soil, surface water, and sediment. Routes of exposure for wildlife include ingestion, inhalation, and dermal uptake. Acrylonitrile is not bioconcentrated by aquatic organisms, and is not bioaccumulated by mammals and birds. Therefore, it does not bioaccumulate in aquatic or terrestrial food chains.

The following is a profile of the fate of acrylonitrile in soil, surface water, and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in surface soil, surface water, and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

Due to its high water solubility, acrylonitrile is highly mobile in moist soils (EPA 1987). Adsorption into the soil is considered insignificant (Kenaga 1980). Evaporation of acrylonitrile from dry soils is expected to occur rapidly because of its high vapor pressure (Norris 1967; EPA 1987) and high Henry's Law constant (Meylan 1991).

Acrylonitrile is readily soluble in water and does not strongly adsorb to soil or sediment (Klein et al. 1957; ATSDR 1990). Acrylonitrile biodegrades rapidly in water (Miller and Villaume 1978; EPA 1987). Aerobic microorganisms readily degrade acrylonitrile, particularly if acclimation time is allowed (Cherry et al. 1956; Stover and Kincannon 1983; Mills and Stack 1954, 1955).

Acrylonitrile rapidly volatilizes from surface water. A volatilization half-life of 1-6 days in water has been estimated (Thomas 1982; HSDB 1997).

3.0 FATE IN ECOLOGICAL RECEPTORS

Based on experimental and estimated bioconcentration factors, the bioconcentration of acrylonitrile in aquatic organisms is not believed to be significant (Kenaga 1980). A steady-state bioconcentration factor

(BCF) of 48 was measured in bluegill sunfish (Barrows et al. 1978). The estimated average BCF for edible portions of freshwater and marine species was approximately 30 based on the relative proportion of fat in sunfish and other organisms (EPA 1980). Also, based on a low log K_{ow}, acrylonitrile is estimated to show low bioconcentration in aquatic organisms (Verschueren 1983; Kenaga 1980).

Acrylonitrile is readily absorbed into the body through lung and intestinal mucosa following inhalation, ingestion, or dermal contact (Clayton and Clayton 1982). Once absorbed into the body, acrylonitrile is distributed throughout the body to the major organs (Pilon et al. 1988a). Following a single oral dose of radiolabeled acrylonitrile, rapid distribution of acrylonitrile and its metabolites was shown in all tissues of rats (Ahmed et al. 1982, 1983; Silver et al. 1987; Young et al. 1968). Another metabolic pathway includes the formation of CO_2 which is excreted via the lungs (Young et al. 1968). The rate of acrylonitrile metabolism is inconclusive; however, evidence suggests that it is rapid (Pilon et al. 1988b; Ghanayem and Ahmed 1982; Miller and Villaume 1978). Values representing the amount of acrylonitrile metabolized range from 4% to 30% (IARC 1979).

No information was available on the fate of acrylonitrile after exposure by birds or plants.

4.0 REFERENCES

Ahmed A, Farooqui M, Upreti R, El-Shabrawy O. 1982. "Distribution and Covalent Interactions of [1(-14)c]acryolontrile in the Rat." Toxicology 23:159-175.

Ahmed A, Farooqui M, Upreti R, El-Shabrawy O. 1983. "Comparative Toxicokinetics of 2,3-(14)c- and 1-(14)c-acrylonitrile in the Rat." J Appl Toxicol 3:39-47.

ATSDR. 1990. *Toxicological Profile for Acrylonitrile*. Agency for Toxic Substances and Disease Registry. December.

Barrows M, Petrocelli S, Macek K, et al. 1978. "Bioconcentration and Elimination of Selected Water Pollutants by Bluegill Sunfish." Proc Am Chem Soc 18:345-346.

Cherry A, Bagaccia A, Senn H. 1956. "The Assimilation Behavior of Certain Toxic Organic Compounds in Natural Water." Sewage Industrial Wastes 28:1137-1146.

Clayton G, Clayton F. 1982. *Patty's Industrial Hygiene and Toxicology*. 3rd ed. Vol 2c. John Wiley & Sons, New York. pp. 4863-4866.

EPA. 1980. *Ambient Water Quality Criteria Document for Acrylonitrile.* EPA 440/5-80-017. Office of Water Regulations and Standards, Washington, DC.

EPA. 1987. *Health Assessment Document for Acrylonitrile.* Cincinnati, OH: US Environmental Protection Agency, Office of Research and Development. EPA 600/8-88/014. NTIS No. PB88-179411.

Ghanayem B, Ahmed A. 1982. "In Vivo Biotransformation and Biliary Excretion of 1-14c-acrylonitrile in Rats." Arch Toxicol 50:175-185.

HSDB. 1997. Hazardous Substance Data Bank.

IARC. 1979. "Acrylonitrile, Acrylic and Modacrylic Fibers, and Acrylonitrile-butadiene-styene and Styrene-acrylonitrile Copolymers." IARC monographs, Vol 19. IARC, Lyon. pp. 73-113.

Kenaga E. 1980. "Predicted Bioconcentration Factors and Soil Sorption Coefficients of Pesticides and Other Chemicals." Ecotoxicol Environ Safety 4:26-38.

Klein E, Weaver J, Webre B. 1957. "Solubility of Acrylonitrile in Aqueous Bases and Alkali Salts." Ind Eng Chem 2:DS72-75.

Meylan W, Howard P. 1991. Environ Toxicol Chem 10:1283-1293. As cited in HSDB 1997.

Miller L, Villaume J. 1978. *Investigation of Selected Potential Environmental Contaminants: Acrylonitrile.* Office of Toxic Substances. U.S. Environmental Protection Agency. Washington, DC.

Mills E, Stack V. 1954. "Biological Oxidation of Synthetic Organic Chemicals." Engineering Bulletin, Proceedings 8th Ind Waste Conf Ext Ser. 83:492-517. As cited in ATSDR 1990.

Mills E, Stack V. 1955. "Acclimation of Microorganisms for the Oxidation of Pure Organic Chemicals." Proceedings 9th Ind Waste Conf Ext Ser. 87:449-464. As cited in ATSDR 1990.

Norris M. 1967. *Acrylonitrile. Encyclopedia of Industrial Chemical Analysis.* Interscience Publ., New York. 4:368-371.

Pilon D, Roberts A, Rickert D. 1988a. "Effect of Glutathione Depletion on the Uptake of Acrylonitrile Vapors and on its Irreversible Association with Tissue Macromolecules." Toxicol Appl Pharmacol 95:265-278.

Pilon D, Roberts A, Rickert D. 1988b. "Effect of Glutathione Depletion on the Irreversible Association of Acrylonitrile with Tissue Macromolecules after Oral Administration to Rats." Toxicol Appl Pharmacol 95:311-320.

Silver E, Szabo S, Cahill M, Jaeger R. 1987. "Time-course Studies of the Distribution of [1-14c]acrylonitrile in Rats after Intravenous Administration." J Appl Toxicol 7:303-306.

Stover E, Kincannon D. 1983. "Biological Treatability of Specific Organic Compounds Found in Chemical Industry Wastewaters." J Water Pollut Control Fed 55:97-109.

Thomas R. 1982. "Volatilization from Water." In: *Handbook of Chemical Property Estimation Methods. Environmental Behavior of Organic Compounds*. McGraw-Hill, New York. pp. 15.1 to 15.34.

Verschueren K. 1983. *Handbook of Environmental Data on Organic Chemicals*. 2nd ed. Van Nostrand Reinhold Co., New York. pp. 162-165.

Young J, Slauter R, Karbowski R. 1968. *The Pharmacokinetic and Metabolic Profile of 14c-acrylonitrile Given to Rats by Three Routes*. Dow Chemical Company, Toxicology Research Laboratory, Midland, MI. As cited in ATSDR 1990.

ALUMINUM

1.0 SUMMARY

In nature, aluminum does not exist in the elemental state, but partitions between the liquid and solid phases by forming complexes with various compounds. Aluminum adsorbs to clays and suspended solids in water. Exposure routes for aquatic organisms include ingestion, gill uptake and dermal contact. Aluminum bioconcentrates in aquatic organisms. Exposure routes for mammals include ingestion, inhalation and dermal exposure; however, regardless of the route of exposure, aluminum is poorly absorbed by mammals. Aluminum is not readily metabolized. Aluminum causes pulmonary and developmental effects. Aluminum uptake by plants varies between species, resulting in differing rates of bioconcentration in plant tissues.

The following is a profile of the fate of aluminum in soil, surface water and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, surface water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER AND SEDIMENT

Aluminum does not exist as a free metal in nature due to its reactivity, but rather partitions between the solid and liquid phases by reacting with water, chloride, fluoride, sulfate, nitrate, phosphate, humic materials and clay (Bodek et al. 1988). Soils with a greater mineral content result in reduced mobility of aluminum (James and Riha 1989).

In water, aluminum forms relatively water-insoluble complexes, or is found as a water-soluble complex. Aluminum adsorbs to suspended solids and sediment. If large amounts of organic matter or fulvic acid are present, aluminum binds to them (Brusewitz 1984). In water, aluminum undergoes hydrolysis to form hydroxy aluminum species (Snoeyink and Jenkins 1980). The pH of the water determines which hydrolysis products are formed.

3.0 ECOLOGICAL RECEPTORS

Exposure routes for aquatic organisms include ingestion, gill uptake, and dermal absorption. Aluminum bioconcentrates in aquatic species (Cleveland et al. 1989).

Exposure routes for mammals include ingestion, inhalation and dermal exposure. Aluminum is poorly absorbed. Aluminum is distributed to the brain (Santos et al. 1987), bone, muscle and kidneys (Greger and Donnaubauer 1986). No studies were located that described excretion of aluminum in animals; however in humans, absorbed aluminum is excreted primarily through the kidney (Gorsky et al. 1979).

Information was not available on the fate of aluminum in birds.

Aluminum is taken up by plants (Brusewitz 1984). Some plants bioaccumulate aluminum in the root tissues. Plant uptake of aluminum and the transport to stems and leaves varies considerably between species (Kabata-Pendias and Pendias 1984).

4.0 REFERENCES

ATSDR. 1992. *Toxicological Profile for Aluminum.* Agency for Toxic Substances and Disease Registry. July.

Bodek I, Lyman W, Reehl W, et al., eds. 1988. *Environmental Inorganic Chemistry-properties, Processes, and Estimation Methods.* Pergamon Press, New York. pp. 6.7-1 to 6.7-9.

Brusewitz S. 1984. *Aluminum. Vol 203.* University of Stockholm, Institute of Theoretical Physics, Stockholm, Sweden. p 138. As cited in ATSDR 1992.

Cleveland L, Little E, Wiedmeyer R, Buckler D. 1989. "Chronic No-observed-effect Concentrations of Aluminum for Brook Trout Exposed in Low-calcium, Dilute Acidic Water." In: Lewis T, ed. *Environmental Chemistry and Toxicology of Aluminum*. Lewis Publishers, Chelsea, MI. pp. 229-246.

Gorsky J, Dietz A, Spencer H, Osis D. 1979. "Metabolic Balance of Aluminum Studied in Six Men." Clin Chem 25:1739-1743.

Greger J, Donnaubauer S. 1986. "Retention of Aluminum in the Tissues of Rats after the Discontinuation of Oral Exposure to Aluminum." Food Chem Toxicol 24:1331-1334.

James B, Riha S. 1989. "Aluminum Leaching by Mineral Acids in Forest Soils: I. Nitric-sulfuric Acid Differences." Soil Sci Soc Am J 53:259-264.

Kabata-Pendias A, Pendias H, eds. 1984. *Trace Elements in Soils and Plants.* CRC Press, Boca Raton, FL. pp. 135-136.

Santos F, Chan J, Yang M, Savory J, Wills M. 1987. "Aluminum Deposition in the Central Nervous System. Preferential Accumulation in the Hippocampus in Weanling Rats." Med Biol 65:53-55.

Snoeyink V, Jenkins D, ed. 1980. *Water Chemistry*. John Wiley and Sons, New York. pp. 209-210.

ANTIMONY

1.0 SUMMARY

Antimony binds to soil and particulates and is oxidized by bacteria in soil. Exposure routes for aquatic organisms include ingestion and gill uptake. Antimony bioconcentrates in aquatic organisms. Exposure routes for mammals include ingestion and inhalation. It does not biomagnify in terrestrial food chains. Antimony is not significantly metabolized and is excreted in the urine and the feces. Antimony causes reproductive, pulmonary and hepatic effects in mammals. Antimony uptake by plants occurs following surface deposition.

The following is a profile of the fate of antimony in soil, surface water and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, surface water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER AND SEDIMENT

Antimony binds to soil, particularly to particles containing iron, manganese, or aluminum Ainsworth 1988). In water, antimony is oxidized when exposed to atmospheric oxygen (Parris and Brinckman 1976).

3.0 ECOLOGICAL RECEPTORS

Exposure routes for aquatic organisms include ingestion and gill uptake. Antimony bioconcentrates in aquatic organisms (ACQUIRE 1989; Callahan et al. 1979; EPA 1980).

Exposure routes for mammals include ingestion and inhalation (Groth et al. 1986, EPA 1988). Dermal absorption is low (Myers et al. 1978) and absorption from the respiratory tract is dependent on particle size (Thomas et al. 1973). Following absorption, antimony is distributed to the liver, kidney, bone, lung, spleen and thyroid (Sunagawa 1981; Ainsworth 1988). Antimony is excreted in the urine and the feces (Felicetti et al. 1974). Antimony does not biomagnify in the food chain (Ainsworth 1988). Data regarding the amount of antimony that reaches the site of action and assimilation efficiency were not available.

Information was not available on the fate of antimony in birds.

Antimony is taken up by plants following surface deposition, with uptake from soil dependent on the solubility of the antimony in the soil (Ainsworth 1988).

4.0 REFERENCES

Acquire. 1989. Acquire database. September 7. As cited in ATSDR 1990.

Ainsworth N. 1988. *Distribution and Biological Effects of Antimony in Contaminated Grassland.* Dissertation. As cited in ATSDR 1990.

ATSDR. 1990. *Toxicological Profile for Antimony.* Agency for Toxic Substances and Disease Registry. October.

Callahan M, Slimak M, Gabel N, et al. 1979. *Water-Related Environmental Fate of 129 Priority Pollutants.* Vol 1. EPA 440/4-79-029a. Office of Water Planning and Standards, Washington, DC. pp. 5-1 to 5-8.

EPA. 1988. *Drinking Water Criteria Document for Antimony.* EPA contract no. 68-03-3417. p. III-16.

EPA. 1980. *Ambient Water Quality Criteria for Antimony.* EPA 440/5-80-020. Office of Water Regulations and Standards Criteria Division, Washington, DC.

Felicetti S, Thomas R, McClellan R. 1974. "Metabolism of Two Valence States of Inhaled Antimony in Hamsters." Am Ind Hyg Assoc J 355:292-300.

Groth D, Stettler L, Burg J. 1986. "Carcinogenic Effects of Antimony Trioxide and Antimony Ore Concentrate in Rats." J Toxicol Environ Health 18:607-626.

Myers R, Homan E, Well C, et al. 1978. *Antimony Trioxide Range-finding Toxicity Studies.* Ots206062. Carnegie-Mellon Institute of Research, Carnegie-Mellon University, Pittsburgh, Pa. Sponsored by Union Carbide. As cited in ATSDR 1990.

Parris G, Brinckman F. 1976. "Reactions Which Relate to the Environmental Mobility of Arsenic and Antimony. Ii. Oxidation of Trimethylarsine and Trimethylstibine." Environ Sci Technol 10:1128-1134.

Sunagawa S. 1981. "Experimental Studies on Antimony Poisoning." Igaku Kenkyu 51: 129-142.

Thomas R, Felicetti S, Lucchino R, McClellan R. 1973. "Retention Patterns of Antimony in Mice Following Inhalation of Particles Formed at Different Temperatures." Proc Exp Biol Med 144:544-550.

ARSENIC

1.0 SUMMARY

Arsenic, because of its complex chemistry, exists in the environment in many different inorganic and organic forms, which have different toxicological and physicochemical properties. Inorganic arsenic exists as either the trivalent (3+) form or the pentavalent (5+) form. The inorganic trivalent arsenic forms are more toxic than the pentavalent forms. Elemental arsenic (the metalloid -0+) is essentially nontoxic even at high intakes.

Arsenic in soil is usually tightly bound. The bioconcentration potential in soil invertebrates and aquatic species is low. Biomagnification through the food chain is minimal because once ingested, arsenic is metabolized to methylated compounds that are rapidly excreted. Absorbed arsenic is distributed to all tissues where it interferes with normal enzymatic activity or disrupts the functioning of other cellular macromolecules. Evaluation of the potential for toxicity from exposure to low levels of arsenic is complicated by the current understanding that arsenic is an essential element in some mammalian species, and that arsenic deficiency may result in adverse reproductive and developmental effects.

The following is a profile of the fate of arsenic in soil, surface water and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, surface water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

The dominant form of arsenic in soil and its transport are largely dependent on the physical characteristics of the soil matrix. Insoluble arsenic compounds, such as arsenic trioxide, bind tightly to organic matter in soil or sediment (EPA 1984; ATSDR 1993). Various forms of arsenic in soil are interconverted by chemical reactions and microbial activity. Soil microorganisms convert small amounts of arsenic to volatile arsines. These volatile arsines are released to the air, become adsorbed to particles, and are redeposited (ATSDR 1993) or, under certain conditions, react to form oxides (Ghassemi et al. 1981).

The bioavailability of arsenic in soil is inversely proportional to the organic carbon and clay content of the soil matrix. Arsenic in soil is directly taken up by plants and soil microbes and invertebrates, and indirectly taken up by terrestrial receptors via ingestion.

In surface water, soluble inorganic arsenate (As5+) predominates under normal conditions and is more stable than arsenite (EPA 1980a). Movement and partitioning of arsenic in water depends on the chemical form of arsenic and on interactions with other materials present (Callahan et al. 1979). Soluble forms of arsenic remain dissolved in the water column or adsorb onto sediments or soils, especially those containing clays, iron oxides, aluminum hydroxides, manganese compounds, and organic matter (Callahan et al. 1979; Welch et al. 1988). Sediment bound arsenic is released back into the water by chemical or biological interconversions. This interconversion is influenced by the Eh (the oxidation-reduction potential), pH, temperature, other metals, salinity, and biota (Callahan et al. 1979). Arsenate is transformed by microbes to arsenite and methylated arsenicals (Benson 1989; Braman and Foreback 1973).

3.0 ECOLOGICAL RECEPTORS

Exposure routes for aquatic organisms include gill uptake, ingestion of arsenic suspended on particles in the water column or deposited in sediment, and ingestion of plant matter and lower trophic level aquatic species. Arsenic bioconcentration in aquatic organisms is low (Spehar et al. 1980; EPA 1980b). Fish and shellfish rapidly metabolize arsenic to non-toxic forms (EPA 1984, Garcia-Vargas and Cebrian 1996; ATSDR 1993). Biomagnification does not readily occur in aquatic food chains (Callahan et al. 1979).

Soil invertebrates are directly exposed to arsenic found in soil and soil pore water. Exposure routes for soil invertebrates include ingestion and dermal absorption. Arsenic bioconcentration in soil invertebrates is low (Rhett et al. 1988).

The majority of ecological mammalian exposure occurs through ingestion. The oral absorption efficiency is dependent on the form of arsenic, its solubility, and the media ingested. Soluble arsenic compounds in aqueous solution are more readily absorbed from the gastrointestinal tract than insoluble compounds. Absorption from water ingested is approximately 85%. Inorganic arsenic in food sources is expected to be readily bioavailable with absorption rates of greater than 85% expected. Once absorbed, arsenic is readily transported throughout the body with little tendency to accumulate preferentially in any one internal organ

(ATSDR 1993). Dermal absorption is a minor route of exposure with absorption estimated at 0.1% (ATSDR 1993).

Metabolism of arsenic occurs primarily in the liver. The methylated metabolites are less toxic than the inorganic precursors, and metabolism results in lower tissue retention of inorganic arsenic (Marafante and Vahter 1984, 1986, 1987; Marafante et al. 1985). Inorganic arsenic and its methylated products are rapidly eliminated.

The toxicokinetic data for arsenic indicate there is little potential for bioaccumulation in animal tissue exposed to doses that are below the level required to saturate detoxifying methylation reactions. The level of biomagnification in mammals depends on the diet of the animal. Herbivores have a low arsenic biomagnification rate due to the general lack of transport of arsenic from soil to above ground plant parts. Omnivores have a higher biomagnification rate based on the higher proportion of soil invertebrates in their diet. Carnivores have the highest biomagnification rate due to their diet of aquatic invertebrates, small mammals, and fish and the incidental ingestion of soil. However, arsenic is rapidly metabolized in mammalian species, therefore, arsenic does not readily bioaccumulate in mammals.

Exposure routes for avian receptors include ingestion of surface water, soil, soil and aquatic invertebrates, and plant material. Absorption studies specific to avian species are not available. Based on mammalian absorption (ATSDR 1993), avian absorption can be assumed to be 85% absorption from water, 30% to 40% absorption from soil, and 85% absorption from food sources.

Arsenic uptake by plants depends on the form of arsenic and the type of soil. The higher the soil's organic carbon and clay content the more the arsenic will bind to the soil and, therefore, less arsenic is available for uptake by plant roots. That which is readily taken up by the plant is accumulated in the roots. Arsenite (3+) is highly toxic to cell membranes and, therefore, not readily translocated once taken up; arsenate (5+) is less toxic and, therefore, more readily translocated after uptake (ORNL 1996; Speer 1973). Rice, most legumes, and members of the bean family are sensitive to arsenic in most forms, with spinach being the most sensitive plant (Woolson et al 1975).

4.0 REFERENCES

ATSDR. 1993. *Toxicological Profile for Arsenic.* Agency for Toxic Substances and Disease Registry. April.

Benson A. 1989. "Arsonium Compounds in Algae." Proc Natl Acad Sci 86:6131-6132.

Braman R, Foreback C. 1973. "Methylated Forms of Arsenic in the Environment." Science 182:1247-1249.

Callahan M, Slimak M, Gabel N et al. 1979. *Water-related Environmental Fate of 129 Priority Pollutants.* Volume 1. EPA/440/4-79-029a. Office of Water Planning and Standards, Washington, DC. As cited in ATSDR 1993.

EPA. 1980a. *Ambient Water Quality Criteria for Arsenic.* EPA 440/5-80-021. Office of Water Regulations and Standards, Washington, DC.

EPA. 1980b. Unpublished laboratory data. Environmental Research Lab, Narragansett, Rhode Island. As cited in EPA 1980a.

EPA. 1984. *Health Assessment Document for Inorganic Arsenic.* EPA/600/8-83-021f. Office of Health and Environmental Assessment, Washington, DC.

Garcia-Vargas G, Cebrian M. 1996. "Health Effects of Arsenic." In: Chang L, ed. *Toxicology of Metals.* Lewis Publ., Boca Raton, FL. pp. 423-438.

Ghassemi M, Fargo L, Painter P, et al. 1981. *Environmental Fates and Impacts of Major Forest Use Pesticides.* Prepared by TRW Environmental Division, Redondo Beach, CA. Prepared for EPA, Office of Pesticides and Toxic Substances, Washington, DC.

Marafante F, Vahter M. 1984. "The Effect of Methyltransferase Inhibition on the Metabolism of [74as] Arsenite in Mice and Rabbits." Chem Biol Interact 50:49-57.

Marafante E, Vahter M. 1986. "The Effect of Dietary and Chemically Induced Methylation Deficiency on the Metabolism of Arsenate in the Rabbit." Acta Pharmacol Toxicol 59(Suppl 7):35-38.

Marafante E, Vahter M. 1987. "Solubility, Retention, and Metabolism of Intratracheally and Orally Administered Inorganic Arsenic Compounds in the Hamster." Environ Res 42:72-82.

Marafante E, Vahter M, Envall J. 1985. "The Role of the Methylation in the Detoxication of Arsenate in the Rabbit." Chem-Biol Interact 56:225-238.

ORNL. 1996. *Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Terrestrial Plants: 1995 Revision*. ES/ER/TM-82-R2. Oak Ridge National Laboratory, Oak Ridge, TN.

Rhett RG, Simmers JW, Lee CR. 1988. *Eisenia Foetida Used as a Biomonitoring Tool to Predict the Potential Bioaccumulation of Contaminants from Contaminated Dredging Material.* SPB Academic Publishing. Pp. 321-328.

Speer, H.L. 1973. "The Effect of Arsenic and Other Inhibitors on Early Events During the Germination of Lettuce Seeds." Plant Physiology 52: 142-146.

Spehar R, Fiandt J, Anderson R, Defoe D. 1980. "Comparative Toxicity of Arsenic Compounds and Their Accumulation in Invertebrates and Fish." Arch Environ Contam Toxicol 9:53-63.

