Antimony

Antimony is a silvery white metal of medium hardness that breaks easily. Antimony is used as a component of lead and zinc alloys which are used in lead storage batteries, solder, sheet and pipe metal, bearings, castings, type metal, ammunition and pewter. Exposure to antimony may occur through the ingestion of food or water, via breathing of air or through contact with soil, water or other substances that contain antimony. Skin contact and inhalation are common occupational exposures. A small amount of ingested antimony is absorbed into the bloodstream after a few hours. An unknown amount of inhaled antimony is absorbed through the lungs within a few days. Most of the absorbed antimony is transported to the liver, lungs, intestines and spleen. Within several weeks, antimony is excreted in the feces and urine (ATSDR, 1990).

Acute symptoms of antimony exposure include, diarrhea, vomiting, gastric discomfort and ulcers following oral ingestion of large quantities (< 19 ppm). Animal studies indicate that acute exposures may result in lung, heart, liver and kidney damage, eye irritation following inhalation of antimony, and skin irritation following dermal contact. Subchronic exposure to antimony via inhalation leads to heart problems, stomach ulcers, pneumoconiosis and eye and skin irritation. Animal studies indicate that subchronic ingestion of antimony may cause diarrhea, weight loss, liver damage and decreased red blood cell count (ATSDR, 1990).

Antimony in the atmosphere is in the form of particulate matter or adsorbed to particulate matter. Transport to land and surface water occurs through gravitational settling and other forms of dry and wet deposition. The fate of antimony in the environment is complicated because it can exist in two oxidation states, 3+ and 5+. In the aquatic environment, antimony is mainly associated with particulate matter and tends to settle out in areas of active sedimentation. Some forms of antimony are strongly sorbed to soil, making it relatively immobile. Antimony may also adsorb strongly to colloidal materials in soil which may become mobilized and transported to groundwater. In general, adsorption is greatest at near neutral pHs. Antimony does not appear to bioconcentrate in fish and aquatic organisms (ATSDR, 1990).

Arsenic

Arsenic is a naturally occurring element and enters the environment as a result of natural forces (volcanoes, weathering) and human activities such as metal smelting, glass manufacturing, pesticide production and application, and fossil-fuel burning (ATSDR, 1991). In general, inorganic arsenic is more toxic than organic arsenic. The most common exposure route is ingestion of arsenic in food or water. Inhalation and skin contact are secondary routes of exposure. Arsenic is quickly absorbed through the lungs or digestive tract into the bloodstream. Within a few hours most of the absorbed arsenic is cleared from the blood and is excreted in the urine (ATSDR, 1991).
Large doses of ingested inorganic arsenic (20 mg/kg or greater) induce death while smaller doses produce systemic effects such as irritation of the digestive tract, nausea, vomiting, and diarrhea. In addition, there are effects on cells that produce blood, abnormal heart function, blood vessel damage, liver or kidney injury, and impaired nerve functioning. The ingestion of arsenic in drinking water has been associated with an elevated incidence of skin cancer. Epidemiological data demonstrate an association between occupational exposure to inhaled arsenic and lung cancer (ATSDR, 1991). The USEPA classifies arsenic as a group A carcinogen (sufficient evidence of carcinogenicity in humans) (IRIS, 1995).

Arsenic is a non-volatile solid. The mobility of arsenic in the environment depends on the solubility of the particular chemical form present. Most arsenic in the air is adsorbed to particulate matter and settles out according to particle size. Arsenic found in the soil is predominantly an insoluble, adsorbed form. Arsenic in soil and water may be reduced and methylated by soil organisms. Bioconcentration of arsenic occurs primarily in aquatic algae and lower invertebrates. Biomagnification varies by species with some fish and invertebrates containing elevated levels of arsenic compounds. Terrestrial plants uptake arsenic from the soil and air (ATSDR, 1991).

1,2-DICHLOROETHANE

1,2-Dichloroethane is used primarily as a starting material in the manufacture of vinyl chloride, tetrachloroethylene, trichloroethylene, and other chlorinated organic compounds. It is also used as a degreasing agent, solvent, fumigant for grain and upholstery, varnish and paint remover, and lead-scavenging agent in gasoline. Primary routes of exposure include breathing air, drinking water, and skin absorption (IRP, 1989).

Short-term exposures to vapor concentrations greater than 125 ppm produce irritation of the eyes, nose and throat. Ingestion or inhalation of the compound causes dizziness, nausea, vomiting, increasing stupor, cyanosis, rapid pulse, loss of consciousness, and injury to the liver, kidneys and lungs. The dermal LD<sub>50</sub> in rabbits is 2800 mg/kg of body weight (IRP, 1989). In addition, reproductive effects have been noted in rats at an oral dose of 120 mg/kg-day (ATSDR, 1992).

Chronic exposures are associated with loss of appetite, nausea, vomiting, gastric pain, neurological disturbances, and liver and kidney impairment. In animal studies, death was outcome for inhalation exposures greater than 400 ppm. The USEPA has classified 1,2-dichloromethane as a Group B2 carcinogen (probable human carcinogen) (IRP, 1989).

1,2-Dichloroethane is expected to be highly mobile in the soil/ground-water system. Adsorption to soil is low; volatilization from soils is the primary transport process. Microbial degradation in soil is not expected to be significant (IRP, 1989).
1,1-DICHLOROETHENE

1,1-Dichloroethene (1,1-DCE), also known as 1,1-dichloroethylene and vinylidene chloride, is a clear, colorless liquid that has a mild, sweet chloroform-like odor. It is a man-made chemical used to make copolymers, modacrylic fibers and other chemicals. It evaporates quickly at room temperature and is flammable. 1,1-DCE may enter the body by inhalation of contaminated air or ingestion of contaminated food or water (ATSDR, 1992).

There have been no studies that reported death of humans following inhalation exposure to 1,1-DCE. Lethality in laboratory animals after inhalation exposure varies considerably depending on species, strain, sex and nutritional status. When experimental animals were exposed to high concentrations of 1,1-DCE, damage to the liver, kidney and central nervous system has been reported. The liquid is moderately irritating to the eyes and skin (ATSDR, 1992).

Long-term effects of 1,1-DCE include liver and kidney damage. There is also evidence of mutagenicity in several test systems. A statistically significant increase in kidney tumors was observed in mice exposed to 1,1-DCE via inhalation. Other tumor types were also reported in this study. The USEPA has classified 1,1-DCE as a Group C carcinogen (possible human carcinogen) (IRP, 1989).

The environmental fate of 1,1-DCE is influenced largely by its high volatility; the majority evaporates to the atmosphere. Because it is water soluble and weakly adsorbed to soil/sediment, the potential for surface water or ground-water contamination and transport is great. Transformation processes are not expected to be great in natural soil (IRP, 1989).

DIELDRIN

Dieldrin is a chlorinated hydrocarbon compound that has been widely used as a domestic pesticide. Its primary use in the past was as an insecticide for corn and for termite control. Human exposure can result from inhalation and ingestion. Dermal exposure is limited to the past for those involved in manufacturing or application of pesticides containing dieldrin. Dermal exposure could occur when contact at hazardous waste sites or contaminated soils/sediments.

Dieldrin is absorbed into the bloodstream from the gastrointestinal tract after ingestion or from the lungs after inhalation. It is quickly distributed throughout the body after intake, but is rapidly concentrated in fatty tissues due to its lipophilic nature where it can remain for years. Other organs which tend to have high concentrations are the liver, kidneys, brain, and blood. Dieldrin is excreted, mainly in the feces, in the form of several metabolites that are more polar than the parent compounds (ATSDR, 1991).

Signs of toxicity include effects on the central nervous system with symptoms of headache, dizziness, nausea, general malaise, and vomiting, followed by muscle twitching, myoclonic jerks,
and even convulsions. These symptoms are reversible with time after removal from the source of exposure. Death may result from anoxemia (ATSDR, 1991).

No chronic effects have been observed in humans exposed to low levels of dieldrin in the workplace. Animal studies, however, have indicated a decrease in immune function and liver damage resulting from dieldrin exposure. In addition, liver cancer has been observed in mice chronically exposed to dieldrin (ATSDR, 1991). Dieldrin has been classified by the USEPA as a probable human carcinogen, class B2. There is sufficient evidence that exposure to dieldrin has resulted in liver cancer in animal studies (IRIS, 1995).