Welch A, Lico M, Hughes J. 1988. "Arsenic in Groundwater of the Western United States." Ground Water 26:333-347.

Woolson E.A., Axley J.H., and Kearney P.C. 1973. "The Chemistry and Phytotoxicity of Arsenic in Soils Ii. Effects of Time and Phosphorus." Soil Science Society of America Proceedings 37:254-259.

BERYLLIUM

1.0 SUMMARY

In environmental media, beryllium usually exists as beryllium oxide. Beryllium has limited solubility and mobility in sediment and soil. Exposure routes for aquatic organisms include ingestion and gill uptake. Beryllium does not bioconcentrate in aquatic organisms. Beryllium is toxic to warm water fish, especially in soft water. Exposure routes for mammalian species include inhalation. Mammals exposed via inhalation exhibit pulmonary effects which may last long after exposure ceases.

The following is a profile of the fate of beryllium in soil, surface water and sediment, and the fate after uptake by biological receptors. Section 2 discusses the environmental fate and transport in soil, surface water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

Beryllium adsorbs to clays at low pH, precipitates as insoluble complexes at higher pH, and has limited solubility in soil (Callahan et al. 1979). Chemical reactions in soil transform one beryllium compound into another (ATSDR 1993). Reactions in soil include hydrolysis of soluble salts, anion exchange, and complexation with ligands such as humic substances (ATSDR 1993).

In water, beryllium is speciated often by hydrolysis in which soluble beryllium salts are hydrolyzed to form relatively insoluble beryllium hydroxide (Callahan et al. 1979). Beryllium is not volatilized from water (ATSDR 1993). Beryllium is retained in an insoluble and immobile form in sediment (EPA 1980).

3.0 ECOLOGICAL RECEPTORS

Beryllium uptake from water is low, resulting in low bioconcentration rates (EPA 1980; Callahan et al. 1979). Biomagnification of beryllium in aquatic food chains does not occur (Fishbein 1981).

In mammals, beryllium compounds are absorbed primarily through the lung (ATSDR 1993). Beryllium is poorly absorbed from the gastrointestinal tract, and is not absorbed through intact skin to any significant degree

(ATSDR 1993). Beryllium is distributed to the liver, skeleton, tracheobronchial lymph nodes, and blood (Finch et al. 1990). Beryllium is not biotransformed, but soluble beryllium salts are partially converted to less soluble forms in the lung (Reeves and Vorwald 1967). Excretion is predominantly via the feces (Finch et al. 1990). Data regarding the amount of beryllium that reaches the site of action or assimilation efficiency were not located.

Information was not available on the fate of beryllium in birds.

Beryllium uptake by plants occurs when beryllium is present in the soluble form. The highest levels of beryllium are found in the roots, with lower levels in the stems and foliage (EPA 1985).

4.0 REFERENCES

ATSDR. 1993. *Toxicological Profile for Beryllium.* Agency for Toxic Substances and Disease Registry.

Callahan M, Slimak M, Gabel N, et al. 1979. *Water-Related Environmental Fate of 129 Priority Pollutants.* EPA-440/4-79-029a. Vol 1. Office of Water Planning and Standards, Washington, DC. pp. 8-1 to 8-7.

EPA. 1980. *Ambient Water Quality Criteria for Beryllium.* EPA 440/5-80-024. Office of Water Regulations and Standards, Washington, DC.

EPA. 1985. *Environmental Profiles and Hazard Indices for Constituents of Municipal Sludge: Beryllium.* Office of Water Regulations and Standards. Washington, DC.

Finch G, Mewhinney J, Hoover M, Eidson A, Haley P, Bice D. 1990. "Clearance, Translocation, and Excretion of Beryllium Following Acute Inhalation of Beryllium Oxide by Beagle Dogs." Fundam Appl Toxicol 15:231-241.

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.

Reeves A, Vorwald A. 1967. "Beryllium Carcinogenesis. Ii. Pulmonary Deposition and Clearance of Inhaled Beryllium Sulfate in the Rat." Cancer Res 27:446-451.

BIS(2-ETHYLHEXYL)PHTHALATE

1.0 SUMMARY

Bis(2-ethylhexyl)phthalate (BEHP) is a high molecular weight, semi-volatile organic compound. BEHP adsorbs strongly to soil and sediment, and it may be biodegraded in aerobic environments. It has a low water solubility and low vapor pressure. It does not undergo significant photolysis, hydrolysis, or volatilization in soil or water. Receptors may be exposed to BEHP by the oral, inhalation, and dermal routes. BEHP bioconcentration in aquatic organisms is generally low, therefore significant food chain biomagnification in upper-trophic-level fish is unlikely. Mammalian and avian wildlife can metabolize and eliminate BEHP, therefore, it does not biomagnify in these receptors.

The following summarizes the fate of BEHP in surface soil, surface water and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate after released to surface soil, surface water, and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER AND SEDIMENT

BEHP adsorbs strongly to soil and does not undergo significant volatilization or photolysis (HSDB 1997). Limited information indicates that, under aerobic conditions, degradation in soil may occur (Hutchins et al. 1983; Mathur 1974). However, because BEHP adsorbs strongly to soil, biodegradation is slow (Wams 1987). Biodegradation in anaerobic conditions is slower than under aerobic conditions (Johnson et al. 1984).

BEHP has a low water solubility. In surface water environments, adsorption is the major mechanism affecting the concentration of BEHP. BEHP strongly adsorbs to suspended solids and sediments (Al-Omran and Preston 1987; Sullivan et al. 1982; Wolfe et al. 1980). However, in marine environments, adsorption to sediments may be decreased because BEHP is not as soluble in salt water when compared to fresh water (Al-Omran and Preston 1987). BEHP may also form complexes with fulvic acid, potentially increasing its mobility in aquatic environments (Johnson et al. 1977).

In aquatic environments, biodegradation is the primary route of degradation. BEHP is biodegraded in aerobic conditions; however, under anaerobic conditions, biodegradation is limited (O'Connor et al. 1989; Tabek et al. 1981; O'Grady et al. 1985). A half-life of approximately one month, due to microbial biodegradation has been reported for BEHP in river water (Wams 1987). BEHP does not undergo significant hydrolysis or photolysis in aquatic environments (Callahan et al. 1979). A hydrolysis half-life of 2,000 years has been estimated (Callahan et al. 1979); and in water a photolysis half-life of 143 days has been reported (Wolfe et al. 1980). BEHP does not significantly volatilize from water, with an half-life of 15 years reported (Callahan et al. 1979).

3.0 FATE IN ECOLOGICAL RECEPTORS

Aquatic receptors may be exposed through ingestion of contaminated food or water, dermal exposure, or in the case of fish, by direct contact of the gills with the surrounding water. Based on its low water solubility and high soil partition coefficient (ATSDR 1993), dietary uptake is the most significant route of exposure anticipated for BEHP.

Based on its high log Kow value, BEHP is expected to accumulate in aquatic species (Barrows et al.1980; Mayer 1977). Invertebrates will bioconcentrate BEHP from surface water and from sediment. The level of bioconcentration is receptor-specific, because some invertebrates can metabolize BEHP, while some have limited capability (Sanders et al. 1973). Under continuous exposure conditions, fish will bioconcentrate BEHP to levels moderately higher than the concentration in surface water (Mehrle and Mayer 1976). BEHP has a short half-life in fish, indicating that it is quickly eliminated (Park et al. 1990). Fish eliminate BEHP by metabolizing it to polar byproducts, which are quickly excreted (Melancon and Lech 1977; Menzie 1980). Therefore, food chain accumulation and biomagnification of BEHP in aquatic food webs is not significant (Callahan et al. 1979; Johnson et al. 1977; Wofford et al. 1981).

BEHP is absorbed by mammals following oral (Astill 1989; Rhodes et al. 1986) or dermal exposure (Melnick et al. 1987), with oral exposure being the route with the greatest absorption efficiency in laboratory animals. In laboratory animals, small amounts of BEHP have been shown to be absorbed following dermal exposure (Melnick et al. 1987). Following oral exposure, it has been reported that a portion of the BEHP is hydrolyzed in the small intestine to 2-ethylhexanol and mono(ethylhexyl)phthalate

which is subsequently absorbed (Albro, et al. 1982). Following absorption, BEHP is distributed primarily to the liver and kidney, and in some species, to the testes (Rhodes et al. 1986).

In mammals, BEHP is metabolized by tissue esterases that hydrolyze one of the ester bonds resulting in the formation of mono(2-ethylhexyl)phthalate and 2-ethylhexanol. Small amounts of mono(2-ethylhexyl)phthalate may be further hydrolyzed to form phthalic acid; however, the majority undergoes aliphatic side chain oxidation followed by alpha- or beta-oxidation. These oxidized products may then be conjugated with glucuronic acid and excreted (Albro 1986). Metabolites of BEHP are excreted in both the urine and the feces (Astill 1989; Short et al. 1987; Ikeda et al. 1980).

BEHP may evaporate from the leaves of plants. In one study, using a closed terrestrial simulation chamber, BEHP was applied to the leaves of *Sinapis alba*. Evaporation rates from the leaves were <0.8 ng/cm^2-hr for a time interval of 0–1 days and <0.5 ng/cm^2-hr for a time interval of 8–15 days (Loekke and Bro-Rasumussen 1981). Uptake of BEHP by plants has also been reported (Overcash et al. 1986).

No data were available on the fate of BEHP in birds.

4.0 REFERENCES

Al-Omran L, Preston M. 1987. "The Interactions of Phthalate Esters with Suspended Particulate Material in Fresh and Marine Waters." Environ Pollut 46:177-186.

Albro P. 1986. "Absorption, Metabolism and Excretion of Di(2-ethyhexyl)phthalate by Rats and Mice." Environ Health Perspect 65:293-298.

Albro PW, Hass JR, Peck CC, et al. 1982. "Identification of Metabolites of Di(2-ethylhexyl)phthalate in Urine from the African Green Monkey." Drug Metab Dispos 9:223-225. As cited in ATSDR 1993.

Astill B. 1989. "Metabolism of Dehp: Effects of Prefeeding and Dose Variation, and Comparative Studies in Rodents and the Cynomolgus Monkey (CMS Studies)." Drug Metab Rev 21:35-53.

ATSDR. 1993. *Toxicological Profile for Di(2-ethylhexyl)phthalate*. Agency for Toxic Substances and Disease Registry. April.

Barrows M, Petrocelli S, Macel K, et al. 1980. "Bioconcentration and Elimination of Selected Water Pollutants by Bluegill Sunfish." In: Haque R, ed. *Dynamics, Exposure Hazard Assessment of Toxic Chemicals.* Ann Arbor Sci., Ann Arbor, MI. pp. 379-392.

Callahan M, Slimak M, Gabel N, et al. 1979. *Water-Related Environmental Fate of 129 Priority Pollutants*. Vol. II. EPA-440/4-79-029b. U.S. EPA, Office of Water Planning and Standards, Washington, DC. pp. 94-6 to 94-14.

HSDB. 1997. Hazardous Substances Data Bank.

Hutchins S, Tomson M, Ward C. 1983. "Trace Organic Contamination of Ground Water from Rapid Infiltration Site: A Laboratory-Field Coordinated Study." Environ Toxicol Chem 2:195-216.

Ikeda G, Sapienza P, Couvillion J, et al. 1980. "Comparative Distribution, Excretion and Metabolism of Di-(2-ethylhexyl)phthalate in Rats, Dogs and Miniature Pigs." Food Cosmet Toxicol 18:637-642. As cited in ATSDR 1993.

Johnson B, Heitkamp M, Jones J. 1984. "Environmental and Chemical Factors Influencing the Biodegradation of Phthalic Acid Esters in Freshwater Sediments." Environ Pollut (Series B)8:101-118.

Johnson B, Stalling D, Hogan J, et al. 1977. "Dynamics of Phthalic Acid Esters in Aquatic Organisms." In: Suffet I, ed. *Fate of Pollutants in the Air and Water Environments*. Part 2. John Wiley, New York. pp. 283-300.

Loekke H, Bro-Rasumussen F. 1981. "Studies of Mobility of Di-iso-butyl Phthalate (Dibp), Di-n-butyl Phthalate (Dbp), and Di-(2-ethyl Hexyl) Phthalate (Dehp) by Plant Foliage Treatment in a Closed Terrestrial Simulation Chamber." Chemosphere 10:1223-1235.

Mathur S. 1974. "Respirometric Evidence of the Utilization of Di-octyl and Di-2-ethylhexyl Phthalate Plasticizers." J Environ Qual 3:207-209.

Mayer F. 1977. J Fish Res Board Can 33:2610.

Mehrle P, Mayer F. 1976. *Trace Substances in Environmental Health*. pp. 518. As cited in HSDB 1997.

Melancon M, Lech J. 1977. "Metabolism of Di-2-ethylhexyl Phthalate by Subcellular Fractions from Rainbow Trout Liver." Drug Metab Dispos 5(1):29.

Melnick R, Morrissey R, Tomaszewski K. 1987. "Studies by the National Toxicology Program on Di(2-ethylhexyl)phthalate." Toxicol Ind Health 3:99-118.

Menzie C. 1980. *Metabolism of Pesticides*. Update III. U.S. Department of Interior, Fish and Wildlife Service. p. 453.

O'Connor O, Rivera M, Young L. 1989. "Toxicity and Biodegradation of Phthalic Acid Esters under Methanogenic Conditions." Environ Toxicol Chem 8:569-576.

O'Grady D, Howard P, Werner A. 1985. *Activated Sludge Biodegradation of 12 Commercial Phthalate Esters*. Report to Chemical Manufacturers Association by Syracuse Research Corporation. Contract no. PE-17.0-ET-SRC. SRC l1553-03. As cited in ATSDR 1993.

Overcash M, Weber J, Tucker W. 1986. *Toxic and Priority Organics in Municipal Sludge Land Treatment Systems*. EPA/600/2-86/010. EPA, ORD, Cincinnati, OH NTIS PB86-50208.

Park, C.W., O. Imamura, and T. Yoshida. 1990. "Uptake, Excretion, and Metabolism of ^{14}C-labeled Di-2-ethylhexyl Phthalate by Mullet, *Mugil cephalus*." Bulletin of Korean Fish. Soc. 22:424-428.

Rhodes C, Orton T, Pratt I, Batten P, Bratt H, Jackson S, Elcombe C. 1986. "Comparative Pharmacokinetics and Subacute Toxicity of Di(2-ethylhexyl)phthalate in Rats and Marmosets: Extrapolation of Effects in Rodents to Man." Environ Health Perspect 65:299-308.

Sanders H, Mayer F, Walsh D. 1973. "Toxicity, Residues Dynamics, and Reproductive Effects of Phthalate Esters in Aquatic Invertebrates." Environ Res 6:84-90.

Short R, Robinson E, Lington A, Chin A. 1987. "Metabolic and Peroxisome Proliferation Studies with Di(2-ethylhexylphthalate in Rats and Monkeys." Toxicol Ind Health 3:185-195.

Sullivan K, Atlas E, Giam C. 1982. "Adsorption of Phthalic Acid Esters from Seawater." Environ Sci Technol 16:428-432.

Tabak H, Quave S, Mashni C, Barth E. 1981. "Biodegradability Studies with Organic Priority Pollutant Compounds." J Water Pollut Contr Fed 53:1503-1518.

Wams T. 1987. "Diethylhexylphthalate as a Environmental Contaminant--a Review." Sci Total Environ 66:1-16.

Wofford HW, Wilsey CD, Neff GS, et al. 1981. "Bioaccumulation and Metabolism of Phthalate Esters by Oysters, Brown Shrimp, and Sheepshead Minnows." Ecotoxicol Environ Safety 5:202-210. As cited in ATSDR 1993.

Wolfe N, Burns L, Steen W. 1980. "Use of Linear Free Energy Relationships and an Evaluative Model to Assess the Fate and Transport of Phthalate Esters in the Aquatic Environment." Chemosphere 9:393-402.

CADMIUM

1.0 SUMMARY

Cadmium exists in the elemental (0+) state or the 2+ valance state in nature. Exposure routes for aquatic organisms include ingestion and gill uptake. Freshwater biota are the most sensitive organisms to cadmium exposure, with toxicity inversely proportional to water hardness. Cadmium bioaccumulates in both aquatic and terrestrial animals, with higher bioconcentration in aquatic organisms. Exposure routes for ecological mammalian species include ingestion and inhalation. Cadmium interferes with the absorption and distribution of other metals and causes renal toxicity in vertebrates.

The following is a profile of the fate of cadmium in soil, surface water and sediment, and the fate after uptake by biological receptors. Section 2 discusses the environmental fate and transport in soil, surface water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER AND SEDIMENT

Cadmium has a low vapor pressure and is released from soil to air by entrainment with soil particles (EPA 1980; OHM/TADS 1997). Cadmium compounds in soil are stable and are not subject to degradation (ATSDR 1993). Cadmium compounds can be transformed by precipitation, dissolution, complexation, and ion exchange (McComish and Ong 1988).

Cadmium compounds in aquatic environments are not affected by photolysis, volatilization, or biological methylation (Callahan et al. 1979). Precipitation and sorption to mineral surfaces and organic materials are important removal processes for cadmium compounds (ATSDR 1993). Concentrations of cadmium are generally higher in sediments than in overlying water (Callahan et al. 1979).

3.0 ECOLOGICAL RECEPTORS

Cadmium bioconcentrates in aquatic organisms, primarily in the liver and kidney (EPA 1985). Cadmium accumulated from water is slowly excreted, while cadmium accumulated from food is eliminated more

rapidly (EPA 1985). Metal-binding, proteinaceous, metallothionens appear to protect vertebrates from deleterious effects of high metal body burdens (Eisler 1985).

Exposure routes in ecological mammalian species include ingestion and inhalation, while dermal absorption is negligible (Goodman and Gilman 1985). Absorption and retention of cadmium decreases with prolonged exposure. Cadmium absorption through ingestion is inversely proportional to intake of other metals, especially iron and calcium (Friberg 1979). Cadmium accumulates primarily in the liver and kidneys (IARC 1973). Cadmium crosses the placental barrier (Venugopal 1978). Cadmium does not undergo direct metabolic conversion, but the ionic (+2 valence) form binds to proteins and other molecules (Nordberg et al. 1985). Absorbed cadmium is excreted very slowly, with urinary and fecal excretion being approximately equal (Kjellstrom and Nordberg 1978).

Freshwater aquatic species are most sensitive to the toxic effects of cadmium, followed by marine organisms, birds, and mammals.

4.0 REFERENCES

ATSDR. 1993. *Toxicological Profile for Cadmium.* Agency for Toxic Substances and Disease Registry.

Callahan M, Slimak M, Gable N, et al. 1979. *Water-Related Fate of 129 Priority Pollutants.* EPA-440/4-79-029a. Vol 1. Office of Water Planning and Standards, Washington, DC. pp. 9-1 to 9-20.

Eisler 1985. *Cadmium Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review.* U. S. Fish and Wildlife Service, U.S. Department of the Interior. Biological Report 85 (1.2).

EPA. 1980. *Fate of Toxic and Hazardous Materials in the Air Environment.* Environmental Sciences Research Laboratory, Research Triangle Park, NC.

EPA. 1985. *Cadmium Contamination of the Environment: an Assessment of Nationwide Risk.* EPA 600/8-83/025f. Office of Water Regulations and Standards, Washington, DC.

Friberg L. 1979. *Handbook of the Toxicity of Metals.* As cited in HSDB 1997.

Goodman L, Gilman A, eds. 1985. *The Pharmacological Basis of Therapeutics.* 7th ed. Macmillan Publ., New York. pp. 1617-1619.

HSDB. 1997. Hazardous Substance Data Base.

IARC. 1973. IARC monographs. 2:74-99.

Kjellstrom T, Nordberg G. 1978. "A Kinetic Model of Cadmium Metabolism in the Human Being." Environ Res 16:248-269.

McComish MF, Ong JH. 1988. "Trace Metals." In: Bodek I, Lyman W, Reehl W, Rosenblatt DH eds. *Environmental Inorganic Chemistry: Properties, Processes, and Estimation Methods.* Pergammon Press, New York. pp. 7.5.1 to 7.5.12. As cited in ATSDR 1993.

Nordberg G, Kjellstrom T, Nordberg M. 1985. "Kinetics and Metabolism." In: Friberg L,Elinder C, Kjellstrom T, et al., eds. *Cadmium and Health: A Toxicological and Epidemiological Appraisal.* Vol 1. CRC Press, Boca Raton, FL. pp. 103-178. As cited in ATSDR 1993.

OHM/TADS. 1997. Oil and Hazardous Materials/Technical Assistance Data System.

Venugopal. 1978. *Metal Toxicity in Mammals 2.* pp. 78, 83. As cited in HSDB 1997.

CHROMIUM

1.0 SUMMARY

Chromium exists primarily in the Cr3+ and Cr6+ valence forms in environmental and biological media. It exists in soil primarily in the form of insoluble oxides with very limited mobility. In the aquatic phase, chromium may be in the soluble state or attached to clay-like or organic suspended solids.

Exposure routes for aquatic organisms include ingestion, gill uptake, and dermal absorption. Bioaccumulation occurs in aquatic receptors; biomagnification does not occur in aquatic food chains. Exposure routes for ecological mammalian species include ingestion, inhalation, and dermal absorption. Chromium is not truly metabolized, but undergoes various changes in valence states and binding with ligands and reducing agents in vivo. Elimination of chromium is slow.

The following is a profile of the fate of chromium in soil, surface water and sediment, and the fate after uptake by biological receptors. Section 2 discusses the environmental fate and transport in soil, surface water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

In soil, chromium 3+ is readily hydrolyzed and precipitated as chromium hydroxide. It exists in soil primarily as insoluble oxide with very limited mobility (EPA 1984a, b).

In water, chromium 6+ occurs in the soluble state or as suspended solids adsorbed onto clay-like materials, organics, or iron oxides. Cr6+ persists in water for long periods of time, but is eventually reduced to chromium 3+ by organic matter or other reducing agents in water (Cary 1982).

3.0 ECOLOGICAL RECEPTORS

Exposure routes for aquatic organisms include ingestion, gill uptake, and dermal absorption. Chromium bioconcentrates in aquatic organisms (ATSDR 1993; OHM/TADS 1997; EPA 1985; EPA 1984a). The

biomagnification and toxicity of chromium 3+ is low relative to chromium 6+ because of its low membrane permeability and noncorrosivity. Chromium is not significantly biomagnified in aquatic food chains.

In vertebrates, chromium 3+ is an essential nutrient needed to produce glucose tolerance factor (GTF), which is required for regulation of glucose levels (ATSDR 1993). Exposure routes for ecological mammalian species include ingestion, inhalation, and dermal absorption. Chromium is poorly absorbed from the gastrointestinal tract after oral exposure, but fasting increases the absorption (Chen et al. 1973). Absorbed chromium is distributed to various organs including the liver and spleen (Maruyama 1982 as cited in ATSDR 1993; Witmer et al. 1989, 1991, as cited in ATSDR 1993).

Following inhalation exposure, chromium is distributed to the lung, kidney, spleen, and erythrocytes (Weber 1983; Baetjer et al. 1959). Following dermal exposure, chromium is readily absorbed and is distributed to the blood, spleen, bone marrow, lymph glands, urine, and kidneys. Chromium is not truly metabolized, but undergoes various changes in valence states and binding with ligands and reducing agents in vivo. Elimination of chromium is slow (Langard et al. 1978).

A large degree of accumulation by aquatic and terrestrial plants and animals in the lower trophic levels has been documented, however, the mechanism of this accumulation remains unknown.

4.0 REFERENCES

ATSDR. 1993. *Toxicological Profile for Chromium.* Agency for Toxic Substances and Disease Registry.

Baetjer A, Damron C, Budacz V. 1959. "The Distribution and Retention of Chromium in Men and Animals." Arch Ind Health 20:136-150.

Cary E. 1982. "Chromium in Air, Soil and Natural." In: Langard S, ed. *Topics in Environmental Health 5: Biological and Environmental Aspects of Chromium.* Elsevier Science, New York. pp. 49-64.

Chen N, Tsai A, Dyer I. 1973. "Effect of Chelating Agents on Chromium Absorption in Rats." J Nutr 103:1182-1186.

EPA. 1985. *Ambient Water Quality Criteria for Chromium*. Office of Water Regulations and Standards. EPA 440/5-84-029.

EPA. 1984a. *Health Assessment Document for Chromium*. Research Triangle Park, NC: Environmental Assessment and Criteria Office. US Environmental Protection Agency. EPA-600/8-81-014F.

EPA. 1984b. *Health Assessment Document for Chromium*. Final report. As cited in ATSDR 1993.