HEPTACHLOR/HEPTACHLOR EPOXIDE

Heptachlor is a man-made pesticide used in homes, buildings, and on food crops, but it is no longer manufactured in the United States. It is a white powder in its pure form, while technical grade heptachlor is a tan powder. It is a component of the pesticide, chlordane. Heptachlor epoxide is a breakdown product of heptachlor, and is a result of bacterial activity (ATSDR, 1992).

Heptachlor is readily absorbed into the gastrointestinal tract and the skin. It is slowly eliminated via the bile duct to the feces. Heptachlor epoxide is an oxidation product which is formed by plants and animals after exposure to heptachlor. Heptachlor epoxide is often detected in human milk, blood and other body tissues and is more harmful than heptachlor itself. Other breakdown products are less harmful (ATSDR, 1992).

Heptachlor is acutely toxic via the oral and dermal routes. No studies are available to show that inhalation of heptachlor is harmful to humans. Hepatotoxicity is the most sensitive noncancer endpoint with animal acute and chronic studies describing evidence of severe liver damage, increased liver weight, and increased levels of serum liver enzymes. Central nervous system disorders are also evident. Long-term oral exposures in animals are also associated with kidney, adrenal, and blood defects. It is also fetotoxic and caused reduced fertility in laboratory rodents. Chronic oral exposure to heptachlor or heptachlor epoxide increased the incidence of liver carcinomas in several species of mice. Studies of pesticide applicators indicate a slight increased incidence in cancers of the lung, skin and bladder (ATSDR, 1992). The USEPA classifies heptachlor and heptachlor epoxide as group B2 carcinogens (probable human carcinogens) (IRIS, 1995).

Heptachlor and its epoxide are persistent in soil with half-lives of two and fourteen years, respectively. Heptachlor and its epoxide can evaporate into the air and ultimately travel long distances. Heptachlor epoxide dissolves more easily in water than does heptachlor and evaporates slowly. Heptachlor does not dissolve easily in water and will bind to sediments. In soil and water heptachlor is broken down by bacteria to its epoxide and other substances. Bioaccumulation of both heptachlor and heptachlor epoxide occur in aquatic and terrestrial organisms, where they are stored in fatty tissues for long periods of time (ATSDR, 1992).
LEAD

Lead is a commonly used, naturally occurring metal which is ubiquitous throughout the environment. Lead is found in construction materials, leaded gasoline, radiation protection gear, paint, ceramics, plastics, antimonial lead storage batteries, and ammunition. Lead is well absorbed from all portions of the respiratory tract including the nasal passages. Absorption from the gastrointestinal tract is less rapid and complete than from the respiratory tract. Dermal absorption is a much less significant route of lead absorption than inhalation or ingestion. Absorbed lead is distributed to the soft tissues of the body with the greatest distribution to the kidneys and the liver. Lead is eventually transferred to the skeleton where 90% of the body’s long-term burden is stored. Approximately 70% of the absorbed lead dose is excreted. The portion of lead that is not absorbed is excreted in the feces. Most of the absorbed lead is excreted by the kidneys or through biliary clearance into the gastrointestinal tract (ATSDR, 1988).

Lead intoxication in humans can occur by ingestion and inhalation of dust or fumes. Lead interferes with the blood making process, production of energy, and transmission of nerve impulses. Symptoms of lead intoxication include anorexia, malaise, headaches, and intestinal spasms. The neuromuscular disease, lead palsy, is a result of advanced subacute poisoning (lead blood levels of 70 Tg/dL and less), and is characterized by muscle weakness leading to paralysis. Lead encephalopathy is the term used for the central nervous system manifestation which is commonly seen in children when lead blood levels reach 90 Tg/Dl. Symptoms include clumsiness, dizziness, delirium, convulsions, and coma. The mortality rate is 25% when the brain is involved, with survivors suffering long-term neurological problems (ATSDR, 1988).