Langard S, Gundersen N, Tsalev D, Gylseth B. 1978. "Whole Blood Chromium Level and Chromium Excretion in the Rat after Zinc Chromate Inhalation." Acta Pharmacol et Toxicol 42:142-149.

Maruyama Y. 1982. "The Health Effect of Mice Given Oral Administration of Trivalent and Hexavalent Chromium over a Long-term." Acta Scholae Med Univ Gifu 31:24-46. As cited in ATSDR 1993.

OHM/TADS. 1997. Oil and Hazardous Materials/Technical Assistance Data System.

Weber H. 1983. "Long-term Study of the Distribution of Soluble Chromate-51 in the Rat after a Single Intratracheal Administration." J Toxicol Environ Health 11:749-764.

Witmer C, Harris R, Shupack S. 1991. "Oral Bioavailability of Chromium from a Specific Site." Environ Health Perspect 92:105-110.

Witmer C, Park H-S, Shupack S. 1989. "Mutagenicity and Disposition of Chromium." Sci Total Environ 86:131-148.

COPPER

1.0 SUMMARY

Copper binds to soils and sediment. Copper is not biodegraded or transformed. Exposure routes for aquatic organisms include ingestion, gill uptake, and dermal absorption. In aquatic organisms, exposures to copper are associated with developmental abnormalities. Copper bioconcentrates in aquatic organisms, however, biomagnification does not occur. Exposure routes for ecological mammalian species include ingestion, inhalation, and dermal absorption. Copper is associated with adverse hematological, hepatic, developmental, immunological, and renal effects in mammals. Copper does not bioaccumulate in mammals.

The following is a profile of the fate of copper in soil, surface water and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, surface water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER AND SEDIMENT

Copper occurs naturally in many animals and plants and is an essential micronutrient. Copper may exist in two oxidation states: +1 or +2. Copper (+1) is unstable and, in aerated water over the pH range of most natural waters (6 to 8), oxidizes to the +2 state. In the aquatic environment, the fate of copper is determined by the formation of complexes, especially with humic substances, and sorption to hydrous metal oxides, clays, and organic materials. The amount of copper able to remain in solution is directly dependent on water chemistry, especially pH and temperature, and the concentration of other chemical species (Callahan et al. 1979; Tyler and McBride 1982; Fuhrer 1986).

The majority of copper released to surface waters settles out or adsorbs to sediments (Harrison and Bishop 1984). Copper is affected by photolysis (Moffett and Zika 1987). Some copper complexes undergo metabolism however, biotransformation of copper is low (Callahan 1979).

3.0 ECOLOGICAL RECEPTORS

Copper bioconcentrates in aquatic organisms. Copper does not biomagnify in aquatic food chains (Heit and Klusek 1985; Perwack et al. 1980).

Copper is absorbed by mammals following ingestion, inhalation, and dermal exposure (Batsura 1969; Van Campen and Mitchell 1965; Crampton et al. 1965). Once absorbed, copper is distributed to the liver (Marceau et al. 1970). Copper is not metabolized. Copper exerts its toxic effects by binding to DNA (Sideris et al. 1988) or by generating free radicals (EPA 1985). Copper does not bioaccumulate in mammals and is excreted primarily in the bile (Bush et al. 1955).

Copper is known to inhibit photosynthesis and plant growth. Because copper is an essential micronutrient for plant nutrition, most adverse effects result from copper deficiency (Adriano 1986).

4.0 REFERENCES

Adriano D.C. 1986. *Trace elements in the terrestrial environment*. Springer-Verlag. New York.

ATSDR. 1990. *Toxicological Profile for Copper*. Agency for Toxic Substances and Disease Registry. December.

Batsura Y. 1969. "Electron-microscopic investigation of penetration of copper oxide aerosol from the lungs into the blood and internal organs." Bull Exp Biol Med 68:1175-1178.

Bush J, Mahoney J, Markowitz H, Gubler C, Cartwright G, Wintrobe M. 1955. "Studies on copper metabolism. XVI. Radioactive copper studies in normal subjects and in patients with hepatolenticular degeneration." J Clin Invest 34:1766-1778. .

Callahan M, Slimak M, Gabel N, et al. 1979. *Water-Related Environmental Fate of 129 Priority Pollutants.* Vol. 1&2. EPA-440/4-79-029. Office of Water Planning and Standards, Washington, DC. 11-1 to 11-19.

Crampton R, Matthews D, Poisner R. 1965. "Observations on the mechanism of absorption of copper by the small intestine." J Physiol 178:111-126.

EPA. 1985. *Drinking Water Criteria Document for Copper*. Final draft. EPA-600/X-84-190-1. P. VII-1.

Fuhrer G. 1986. "Extractable cadmium, mercury, copper, lead, and zinc in the lower Columbia River estuary, Oregon and Washington." In: *U.S. geological survey water-resources investigations report*. U.S. Department of the Interior. 86:4088.

Harrison F, Bishop D. 1984. *A review of the impact of copper released into freshwater environments*. Prepared for Division of Health, Siting and Waste Management, Office of Nuclear Regulatory Research. U.S. Nuclear Regulatory Commission, Washington, DC. As cited in ATSDR 1990.

Heit M, Klusek C. 1985. "Trace element concentrations in the dorsal muscle of white suckers and brown bullheads from two acidic Adirondack lakes." Water Air Soil Pollut 25:87-96.

HSDB. 1997. Hazardous Substance Data Base.

Marceau N, Aspin N, Sass-Kortsak A. 1970. "Absorption of copper 64 from gastrointestinal tract of the rat." Am J Physiol 218:377-383.

Moffett J, Zika R. 1987. "Photochemistry of copper complexes in sea water." In: Zika R, Copper W, ed. *ACS Symposium Series*, Washington, DC. 327:116-130. As cited in ATSDR 1990.

Perwak J, Bysshe S, Goyer M, et al. 1980. *Exposure and risk assessment for copper*. EPA 400/4-81-015. EPA, Cincinnati, OH. NTIS PB85-211985. As cited in ATSDR 1990.

Sideris E, Sylva C, Charalambous AT, and Katsaros N. 1988. "Mutagenesis; Carcinogenisis and the metal elements - DNA interaction." Prog Clin Biol Res 259:13-25.

Tyler L, McBride M. 1982. "Mobility and extractability of cadmium, copper, nickel, and zinc in organic and mineral soil columns." Soil Sci 134:198-205.

Van Campen D, Mitchell E. 1965. "Absorption of Cu^{64}, Zn^{66}, Mo^{99}, and Fe^{59} from ligated segments of the rat gastrointestinal tract." J Nutr 86:120-124.

CROTONALDEHYDE

1.0 SUMMARY

Crotonaldehyde is a highly volatile, water-soluble, low molecular weight, organic compound. Volatilization is the major fate process for crotonaldehyde in surface water and surface soil. Crotonaldehyde does not bioconcentrate in aquatic organisms and does not accumulate in wildlife. Therefore, food chain transfer is insignificant.

The following summarizes information about the fate of crotonaldehyde in soil, surface water, and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

Crotonaldehyde has a low K_{oc} value, therefore it will not strongly adsorb to soils (Irwin 1988 as cited in ATSDR 1990), and may dissolve in soil water. Crotonaldehyde has a short half-life (Lyman 1982) and it will quickly volatilize from surface soils.

Crotonaldehyde is completely miscible in water and does not dissolve in oils. However, based on its volatilization half-life of about 1 to 2 days (Bowmer et al. 1974; Thomas 1982), crotonaldehyde is expected to quickly volatilize from surface water. The adsorption of crotonaldehyde to suspended solids and sediment is not expected to be significant because of its low K_{oc} value (Lyman 1982).

Aerobic biodegradation may degrade crotonaldehyde at low concentrations in natural water (Bowmer and Higgins 1976; Callahan et al. 1979; Tabak et al. 1981). In addition, data suggest that persistence of crotonaldehyde in aerobic aquatic environments for moderate to long periods of time will not occur (Jacobson and Smith 1990 as cited in ATSDR 1990).

3.0 FATE IN ECOLOGICAL RECEPTORS

Based on its short volatilization half life and low bioconcentration factor (Bysshe 1982; Hansch and Leo 1985), crotonaldehyde will not concentrate in aquatic organisms.

Little information was available on the fate of crontonaldehyde in mammals. Because crotonaldehyde has a low soil adsorption coefficient and strongly volatilizes, inhalation is the primary exposure route for mammals. Studies have indicated that inhaled crotonaldehyde is quickly absorbed by the upper and lower respiratory tracts (Egle 1972). Studies also suggest that absorbed crotonaledhyde is quickly metabolized (Alarcon 1976; Kaye 1973; Patel et al. 1980).

No information was available on the fate of crotonaldehyde in birds or plants.

4.0 REFERENCES

Alarcon R. 1976. "Studies on the in vivo formation of acrolein. 3-hydroxypropylmercapturic acid as an index of cyclophosphamide (nsc-26271) activation." Cancer Treat Rep 60:327-335.

ATSDR. 1990. *Toxicological Profile for Acrolein*. Agency for Toxic Substances and Disease Registry, Atlanta, GA. December.

Bowmer K, Higgins M. 1976. "Some aspects of the persistence and fate of acrolein herbicide in water." Arch Environ Contam Toxicol 5:87-96.

Bowmer K, Lang A, Higgins M, et al. 1974. "Loss of acrolein from water by volatilization and degradation." Weed Res 14:325-328.

Bysshe S. 1982. "Bioconcentration factor in aquatic organisms." In: Lyman W, Reehl W, Rosenblatt D, eds. *Handbook of Chemical Property Estimation Methods*. McGraw-Hill Book Co., New York. pp 5-1 to 5-30. As cited in ATSDR 1990.

Callahan M, Slimak M, Gabel N, et al. 1979. *Water-Related Environmental Fate of 129 Priority Pollutants.* Vol 1 & 2. EPA-440/4-79-029a. USEPA, Washington, DC.

Egle J. 1972. "Retention of inhaled formaldehyde, propionaldehyde, and acrolein in the dog." Arch Environ Health 25:119-124.

Hansch C, Leo A. 1985. *Medchem Project Issue No. 26*, Pomona College, Claremont, CA. As cited in ATSDR 1990.

Irwin K. 1988. *Soil Adsorption Coefficient For Acrolein (Magnicide, H Herbicide And Magnicide, B Microbiocide)*. Prepared by SRI International, Menlo Park, CA, for Baker Performance Chemicals, Houston, TX. SRI Project No. PYU 3562. As cited in ATSDR 1990.

Jacobson B, Smith J. 1990. *Aquatic Dissipation for Acrolein.* Prepared by Analytical Bio-Chemistry Laboratories, Inc., Columbia, MI, for Baker Performance Chemicals, Houston, TX. ABC Final Report No. 37891. As cited in ATSDR 1990.

Kaye C. 1973. "Biosynthesis of mercapturic acids from allyl alcohol, allyl esters, and acrolein." Biochem J 134:1093-1101.

Lyman W. 1982. "Adsorption coefficient for soils and sediments." In: Lyman W, Reehl W, Rosenblatt D, eds. *Handbook of Chemical Property Estimation Methods.* McGraw-Hill Book Co., New York. pp 4-1 to 4-33.

Patel J, Wood J, Leibman K. 1980. "The biotransformation of allyl alcohol and acrolein in rat liver and lung preparations." Drug Metab Dispos 8:305-308.

Tabak H, Quave S, Mashni C, et al. 1981. "Biodegradability studies with organic priority pollutant compounds." J Water Pollut Cont Fed 53:1503-1518.

Thomas R. 1982. "Volatilization from water." In: Lyman W, Reehl W, Rosenblatt D, eds. *Handbook of Chemical Property Estimation Methods.* McGraw-Hill Book Company, New York. pp 15-1 to 15-34.

CUMENE (ISOPROPYLBENZENE)

1.0 SUMMARY

1-methylethylbenzene is also called cumene. Cumene and its superoxidized form, cumene hydroperoxide, are moderately volatile organic compounds. Cumene released to soil and surface water will rapidly dissipate through biodegradation and volatilization. Routes of exposure for cumene and cumene hydroperoxide include inhalation, ingestion, and dermal exposure. However, due to its high potential to volatilize, inhalation is the major exposure route for wildlife receptors. Bioconcentration of cumene is not likely in aquatic organisms. No information was available regarding the environmental fate of cumene hydroperoxide in air, water, or soil. However, degradation in soil and water is expected to be very rapid based on the high reactivity of cumene hydroperoxide with multivalent metal ions and free radicals.

The following is a profile of the fate of cumene and cumene hydroperoxide in soil, surface water and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, surface water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

The primary removal process for cumene in soil is expected to be through biodegradation in surface soil, and volatilization (HSDB 1997). Based on its log K_{oc} value (Lyman 1982), cumene that does not volatilize is expected to strongly adsorb to soil.

The environmental fate of cumene hydroperoxide in soil is unknown. However, based on its high reactivity with multivalent metal ions and free radicals, degradation in soil is expected to be very rapid (HSDB 1997).

In surface water, cumene is expected to have a relatively short half-life. The primary removal processes for cumene when released in water are volatilization and biodegradation (GEMS 1986; HSDB 1997). Based on different water characteristics, volatilization half-lives ranging from a few hours to a few days have been estimated (GEMS 1986). Cumene is amenable to biodegradation (Price et al. 1974; Kappeler and Wuhrmann 1978), and biodegrades in 10 to 30 days (Walker and Colwell 1975; Price et al. 1974).

The environmental fate of cumene hydroperoxide in water is unknown. However, based on its high reactivity with multivalent metal ions and free radicals, degradation in water is expected to be very rapid (HSDB 1997).

3.0 FATE IN ECOLOGICAL RECEPTORS

Cumene is reported to have relatively low bioconcentration in fish (ITC/EPA 1984; Geiger 1986;).

In wildlife, cumene and cumene hydroperoxide enter the body primarily via inhalation and dermal absorption (Lefaux 1968; HSDB 1997). Cumene is readily absorbed in mammalian systems and oxidized (Clayton and Clayton 1982). In the event that cumene is ingested, it is readily metabolized and excreted (Robinson et al. 1955). Long-term exposure by mammals results in cumene distribution to many tissues and organs (Gorban et al. 1978).

4.0 REFERENCES

Clayton G, Clayton F, eds. 1982. *Patty's Industrial Hygiene and Toxicology*. 3rd ed. Vol 2. John Wiley & Sons, New York. pp. 3309-3310.

Geiger. 1986. *Acute Tox Org Chem to Minnows.* Vol III. p.213. As cited in HSDB 1997.

GEMS. 1986. *Graphical Exposure Modeling System. Fate of atmospheric pollution*. EPA, Office of Toxic Substances.

Gorban G, et al. 1978. Gig Sanit 10:113. As cited in HSDB 1997.

HSDB. 1997. Hazardous Substance Data Bank.

ITC/EPA. 1984. *Information review #464*. Draft. Cumene. pp. 10; 23. As cited in HSDB 1997.

Kappeler T, Wuhrmann K. 1978. "Microbial degradation of the water-soluble fraction of gas oil--II. Bioassayw with pure strains." Water Res 12:335-342.

Lefaux. 1968. *Prac tox of plastics*. p. 166. As cited in HSDB 1997.

Lyman W. 1982. "Adsorption coefficient for soils and sediments." In: Lyman W, Reehl W, Rosenblatt D, eds. *Handbook of Chemical Property Estimation Methods.* McGraw Hill Book Co., New York. pp. 4-1 to 4-33.

Price K, Waggy G, Conway R. 1974. "Brine shrimp bioassay and seawater BOD of petrochemicals." J Water Pollut Cont Fed 46:63-77.

Robinson D, Smith J, Williams R. 1955. "Studies in detoxication." Biochem J 59:153-159.

Walker J, Colwell R. 1975. J Gen Appl Microbiol 21:27-39.

DDE

1.0 SUMMARY

Dichlorodiphenyldichloroethane (DDE) is a high molecular weight, chlorinated pesticide. It is also a congener of dichlorodiphenyltrichloroethane (DDT), a full-spectrum pesticide. DDE is stable, accumulates in soil and sediment, and concentrates in fatty tissue. DDE has a low water solubility, and is adsorbed strongly in soils and sediments. Soil and benthic organisms accumulate DDE from soil and sediment. Wildlife will accumulate DDE in fatty tissue. Following chronic exposure by wildlife to DDE, an equilibrium between absorption and excretion may occur; however, concentrations will continue to increase because accumulation is related to fat content, which increases with age.

The following summarizes the fate of DDE in surface soil, surface water, and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, water, and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

DDE absorbs strongly to soil and is only slightly soluble in water. Under normal environmental conditions, DDE does not hydrolyze or biodegrade. In soils with low organic content, evaporation from the surface of soil may be significant (HSDB 1997).

DDE is bioavailable to plants and soil invertebrates despite being highly bound to soil. DDT has been found to accumulate in grain, maize, and rice plants with the majority located in the roots. Mobilization of soil-bound DDT by earthworms to more bioavailable forms has also been reported (Verma and Pillai 1991).

DDE is very persistent in the aquatic environment, has a very low water solubility, and is highly soluble in lipids. Compounds with these characteristics tend to partition to the organic carbon fraction of sediments and lipid fraction of biota (EPA 1986). DDE absorbs very strongly to sediment, and bioconcentrates in aquatic organisms (HSDB 1997). In aquatic environments, the small fraction of dissolved DDE may be photolyzed.

3.0 FATE IN ECOLOGICAL RECEPTORS

In general, DDE will bioconcentrate in lower-trophic-level organisms and will accumulate in food chains. Fish and other aquatic organisms readily take up pesticides, including DDE. Pesticides are taken up by organisms through the gills, by direct contact with the contaminant in the water, or by ingestion of contaminated food, sediment, or water. The lipophilic nature and extremely long half life of DDE result in bioaccumulation when it is present in ambient water. DDE will bioconcentrate in freshwater and marine plankton, insects, mollusks and other invertebrates, and fish (Oliver and Niimi 1985). When these organisms are consumed by other receptors, DDE is transferred up food chains. Following absorption, either through the gills or by ingestion, pesticides appear in the blood and may be distributed to tissues of all soft organs (Nimmo 1985).

DDE is accumulated to high concentrations in fatty tissues of carnivorous receptors. Elimination and absorption of DDE may occur simultaneously once an equilibrium is reached. This equilibrium may be disturbed by high concentrations of DDE, but termination of exposure usually results in elimination of the stored substance. This elimination occurs in two phases—an initial rapid phase followed by a much slower gradual loss (Nimmo 1985).

DDE can be introduced into mammals through oral, dermal, and inhalation exposure. Inhalation absorption is considered minor because the large particle size of DDE precludes entry to the deeper spaces of the lung; DDE is deposited in the upper respiratory tract and, through mucociliary action, is eventually swallowed and absorbed in the gastrointestinal tract. Gastrointestinal absorption following oral exposure has been shown in experimental animals (Hayes 1982). Dermal absorption is limited and the toxic effects are less than those seen following oral exposure. The highest concentration of DDE and metabolites has been found in adipose tissue, followed by reproductive organs, liver, kidneys, and brain (EPA 1980).

The metabolism of DDE in animals is similar to that in humans. DDE metabolism and elimination occurs very slowly. The primary route of elimination is in the urine (Gold and Brunk 1982, 1983, 1984); however, DDE may also be eliminated through the feces, semen, or breast milk. When exposure ceases, DDE is slowly eliminated from the body (Murphy 1986). The biological half-life of DDE is 8 years (NAS 1977).

Bioaccumulation has been reported in one Alaskan study of two raptor species—the Rough-legged hawk and the Peregrine falcon. Higher tissue residues were reported in the peregrine falcon than in the rough-legged hawk. It was believed that these differences may have been due to the different feeding habits of the birds (Matsumura 1985).

No information was available on the fate of DDE taken up by plants.

4.0 REFERENCES

ATSDR. 1994. *Toxicological Profile for p,p'-DDT, p,p'-DDE, and p,p'-DDD.* Agency for Toxic Substances and Disease Registry. April.

EPA. 1980. *Ambient Water Quality Criteria for DDT*. EPA 440/5-80-038. EPA, Office of Water Regulations and Standards, Washington, DC. October. 95 PP.

EPA. 1986. *Superfund Public Health Evaluation Manual*. EPA 540/1-86/000. Office of Emergency and Remedial Response, Washington, DC.

Gold B, Brunk G. 1982. "Metabolism of 1,1,1-trichloro-2,2-bis(p-chlorophenyl)-ethane and 1,1-dichloro-2,2-bis(p-chlorophenyl)ethane in the mouse." Chem-Biol Interact 41:327-339.

Gold B, Brunk G. 1983. "Metabolism of 1,1,-trichloro-2,2-bis(p-chlorophenyl)ethane (DDT), 1,1-dichloro-2,2-bis(p-chlorphenyl)ethane, and 1-chloro-2,2-bis(p-chlorophenyl)ethene in the hamster." Cancer Res 43:2644-2647.

Gold B, Brunk G. 1984. "A mechanistic study of the metabolism of 1,1-dichloro-2,2-bis(p-chloropheny l)ethane (DDD) to 2,2-bis(p-chlorophenyl)acetic acid (DDA)." Biochem Pharmacol 33:979-982.

Hayes W. 1982. "Chlorinated hydrocarbon insecticides." In: *Pesticides Studied in Man*. Williams and Wilkins, Baltimore, MD.. pp. 180-195.

HSDB. 1997. Hazardous Substances Data Bank.

Matsumura F. 1985. *Toxicology of Insecticides.* 2nd ed. Plenum Press, New York.

Murphy S. 1986. "Toxic effects of pesticides." In: Klaassen C, et al., eds. *Casarett and Doull's Toxicology.* 3rd ed. MacMillan Publishing Company, New York. pp 519-580.

NAS. 1977. *Drinking Water and Health.* Safe Drinking Water Committee, National Research Council. National Academy of Sciences, Washington, DC. p. 576.

Nimmo D. 1985. "Pesticides." In: *Fundamentals of Aquatic Toxicology Methods and Applications.* Hemisphere Publishing Corp. pp. 335-373.

Oliver B, Niimi A. 1985. "Bioconcentration factors of some halogenated organics for rainbow trout: Limitations in their use for prediction of environmental residues." Environ Sci Technol 19:842-849.

Verma A, Pillai M. 1991. "Bioavailability of soil-bound residues of DDT and HCH to earthworms." Curr Sci 61(12):840-843. As cited in ATSDR 1994.

DICHLOROFLUOROMETHANE

1.0 SUMMARY

Dichlorofluoromethane (DCFM) is a highly volatile hydrocarbon. It has a high vapor pressure and low soil adsorption coefficient; therefore, volatilization is the main fate process for DCFM released to surface soil and surface water. For terrestrial animals, inhalation is the main exposure route and ingestion is a minor exposure route. DCFM is not expected to bioconcentrate in fish; however, it can accumulate in tissues of mammals. DCFM is not expected to move up food chains.

The following information summarizes the fate of dichlorofluoromethane in soil, surface water and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

If released to soil, DCFM, an inert gas at room temperature, is expected to volatilize into the air due to its low soil adsorption coefficient (K_{oc}) value (Lyman et al. 1982). Because it does not have a strong affinity for organic carbon, it may dissolve in soil pore water, thus becoming bioavailable. Photooxidation, hydrolysis, and biodegradation are not likely to be significant removal processes for DCFM in soil due to its high volatility and minimal reactivity (HSDB 1997).

Based on its high water solubility and low soil adsorption coefficient, DCFM does not adsorb strongly to suspended solids or sediment. Based on a reported half-life of less than 1 day, DCFM is expected to rapidly volatilize from water (Lyman et al. 1982). The hydrolysis of DCFM is reported to be very low (<0.01 g/l of water-yr) (Du Pont de Nemours Co. 1980).

3.0 FATE IN ECOLOGICAL RECEPTORS

DCFM is not expected to bioconcentrate in aquatic organisms, based on its low log K_{ow} value (Hansch and Leo 1985) and low estimated BCF value (Lyman et al. 1982).

Information was not available on the fate of DCFM in mammals, birds, or plants.

4.0 REFERENCES

Du Pont de Nemours Company. 1980. *Freon Product Information B-2.* DuPont de Nemours and Company, Wilmington, DE. As cited in HSDB 1997.

Hansch C, Leo A. 1985. *Medchem Project Issue No. 26,* Pomona College, Claremont, CA. As cited in HSDB 1997.

HSDB. 1997. Hazardous Substance Data Bank.

Lyman W. 1982. "Adsorption coefficient for soils and sediments." In: Lyman W, Reehl W, Rosenblatt D, eds. *Handbook of Chemical Property Estimation Methods.* McGraw-Hill Book Co., New York. pp 4-1 to 4-33.

DICHLOROETHENE, 1,1-

1.0 SUMMARY

1,1-dichloroethene is a hydrophillic, low molecular weight, chlorinated hydrocarbon. It has a short half-life in the environment, thus acute exposures by ecological receptors are the main concern. Evaporation and biodegradation are major fate processes for 1,1-dichloroethene in soil, surface water, and sediment. It will also adsorb to detritus in soils and sediments. Ingestion and respiratory uptake are the significant direct exposure routes for ecological receptors exposed to 1,1-dichloroethene. Metabolic intermediates are responsible for the toxicity of 1,1-dichloroethene to upper trophic level receptors. Indirect (food chain) exposure through ingestion of contaminated food is minor because it is readily biotransformed and excreted. Hence, the biomagnification potential is very low.

The following is a profile of the fate of 1,1-dichloroethene in soil, surface water and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER AND SEDIMENT

If released onto the soil surface, the majority of 1,1-dichloroethene will quickly evaporate. Depending on the hydrogeology of a site, some may leach into ground water. Based on its high water solubility and small K_{oc} value, 1,1-dichloroethene may migrate through soils by adsorbing to dissolved organic carbon (EPA 1982). Studies have also documented that 1,1-dichloroethene will biodegrade in soils (HSDB 1997). A bioaccumulation factor for 1,1-dichloroethene in soil was not reported. However, based on its volatility and polarity, 1,1-dichloroethene is not expected to significantly bioaccumulate in soil (Callahan et al. 1979).

Evaporation is the major fate of 1,1-dichloroethene in surface water, with a short half-life of 1-6 days. Only a small quantity of 1,1-dichloroethene will be lost by adsorption onto the sediment (HSDB 1997). 1,1-dichloroethene also quickly biodegrades in aqueous environments. Degradation studies showed that 45–78% was lost in 7 days, when incubated with a wastewater inoculum. A large amount was also lost due to volatilization (Patterson and Kodukala 1981). In anaerobic environments, 1,1-dichloroethene

degrades (through reductive dechlorination) to vinyl chloride. Anaerobic degredation is slower that aerobic degradation. Approximately 50-80% of 1,1-dichloroethene underwent degradation in 6 months in a simulated groundwater environment (Barrio-Lage et al. 1986; Hallen et al. 1986). Photo-oxidation and hydrolysis are not expected to be significant removal processes for 1,1-dichloroethene (Callahan et al. 1979; Mabey et al. 1981; Cline and Delfino 1987). A bioaccumulation factor for 1,1-dichloroethene in water and sediment was not reported. However, based on its volatility and polarity, 1,1-dichloroethene is not expected to significantly bioaccumulate in water or sediment (Callahan et al. 1979).

3.0 FATE IN ECOLOGICAL RECEPTORS

Aquatic receptors may be directly exposed to dissolved 1,1-dichloroethene through gill respiration or through ingestion of suspended particles. Because 1,1-dichloroethene generally is not persistent in surface water, exposures are expected to be of short duration. 1,1-dichloroethene is not expected to bioconcentrate in fish or aquatic invertebrates, based on its low log K_{ow} value (Tute 1971; HSDB 1997). Due to limited bioconcentration, 1,1-dichloroethene is not expected to biomagnifiy in terrestrial or aquatic food chains (Barrio-Lage et al. 1986; Wilson et al. 1986).

1,1-dichloroethene is readily absorbed following inhalation (Dallas et al. 1983; McKenna et al. 1978a) or oral exposure, and is rapidly distributed in the body. Following inhalation exposure to 1,1-dichloroethene, uptake is dependent upon the duration of the exposure and the dose. Until equilibrium is reached, as exposure concentration increases, the percentage of 1,1-dichloroethene uptake decreases. Studies show that 2 minutes after inhalation exposure, substantial amounts of 1,1-dichloroethene were found in the venous blood of rats. Concentrations of 150 ppm or less of 1,1-dichloroethene showed a linear cumulative uptake. However, at 300 ppm steady state was not achieved, indicating saturation at high concentrations (Dallas et al. 1983).

Following oral administration of 1,1-dichloroethene in corn oil, rapid and almost complete absorption from the gastrointestinal tract of rats and mice was observed (Jones and Hathway 1978a; Putcha et al. 1986). Recovery of radio-labeled 1,1-dichloroethene was 43.55, 53.88, and 42.11%, 72 hours following oral administrations of 0.5, 5.0, and 50 mg/kg, respectively, to rats (Reichert et al. 1979). Also, 14.9-22.6% 1,1 dichloroethene was recovered in expired air, 42.11-53.88% in urine, 7.65-15.74% in feces, 2.77-5.57% in the carcass, and 5.91-9.8% in the cage rinse (Reichert et al. 1979).

1,1-dichloroethene is distributed mainly to the liver and kidneys following inhalation or oral exposure. In rodents, the highest levels of 1,1-dichloroethene are found in the liver and kidneys. Rats that were fasted and exposed to 1,1-dichloroethene showed significantly greater tissue burden than nonfasted rats (McKenna et al. 1978b; Jones and Hathway 1978b).

1,1-dichloroethene does not appear to be stored or accumulated in tissues, but is metabolized by the hepatic microsomal cytochrome P-450 system. This reaction produces reactive intermediates responsible for the toxicity of 1,1-dichloroethene. These reactive intermediates are detoxified through hydroxylation or conjugation with GSH, which is the primary biotransformation pathway in the rat. Excretion of unmetabolized 1,1-dichloroethene is through exhaled air, and metabolites are excreted via urine and exhaled air (Fielder et al. 1985; ATSDR 1994).

Avian receptors may be directly exposed to 1,1-dichloroethene through the ingestion of surface water and soil. Absorption studies specific to avian species were not identified in the literature.

Data on the fate of 1,1-dichloroethene in plant receptors were not identified in the literature. However, based on the low probability of significant bioaccumulation, uptake by plant receptors is expected to be minimal.

4.0 REFERENCES

ATSDR. 1994. *Toxicological Profile for 1,1-Dichloroethene.* Agency for Toxic Substances and Disease Registry.

Barrio-Lage G, Parsons F, Nassar R, Lorenzo P. 1986. "Sequential Dehalogenation of Chlorinated Ethenes." Environ Sci Technol 20:96-99.

Callahan M, Slimak M, Gabel N, et al. 1979. *Water-Related Environmental Fate of 129 Priority Pollutants.* Vol 2. EPA-440/4-79-029b. USEPA, Washington, DC. pp. 50-1 to 50-10.

Cline P, Delfino J. 1987. *Am Chem Soc Div Environ Chem preprint*. New Orleans, LA. 27:577-579. As cited in HSDB 1997.

Dallas C, Weir R, Feldman S, et al. 1983. "The Uptake and Disposition of 1,1-dichloroethene in Rats During Inhalation Exposure." Toxicol Appl Pharmacol 68:140-151.

EPA. 1982. *Aquatic Fate Process Data for Organic Priority Pollutants.* Washington, DC: US Environmental Protection Agency. Code of Federal Regulations 40 CFR 61.65.

Fielder R, Dale E, Williams S. 1985. *Toxicity Review 13: Vinylidene Chloride.* Her Majesty's Stationary Office, London, England. As cited in ATSDR 1994.

Hallen R, et al. 1986. "Am Chem Soc Div Environ Chem, 26th Natl Mtg." 26:344-346. As cited in HSDB 1997.

HSDB 1997. Hazardous Substance Data Base. June 1997.

Jones B, Hathway D. 1978a. "Differences in Metabolism of Vinylidene Chloride Between Mice and Rats." Br J Cancer 37:411-417.

Jones B, Hathway D. 1978b. "The Biological Fate of Vinylidene Chloride in Rats." Chem-Biol Interact 20:27-41.

Mabey W, Smith J, Podoll R, et al. 1981. *Aquatic Fate Process Data for Organic Priority Pollutants.* EPA 440/4-81-014. EPA Office of Water Regulations and Standards, Washington, DC.

McKenna M, Zempel J, Madrid E, et al. 1978a. "Metabolism and Pharmacokinetic Profile of Vinylidene Chloride in Rats Following Oral Administration." Toxicol Appl Pharmacol 45:821-835.

McKenna M, Zempel J, Madrid E, et al. 1978b. "The Pharmacokinetics of [14c]vinylidene Chloride in Rats Following Inhalation Exposure." Toxicol Appl Pharmacol 45:599-610.

Patterson J, Kodukala P. 1981. "Biodegradation of Hazardous Organic Pollutants." Chem Eng Prog 77:48-55.

Putcha L, Bruchner J, D'Soyza R, et al. 1986. "Toxicokinetics and Bioavailability of Oral and Intravenous 1,1-dichloroethene." Fundam Appl Toxicol 6:240-250.

Reichert D, Werner H, Metzler M, et al. 1979. "Molecular Mechanism of 1,1-dichloroethene Toxicity: Excreted Metabolites Reveal Different Pathways of Reactive Intermediates." Arch Toxicol 42:159-169.

Tute M. 1971. Adv Drug Res 6:1-77. As cited in HSDB 1997.

Wilson B, Smith G, Rees J. 1986. "Biotransformations of Selected Alkylbenzenes and Halogenated Aliphatic Hydrocarbons in Methanogenic Acquifer Material; a Microcosm Study." Environ Sci Technol 20:997-1002.

DINITROTOLUENES

1.0 SUMMARY

2,4-dinitrotoluene and 2,6-dinitrotoluene are semi-volatile, nitrogen-substituted, organic compounds. They are moderately persistent in soil and have short half-lives in aqueous environments due to high rates of photolysis. Evidence also indicates that they are biodegraded in soil, surface waters and sediment. For wildlife, all routes of exposure are significant. Dinitrotoluenes are not expected to bioconcentrate in aquatic organisms and bioaccumulation is not expected in animal tissues. The major target organs following exposure to 2,4-dinitrotoluene are the liver and kidney. 2,6-dinitrotoluene is distributed to various organs following uptake. Evidence indicates that upper-trophic-level receptors rapidly metabolize 2,4-dinitrotoluene to innocuous by-products that are readily excreted. 2-6-dinitrotoluene is metabolized to a highly electrophilic ion that is capable of reacting with DNA and other biological nucleophiles.

The following summarizes the fate of 2,4-dinitrotoluene and 2,6-dinitrotoluene in soil, surface water and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

2,4-dinitrotoluene is expected to be slightly mobile in soil, based on its estimated K_{oc} value (Lyman et al 1982; Kenaga 1980). Information on the biodegradation of 2,4-dinitrotoluene in soil was not located; however, biodegradation is thought to occur in both aerobic and anaerobic zones of soil, based on aqueous biodegradation experiments (HSDB 1997).

2,6-dinitrotoluene readily biodegrades when released into the soil. Half-lives of 73 and 92 days were reported, when tested in two soils, with degradation rates of 0.5 to 0.7 mg/kg/day reported (Loehr 1989). Based on the calculated K_{oc} value (Lyman et al. 1982) and the estimated log K_{ow} value (GEMS 1984), 2,6-dinitrotoluene is expected to be slightly mobile in soil (Kenaga 1980).

Volatilization of dinitrotoluenes from surface soil is expected to be negligible due to very low vapor pressures of these compounds (Banerjee et al. 1990). Hydrolysis is not a significant removal process for nitroaromatic hydrocarbons (Lyman et al. 1982).

2,4-dinitrotoluene and 2,6-dinitrotoluene have a slight tendency to sorb to sediments, suspended solids, and biota, based on measured log K_{ow} values (GEMS 1984). In surface water, photolysis is the primary removal process for 2,4-dinitrotoluene and 2,6-dinitrotoluene. Reported half-lives range from a few minutes to a few hours (Spanggord et al. 1980; Zepp et al. 1984). Hydrolysis is not a removal process for nitroaromatics (Lyman et al. 1982).

Dinitrotoluenes do not readily volatilize in surface water. Volatilization half-lives of 2-4 dinitrotoluene from distilled water were 248 and 133 hours, which correspond to the volatilization rate constants of 0.0028 and 0.0052/hour (Smith et al. 1981). Davis et al. (1981), reported a 0.3 percent loss of 2,6-dinitrotoluene in a model waste stabilization pond. Empirical evidence indicates that dinitrotoluenes are expected to biodegrade in surface waters (Uchimura and Kido 1987; Umeda et al. 1985; Kondo et al. 1988; Tabak et al. 1981).

3.0 FATE IN ECOLOGICAL RECEPTORS

Aquatic organisms take up 2,4-dinitrotoluene, however, it does not bioconcentrate because it is readily eliminated. Measured BCF values for dinitrotoluenes are low indicating that bioconcentration does not occur in aquatic organisms (Deneer et al. 1987; EPA 1980).

Evidence indicates that once it is ingested by wildlife, 2,4-dinitrotoluene is rapidly absorbed into the bloodstream (Rickert et al. 1983). 2,4-dinitrotoluene is quickly distributed, with the highest concentrations in the liver and kidney (Rickert and Long 1981). The metabolism of 2,4-dinitrotoluene occurs in the liver and the intestine (via intestinal microflora), and it is quickly eliminated through the urine and feces (Lee et al. 1978; Long and Rickert 1982; Rickert and Long 1981; Schut et al. 1983). Based on the low log P value for 2,4-dinitrotoluene, bioaccumulation in animal tissues is not expected (Callahan et al. 1979; Mabey et al. 1981).

Dinitrotoluenes are expected to be readily taken up by plants, based on structural analogies with 1,3-dinitrobenzene and p-nitrotoluene (McFarlane et al. 1987; Nolt 1988).

4.0 REFERENCES

ATSDR. 1989. *Toxicological Profile for 2,4-Dinitrotoluene, 2,6-Dinitrotoluene.* Agency for Toxicological Substances and Disease Registry.

Banerjee S, et al. 1990. Chemosphere 21:1173-1180. As cited in HSDB 1997.

Callahan M, Slimak M, Gabel N, et al. 1979. *Water-Related Environmental Fate of 129 Priority Pollutants.* Vol 2. EPA-440/4-79-029b. USEPA, Washington, DC.. PP. 81-1 TO 82-8.

Davis E, et al. 1981. Water Res 15:1125-1127. As cited in HSDB 1997.

Deneer J, et al. 1987. Aquatic Toxicol 10:115-129. As cited in HSDB 1997.

EPA. 1980. *Ambient Water Quality Criteria for Dinitrotoluene.* EPA 440/5-80-045. Office of Water Regulations and Standards, Washington, DC. P. C-6.

GEMS. 1984. *Graphical Exposure Modeling System.* CLOGP3. Office of Toxic Substances. As cited in HSDB 1997.

HSDB. 1997. Hazardous Substances Data Bank.

Kenaga E. 1980. "Predicted bioconcentration factors and soil sorption coefficients of pesticides and other chemicals." Ecotoxicol Environ Safety 4:26-38. As cited in HSDB 1997.

Kondo M, et al. 1988. Eisei Kagaku 34:115-122. As cited in HSDB 1997.

Lee C, Ellis H, Kowalski J, et al. 1978. *Mammalian Toxicity of Munitions Compounds. Phase II. Effects of Multiple Doses. Part II: 2,4-Dinitrotoluene.* DAMD 17-74-c-4073. Midwest Research Institute, Kansas City, MO. As cited in ATSDR 1989.

Loehr R. 1989. *Treatability Potential for EPA Listed Hazardous Wastes in Soil.* EPA 600/2-89-011. Robert S. Kerr Environ Res Lab, Ada, OK. As cited in HSDB 1997.

Long L, Rickert D. 1982. "Metabolism and Excretion of 2,6-dinitro-[14c]toluene in Vivo and in Isolated Perfused Rat Livers." Drug Metab Dispos 10:455-458. As cited in ATSDR 1989.

Lyman W, Reehl W, Rosenblatt D, eds. 1982. *Handbook of Chemical Property Estimation Methods.* McGraw-Hill, New York.

Mabey W, Smith J, Podoll R, et al. 1982. *Aquatic Fate Process Data for Organic Priority Pollutants.* EPA 440/4-81-014. EPA Office of Water Regulations and Standards, Washington, DC.

McFarlane C, Nolt C, Wickliff C, et al. 1987. "The uptake, distribution and metabolism of four organic chemicals by soybean plants and barley roots." Environ Toxicol Chem 6:874-856. As cited in ATSDR 1989.

Nolt C. 1988. *Uptake and Translocation of Six Organic Chemicals in a Newly-Designed Plant Exposure System and Evaluation of Plant Uptake Aspects of the Prebiologic Screen for Ecotoxicologic Effects*. Master's Thesis. Cornell Univ., Ithaca, NY. As cited in ATSDR 1989.

Rickert D, Long R. 1981. "Metabolism and excretion of 2,4-dinitrotoluene in male and female Fischer-344 rats after different doses." Drug Metab Dispos 9(3):226-232. As cited in ATSDR 1989.

Rickert D, Schnell S, Long R. 1983. "Hepatic macromolecular covalent binding and intestinal disposition of 2,4-(14C)dinitrotoluene." J Toxicol Environ Health 11:555-568. As cited in ATSDR 1989.

Schut H, et al. 1983. J Toxicol Environ Health 12(4-6):659-670. As cited in ATSDR 1989.

Smith J, et al. 1981. Chemosphere 10:281-289. As cited in HSDB 1997.

Spanggord R, et al. 1980. *Environmental Fate Studies on Certain Munitions Wastewater Constituents.* NTIS AD A099256.

Tabak H, Quave S, Mashni C, et al. 1981. "Biodegradability studies with organic priority pollutant compounds." J Water Pollut Cont Fed 53:1503-1518. .

Uchimura Y, Kido K. 1987. Kogai to Taisaku 23:1379-1384. As cited in HSDB 1997.

Umeda H, et al. 1985. Hyogo-Ken Kogai Kenkysho Kenkyu Hokoku 17:76-82.

Zepp R, et al. 1984. "Dynamics of pollutant photoreactions in the hydrosphere." Fresenius Z Anal Chem 319:119-125.

DI(N)OCTYLPHTHALATE

1.0 SUMMARY

Di(n)octylphthalate (DOP) is a high-molecular-weight, semi-volatile compound. It has a low water solubility and low vapor pressure, therefore it adsorbs strongly to the soil and sediment. Biodegradation is possible under aerobic conditions, but is slow under anaerobic conditions. DOP also undergoes hydrolysis in water. DOP may be absorbed following oral (dietary), inhalation, or dermal exposures, however dietary exposure is the most significant route of exposure. DOP may accumulate to increasing concentrations in algae, aquatic invertebrates, and fish, and accumulate to low levels in terrestrial wildlife. However, higher-trophic-level receptors will quickly metabolize it, therefore it does not biomagnify in food chains.

The following is a profile of the fate of DOP in soil, surface water and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER AND SEDIMENT

DOP has a very high K_{oc} value; therefore, it should adsorb strongly and remain immobile in soil (Wolf et al. 1980). Degradation in soil is slow, especially under anaerobic conditions (HSDB 1997).

Following release into aquatic environments, DOP adsorbs strongly to sediments and particulate material suspended in the water column (HSDB 1997). DOP has a moderate half-life in aquatic environments; losses are due to both volatilization and microbial degradation. Slow degradation is possible in aerobic conditions; however, DOP is resistant to anaerobic degradation (HSDB 1997). Approximately 50% degradation was observed within 5 days in a model terrestrial-aquatic ecosystem, with the monoester and phthalic acids the primary degradation products (Sanborn et al. 1975). DOP may bioconcentrate in aquatic organisms (Sanborn et al. 1975).

3.0 FATE IN ECOLOGICAL RECEPTORS

Sanborn et al. (1975) evaluated the bioconcentration and trophic transfer of DOP in model aquatic ecosystems containing phytoplankton, zooplankton, snails, insects, and fish. Evidence showed that the algae and invertebrates bioconcentrated DOP. Fish accumulated DOP to low levels, indicating that these receptors readily eliminate DOP.

DOP may be absorbed following oral, inhalation or dermal exposures (EPA 1980a); however, due to low volatility of DOP, inhalation is not a significant route of exposure (Meditext 1997). Following absorption, DOP is rapidly distributed with the highest amounts concentrated in the liver, kidney and bile (EPA 1980b). DOP is rapidly metabolized to water-soluble derivatives (Gosselin et al. 1984) prior to and after absorption (EPA 1980b). These metabolites are then excreted through the urine and the bile (Ikeda et al. 1978).

No information was available on the fate of DOP in birds or plants.

4.0 REFERENCES

EPA. 1980a. *Ambient Water Quality Criteria Document for Phthalate Esters.* EPA 440/5-80-067. Office of Water Regulations and Standards, Washington, DC. pp. B-8; C-12. As cited in HSDB 1997.

EPA. 1980b. *Atlas Document for Phthalate Esters.* EPA/ECAO. XI-2; XI-5; XI-21. As cited in HSDB 1997.

Gosselin R, Smith R, Hodge H. 1984. *Clinical Toxicology of Commercial Products.* Vol II. 5th ed. Williams and Wilkins, Baltimore, MD. p. 204. As cited in ATSDR 1993, Meditext 1997, and ATSDR 1993.

HSDB. 1997. Hazardous Substances Data Bank.

Ikeda G, Sapienza P, Couvillion J, Farber T, Smith C, Inskeep P, Marks E, Cerra F, van Loon E. 1978. "Distribution and excretion of two phthalate esters in rats, dogs and miniature pigs." Fd Cosmet Toxicol 16:409-413. As cited in HSDB 1997.

Meditext. 1997. Medical Management Data Base.

Sanborn J, Metcalf R, Yu C-C, Lu P-Y. 1975. "Plasticizers in the environment: The fate of di-n-octyl phthalate (DOP) in two model ecosystems and uptake and metabolism of DOP by aquatic organisms." Arch Environ Contam Toxicol 3:244-255.

Wolfe N, Burns L, Steen W. 1980. "Use of linear free energy relationships and an evaluative model to assess the fate and transport of phthalate esters in the aquatic environment." Chemosphere 9:393-02.

DIOXANE, 1,4-

1.0 SUMMARY

1,4-dioxane is a highly water-soluble, moderately volatile organic compound. In soil, surface water, and sediment environments, 1,4-dioxane is not persistent because it is volatile and because it has a low affinity for adsorption to organic carbon. It has a low potential to bioconcentrate in aquatic receptors. Wildlife can be exposed to 1,4-dioxane through ingestion, inhalation, and dermal contact. It does not bioaccumulate in food chains.

The following is a profile of the fate of 1,4-dioxane in soil, surface water and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER AND SEDIMENT

Based on an estimated log K_{oc} value (Lyman et al. 1982), 1,4-dioxane is expected to have a low affinity for organic carbon in soil, thus having a high potential to leach out of surface soils (HSDB 1997). This reduces the exposure potential for vegetation (through root uptake) and soil invertebrates. In addition, because of its moderate vapor pressure, volatilization is expected to be a significant fate process in soil (Verschueren 1983). Based on the volatility of 1,4-dioxane, biaccumulation is not considered to be a significant fate process in soil.

1,4-dioxane is infinitely soluble in water (Lange 1967). However, because 1,4-dioxane has a moderate vapor pressure at 25°C, volatilazation from water is a significant removal process (Verschueren 1983; HSDB 1997). 1,4-dioxane is not expected to adsorb to suspended sediments or detritus due to the estimated K_{oc} value (HSDB 1997). Based on its high volatility in water and low absorption to sediments, bioaccumulation is not expected to be a significant fate process for 1,4-dioxane in water and sediment.

3.0 FATE IN ECOLOGICAL RECEPTORS

Because it is highly soluble in water, aquatic receptors can take up 1,4-dioxane through direct exposure, however, it is not expected to bioconcentrate based on its low K_{ow} value (Hansch and Leo 1985).

Information suggests that 1,4-dioxane has a low potential to be biodegraded in aerobic aquatic environments. Biodegradation experiments with activated sludge showed a negligible biochemical oxygen demand for 1,4-dioxane, therefore, classifying 1,4-dioxane as relatively undegradable (Mills 1954; Alexander 1973; Heukelekian and Rand 1955; Fincher and Payne 1962; Lyman et al. 1982; Kawasaki 1980).

No information was available on the fate of 1,4-dioxane after uptake by aquatic receptors. However, its low bioconcentration factor suggests that 1,4-dioxane is readily eliminated after uptake (Hansch 1985).

The metabolism of 1,4-dioxane in rats has been studied, and information indicates that at high daily doses, 1,4-dioxane can induce its own metabolism. There is an apparent threshold of toxic effects of 1,4-dioxane that coincides with saturation of the metabolic pathway for its detoxification (Young et al. 1978). 1,4-dioxane is highly toxic via all routes of exposure (OHM/TADS 1997), and is readily absorbed through intact skin (Gosselin 1984). Once 1,4-dioxane enters the body, it is distributed throughout the tissues, including the liver, kidney, spleen, lung, colon, and skeletal muscle (Woo et al. 1977). The excretion of 1,4-dioxane is primarily through the urine, in which approximately 85% of excreted material is in the form of beta-hydroxyethoxyacetic acid, a metabolic byproduct. The remaining material is excreted as unchanged dioxane (Braun & Young 1977).

Information was not available on the fate of 1,4-dioxane in birds or plants.

4.0 REFERENCES

Alexander M. 1973. "Nonbiodegradable and Other Recalcitrant Molecules." Biotechnol Bioeng 15:611-647.

Braun W, Young J. 1977. "Identification of B-hydroxyethoxyacetic Acid as the Major Urinary Metabolite of 1,4-dioxane in the Rat." Toxicol Appl Pharmacol 39:33-38.

Fincher E, Payne W. 1962. "Bacterial Utilization of Ether Glycols." Appl Microbiol 10:542-547.

Gosselin R, Smith R, Hodge H. 1984. *Clinical Toxicology of Commercial Products.* 5th ed. Vol II. Williams and Wilkins, Baltimore, MD. p. 408.

Hansch C, Leo A. 1985. *Medchem Project Issue No. 26*, Pomona College, Claremont, CA. As cited in ATSDR 1990.

Heukelekian H, Rand M. 1955. "Biochemical Oxygen Demand of Pure Organic Compounds." J Water Pollut Contr Assoc 27:1040-1053.

HSDB. 1997. Hazardous Substances Data Bank. June 1997.

Kawasaki M. 1980. "Experiences with the Test Scheme under the Chemical Control Law of Japan: an Approach to Structure-activity Correlations." Ecotox Environ Safety 4:444-454.

Lange N. 1967. *Handbook of Chemistry*. 10th ed. McGraw-Hill, New York. p. 523.

Lyman W, Reehl W, Rosenblatt D, eds.. 1982. *Handbook of Chemical Property Estimation Methods.* McGraw-Hill, New York. pp. 7-4; 9-64.

Mills E, Stack V. 1954. Proceedings 8th Ind Waste Conf Ext Ser. 83:492-517.

OHM/TADS. 1997. Oil and Hazardous Materials/Technical Assistance Data System. June 1997.

Verschueren K. 1983. *Handbook of Environmental Data on Organic Chemicals.* 2nd ed. Van Nostrand Reinhold, New York. pp. 578-580.

Woo Y-T, Argus M, Arcos J. 1977. "Tissue and Subcellular Distribution of 3h-dioxane in the Rat and Apparent Lack of Microsome-catalyzed Covalent Binding in the Target Tissue." Life Sci 21(10):1447-1456.

Young J, Braun w, Gehring P. 1978. "Dose-dependent Fate of 1,4-dioxane in Rats." J Toxicol Enivorn Health 4(5-6):709-726.

DIBENZO-*p*-DIOXINS

1.0 SUMMARY

Dibenzo-p-dioxins (dioxins) are a group of high molecular weight chlorinated compounds that are highly soluble in fatty tissues. The congener tetrachlorodibenzodioxin (TCDD) is commonly used as a surrogate for estimating the fate of dioxins in the environment and in ecological receptors. Dioxins have low water solubilities and adsorb strongly to organic carbon in sediment and soil. Dioxins bioaccumulate in aquatic organisms and wildlife, and biomagnify in food chains because of their affinity for lipids. Biomagnification of TCDD appears to be significant between fish and fish-eating birds, but not between fish and their food (other fish).

The following is a profile of the fate of dioxins in soil, surface water, and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, water, and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

TCDD adsorbs strongly to soils (HSDB 1997). TCDD in soil may be susceptible to photodegradation. Volatilization from soil surfaces during warm months may be a major mechanism by which TCDD is removed from soil. Various biological screening studies have demonstrated that TCDD is generally resistant to biodegradation. The half-life of TCDD in surface soil varies from less than 1 year to 3 years. Half-lives in deeper soils may be as long as 12 years (EPA 1993).

TCDD is very persistent in the aquatic environment, has a very low aqueous solubility, and is highly soluble in lipids. Aquatic sediments are an important reservoir for dioxins, and may be the ultimate environmental sink for all global releases of TCDD (HSDB 1997). TCDD may be removed from water through either photolysis or volatilization. The photolysis half-life at surface level has been estimated to range from 21 hours in summer to 118 hours in winter (HSDB 1997). These rates increase significantly with increasing water depths. Therefore, many bottom sediments may not be susceptible to significant photodegradation. The volatilization half-life from the water column of an environmental pond has been estimated to be 46 days, and may be as high as 50 years if adjusted for the effects of sediment adsorption.

Various biological screening studies have demonstrated that TCDD is generally resistant to biodegradation. The persistent half-life of TCDD in lakes has been estimated to be in excess of 1.5 years (HSDB 1997).

3.0 FATE IN ECOLOGICAL RECEPTORS

Ecological exposures to TCDD can occur via ingestion of contaminated soils, water, and sediment, dermal exposure to soil and water, and to a much lesser extent via inhalation of airborne vapors and particulates. It should be noted that, unlike toxicokinetic and toxicodynamic studies where exposures are closely controlled, environmental exposure to dioxin occurs as a complex mixture of congeners, including TCDD. It is generally understood that persistent, lipophilic compounds accumulate in fish in proportion to the lipid content and age of each animal (Gutenmann et al. 1992). Also, it has been demonstrated that the influence of biotransformation on bioaccumulation increases as a function of the K_{ow} of the compound (de Wolf et al. 1992). The dependence of metabolic rate on TCDD dose and length of exposure is not well understood, but time-course studies of P-450 induction in rainbow trout by β-napthoflavone demonstrate that different toxicity responses can occur over time depending on the frequency and duration of exposure (Zhang et al. 1990).

Dioxins readily bioconcentrate in aquatic organisms (Branson et al. 1985; Mehrle et al. 1988; Cook et al. 1991; and Schmieder et al. 1992). Evidence indicates that dioxins will distribute in fish tissues in proportion to the total lipid content of the tissues (Cook et al 1993). Dioxins are metabolized and eliminated very slowly from fish (Kleeman et al. 1986a,b; Opperhuizen and Sijm 1990; Kuehl et al. 1987).

Several studies in a wide range of mammalian and aquatic species indicate that TCDD is metabolized to more polar metabolites (Ramsey et al. 1979; Poiger and Schlatter 1979; Olson et al. 1980; Olson 1986; Poiger et al. 1982; Sijm et al. 1990; Kleeman et al. 1986a,b, 1988; Gasiewicz et al. 1983; Ramsey et al. 1982). The metabolism of TCDD and related compounds is required for urinary and biliary elimination and plays an important role in regulating the rate of excretion of these compounds.

Dioxins are transferred through food chains, biomagnifying in upper-trophic-level receptors, especially birds. Biomagnification of TCDD appears to be significant between fish and fish-eating birds but not between fish and their food (Carey et al. 1990). The lack of apparent biomagnification between fish and their prey is probably due to the influence of biotransformation of TCDD by the fish. Limited data for the

base of the Lake Ontario lake trout food chain indicates little or no biomagnification between zooplankton and forage fish (Whittle et al. 1992). BMFs based on fish consuming invertebrate species probably are close to 1.0 because of the TCDD biotransformation by forage fish.

Oral absorption of dioxin related compounds in laboratory animals has been reported to be contingent on species, test compound, administered dose, and vehicle. Typical oral absorption values range from 50 to 90 percent (EPA 1994). Because TCDD in the environment is likely to be adsorbed strongly to soil, the oral bioavailability of TCDD varies significantly from laboratory values. Studies have shown that oral bioavailability of TCDD in soil is lower by as much as 50 percent as compared to oral bioavailability of TCDD administered in corn oil over a 500-fold dose range (EPA 1994). Moreover, oral bioavailability of TCDD may be significantly lower in different soil types, with values as low as 0.5 percent bioavailability reported (Umbreit et al. 1986 a,b).

Dermal absorption of TCDD has been studied extensively in laboratory animals. Dermal absorption has been demonstrated to depend on applied dose, with lower relative absorption (percentage of administered dose) decreasing at higher doses (Brewster et al. 1989). Dermal absorption rates in laboratory rats ranged from 17 to 40 percent of administered dose (Brewster et al. 1989). Percent bioavailability of TCDD following dermal absorption is significantly lower than bioavailability following oral absorption by as much as 60 percent (Poiger and Schlatter 1980). As with oral absorption of TCDD in soil, percent bioavailability following dermal exposure to TCDD in soil was significantly lower than percent bioavailability following an equivalent oral dose (approximately 1 percent of an administered dose) (Shu et al. 1988).

Transpulmonary absorption of TCDD has been studied in laboratory animals following intratracheal instillation of the compound in various vehicles (Nessel et al. 1990, 1992). Systemic effects characteristic of TCDD exposures, including hepatic microsomal cytochrome p-450 induction, were observed after inhalation exposures, indicating that transpulmonary absorption does occur and that inhalation may be an important route of TCDD exposure. Transpulmonary bioavailability was estimated at approximately 92 percent of administered dose, very similar to that observed after oral exposures (Diliberto et al. 1992). It should be noted that in an environmental setting, inhalation exposures to TCDD in fly ash, dust and soil particulates may be associated with very different absorption and bioavailability patterns.

Tissue distribution studies in laboratory rats and mice indicate that TCDD is distributed preferentially to adipose tissue and liver (EPA 1994). TCDD is distributed to other organs as well, but to a lesser extent. Also, tissue distribution of TCDD has been demonstrated to be time and dose-dependent, with increasing levels of TCDD distributing to adipose and liver associated with higher doses and increased latency period from time of dosage (EPA 1994).

Plants will take up TCDD through root uptake from soil and through foliar uptake from air (EPA 1994). No other information was available on the fate of dioxins after uptake by plants.

No information was available on the fate of dioxins in birds.

4.0 REFERENCES

Branson D, Takahashi I, Parker W, Blau G. 1985. "Bioconcentration of 2,3,7,8-tetrachlorodibenzo-p-dioxin in rainbow trout." Environ Toxicol Chem 4:779-788.

Brewster D, Banks Y, Clark A-M, Birnbaum L. 1989. "Comparative dermal absorption of 2,3,7,8-tetrachlorodibenzo-p-dioxin and three polychlorinated dibenzofurans." Toxicol Appl Pharmacol 97:156-166.

Carey A, Shifrin N, Cook P. 1990. "Derivation of a Lake Ontario bioaccumulation factor for 2,3,7,8-TCDD." In: *Lake Ontario TCDD bioaccumulation study, final report, Chapter 9.* EPA, Region II, New York. As cited in EPA 1993.

Cook et al. 1991. As cited in EPA 1993. *Interim Report on Data and Methods for Assessment of 2,3,7,8-Tetracholorodibenzo-p-dioxin Risks to Aquatic Life and Associated Wildlife.* EPA 600/R-93/055. Office of Research and Development, Washington, DC.

Cook P, Nichols J, Berini C, Libal J. 1993. *Disposition of 2,3,7,8-tetrachlorodibenzo-p-dioxin and co-planar chlorinated biphenyls in tissue of male lake trout following ingestion of food.* EPA, Duluth, MN. As cited in EPA 1993.

de Wolf W, de Bruijn J, Seinen W, Hermens J. 1992. "Influence of biotransformation on the relationship between bioconcentration factors and octanol-water partition coefficients." Environ Sci Technol 26:1197-1201.

Diliberto J, Jackson J, Birnbaum L. 1992. "Disposition and absorption of intratracheal, oral, and intravenous 3H-TCDD in male Fischer rats." Toxicologist 12:79.

EPA. 1993. *Interim report on data and methods for assessment of 2,3,7,8-tetrachlorodibenzo-p-dioxin risks to aquatic life and associated wildlife.* EPA 600/r-93/055. Office of Research and Development, Washington, DC.

EPA. 1994. *Health Assessment Document for 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and related compounds*. EPA/600/bp-92/001. Office of Research and Development, Washington, DC.

Gasiewicz T, Olson J, Geiger L, Neal R. 1983. "Absorption, distribution and metabolism of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in experimental animals." In: Tucker R, Young A, Gray A, eds. *Human and Environmental Risks of Chlorinated Dioxins and Related Compounds.* Plenum Press, New York pp. 495-525. As cited in EPA 1994.

Gutenmann W, Ebel J, Kuntz H, Yourstone K, Lisk D. 1992. "Residues of p,p'-DDE and mercury in lake trout as a function of age." Arch Environ Contam Toxicol 22:452-455.

HSDB. 1997. Hazardous Substance Data Bank.

Kleeman J, Olson J, Chen S, Peterson R. 1986a. "2,3,7,8-tetrachlorodibenzo-p-dioxin metabolism and disposition in yellow perch." Toxicol Appl Pharmacol 83:401-411.

Kleeman J, Olson J, Chen S, Peterson R. 1986b. "Metabolism and disposition of 2,3,7,8-tetrachlorodibenzo-p-dioxin in rainbow trout." Toxicol Appl Pharmacol 83:391-401.

Kleeman J, Olson J, Peterson R. 1988. "Species differences in 2,3,7,8-tetrachlorodibenzo-p-dioxin toxicity and biotransformation in fish." Fundam Appl Toxicol 10:206-213.

Kuehl D, Cook P, Batterman A, Lothenbach D, Butterworth B. 1987. "Bioavailability of polychlorinated dibenzo-p-dioxins and dibenzofurans from contaminated Wisconsin river sediment to carp." Chemosphere 16(4):667-679.

Mehrle P, Buckler D, Little E, et al. 1988. "Toxicity and bioconcentration of 2,3,7,8-tetrachlorodibenzodioxin and 2,3,7,8-tetrachlorodibenzofuran in rainbow trout." Environ Toxicol Chem 7:47-62.

Nessel C, Amoruso M, Umbreit T, Gallo M. 1990. "Hepatic aryl hydrocarbon hydroxylase and cytochrome P450 induction following the transpulmonary absorption of TCDD from intratracheally instilled particles." Fundam Appl Toxicol 15:500-509.

Nessel C, Amoruso M, Umbreit T, Meeker R, Gallo M. 1992. "Pulmonary bioavailability and fine particle enrichment of 2,3,7,8-tetrachlorodibenzo-p-dioxin in respirable soil particles." Fundam Appl Toxicol 19:279-285.

Olson J. 1986. "Metabolism and disposition of 2,3,7,8-tetrachlorodibenzo-p-dioxin in guinea pigs." Toxicol Appl Pharmacol 85:263-273.

Olson J, Gasiewicz T, Neal R. 1980. "Tissue distribution, excretion, and metabolism of 2,3,7,8-tetrachlorodibenz o-p-dioxin (TCDD) in the golden syrian hamster." Toxicol Appl Pharmacol 56:78-85.

Opperhuizen A, Sijm D. 1990. "Bioaccumulation and biotransformation of polychlorinated dibenzo-p-dioxins and dibenzofurans in fish." Environ Toxicol Chem 9:175-186.

Poiger H, Buser H-R, Weber H, Zweifel U, Schlatter C. 1982. "Structure elucidation of mammalian TCDD-metabolites." Experientia 38:484-486.

Poiger H, Schlatter C. 1979. "Biological degradation of TCDD in rats." Nature 281:706-707.

Poiger H, Schlatter C. 1980. "Influence of solvents and adsorbents on dermal and intestinal absorption of TCDD." FD Cosmet Toxicol 18:477-481.

Ramsey J, Hefner J, Karbowski R, Braun W, Gehring P. 1979. "The in vivo biotransformation of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in the rat." Toxicol Appl Pharmacol 42:A162.

Ramsey J, Hefner J, Karbowski R, Braun W, Gehring P. 1982. "The in vivo biotransformation of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in the rat." Toxicol Appl Pharmacol 65:180-194.

Schmieder P, Lothenbach D, Johnson R, Erickson R, Tietge J. 1992. "Uptake and elimination kinetics of 3H-TCDD in medaka." Toxicologist 12:138. As cited in EPA 1993.

Shu H, Teitelbaum P, Webb A, et al. 1988. "Bioavailability of soil-bound TCDD: Dermal bioavailability in the rat." Fund Appl Toxicol 10:335-343.

Sijm D, Tarechewski A, Muir D, Webster G, Seinen W, Opperhuizen A. 1990. "Biotransformation and tissue distribution of 1,2,3,7-tetrachlorodibenzo-p-dioxin, 1,2,3,4,7-pentachlorodibenzo-p-dioxin, 2,3,4,7,8-pentachlorodibenzofuran in rainbow trout." Chemosphere 21(7):845-866.

Umbreit T, Hesse E, Gallo M. 1986a. "Bioavailability of dioxin in soil from a 2,4,5-T manufacturing site." Science 232:497-499.

Umbreit T, Hesse E, Gallo M. 1986b. "Comparative toxicity of TCDD contaminated soil from Times Beach, Missouri, and Newark, New Jersey." Chemosphere 15:2121-2124.

Whittle D, Sergent D, Huestis S, Hyatt W. 1992. "Foodchain accumulation of PCDD and PCDF isomers in the Great Lakes aquatic community." Chemosphere 25:181-184.

Zhang Y, Anderson T, Forlin L. 1990. "Induction of hepatic xenobiotic biotransformation enzymes in rainbow trout by beta-napthoflavone." Time-course studies. Comp Biochem Physiol 95B:247-253.

DIBENZOFURANS

1.0 SUMMARY

Polychlorinated dibenzofurans (PCDF) are a class of hydrophobic chlorinated compounds that adsorb strongly to soils and sediments. Like dioxins, PCDFs are persistent in the environment, bioconcentrate in aquatic organisms, and biomagnify in some food chains. Because PCDFs are associated with organic material in abiotic media, direct contact by soil and sediment receptors, and ingestion by bottom-feeding fish and upper trophic level wildlife, are the most important exposure routes.

Since PCDFs are structurally similar to, and behave in the environment like dioxins, fate of PCDFs is inferred from information about dioxins. Most of the description on the fate of PCDFs is based on the behavior of tetrachlorodibenzofuran (TCDF), one of the most toxic PCDF congeners. The following is a profile of the fate of polychlorinated dibenzofurans (PCDFs) in soil, water, and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, surface water, and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

TCDF adsorbs strongly to soils. Based upon its high K_{oc} value, TCDF is expected to sorb very strongly in soil and not be susceptible to leaching under most soil conditions. No data are available regarding the biological degradation of TCDF in soil (HSDB 1997).

TCDF in the water column can be expected to partition strongly to sediment and suspended particulate matter. Volatilization from the water column can be important, however the significance of this fate process is limited by strong sorption to sediments (HSDB 1997). Bioconcentration in aquatic organisms may be significant. Aquatic hydrolysis is not expected to be important. Data on biodegradation of TCDF are unavailable (HSDB 1997).

3.0 FATE IN ECOLOGICAL RECEPTORS

Based on high Kow values, PCDFs are expected to accumulate in aquatic receptors (Gutenmann et al. 1992).

Based on its similar structure to dioxins, PCDFs are expected to accumulate to high concentrations in aquatic and semi-aquatic mammals and in fish-eating birds.

Information was not available on the disposition of PCDFs in plants.

4.0 REFERENCES

Gutenmann W, Ebel J, Kuntz H, Yourstone K, Lisk D. 1992. "Residues of p,p'-DDE and mercury in lake trout as a function of age." Arch Environ Contam Toxicol 22:452-455.

HSDB. 1997. Hazardous Substance Data Bank.

HEXACHLOROBENZENE

1.0 SUMMARY

Hexachlorobenzene (HCB) is a persistent chemical that adsorbs strongly to soil and sediment. It is relatively stable in the environment and is resistant to hydrolysis, photolysis, and oxidation, with relatively no metabolism by microorganisms. Due to its high affinity for organic carbon, HCB will accumulate in sediments. Soil invertebrates and benthic invertebrates will take up HCB directly from these media. For higher-trophic-level receptors, indirect (food chain) exposure is anticipated to be the most significant pathway because HCB is resistant to metabolism and is very soluble in fat. The major toxic effect that has been observed across all species tested is porphyria.

The following is a profile of the fate of HCB in soil, surface water and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

Due to a long half-live in soil and its strong affinity for organic carbon, HCB released to soil is likely to remain there for extended periods of time (Beck and Hansen 1974). Minimal biodegradation occurs, depending on the organic carbon content of the soil. Some evaporation from surface soil to air may occur, again depending on the organic carbon content of the soil (Gile and Gillett 1979).

Once released to water, HCB will either evaporate rapidly or adsorb to sediments, with very little dissolved in water (HSDB 1997; Kelly et al. 1991). Limited degradation of HCB is expected, since it appears to be stable to hydrolysis, photolysis, and oxidation (Callahan et al. 1979). Since HCB adsorbs strongly to sediments, it may build up in bottom sediments.

3.0 FATE IN ECOLOGICAL RECEPTORS

Aquatic organisms may be exposed to HCB through ingestion of contaminated water, soil, sediment, or food. Empirical information indicates that HCB bioconcentrates in fish and invertebrates (Giam et al.

1980; Konemann and Vanleeuwen 1980; Veith et al. 1979; Oliver and Niimi 1983; Parrish et al. 1978; Kosian et al. 1978; Neely et al. 1974; Zitko and Hutzinger 1976; Laseter et al. 1976).

HCB can be transferred through aquatic food chains. Knezovich and Harrison (1988) reported that chironomid larvae, a common food item of young fish and other aquatic receptors, rapidly bioaccumulate HCB and other chlorobenzenes from contaminated sediments, achieving steady state within 48 hours. Information was not available about metabolism of HCB by fish.

Ingestion of contaminated media and food is the main route of mammalian exposure to HCB (HSDB 1997; ATSDR 1994; Edwards et al. 1991). Following ingestion, HCB is readily absorbed and is distributed through the lymphatic system to all tissues. It accumulates in fatty tissues and persists for many years since it is highly lipophilic and is very slowly metabolized (Weisenberg 1986; Mathews 1986).

HCB is slowly metabolized by the hepatic cytochrome P-450 system, conjugated with glutathione, or reductively dechlorinated (ATSDR 1994). The metabolites of HCB in laboratory animals include pentachlorophenol, pentachlorobenzene, tetrachlorobenzene, traces of trichlorophenol, a number of sulfur containing compounds, and some unidentified compounds (Mehendale et al. 1975; Renner and Schuster 1977, 1978; Renner et al. 1978; Edwards et al. 1991).

Plants take up relatively minimal amounts of HCB from soils (EPA 1985; Carey et al. 1979). Information was not available on the fate of HCB in birds.

4.0 REFERENCES

ATSDR. 1994. Toxicological Profile for Hexachlorobenzene. Agency for Toxic Substances and Disease Registry. August.

Beck J, Hansen K. 1974. The degradation of quintozene, pentachlorobenzene, hexachlorobenzene and pentachloraniline in soil. Pestic Sci 5:41-48. As cited in ATDSR 1994.

Callahan M, Slimak M, Gabel N, et al. 1979. Water-Related Environmental Fate of 129 Priority Pollutants. EPA-440/4-79-029b. Office of Water Planning and Standards, Washington, DC. p. 77-1 to 77-13.

Carey A, Gowen J, Tai H, Mitchell W, Wiersma G. 1979. Pesticide residue levels in soils and crops from 37 states, 1972--National soils monitoring program (IV). Pestic Monit J 12:209-229.

Edwards I, Ferry D, Temple W. 1991. Fungicides and related compounds. In: Hayes W, laws E,eds. Handbook of Pesticide Toxicology. Vol 3. Classes of Pesticides. Academic Press, New York. pp. 1409-1470.

EPA. 1985. Environmental Profiles and Hazard Indices for Constituents of Municipal Sludge: Hexachlorobenzene. Office of Water Regulations and Standards, Washington, DC. June.

Giam C, Murray HE, Lee ER, Kira S. 1980. Bioaccumulation of hexachlorobenzene in killifish (Fundulus similis). Bull Environ Contam Toxicol 25:891-897.

Gile J, Gillett J. 1979. Fate of selected fungicides in a terrestrial laboratory ecosystem. J Agric Food Chem 27(6):1159-1164.

HSDB. 1997. Hazardous Substance Data Bank.

Kelly T, Czuczwa J, Sticksel P, Sverdrup G. 1991. Atmospheric and tributary inputs to toxic substances to Lake Erie. J Great Lakes Res 14(4):504-516.

Knezovich P, Harrison F. 1988. The bioavailability of sediment-sorbed chlorobenzenes to larvae of the midge, Chironomus decorus. Ecotoxicol Environ Saf 15(2):226-241.

Konemann H, Van Leeuwen K. 1980. Toxicokinetics in fish: Accumulation and elimination of six chlorobenzenes by guppies. Chemosphere 9:3-19.

Kosian P, Lemke A, Studders K, Veith G. 1981. The precision of the ASTM bioconcentration test. EPA 600/3-81-022. Environmental Research Lab, Duluth, MN. As cited in HSDB 1997.

Laseter J, et al. 1976. Govt rept announce index. NTIS PB-252671. 76:66. As cited in HSDB 1997.

Mathews H. 1986. IARC Sci Publ 77:253-260. As cited in HSDB 1997.

Mehendale H, Fields M, Matthews H. 1975. Metabolism and effects of hexachlorobenzene on hepatic microsomal enzymes in the rat. J Agric Food Chem 23:261-265.

Neely W, Branson D, Blau G. 1974. Partition coefficient to measure bioconcentration potential of organic chemicals in fish. Environ Sci Technol 8:1113-1115.

Oliver B, Niimi A. 1983. Bioconcentration of chlorobenzenes from water by rainbow trout: Correlations with partition coefficients and environmental residues. Environ Sci Technol 17:287-291.

Parrish P, et al. 1978. Chronic toxicity of chlordane, trifluralin, and pentachlorophenol to sheepshead minnows (cyprinodon variegatus). EPA-600/3-78-010. Environmental Research Laboratory. pp. 35-40.

Renner G, Schuster K. 1977. 2,4,5-trichlorophenol, a new urinary metabolite of hexachlorobenzene. Toxicol Appl Pharmacol 39:355-356.

Renner G, Richter E, Schuster K. 1978. N-acetyl-s-(pentachlorophenyl)cysteine, a new urinary metabolite of hexachlorobenzene. Chemosphere 8:663-668.

Renner G, Schuster K. 1978. Synthesis of hexachlorobenzene metabolites. Chemosphere 8:669-674.

Veith G, Defoe D, Bergstedt B. 1979. Measuring and estimating the bioconcentration factor of chemicals in fish. J Fish Res Board Can 36:1040-1048.

Weisenberg E. 1986. Hexachlorobenzene in human milk: A polyhalogenated risk. IARC Sci Publ 77:193-200.

Zitko V, Hutzinger D. 1976. Bull Environ Contam Toxicol 16:665-673.

HEXACHLOROBUTADIENE

1.0 SUMMARY

Hexachlorobutadiene (HCBD) is a moderately volatile, high molecular weight, chlorinated compound. In surface soil and sediment, it will adsorb to organic carbon. It is moderately soluble in water. In surface water, it will adsorb to suspended material; however, it has a tendency to volatilize. In aerobic environments, in will biodegrade. Exposure routes for aquatic organisms include ingestion, gill uptake, and dermal contact. HCBD bioconcentrates in aquatic life. For mammalian and avian wildlife, HCBD can be taken up through oral, inhalation, and dermal exposure routes. HCBD is not expected to bioaccumulate to high levels in upper-trophic-level receptors. HCBD metabolites cause adverse effects.

The following is a profile of the fate of HCBD in soil, surface water and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

HCBD has a high soil partition coefficient, and would, therefore, be expected to adsorb to soils with a high organic content (Montgomery and Welkom 1990); however, in sandy soils with a low organic content, HCBD is more mobile and will be found in soil pore water (Piet and Zoeteman 1980). HCBD also has a moderate potential to evaporate from surface soils, unless it is bound to organic carbon (Pearson and McConnel 1975). HCBD is expected to biodegrade in aerobic soils (Tabak et al. 1981), but not in anaerobic environments (Johnson and Young 1983).

Following release into water, HCBD will either quickly volatilize or adsorb to sediments and suspended material (Montgomery and Welkom 1990). HCBD will accumulate concentrations in sediments (Elder et al. 1981; EPA 1976; Oliver and Charlton 1984). Biodegradation is a significant removal process for HCBD in aerobic environments (Tabak et al. 1981). However, under anaerobic conditions biodegradation does not occur (Johnson and Young 1983).

3.0 FATE IN ECOLOGICAL RECEPTORS

HCBD dissolved in surface water is expected to bioconcentrate in aquatic organisms, including algae, benthic macroinvertebrates (such as worms and bivalves), detritivore (crayfish), and plantivorous fish (EPA 1976, Oliver and Niimi 1983). HCBD also accumulates in carnivorous fish (EPA 1976). In fish, HCBD will distribute to fatty tissue, especially the liver (Pearson and McConnell 1975 as cited in ATSDR 1994).

Mammals may be exposed to HCBD through (1) ingestion of soil and exposed sediment while foraging for food, grooming, and soil covering plant matter, (2) ingestion of drinking water, and (3) indirect ingestion of contaminated plant and animal matter. Based on HCBD's affinity for soil and sediment, and its potential to be bioconcentrated, it is anticipated that indirect exposure will be the most significant exposure route for mammals. Once ingested, HCBD is readily absorbed in the gastrointestinal tract (Reichert et al. 1985). Following absorption, HCBD is distributed primarily to the kidney, liver, adipose tissue, and brain (Dekant et al. 1988; Nash et al. 1984; Reichert et al. 1985).

HCBD does not appear to be metabolized by the hepatic mixed function oxidase system; however, it does undergo conjugation with glutathione in the liver (Garle and Fry 1989). Metabolic derivatives of these conjugates are believed to be responsible for the renal damage associated with exposure to HCBD (Dekant et al. 1991; Koob and Dekant 1992).

In gravid birds, low levels of HCBD will be transferred to eggs (Dow Chemical Co. 1972).

Information was not available on the fate of HCBD in plants.

4.0 REFERENCES

ATSDR. 1994. *Toxicological Profile for Hexachlorobutadiene.* Agency for Toxic Substances and Disease Registry, Atlanta, GA.

Dekant W, Schrenk D, Vamvakas S, et al. 1988. "Metabolism of hexachloro-1,3-butadiene in mice: in vivo and in vitro evidence for activation by glutathione conjugation." Xenobiotica 18:803-816. As cited in ATSDR 1994.

Dekant W, Urban G, Gorsman C, et al. 1991. "Thioketene formation from haloalkenyl 2-nitrophenyl disulfides: models for biological reactive intermediates of cytotoxic S-conjugates." J Am Chem Soc 113:5120-5122.

Dow Chemical Company. 1972. *Analysis of Quail Eggs for Hexachlorobutadiene by Gas Liquid Chromatography. EPA Document No. 878211372, Fiche No. OTS0206136.* As cited in HSDB 1997.

Elder V, Proctor B, Hites R. 1981. "Organic compounds found near dump sites in Niagara Falls, New York." Environ Sci Technol 15:1237-1243.

EPA. 1976. *An Ecological Study of Hexachlorobutadiene (HCBD).* EPA/560/6-76-010. Office of Toxic Substances, Washington, DC.

Garle M, Fry J. 1989. "Detection of reactive metabolites in vitro." Toxicology 54:101-110. As cited in ATSDR 1994.

HSDB. 1997. Hazardous Substances Data Base.

Johnson L, Young J. 1983. "Inhibition of anaerobic digestion by organic priority pollutants." J Water Pollut Control Fed 55:1141-1149.

Koob M, Dekant W. 1992. "Biotransformation of the hexachlorobutadiene metabolites 1-(glutathione-S-yl)-pentachlorobutadiene and 1-(cystein-S-yl)-pentachlorobutadiene in the isolated perfused rat liver." Xenobiotica 22:125-138. As cited in ATSDR 1994.

Montgomery J, Welkom L. 1990. *Groundwater Chemicals Desk Reference.* Lewis Publications, Chelsea, MI. pp. 334-336. As cited in ATSDR 1994.

Nash J, King L, Lock E, et al. 1984. "The metabolism and disposition of hexachloro-1,3-butadiene in the rat and its relevance to nephrotoxicity." Toxicol Appl Pharmacol 73:124-137. As cited in ATSDR 1994.

Oliver B, Charlton M. 1984. "Chlorinated organic contaminants on settling particulates in the Niagara River vicinity of Lake Ontario." Environ Sci Technol 18:903-908.

Oliver B, Niimi A. 1983. "Bioconcentration of chlorobenzenes from water by rainbow trout: Correlations with partition coefficients and environmental residues." Environ Sci Technol 17:287-291.

Pearson C, McConnell G. 1975. "Chlorinated C1 and C2 hydrocarbons in the marine environment." Proc Royal Soc Lond Biol 189:305-332. As cited in HSDB 1997 and ATSDR 1994.

Piet G, Zoeteman B. 1980. "Organic water quality changes during sand bank and dune filtration of surface waters in the Netherlands." J Am Water Works Assoc 72:400-404. As cited in ATSDR 1994.

Reichert D, Schutz S, Metzler M. 1985. "Excretion pattern and metabolism of hexachlorobutadiene in the rats: Evidence for metabolic activation by conjugation reactions." Biochem Pharmacol 34:499-505. As cited in ATSDR 1994.

Tabak H, Quave S, Mashni C, et al. 1981. "Biodegradability studies with organic priority pollutant compounds". J Water Pollut Cont Fed 53:1503-1518.

HEXACHLOROCYCLOPENTADIENE

1.0 SUMMARY

Hexachlorocyclopentadiene (HCCP) is a semi-volatile, chlorinated compound. If HCCP is released as an emission product, it has been shown to exist mostly in the vapor phase, with photolysis resulting in rapid degradation. HCCP in soil will adsorb to soil particles. Degradation of HCCP may also occur in the environment by chemical hydrolysis and biodegradation by soil biota. Depending on the route of exposure, HCCP may distribute mainly to the lungs, kidneys, and liver. HCCP could potentially bioaccumulate in some aquatic organisms depending upon the species. The respiratory system is the major site of toxicity following inhalation exposure, while, depending on the species, the kidney or the liver are the major sites of toxicity following oral exposure.

The following is a profile of the fate of HCCP in soil, surface water and sediment, and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

HCCP deposited to soil is expected to adsorb strongly to organic carbon in the soil (HSDB 1997). Volatilization from soil surfaces is expected to be minor. In moist soil, hydrolysis and biodegradation under aerobic and anaerobic conditions may occur (HSDB 1997). HCCP on the surface of soil may be subject to photolysis.

HCCP present in surface water will degrade primarily by photolysis and chemical hydrolysis. The half-life of HCCP from photodegradation is very short ; Wolfe et al.(1982) reported a half-life of less than 15 minutes in the top of the water column. In unlit or deep, turbid water, the degradation of HCCP occurs by chemical hydrolysis. Hydrolytic half-lives for HCCP range from several hours to 2-3 weeks, depending on the temperature of the water (Chou et al. 1981; Zepp and Wolfe 1987). HCCP has the potential to adsorb to suspended solids in surface water and sediments; however, this adsorption does not affect the rate of hydrolysis (Wolfe et al. 1982).

Volatilization from water is also expected to be a significant removal mechanism; however, adsorption to suspended solids and sediments may interfere with this process. (EPA 1987).

3.0 FATE IN ECOLOGICAL RECEPTORS

HCCP is expected to be moderately bioconcentrated by algae, invertebrates, and fish. (Lu et al. 1975; Spehar et al. 1979; Veith et al. 1979; Podowski and Khan 1984; Freitag et al. 1982) (Geyer et al. 1981). HCCP taken up by freshwater fish (goldfish) is readily distributed, stored, and metabolized (Podowski et al. 1991). In fish, HCCP is excreted in the bile. The biological half-life of HCCP in the goldfish was approximately 9 days (Podowski and Khan 1984).

Inhalation is the main exposure route for HCCP toxicity in mammals. HCCP is less absorbed following ingestion (Lawrence and Dorough 1981). Following ingestion, HCCP will move primarily to the liver and the kidney (Lawrence and Dorough 1981), which appear to be the main sites of toxicity (Abdo et al. 1984; Southern Research Inst 1981).

Limited information was available regarding the metabolism of HCCP. Some degradation may occur in the gut following oral administration (Dorough and Ranieri 1984; Mehendale 1977).

Information was not available on the fate of HCCP in birds or plants.

4.0 REFERENCES

Abdo K, Montgomery C, Kluwe W, Farnell D, Prejean J. 1984. "Toxicity of Hexachlorocyclopentadiene: Subchronic (13-week) administration by gavage to F344 rats and B6C3F mice." J Appl Toxicol 42(2):75-81.

Chou S, et al. 1981. *Aqueous chemistry and adsorption of hexachlorocyclopentadiene by earth materials.* NTIS PB81-173882. As cited in HSDB 1997.

Dorough H, Ranieri T. 1984. "Distribution and elimination of hexachlorocyclopentadiene in rats and mice." Drug Chem Toxicol 7(1):73-89.

EPA. 1987. Exams II. Computer simulation. As cited in HSDB 1997.

Freitag D, Geyer H, Kraus A, Viswanathan R, Kotzias D, Attar A, Klien W, Korte F. 1982. "Ecotoxicological profile analysis. VII. Screening chemicals for their environmental behavior by comparative evaluation." Ecotox Environ Safety 6:60-81.

Geyer H, Viswanathan R, Freitag D, Korte F. 1981. "Relationship between water solubility of organic chemicals and their bioaccumulation by the alga chlorella." Chemosphere 10:1307-1313.

HSDB. 1997. Hazardous Substance Data Bank.

Lawrence L, Dorough H. 1981. "Retention and fate of inhaled hexachlorocyclopentadiene in the rat." Bull Environ Contam Toxicol 26(5):663-668.

Lu P, Metcalf R, Hirwe A, Williams J. 1975. "Evaluation of environmental distribution and fate of hexachlorocyclopentadiene, chlordane, heptachlor, and heptachlor epoxide in a laboratory model ecosystem." J Agric Food Chem 23:967-973.

Meditext. 1997. Medical Management Data Base.

Mehendale H. 1977. "Chemical reactivity-absorption, retention, metabolism, and elimination of hexachlorocyclopentadiene." Environ Health Perspect 21:275-278.

Podowski A, Khan M. 1984. "Fate of hexachlorocyclopentadiene in water and goldfish." Arch Environ Contam Toxicol 13(4):471-481.

Podowski A, Sclove S, Pilipowicz A, Khan M. 1991. "Biotransformation and disposition of hexachlorocyclopentadiene in fish." Arch Environ Contam Toxicol 20(4):488-496.

Southern Research Institute. 1981. *Subchronic toxicity report on report hexachlorocyclopentadiene (C53607) in rats and mice.* EPA Document No. 40-8349130, Fiche No. OTS0507497. As cited in HSDB 1997.

Spehar R, Veith G, DeFoe D, Bergstedt B. 1979. "Toxicity and bioaccumulation of hexachlorocyclopentadiene, hexachloronorbornadiene and heptachloronorbornene in larval and early juvenile fathead minnows, Pimephales promelas." Bull Environ Contam Toxicol 21(4-5):576-583.

Veith G, Defoe D, Bergstedt B. 1979. "Measuring and estimating the bioconcentration factor of chemicals in fish." J Fish Res Board Can 36:1040-1048.

Wolfe N, Zepp RG, Schlotzhauer, Sink M. 1982. "Transformation pathways of hexachlorocyclopentadiene in the aquatic environment." Chemosphere 11:91-101.

Zepp R, Wolfe N. 1987. Aquatic Surf Chem Vol:423-455. As cited in HSDB 1997.

HEXACHLOROPHENE

1.0 SUMMARY

Hexachlorophene is a persistent organic chemical that is highly soluble in lipids and adsorbs strongly to soil and sediment In surface soils and the euphotic (light-penetrating) zone of surface waters, hexachlorophene is degraded by photolysis. Hexachlorophene may be bioconcentrated by aquatic and soil organisms. In upper-trophic-level receptors, hexachlorophene may be absorbed following oral or dermal exposure and is distributed throughout all body tissues. Due to its high lipid solubility, hexachlorophene has the potential to be transferred significantly in food chains. In mammals, the nervous system is the major site of toxicity for hexachlorophene; however, reproductive and developmental effects have also been reported. Exposure to hexachlorophene may result in decreased egg production in birds.

The following is a profile of the fate of hexachlorophene in soil, surface water, and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, water, and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

Hexachlorophene adsorbs strongly to soil and once bound does not tend to leach from soil or mobilize in soil. Hexachlorophene does not undergo significant hydrolysis or evaporation from the soil; however, slow photodegradation may occur if exposed to light above 290 nm (Kotzias et al. 1982).

Hexachlorophene does not undergo hydrolysis, evaporation or volatilization in water; however, slow photodegradation may occur. Hexachlorophene adsorbs strongly to sediments and has been identified in the humic acid portion of sediment. The half-life of hexachlorophene in water is expected to be greater than 50 years with a half-life of 290 days reported in sediment. Hexachlorophene has been reported to bioconcentrate in aquatic organisms (Kotzias et al. 1982; Hansch and Leo 1985; Lyman et al. 1982).

3.0 FATE IN ECOLOGICAL RECEPTORS

Based on its high octanol-water partition coefficient, hexachlorophene is expected to bioconcentrate in aquatic life living in the water column and in the sediment. Bioconcentration has been measured in mosquito fish and snail (Hansch and Leo 1985; Lyman et al. 1982).

Hexachlorophene is absorbed rapidly following oral exposure (Hatch 1982). Hexachlorophene may also be absorbed following dermal exposure with blood levels peaking approximately 6 to 10 hours post-application (Meditext 1997). Hexachlorophene is highly lipid-soluble. After entering the bloodstream, it distributes into adipose tissue and tissue with a high lipid content including the central nervous system. Hexachlorophene binds preferentially to myelin (Meditext 1997). Transplacental transfer of hexachlorophene has also been reported (Hatch 1982). Target organs include the nervous system, the gastrointestinal system, and skin (Meditext 1997).

Hexachlorophene has been reported to have low volatility from plant leaves (Goetchius et al. 1986). Additional data regarding the potential effects of hexachlorophene on plants were not located. Information was not available on the fate of hexachlorophene in exposed birds.

4.0 REFERENCES

Goetchius P, et al. 1986. *Health and environmental effect profile on hexachlorophene.* SR-TR-220. Syracuse Research Corporation. pp. 2-1 to 3-1. As cited in HSDB 1997.

Hansch C, Leo A. 1985. *Medchem project issue no. 26*, Pomona College, Claremont, CA.

Hatch R. 1982. *Veterinary toxicology.* In: Booth N, McDonald L, eds. *Veterinary Pharmacology and Therapeutics.* 5th ed. Iowa State University Press, Ames, IA. pp. 927-1021.

HSDB. 1997. Hazardous Substance Data Base.

Kotzias D, Parlar H, Korte F. 1982. "Photoreaktivitat organischer chemikalien in wabrigen systemen in gegenwart von nitraten und nitriten." Naturwiss 69:444-445. As cited in HSDB 1997.

Lyman W, Reehl W, Rosenblatt D, eds. 1982. *Handbook of Chemical Property Estimation Methods.* McGraw Hill Book Company, New York.

Meditext (r). 1997. Medical Management Data Base..

HYDRAZINE

1.0 SUMMARY

Hydrazine is a reactive, nitrogen-containing compound. It is readily biodegraded after release to soil and surface water. Volatilization may also be a significant removal process. Hydrazine is readily absorbed following inhalation, ingestion, and dermal absorption. Mammals rapidly break down and excrete hydrazine.

The following is a profile of the fate of hydrazine in soil, surface water and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, surface water, and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

Studies show that hydrazine is expected to biodegrade in soils high in organic carbon, and to adsorb to soils high in clay content (Braun and Zirrolli 1983; Sun et al. 1992). For dry surface soil, volatilization may be a significant process (HSDB 1997).

Hydrazine is expected to have a relatively short half-life of 8.3 days in pond water (Braun and Zirrolli 1983). Hydrazine has been reported to react with dissolved oxygen at a rate inversely proportional to its concentration (Slonim and Gisclard 1976); its degradation rate increases with increasing temperature, dissolved oxygen, and the presence of microorganisms (Sun et al. 1992).

3.0 FATE IN ECOLOGICAL RECEPTORS

Hydrazine is absorbed rapidly from the lungs, gastrointestinal tract, and through skin (ACGIH 1991). Hydrazine is reported to be neurotoxic, hepatotoxic and nephrotoxic in rodents (Lambelt and Shank 1988). Hydrazine is rapidly metabolized in the liver and eliminated (Jenner and Timbrell 1995).

Information was not available on the fate of hydrazine in exposed birds, aquatic life, or plants.

4.0 REFERENCES

ACGIH. 1991. *Documentation of TLVs.* 6th ed. p. 761.

Braun B, Zirrolli J. 1983. *Environmental fate of hydrazine fuels in aqueous and soil environments.* Air Force Report No. ESLTR-82-45. NTIS AD-A125813. As cited in HSDB 1997.

HSDB. 1997. Hazardous Substance Data Bank.

Jenner A, Timbrell J. 1995. "In vitro microsomal metabolism of hydrazine." Xenobiotica 25(6):599-609.

Lambelt C, Shank R. 1988. "Role of formaldehyde hydrazone and catalase in hydrazine-induced methylation of DNA guanine." Carcinogenesis 9(1):65-70.

Slonim A, Gisclard J. 1976. Bull Environ Contam Toxicol 16:301-309. As cited in HSDB 1997.

Sun H, et al. 1992. Huanjing Kexue 13:35-39. As cited in HSDB 1997.

MERCURY

1.0 SUMMARY

Mercury is a highly toxic compound with no known natural biological function. Mercury exists in three valence states: mercuric (Hg2+), mercurous (Hg1+), and elemental (Hg0+) mercury. It is present in the environment in inorganic and organic forms. Inorganic mercury compounds are less toxic than organomercury compounds, however, the inorganic forms are readily converted to organic forms by bacteria commonly present in the environment. The organomercury compound of greatest concern is methylmercury.

Mercury sorbs strongly to soil and sediment. Elemental mercury is highly volatile. In aquatic organisms, mercury is primarily absorbed through the gills. In aquatic and terrestrial receptors, some forms of mercury, especially organomercury compounds, bioaccumulate significantly and biomagnify in the food chain. In all receptors, the target organs are the kidney and central nervous system. However, mercury causes numerous other effects including teratogenicity and mutagenicity.

The following is a profile of the fate of mercury in soil, surface water and sediment, and the fate after uptake by biological receptors. Section 2 discusses the environmental fate and transport in soil, surface water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

In soil, mercury exists in the mercuric (Hg2+) and mercurous (Hg1+) states. Mercury adsorbs to soil or is converted to volatile forms (Krabbenhoft and Babiarz 1992; Callahan et al. 1979). Mercury can migrate by volatilization from aquatic and terrestrial sources through the reduction of metallic mercury to complex species and by the deposition in reducing sediments. Atmospheric transport is a major environmental distribution pathway.

Mercury 2+ is the predominant form of mercury in surface waters (ATSDR 1993). Nonvolatile mercury in surface water binds to organic matter and sediment particles (Lee and Iverfeldt 1991).

Sorption to suspended and bed sediments is one of the most important processes determining the fate of mercury in aquatic systems; sorption onto organic materials is the strongest for mercury 2+. As a result, mercury is generally complexed to organic compounds and is not readily leached from either organic-rich or mineral-rich soils (Rosenblatt et al 1975). Most mercury compounds can be remobilized in aquatic systems by microbial conversion to methyl and dimethyl forms. Conditions reported to enhance microbial conversion include large amounts of available mercury, large numbers of bacteria, absence of strong complexing agents, near neutral pH, high temperatures, and moderately aerobic conditions.

3.0 ECOLOGICAL RECEPTORS

Sorption at the gill surface is the major pathway of mercury entry in aquatic organisms (EPA 1984). In aquatic organisms, bioaccumulation is rapid and elimination is slow. Biomagnification occurs in the aquatic food chain (NRCC 1979). Absorbed mercury is distributed to the blood and ultimately the internal organs. Mercury which is not absorbed is eliminated rapidly in the feces (Eisler 1987). The biological half-life of mercury in fish is approximately 2 to 3 years (EPA 1985). In general, mercury accumulation is enhanced by elevated water temperatures, reduced water hardness or salinity, reduced water pH, increased age of the organism, reduced organic matter content of the medium, and the presence of zinc, cadmium, or selenium in solution.

Mercury is readily absorbed by terrestrial species following oral and inhalation exposure. Elemental and organomercury compounds are readily transferred across the placenta and blood-brain barrier. Mercury is bioaccumulated primarily in the kidney (Rothstein and Hayes 1964; Nielsen and Andersen 1991), and mercury is biomagnified in mammals (Eisler 1987). Retention of mercury in mammals is longer for organomercury compounds (especially methylmercury) than for inorganic forms. Mercury elimination occurs via the urine, feces, expired air, and breast milk (Clarkson 1989; Yoshida et al. 1992).

All mercury compounds interfere with metabolism in organisms, causing inhibition or inactivation of proteins containing thiol ligands and ultimately leading to miotic disturbances (Das et al 1982; Elhassani 1983). Mercury also binds strongly with sulfhydryl groups. Phenyl and methyl mercury compounds are among the strongest known inhibitors of cell division (Birge et al 1979). In mammals, methyl mercury irreversibly destroys the neurons of the central nervous system.

Information was not available on the fate of mercury in birds.

Mercury in soils is generally not available for uptake by plants due to the high binding capacity to clays and other charged particles (Beauford et al 1977). However, mercury levels in plant tissues increase as soil levels increase with 95% of the accumulation and retention in the root system (Beauford et al 1977; Cocking et al 1991). Mercury is reported to inhibit protein synthesis in plant leaves and may affect water-adsorbing and transporting mechanisms in plants (Adriano 1986).

4.0 REFERENCES

Adriano D.C. 1986. *Trace elements in the terrestrial environment*. Springer-Verlag. New York.

ATSDR. 1993. *Toxicological Profile for Mercury*. Agency for Toxic Substances and Disease Registry, Atlanta, GA.

Beauford, W. et al. 1977. "Uptake and distribution of mercury within higher plants." Physiol. Plant 39:261-265.

Birge W.J., Black J.A, Westerman A.G, and Hudson J.E. 1979. *The effect of mercury on reproduction of fish and amphibians.* In: *The biogeochemistry of mercury in the environment.* Editor J.O. Nriagu. Elsevier/North Holland Biomedical Press. New York.

Callahan M, Slimak M, Gabel N, et al. 1979. *Water-Related Environmental Fate of 129 Priority Pollutants. Vol 1 & 2*. Office of Water and Waste Management, U.S. Environmental Protection Agency, Washington, DC. EPA-440/4-79-029a, EPA-440/4-79-029b. pp. 14-1 to 14-15.

Clarkson T. 1989. "Mercury." J Am Coll Toxicol 8:1291-1295.

Cocking D.R., Hayes M.L., Rohrer M.J., Thomas R., and Ward D. 1991. "Compartmentalization of mercury in biotic components of terrestrial floodplain ecosystems adjacent to the south river at Wayneboro, Virginia." Water, Air and Soil Pollution 57-58: 159-170.

Das S.K., Sharma A, and Talukder G. 1982. "Effects of mercury on cellular systems in mammals - A review." Nucleus (Calcutta) 25: 193-230.

Eisler R. 1987. *Mercury Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review.* U.S. Fish and Wildlife Service. Biological Report 85(1.10).

Elhassani S.B. 1983. "The many faces of mercury poisoning." Journal of Toxicology 19: 875-906.

EPA. 1984. *Ambient Water Quality Criteria Document for Mercury.* EPA 440/5-84-026. p. 10-11.

EPA. 1985. *Ambient Water Quality Criteria Document for Mercury.* Office of Water Regulations and Standards. Washington D.C. EPA 440/5-84-026.

HSDB. 1997. Hazardous Substances Data Bank.

Krabbenhoft D, Babiarz C. 1992. "The role of groundwater transport in aquatic mercury cycling." Water Resour Res 28(12):3119-3129. As cited in ATSDR 1993.

Lee Y, Iverfeldt A. 1991. "Measurement of methylmercury and mercury in run-off, lake and rain waters." Water Air Soil Pollut 56:309-321. As cited in ATSDR 1993.

NRCC. 1979. "Effects of Mercury in the Canadian Environment." National research Council of Canada. NRCC No. 16739. pp. 89, 101. As cited in HSDB 1997.

Nielsen J, Andersen O. 1991. "Methyl mercuric chloride toxicokinetics in mice. I: Effects of strain, sex, route of administration and dose." Pharmacol Toxicol 68:201-207. As cited in ATSDR 1993.

Rosenblatt D.H., Miller T.A., Dacre J.C., Mull I. And Cogley D.R. 1975. *Problem definition studies on potential environmental pollutants II. Physical, chemical, toxicological, and biological properties of 16 substances*. Technical Report 7509. U.S. Army Medical Bioengineering Research and Development Laboratory. Fort Detrick, Frederick, Maryland.

Rothstein A, Hayes A. 1964. "The turnover of mercury in rats exposed repeatedly to inhalation of vapor." Health Phys 10:1099-1113.

Yoshida M, Satoh H, Kishimoto T, Yamamura Y. 1992. "Exposure to mercury via breast milk in suckling offspring of maternal guinea pigs exposed to mercury vapor after parturition." J Toxicol Environ Health 35:135-139. As cited in ATSDR 1993.

METHANOL

1.0 SUMMARY

Methanol is a highly water soluble hydrocarbon. It does not adsorb to organic carbon. The primary removal process for methanol in soil and water is biodegradation. Aquatic, soil, and sediment communities can be exposed to methanol through direct contact. Upper-trophic-level receptors may be directly exposed through ingestion, inhalation, or dermal exposure. Methanol does not bioconcentrate or move through food chains.

The following is a profile of the fate of methanol in soil, surface water, and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, surface water, and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

Based on biological screening studies, including soil microcosm studies, methanol undergoes biodegradation if released to the soil. Methanol is expected to be highly mobile in soil, based on its miscibility in water and low log K_{ow} value. Evaporation from dry surfaces is also expected to occur, based on the high vapor pressure of methanol (Weber et al. 1981; Hansch and Leo 1985; HSDB 1997).

Methanol is completely soluble in water. Methanol is significantly biodegradable in water, based on screening studies (HSDB 1997). Volatilization is expected to be a significant removal process (Lyman 1982). Aquatic hydrolysis, oxidation, photolysis, adsorption to sediment, and bioconcentration are not considered significant removal processes for methanol (HSDB 1997).

3.0 FATE IN ECOLOGICAL RECEPTORS

Methanol uptake across gill epithelia is the most significant exposure route. However, based on its low bioconcentration factor for fish, methanol does not bioconcentrate (Freitag et al. 1985; Bysshe 1982) (Hansch and Leo 1985).

Mammals are exposed to methanol through ingestion, inhalation, and dermal contact. Methanol is reported to readily absorb from the gastrointestinal and respiratory tracts (Gosselin et al. 1984), and rapidly distribute within tissues (Clayton and Clayton 1982). Following absorption, methanol is widely distributed in body tissue. Small amounts are excreted in the urine and expired air; however, methanol is mostly oxidized to formaldehyde and formic acid (Goodman and Gillman 1985).

Information was not available on the fate of methanol in exposed birds or plants.

4.0 REFERENCES

Bysshe S. 1982. *Bioconcentration factor in aquatic organisms.* In: Lyman W, Reehl W, Rosenblatt D, eds. *Handbook of Chemical Property Estimation Methods.* McGraw-Hill Book Co., New York. pp 5-1 to 5-30.

Clayton G, Clayton F, eds. 1982. *Patty's Industrial Hygiene and Toxicology.* 3rd ed. Vol 2. John Wiley & Sons, New York. pp. 4531-4534. As cited in HSDB 1997.

Freitag D, Ballhorn L, Geyer H, Korte F. 1985. "Environmental hazard profile of organic chemicals: An experimental method for the assessment of the behavior of organic chemicals in the ecosphere by means of simple laboratory tests with 14C labeled chemicals." Chemosphere 14:1589-1616.

Goodman L, Gilman A, eds. 1985. *The Pharmacological Basis of Therapeutics.* 7th ed. Macmillan Publ., New York. p. 381-382.

Gosselin R, Smith R, Hodge H. 1984. *Clinical toxicology of commercial products.* Vol II. 5th ed. Williams and Wilkins, Baltimore, MD. p. III-275.

Hansch C, Leo A. 1985. *Medchem Project Issue No. 26,* Pomona College, Claremont, CA. As cited in HSDB 1997.

HSDB. 1997. Hazardous Substance Data Bank.

Lyman W, Reehl W, Rosenblatt D, eds.. 1982. *Handbook of Chemical Property Estimation Methods.* McGraw Hill Book Company, New York.

Weber R, Parker P, Bowser M. 1981. *Vapor pressure distribution of selected organic chemicals.* EPA 600/2-81-021. Industrial Environmental Research Laboratory, Cincinnati, OH. As cited in HSDB 1997.

NITROPROPANE, 2-

1.0 SUMMARY

2-nitropropane is a highly volatile, low molecular weight hydrocarbon. Generally, it does not adsorb to soil or sediment, and rapidly volatilizes from soil and surface water. Wildlife may be exposed to 2-nitropropane through ingestion or inhalation. Due to its high water solubility, 2-nitropropane does not bioconcentrate in fish, and does not bioaccumulate in wildlife. 2-nitropropane is rapidly metabolized and excreted by mammals.

The following summarizes information on the fate of 2-nitropropane in soil, surface water and sediment, and its fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

2-nitropropane rapidly volatilizes from soil, and also has the potential to leach in moist soils. 2-nitropropane undergoes minimal degradation in soil (Freitag et al. 1988).

2-nitropropane is highly soluble in water (Baker and Bollmeier 1981). It is expected to have a short half-life in surface water because of its propensity for rapid volatilization, based on its high vapor pressure (Dougan et al. 1976). Adsorption of 2-nitropropane to suspended solids or sediment is not expected, based on its low K_{oc} value (Lyman 1982).

3.0 FATE IN ECOLOGICAL RECEPTORS

2-nitropropane does not bioconcentrate in aquatic organisms (Baker and Bollmeier 1981; Freitag et al. 1988). 2-nitropropane is readily absorbed by the gastrointestinal tract and the lungs, when inhaled. Accumulation of 2-nitropropane in tissues of mammals is low because it is rapidly metabolized and eliminated after uptake (Nolan et al. 1982). 2-nitropropane may be excreted unchanged in expired air or as nitrite and nitrate in the urine (Browning 1965).

No information was available on the fate of 2-nitropropane in birds or plants.

4.0 REFERENCES

Baker B, Bollmeier A. 1981. "Nitroparaffins." In: *Kirk-Otmer Encyclopedia of Chemical Technology.* 3rd ed. John Wiley & Sons, New York. 15:969-987.

Browning E. 1965. *Toxicology and Metabolism of Industrial Solvents.* Elsevier, New York. pp. 285-288.

Dougan J, et al. 1976. *Preliminary Scoring of Selected Organic Air Pollutants.* Apd III. EPA 450/3-77-008d. pp. 303. As cited in HSDB 1997.

Freitag D, et al. 1988. "Ecotoxicological Profile Analysis of Nitroparaffins According to Oecd Guidelines with C14-labelled Compounds." In: *Tsca Set 8d Submissions to EPA for Nitromethane* (Fiche No. ITS516767). As cited in HSDB 1997.

HSDB. 1997. Hazardous Substance Data Bank.

Lyman W. 1982. "Adsorption Coefficient for Soils and Sediments." In: Lyman W, Reehl W, Rosenblatt D, eds. *Handbook of Chemical Property Estimation Methods.* McGraw-Hill Book Co., New York. pp 4-1 to 4-33.

Nolan R, Unger A, Muller C. 1982. "Pharmacokinetics of Inhales [14c]-2-nitropropane in Male Sprague-dawley Rats." Ecotoxicol Environ Safety 6(4):388-397.

POLYNUCLEAR AROMATIC HYDROCARBONS (PAHS)

1.0 SUMMARY

Polynuclear aromatic hydrocarbons (PAH) are a class of semi-volatile compounds that have a high affinity for soil and sediment particles. PAHs have low water solubility. Low molecular weight PAHs volatilize and photolyze from soil and surface water, and may be biodegraded as well. High molecular weight PAHs are resistant to volatilization, photolysis, and biodegradation. PAHs can be bioconcentrated to high concentrations by some aquatic organisms. However, many aquatic organisms can metabolize PAHs. The main PAH exposure route for upper-trophic-level receptors is ingestion. However, wildlife can readily metabolize PAHs and eliminate the by-products. Therefore, food chain transfer and biomagnification are anticipated to be minimal.

The following is a profile of the fate of PAHs in soil, surface water and sediment; and the fate after uptake by ecological receptors. The PAHs considered are benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, and indeno(1,2,3-cd)pyrene. Section 2 discusses the environmental fate and transport in soil, surface water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

PAHs strongly adsorb to the soil; therefore, leaching to groundwater and volatilization are slow insignificant processes in most instances (HSDB 1997). However, the persistence of PAHs in soil is dependent upon the number of condensed rings that a PAH contains. The major source of degradation of PAHs in soil is microbial metabolism (ATSDR 1995). Volatilization and photolysis were determined to be important processes for the degradation of PAHs containing less than four aromatic rings, when analyzed from four surface soils amended with PAHs in sewage sludge. However, PAHs containing four or more aromatic rings showed insignificant abiotic losses (Wild and Jones 1993).

Within aquatic systems, PAHs are found sorbed to particles suspended in the water column or particles which have settled to the bottom. This is due to the low solubility and high affinity PAHs have for organic carbon. Studies have estimated that two-thirds of PAHs found in aquatic systems are in particle form and

only one-third are in dissolved form (Eisler 1987). Low molecular weight PAHs (2 to 3 rings) studied in estuaries show that the primary removal processes are volatilization and biodegradation, while high molecular weight PAHs (4 or more rings) volatilize and adsorb to suspended sediments (Thomas 1982; Southworth et al. 1978; Southworth 1979).

Photo-oxidation, chemical oxidation, and biodegradation by aquatic microorganisms are the primary degradation processes associated with PAHs in water (Neff 1979). The process of photo-oxidation varies widely among PAHs when considering the rate and extent of degradation. Benzo(a)pyrene is the most resistant to photo-oxidation, while benzo(a)anthracene is the most sensitive (Neff 1979). Microbial degradation of PAHs in water is very rapid under oxygenated conditions, but extremely slow under anoxic conditions (Neff 1979).

3.0 FATE IN ECOLOGICAL RECEPTORS

Sources of PAH accumulation in aquatic organisms include water, sediment, and food. Bioconcentration factors can range from low to very high, depending on the PAH and the receptor. Invertebrates and bottom-dwelling fish may accumulate PAHs through ingestion of sediment (Eisler 1987).

Studies indicate that fish are capable of metabolizing PAHs by the mixed function oxidase (MFO) system in the liver. The breakdown products are then eliminated through the urine and feces. Half-lives ranging from 2 to 9 days have been reported for the elimination of PAHs in fish (Niimi 1987). Chrysene has a near-surface half-life computed for sunlight at latitude 40°N of 4.4 hours (Zepp and Schlotzhauer 1979). Assimilation of PAHs from contaminated food is readily achieved by fish and crustaceans; however, this process is limited for mollusks and polychaete worms (Eisler 1987). It is also noted that aquatic organisms such as phytoplankton, certain zooplankton, mussels, scallops, and snails lack a metabolic detoxification enzyme system. Therefore, these organisms have potential for PAH accumulation (Malins 1977).

PAHs can be introduced into mammals through ingestion, inhalation, and dermal exposure. Because PAHs are highly lipid soluble and can cross epithelial membranes, they are readily absorbed from the gastrointestinal tract and lung (HSDB 1997). PAHs are absorbed through the mucous lining of bronchi when inhaled (Bevan and Ulman 1991) and taken up by the gastrointestinal tract in fat-soluble compounds when ingested. Passive diffusion is the process in which PAHs are distributed following percutaneous

absorption (Ng et al. 1991). Once absorbed into the body, PAHs are distributed to the lymph fluid and then the blood stream. Following oral or inhalation exposure, PAHs are widely distributed in animal tissue (Bartosek et al. 1984; Withey et al. 1991; Yamazaki and Kakiuchi 1989).

PAHs have limited transfer across the placenta; therefore, PAH levels are generally lower in the fetus, when compared to maternal levels (Neubert and Tapken 1988; Withey et al. 1992). The major metabolism sites for PAHs are the liver and kidneys. Additional sites of metabolism include the adrenal glands, testes, thyroid, lungs, skin, sebaceous glands, and placenta (Meditext 1997). PAHs are primarily excreted through the urine and bile (Bevan and Weyand 1988; Grimmer et al. 1988; Petridou-Fischer et al. 1988; Weyand and Bevan 1986; Wolff et al. 1989).

PAHs may be taken up by terrestrial plants from the soil or air depending on the concentration, solubility, and molecular weight of the PAHs. Lower molecular weight PAHs are absorbed by plants more readily than higher molecular weight PAHs (USFWS 1987). Some plants are capable of producing benzo(b)fluoranthene (HSDB 1997). The partitioning of PAHs between vegetation and the atmosphere was found to be primarily dependent upon the atmospheric gas-phase PAH concentration and the ambient temperature, when studied throughout the growing season under natural conditions (Simonich and Hites 1994). Above-ground parts of vegetables have been found to contain more PAHs than underground parts, mainly attributable to airborne deposition and subsequent adsorption (USFWS 1987). Growth promoting effects were observed in higher plants, as well as cultures of lower plants, when benzo(a)anthracene, indeno(1,2,3-cd)pyrene, and benzo(b)fluoranthene were tested in a series of soil and hydrocultures (Graf and Nowak 1968).

Information was not available on the fate of PAHs in exposed birds.

4.0 REFERENCES

ATSDR. 1995. *Toxicological Profile for Polycyclic Aromatic Hydrocarbons.* Agency for Toxic Substances and Disease Registry, U.S. Public Health Service. August.

Bartosek I, Guaitani A, Modica R, Fiume M, Urso R. 1984. "Comparative kinetics of oral benz(a)anthracene, chrysene and triphenylene in rats: Study with hydrocarbon mixtures." Toxicol Lett 23:333-339.

Bevan D, Ulman M. 1991. "Examination of factors that may influence disposition of benzo(a)pyrene in vivo: Vehicles and asbestos." Cancer Lett 57(2):173-180.

Bevan D, Weyand E. 1988. "Compartmental analysis of the disposition of benzo(a)pyrene in rats." Carcinogenesis 9(11):2027-2032.

Eisler R. 1987. *Polycyclic Aromatic Hydrocarbon Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review.* U.S. Fish and Wildlife Service, U.S. Department of the Interior. Biological report 85(1.11). As cited in ATSDR 1995.

Graf W, Nowak W. 1968. "Wachstumsforderung bei niederen und hoheren pflanzen durch kanzerogene polyzyklische aromate." Arch Hyg Bakt 150:513-528.

Grimmer G, Brune H, Dettbarn G, Heinrich U, Jacob J, Mohtashamipur E, Norpoth K, Pott F, Wenzel-Hartung R. 1988. "Urinary and fecal excretion of chrysene and chrysene metabolites by rats after oral, intraperitoneal, intratracheal or intrapulmonary application." Arch Toxicol 62(6):401-405.

HSDB. 1997. Hazardous Substances Data Bank.

Malins D. 1977. "Metabolism of aromatic hydrocarbons in marine organisms." Ann NY Acad Sci 298:482-496.

Meditext. 1997. Medical Management Data Base. June.

Neff J. 1979. *Polycyclic aromatic hydrocarbons in the aquatic environment. Sources, fates and biological effects.* Applied Science Publishers, Ltd. London, England.

Neubert D, Tapken S. 1988. "Transfer of benzo(a)pyrene into mouse embryos and fetuses." Arch Toxicol 62(2-3):236-239.

Ng K, Chu I, Bronaugh R, Franklin C, Somers D. 1991. "Percutaneous absorption/metabolism of phenanthrene in the hairless guinea pig: Comparison of in vitro and in vivo results." Fundam Appl Toxicol 16(3):517-524.

Niimi A. 1987. "Biological half-lifes of chemicals in fishes." Rev Environ Contam Toxicol 99:1-46.

Petridou-Fischer J, Whaley S, Dahl A. 1988. "In vivo metabolism of nasally instilled benzo(a)pyrene in dogs and monkeys." Toxicology 48(1):31-40.

Simonich S, Hites R. 1994. "Importance of vegetation in removing polycyclic aromatic hydrocarbons from the atmosphere." Nature 370:49-51.

Southworth G. 1979. "The role of volatilization on removing polycyclic aromatic hydrocarbons from aquatic environments." Bull Environ Contam Toxicol 21:507-514.

Southworth G, Beauchamp J, Schmeider P. 1978. "Bioaccumulation potential of polycyclic aromatic hydrocarbons in Daphnia pulex." Water Research 12:973-977.

Thomas R. 1982. *Volatilization from water.* In: Lyman W, Reehl W, Rosenblatt D, eds. *Handbook of Chemical Property Estimation Methods.* McGraw-Hill Book Company, New York. pp 15-1 to 15-34.

U.S. Fish and Wildlife Service (USFWS). 1987. *Polycyclic aromatic hydrocarbon hazards to fish, wildlife, and invertebrates: A synoptic review.* Biological Report 85 (1.11). Washington D.C.

Weyand E, Bevan D. 1986. "Benzo(a)pyrene disposition and metabolism in rats following intratracheal instillation." Cancer Res 46:5655-5661.

Wild S, Jones K. 1993. "Biological and abiotic losses of polynuclear aromatic hydrocarbons (PAHs) from soils freshly amended with sewage sludge." Environ Toxicol Chem 12:5-12.

Withey J, Law F, Endrenyi L. 1991. "Pharmacokinetics and bioavailability of pyrene in the rat." J Toxicol Environ Health 32(4):429-447.

Withey J, Shedden J, Law F, Abedini S. 1992. "Distribution to the fetus and major organs of the rat following inhalation exposure to pyrene." J Appl Toxicol 12(3):223-231.

Wolff M, Herbert R, Marcus M, Rivera M, Landrigan P, Andrews L. 1989. "Polycyclic aromatic hydrocarbon (PAH) residues on skin in relation to air levels among roofers." Arch Environ Health 44(3):157-163.

Yamazaki H, Kakiuchi Y. 1989. "The uptake and distribution of benzo(a)pyrene in rat after continuous oral administration." Toxicol Environ Chem 24(½):95-104.

Zepp R, Schlotzhauer P. 1979. In: Jones P, Leber P, eds. *Polynuclear aromatic hydrocarbons.* Ann Arbor Science Publ., Ann Arbor MI. pp. 141-158. As cited in HSDB 1997.

POLYCHLORINATED BIPHENYLS (PCBs)

1.0 SUMMARY

Polychlorinated biphenyls (PCB) are mixtures of different congeners of chlorobiphenyl. PCBs are a group of highly fat-soluble, semi-volatile compounds that readily bioaccumulate and biomagnify in ecological receptors, especially upper-trophic-level carnivores in aquatic food webs. In general, PCBs adsorb strongly to soil and sediment, and are soluble in fatty tissues. Volatilization and biodegradation of the lower chlorinated congeners also occur. The toxicological properties of individual PCBs are influenced primarily by: (1) lipophilicity, which is correlated with log K_{ow}, and (2) steric factors resulting from different patterns of chlorine substitution on the biphenyl molecule. In general, PCB isomers with high K_{ow} values and high numbers of substituted chlorines in adjacent positions constitute the greatest environmental concern. Biological responses to individual isomers or mixtures vary widely, even among closely related taxonomic species.

The following is a profile of the fate of PCBs in soil, surface water, and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, surface water, and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

The environmental fate of PCBs in soil depends on the degree of chlorination of the molecule. In general, adsorption and the persistence of PCBs increases with an increase in the degree of chlorination (EPA 1988). Mono-, di-, and trichlorinated biphenyls (Aroclors 1221 and 1232) biodegrade relatively rapidly. Tetrachlorinated biphenyls (Aroclors 1016 and 1242) biodegrade slowly, and higher chlorinated biphenyls (Aroclors 1248, 1254, and 1260) are resistant to biodegradation (HSDB 1997). Although biodegradation of higher chlorinated congeners may occur very slowly, no other degradation mechanisms have been shown to be significant in soil (HSDB 1997). Vapor loss of PCBs from soil surfaces appears to be an important mechanism with the rate of volatilization decreasing with increasing chlorination. Although the volatilization rate may be low, the total loss by volatilization over time may be significant because of persistence and stability of PCBs (Sklarew and Girvin 1987).

In water, adsorption to sediments and organic matter is a major fate process for PCBs (EPA 1988; Callahan et al. 1979). Volatilization of dissolved PCBs is an important aquatic process. Strong PCB adsorption to sediment significantly decreases the rate of volatilization, with higher chlorinated PCBs having longer half-lives than the lower chlorinated PCBs (EPA 1988).

3.0 FATE IN ECOLOGICAL RECEPTORS

Diet is a major route of PCB uptake in many aquatic species (Eisler 1986). However, some species accumulate PCBs from the water column to a much larger extent than the diet, even when comparing closely-related species. Based on its high log K_{ow} value, receptors are expected to bioconcentrate and bioaccumulate PCBs to tissue levels much greater than the concentrations in water and sediment (Eisler 1986). Due to their high lipophilicity, PCBs concentrate mostly in fatty tissues. For upper-trophic-level receptors, diet is the main exposure pathway for PCB exposure (Eisler 1986). In aquatic food webs, evidence indicates that PCBs biomagnify in upper trophic levels, but not in lower trophic levels (Shaw and Connell 1982).

Among mammals, aquatic predators (e.g., mink, otters, seals, etc.) have been found to accumulate PCBs to significant levels. Lower chlorinated PCBs are eliminated more rapidly from lipids than higher chlorinated PCBs. Placental transfer of PCBs occurs in mammals (Hidaka et al. 1983).

The primary biochemical effect of PCBs is to induce hepatic mixed function oxidase systems, increasing an organism's capacity to biotransform or detoxify xenobiotic chemicals. PCBs also induce hepatic enzymes that metabolize naturally occurring steroidal hormones (Peakall 1975). These hepatic microsomal enzyme systems are most likely correlated with observed adverse reproductive effects (Tanabe 1988).

PCBs accumulate in bird tissues and eggs (Eisler 1986). Residues of PCBs in birds are affected by numerous biotic factors including fat content, tissue specificity, sex, and the developmental stage of an organism (Eisler 1986). Sexual differences in PCB bioaccumulation are pronounced due to the female's ability to pass a significant portion of the PCB burden to eggs (Lemmetyinen and Rantamaki 1980).

Water snakes (*Nerodia spp.*) and turtles accumulate PCB levels similar to those of PCB residues in their prey. Aroclor 1260 accounted for most of the PCBs detected in water snakes (Sabourin et al. 1984;

Olafsson et al. 1983). These data suggest diet is an important route of PCB transfer in reptiles (McKim and Johnson 1983).

Organic matter and clay content of soil influences the bioavailability of PCBs to plants (Strek and Weber 1982). Uptake of PCBs from soils by plants has been documented, however, only very low amounts are typically accumulated (Iwata et al 1974, Iwata and Gunther 1976, Weber and Mrozek 1979). Effects of PCBs on plants include reduced growth and chlorophyll content, and negative effects on photosynthesis (Strek and Weber 1982).

Terrestrial and aquatic plants bioconcentrate PCBs (Sawhney and Hankin 1984). Aquatic plants also bioaccumulate PCBs from both the water column and sediments. Transfer of PCBs on microparticulate materials to phytoplankton is well documented, as is partitioning from aqueous solution into algal lipids (Rohrer et al. 1982).

4.0 REFERENCES

Callahan M, Slimak M, Gabel N, et al. 1979. *Water-Related Environmental Fate of 129 Priority Pollutants. Vol 1 & 2.* Office of Water and Waste Management, U.S. Environmental Protection Agency, Washington, DC. EPA-440/4-79-029a, EPA-440/4-79-029b. pp. 36+.

Eisler R. 1986. *Polychlorinated Biphenyl Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review.* U.S. Fish and Wildlife Service. Biological Reports 85(1.7).

EPA. 1988. *Drinking Water Criteria Document for Polychlorinated Biphenyls (PCBs).* ECAO-CIN-414. Environmental Criteria and Assessment Office, Cincinnati, OH.

Hidaka H, Tanake S, Tatsukawa R. 1983. "DDT compounds and PCB isomers and congeners in Weddel seals and their fate in the Antarctic marine ecosystem." Agric Biol Chem 47:2009-2017. As cited in Eisler 1986.

HSDB. 1997. Hazardous Substance Data Bank.

Iwata Y. And Gunther F.A. 1976. "Translocation of the polychlorinated biphenyl Aroclor 1254 from soil into carrots under field conditions." Archives of Environmental Contamination and Toxicology. 4: 44-59.

Iwata Y., Gunther F.A., and Westlake W.E. 1974. "Uptake of a PCB (Aroclor 12554) from soil by carrots under field conditions." Bulletin of Environmental Contamination and Toxicology. 11:523-528.

Lemmetyinen R, Rantamaki P. 1980. "DDT and PCB residues in the arctic tern (*Sterna paradisaea*) nesting in the archipelago of southwestern Finland." Ann Zool Fennici 17:141-146. As cited in Eisler 1986.

McKim J, Johnson K. 1983. "Polychlorinated biphenyls and p,p'-DDE in loggerhead and green postyearling Atlantic sea turtles." Bull Environ Contam Toxicol 31:53-60.

Olafsson P, Bryan A, Bush B, Stone W. 1983. "Snapping turtles -- a biological screen for PCBs." Chemosphere 12:1525-1532. As cited in Eisler 1986.

Peakall D.B. 1975. "PCBs and their environmental effects." CRC Critical Reviews in Environmental Control. 5: 469-508.

Rohrer T, Forney J, Hartig J. 1982. "Organochlorine and heavy metal residues in standard fillets of coho and chinook salmon of the Great Lakes-1980." J Great Lakes Res 8:623-634.

Sabourin T, Stickle W, Michot T, Villars C, Garton D, Mushinsky H. 1984. "Organochlorine residue levels in Mississippi River water snakes in southern Louisiana." Bull Environ Contam Toxicol 32:460-468.

Sawhney B, Hankin L. 1984. "Plant contamination by PCBs from amended soils." J Food Prot 47:232-236.

Shaw G, Connell D. 1982. "Factors influencing polychlorinated biphenyls in organisms from an estuarine ecosystem." Aust J Mar Freshwater Res 33:1057-1010. As cited in Eilser 1986.

Sklarew D, Girvin D. 1987. Rev Environ Contam Toxicol 98:1-41. As cited in HSDB 1997.

Strek H.J. and Weber J.B. 1982. "Behavior of polychlorinated biphenyls (PCBs) in soils and plants." Environmental Pollution (Series A). 28: 291-312.

Tanabe S. 1988. "PCB problems in the future: foresight from current knowledge." Environmental Pollution. 50: 5-28.

Weber J.B. and Mrozek E. 1979. "Polychlorinated biphenyls: absorption and translocation by plants and inactivation by activated carbon." Bulletin of Environmental Contamination and Toxicology. 23:412-417.

PENTACHLOROPHENOL

1.0 SUMMARY

Pentachlorophenol (PCP) has a strong affinity for soil, with sorption higher at lower pH and with increased organic content. Microorganisms readily metabolize PCP in soil, surface water, and sediment. Photolysis rapidly breaks down PCP in surface water. Ecological receptors will rapidly absorb PCP, but will also rapidly excrete it. Therefore, the potential for bioconcentration and bioaccumulation is only moderate. PCP biomagnification has not been observed.

The following is a profile of the fate of PCP in soil, surface water, and sediment, and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

PCP adsorbs strongly to soil, with adsorption higher in acidic conditions (Callahan et al. 1979). The amount of PCP adsorbed to soil at a given pH also increases with increasing organic content of the soil (Chang and Choi 1974). The half-life of PCP in soil ranges from weeks to months (Ide et al. 1972; Murthy 1979; Rao and Davidson 1982). Photolysis and hydrolysis do not appear to be significant processes of degradation in soil (Ball 1987). In certain soil environments, PCP may volatilize; however, in general, mobility of PCP in soil is limited (Arsenault 1976).

Biodegradation is considered the major transformation mechanism for PCP in soil, with PCP metabolized rapidly by acclimated microorganisms (Kaufman 1978). The main degradation products of PCP in soil are 2,3,7,8-tetrachlorophenol and carbon dioxide (Knowlton and Huckins 1983).

The fate of PCP in water and sediment is heavily dependent upon the pH of the water. At lower pH, more of the PCP dissociates and is available for degradation (Weiss et al. 1982). PCP also adsorbs to sediment more readily under acidic conditions, and is more mobile under neutral or alkaline conditions (Kuwatsuka and Igarashi 1975).

In surface water, photolysis and biodegradation are the predominant transformation processes for PCP (ATSDR 1994). Photolysis occurs mainly at the water surface, with its impact decreasing with increasing depth (Callahan et al. 1979). The reported half-life for the photolysis of PCP is about 1 hour (Callahan et al. 1979). Biodegradation of PCP can occur under both aerobic and anaerobic conditions, with more rapid degradation under aerobic conditions (Pignatello et al. 1983). The greatest biodegradation of PCP was observed in the top 0.5 to 1 cm layer of sediment.

3.0 FATE IN ECOLOGICAL RECEPTORS

The aquatic toxicity of PCP depends on water pH; at low pH, PCP is more lipophilic, with a high potential for accumulation. At alkaline pH, PCP is more hydrophilic, with a decreased potential for bioconcentration (Eisler 1989). Fish and bivalves may moderately bioconcentrate PCP (Makela et al. 1991). Accumulation of PCP in fish is rapid, and occurs primarily by direct uptake from water rather than through the food chain or diet. In fish, PCP residues are found in the liver, gill, muscle, and hepatopancreas. PCP is readily metabolized in the liver and hepatopancreas. (Menzie 1978). Half-lives in tissues are less than 24 hours (Eisler 1989).

In mammals, PCP may be absorbed into the body through inhalation, diet or skin contact (Eisler 1989). The degree of accumulation is small, since PCP is efficiently and rapidly excreted. The highest residuals are found in the liver and kidneys, likely reflecting that these organs are the principal organs for metabolism and excretion (Gasiewicz 1991). Small amounts of PCP have been shown to cross the placenta (Shepard 1986).

Uptake into rice has been demonstrated in a 2-year study under flooded conditions. After a single application of radiolabeled PCP, 12.9% of the application was taken up by the plants within the first year, with the highest levels found in the roots (Eisler 1989).

4.0 REFERENCES

Arsenault R. 1976. *Pentachlorophenol and Contained Chlorinated Dibenzodioxins in the Environment.* American Wood-Preservers Association, Alexandria, VA. pp. 122-147. As cited in ATSDR 1994.

ATSDR. 1994. *Toxicological Profile for Pentachlorophenol*. Agency for Toxic Substances and Disease Registry, Atlanta, GA.

Ball J. 1987. *Proc Ind Waste Conference*. 41:347-351. As cited in HSDB 1997.

Callahan M, Slimak M, Gabel N, et al. 1979. *Water-Related Environmental Fate of 129 Priority Pollutants. Vol 1 & 2*. Office of Water and Waste Management, U.S. Environmental Protection Agency, Washington, DC. EPA-440/4-79-029a, EPA-440/4-79-029b. pp. 87-1 to 87-13.

Chang N, Choi J. 1974. "Studies on the adsorption of pentachlorophenol (PCP) in soil." Hanguk Touang Bilyo Hakkhoe Chi 7:197-220. As cited in ATSDR 1994.

Eisler R. 1989. *Pentachlorophenol Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review.* US Fish and Wildlife Service. Biological Rep 85(1.17).

Gasiewicz T. 1991. *Nitro compounds and related phenolic pesticides.* In: Hayes W, Laws E, eds. *Handbook of Pesticide Toxicology*. Vol 3. Academic Press, New York. pp. 1191-1269.

HSDB. 1997. Hazardous Substances Data Bank.

Ide A, et al. 1972. Agric Biol Chem 36:1937-1944. As cited in HSDB 1997.

Kaufman D. 1978. *Degradation of pentachlorophenol in soil, and by soil organisms*. In: Rao K, ed. *Pentachlorophenol: Chemistry, Pharmacology, and Environmental Toxicology*. Plenum Press, New York. pp. 27-39.

Knowlton M, Huckins J. 1983. "Fate of Radiolabeled Sodium Pentachlorophenate in Littoral Microcessing." Bull Environ Contam Toxicol 30:206-213.

Kuwatsuka S, Igarashi M. 1975. "Degradation of PCP in soil: II. The relationship between the degradation." Soil Sci Plant Nutr 21:405-414. As cited in ATSDR 1994.

Makela T, Petanan T, Kukkonen J, et al. 1991. "Accumulation and depuration of chlorinated phenolics in the freshwater mussel (Anodonta anatina L.)." Ecotoxicol Environ Safety 22:153-163. As cited in ATSDR 1994.

Menzie C. 1978. *Metabolism of Pesticides*. U.S. Department of Interior, Fish and Wildlife Service. p. 221.

Murthy B. 1979. "Degradation of pentachlorophenol (PCP) in aerobic and anaerobic soil." J Environ Sci Health B 14:1-14. As cited in HSDB 1997.

Pignatello J, Martinson M, Steiert J, et al. 1983. "Biodegradation and photolysis of pentachlorophenol in artificial freshwater streams." Appl Environ Microbiol 46:1024-1031.

Rao P, Davidson J. 1982. *Retention and Transformation of Selected Pesticides and Phosphorus in Soil-Water Systems*. EPA 600/S3-82-060. As cited in HSDB 1997.

Shepard T. 1986. *Catalog of Teratogenic Agents*. 5th ed. Johns Hopkins University Press, Baltimore, MD. p. 443. As cited in HSDB 1997.

Weiss U, et al. 1982. J Agric Food Chem 30:1191-1194.

THALLIUM

1.0 SUMMARY

In the environment, thallium exists in either the monovalent (thallous) or trivalent (thallic) form. Thallium is chemically reactive with air and moisture, undergoing oxidation. Thallium is relatively insoluble in water, although thallium compounds exhibit a wide range of solubilities. Thallium adsorbs to soil and sediment and is not transformed or biodegraded. In aquatic organisms, thallium is absorbed primarily from ingestion and thereafter bioconcentrates in the organism. In mammals, thallium is absorbed primarily from ingestion and is distributed to several organs and tissues, with the highest levels reported in the kidneys. Thallium exposure in mammals causes cardiac, neurologic, reproductive and dermatological effects. Thallium is taken up by plants and inhibits chlorophyll formation and seed germination.

The following is a profile of the fate of thallium in soil, surface water and sediment; and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, surface water and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

In soil, thallium exists in either the monovalent (thallous) or trivalent (thallic) form, with the monovalent form being more common and stable and, therefore, forming more numerous salts (Hampel 1968). Thallium is reactive with air and moisture, oxidizing slowly in air at 20°C and more rapidly with increasing temperatures (Standen 1967). Moisture increases the oxidation of thallium. Thallium adsorbs to soil and is not transformed or biodegraded (Callahan et al. 1979).

Elemental thallium is relatively insoluble in water (Windholz 1976). However, thallium compounds exhibit solubilities ranging from 220 mg/L to more than 700,000 mg/L (Standen 1967; Weast 1975).

Thallium adsorbs to sediments and micaceous clays (Callahan et al. 1979; Frantz and Carlson 1987). Data regarding the transformation or biodegradation of thallium in water were not located.

3.0 ECOLOGICAL RECEPTORS

The primary exposure route for aquatic organisms exposed to thallium is ingestion. Thallium bioconcentrates in aquatic organisms (Zitko and Carson 1975). Toxic effects have been observed in numerous aquatic organisms including daphnia, fat-head minnow, sheepshead minnow, saltwater shrimp, atlantic salmon, bluegill sunfish, and others (USEPA 1980).

Birds and mammals are exposed to thallium via ingestion of soil, water, and plant material (Lie et al. 1960). Following absorption, thallium is distributed to numerous organs including the skin, liver, and muscle, with the greatest amount found in the kidneys (Downs et al. 1960; Manzo et al. 1983). Thallium is excreted primarily in the urine, with some excretion in the feces (Lehman and Favari 1985). Thallium is distributed from the maternal circulation to the fetus (Gibson et al. 1967; Gibson and Becker 1970). Various effects and toxic responses have been reported. Tikhonova (1967) reported paralysis and pathological changes in the liver, kidneys, and stomach mucosa in rabbits chronically exposed to thallium. Formigli et al. (1986) reported testicular toxicity in rats exposed to thallium. Grunfeld et al. (1963) reported changes in the electrocardiographs of rabbits following oral exposure to thallium.

Some levels of thallium occurs naturally in plants (Seiler 1988). Thallium is taken up by the roots of higher plants (Cataldo and Wildung 1983). Thallium has been shown to inhibit chlorophyll formation and seed generation (OHM/TADS 1997).

4.0 REFERENCES

ATSDR. 1992. *Toxicological Profile for Thallium*. Agency for Toxic Substances and Disease Registry. July.

Callahan M, Slimak M, Gabel N, et al. 1979. *Water-Related Environmental Fate of 129 Priority Pollutants.* Vol 1. EPA-440/4-79-029. Office of Water Planning and Standards, Washington, DC. pp. 18-1 to 18-8.

Cataldo D, Wildung R. 1983. "The role of soil and plant metabolic processes in controlling trace element behavior and bioavailability to animals." Sci Total Environ 28:159-168.

Downs, W.L., Scott J.K., Steadman L.T., Maynard E.A. 1960. "Acute and Sub-acute Toxicity Studies of Thallium Compounds." American Industrial Hygiene Association Journal. 21: 399-406.

Formigli L., Scelsi R., Poggi P., Gregotti C., DiNucci A., Sabbioni E., Gottardi L., Manzo L. 1986. "Thallium-Induced Testicular Toxicity in the Rat." Env. Res. 40: 531-539.

Frantz G, Carlson R. 1987. "Division S-2-soil chemistry: Effects of rubidium, cesium, and thallium on interlayer potassium release from transvaal vermiculite." Soil Sci Soc Am J 51:305-308.

Grunfeld O, Battilana G., Aldana L., Hinostroza G., Larrea P. 1963. "Electrocardiographic Changes in Experimental Thallium Poisoning." Am. Journal Vet Res. 24: 1291-1296.

Gibson J.E. and Becker B.A. 1970. "Placental transfer, embryo toxicity and teratogenicity of thallium sulfate in normal and potassium-deficient rats." Toxicol. Appl. Pharmacol. 16: 120. As cited in USEPA 1980.

Gibson J.E. et al. 1967. "Placental transport and distribution of thallium-204 sulfate in newborn rats and mice." Toxicol. Appl. Pharmacol. 10: 408 (abst.). As cited in USEPA 1980.

Hampel C.A. (ed.). 1968. *The Encyclopedia of Chemical Elements*. Reinhold Publishers, New York. As cited in USEPA 1980.

HSDB. 1997. Hazardous Substance Data Base

Lehman P, Favari L. 1985. "Acute thallium intoxication: Kinetic study of the relative efficacy of several antidotal treatments in rats." Arch Toxicol 57:56-60.

Lie R, Thomas R, Scott J. 1960. "The distribution and excretion of thallium-204 in the rat, with suggested mpc's and a bioassay procedure." Health Phys 2:334-340.

Manzo L, Scelsi R, Moglia A, Poggi P, Alfonsi E, Pietra R, Mousty F, Sabbioni E. 1982. "Long-term toxicity of thallium in the rat". In: *Chemical Toxicology and Clinical Chemistry of Metals.* Academic Press, London. pp. 401-405.

OHM/TADS. 1997. Oil and Hazardous Materials/Technical Assistance Data System. June.

Seiler. 1988. *Handbook of the Toxicity of Inorganic Compounds.* p. 678. As cited in HSDB 1997.

Standen A. (ed.). 1967. *Kirk-Othmer Encyclopedia of Chemical Technology*. Interscience Publishers, New York. As cited in USEPA 1980.

Tikhonova T.S. 1967. "Toxicity of thallium and its compounds in workers." Nov. Dannye Toksikol. Redk. Metal. Ikh Soedin. Chem. Abstr. 71: 53248j, 1969. As cited in USEPA 1980.

U.S. Environmental Protection Agency (USEPA). 1980. *Ambient Water Quality Criteria for Thallium*. EPA 440/5-80-074. October.

Weast R.C. (ed.). 1975. *Handbook of Chemistry and Physics. 56th ed*. CRC Press. Cleveland, Ohio. As cited in USEPA 1980.

Windholz M. (ed.). 1976. *The Merck Index. 9th Edition*. Merck and Co., Inc. Rathway, New Jersey. As cited in USEPA 1980.

Zitko V, Carson W, Carson W. 1975. "Thallium: Occurrence in the environment and toxicity to fish." Bull Environ Contam Toxicol 13:23-30. As cited in ATSDR 1992.

VINYL CHLORIDE

1.0 SUMMARY

Vinyl chloride is a low molecular weight organic compound that rapidly volatilizes after released to soil and surface water. Aquatic organisms may take up vinyl chloride, however it is rapidly depurated because it is highly water-soluble. Routes of exposure for wildlife include inhalation, ingestion, and dermal exposure. Bioaccumulation in terrestrial and aquatic organisms is not an important process in the environmental fate of vinyl chloride because of its high volatility and the rapid metabolism by higher-tropic-level receptors.

The following is a profile of the fate of vinyl chloride in soil, surface water and sediment, and the fate after uptake by ecological receptors. Section 2 discusses the environmental fate and transport in soil, surface water, and sediment. Section 3 discusses the fate in ecological receptors.

2.0 FATE IN SOIL, SURFACE WATER, AND SEDIMENT

Vinyl chloride in dry soil has a very short half-life (less than 1 day) (Jury et al. 1984). Vinyl chloride has a high vapor pressure, indicating rapid volatilization from dry soil surfaces (Riddick et al. 1986; Verschueren 1983). Vinyl chloride is also biodegraded and photolyzed in surface soil (ATSDR 1995; Nelson and Jewell 1993). Vinyl chloride does not adsorb to soil in significant amounts.

Vinyl chloride in surface water has a half-life of a few hours (Thomas 1982). An estimated half-life in fresh water for vinyl chloride of 2.5 hours was reported (Mabey et al. 1981). Vinyl chloride is slightly soluble (Cowfer and Magistro 1983). However, vinyl chloride released to surface water will quickly volatilize, negating other fate processes that might be significant based on physical and chemical parameters.

3.0 FATE IN ECOLOGICAL RECEPTORS

Vinyl chloride is not expected to significantly bioconcentrate in aquatic organisms because it has a very low log K_{ow} value. Bioconcentration and accumulation in aquatic carnivores is not expected because of the

high volatility of vinyl chloride and the rapid metabolism of vinyl chloride by higher-tropic-level organisms (Freitag et al. 1985; Lu et al. 1977).

In mammals, vinyl chloride may be absorbed by the body via inhalation (Bolt et al. 1977; Krajewski et al. 1980; Withey 1976), ingestion (Feron et al. 1981; Watanabe et al. 1976; Withey 1976) and dermal contact (Hefner et al. 1975). It is rapidly absorbed and distributed throughout the tissues following uptake. Because of the rapid metabolism and excretion of vinyl chloride, storage within the body is limited.

Information was not available on the fate of vinyl chloride in birds or plants.

4.0 REFERENCES

ATSDR. 1995. *Toxicological Profile for Vinyl Chloride.* Agency for Toxic Substances and Disease Registry. August.

Bolt H, Laib R, Kappus H, Buchter A. 1977. "Pharmacokinetics of vinyl chloride in the rat." Toxicology 7:179-188.

Cowfer J, Magistro A. 1983. *Vinyl chloride. In: Kirk-Othmer Encyclopedia of Chemical Technology.* Wiley Interscience, New York. 23:865-885.

Feron V, Hendriksen C, Speek A, Til H, Spit B. 1981. "Lifespan oral toxicity of vinyl chloride in rats." FD Cosmet Toxicol 19:317-333.

Freitag D, Ballhorn L, Geyer H, Korte F. 1985. "Environmental hazard profile of organic chemicals: An experimental method for the assessment of the behavior of organic chemicals in the ecosphere by means of simple laboratory tests with 14C labeled chemicals." Chemosphere 14:1589-1616.

Hefner R, Watanabe P, Gehring P. 1975. "Percutaneous absorption of vinyl chloride." Toxicol Appl Pharmacol 34:529-532.

HSDB. 1997. Hazardous Substance Data Bank.

Jury W, Spencer W, Farmer W. 1984. "Behavior assessment model for trace organics in soil: III. Application of screening model." J Environ Qual 13:573-579.

Krajewski J, Dobecki M, Gromiec J. 1980. "Retention of vinyl chloride in the human lung." Br J Ind Med 37:373-374.

Lu P, Metcalf R, Plummer N, Mandel D. 1977. "The environmental fate of three carcinogens: Benzo-a-pyrene, benzidine, and vinyl chloride evaluated in lab model ecosystems." Arch Environ Contam Toxicol 6:129-142.

Mabey W, Smith J, Podoll R, et al. 1981. *Aquatic Fate Process Data for Organic Priority Pollutants.* EPA 440/4-81-014. EPA Office of Water Regulations and Standards, Washington, DC. As cited in HSDB 1997.

Nelson Y, Jewell W. 1993. "Vinyl chloride biodegradation with methanotrophic attached films." J Environ Eng 119(5):890-907.

Riddick J, Bunger W, Sakano T. 1986. *Organic solvents: Physical properties and methods of purification, techniques of chemistry*. Vol II. 4th ed. John Wiley & Sons, New York. pp. 488-489. As cited in HSDB 1997.

Thomas R. 1982. *Volatilization from water*. In: Lyman W, Reehl W, Rosenblatt D, eds. *Handbook of Chemical Property Estimation Methods*. McGraw-Hill Book Company, New York. PP 15-1 TO 15-34.

Verschueren K. 1983. *Handbook of Environmental Data on Organic Chemicals*. 2nd ed. Van Nostrand Reinhold Co., New York. pp. 1185-1186.

Watanabe P, McGowan G, Gehring P. 1976. "Fate of [14C]vinyl chloride after single oral administration in rats." Toxicol Appl Pharmacol 36:339-352. As cited in ATSDR 1995.

Withey J. 1976. "Pharmacodynamics and uptake of vinyl chloride monomer administered by various routes to rats." J Toxicol Environ Health 1:381-394.