Chronic low level lead exposure (lead blood levels of 30-50 Tg/Dl) is associated with learning disabilities. Lead toxicity is defined by the Centers for Disease Control as a blood level of 10 Tg/Dl or greater (child). Kidney damage occurs after prolonged exposures, and is apparently reversible. In epidemiological studies, lead intoxication is also associated with increased blood pressure which is symptomatic of kidney damage. Lead exposure is associated with reproductive effects such as miscarriages and temporary sterility. Lead readily crosses the placenta. In all systems, the concentrations of essential nutrients and elements have a significant impact on the degree of toxicity seen with lead exposures. Occupational exposure to airborne lead is associated with an increased incidence of total malignant neoplasms, cancers of the digestive tract and cancers of the respiratory tract. An increased incidence in kidney cancer was seen in lead smelter workers exposed by inhalation and in various animal species exposed by ingestion at levels of 500 ppm and above. The USEPA has classified lead as a group B2 carcinogen based on animal studies (probable human carcinogen with inadequate or no evidence in humans) (ATSDR, 1988; IRIS, 1995; USEPA, 1994).

The mobility of lead in soil is dependent on the chemical properties of the soil. Lead can react with sulfates, carbonates, and phosphates or combine with clays and organic matter which limits the further migration of lead through the soil matrix. Lead in surface waters is usually present as suspended solids. Atmospheric lead is removed by dry deposition and rainout. Lead does not
significantly bioaccumulate in fish. Lead localizes in fish skin which serves to reduce human exposures by fish consumption. Lead is toxic to wildlife, particularly water fowl, through their consumption of lead shot. Tetraethyl lead is biodegradable, but inorganic lead concentrations above 5 Tg/L can be toxic to microorganisms. As water hardness increases, the acute toxicity of lead to freshwater aquatic species decreases (ATSDR, 1988).

POLYCYCLIC AROMATIC HYDROCARBONS

Polycyclic aromatic hydrocarbons (PAHs) are a diverse class of compounds formed as a result of incomplete combustion of organic compounds with insufficient oxygen. As pure chemicals, PAHs generally exit as colorless, white, pale yellow, or green solids. This leads to the formation of C-H free radicals which can polymerize to form various PAHs. Although the health effects of the individual PAHs are not exactly alike, the following PAHs are considered as three groups in this profile (ATSDR, 1993):

**Low Molecular Weight Compounds** (152-178 g/mol)
- acenaphthene
- acenaphthylene
- anthracene
- fluorene
- phenanthrene

**Medium Molecular Weight Compounds** (202 g/mol)
- fluoranthene
- pyrene

**High Molecular Weight Compounds** (228-278 g/mol)
- benzo(a)anthracene (B[a]A)
- benzo(b)fluoranthene (B[b]F)
- benzo(j)fluoranthene (B[j]F)
- benzo(k)fluoranthene (B[k]F)
- benzo(g,h,i)perylene (B[ghi]P)
- benzo(a)pyrene (B[a]P)
- benzo(e)pyrene (B[e]P)
- chrysene
- dibenz(a,h)anthracene (D[ah]A)
- indeno(1,2,3-cd)pyrene (I[123cd]P)
PAHs are present in the environment from both natural and anthropogenic sources. As a group, they are widely distributed in the environment. Humans may be exposed to PAHs in the environment, in tobacco smoke, in cooked food, and in the workplace. Typically, individuals are not exposed to a single PAH, but to a mixture of related chemicals (ATSDR, 1993). PAHs are readily absorbed into the bloodstream from the gastrointestinal tract after ingestion or the lungs after inhalation. PAHs are metabolized primarily in the liver and excreted in the feces.

Most of the information available for PAHs are from studies on experimental animals. Adverse effects in humans are generally not observed, but have been documented. Hematologic effects (myelosuppression) were produced in people after intravenous administration of anthracene-containing chemotherapeutic agents. Dermal effects have been documented. Regressive verrucae followed repeated topical application of B[a]P over a four-month period. In animals, oral administration of PAHs affect proliferating organs and tissue such as bone marrow, lymphoid organs, and intestinal epithelium (ATSDR, 1993).

PAHs are well established as experimental carcinogens for all routes through which humans would normally be expected to be exposed. In human occupational studies, lung and skin cancer have been demonstrated after inhalation exposure to PAHs. These workers were employed in coke production plants as roofers and as oil refinery workers. In experimental animals, the site of tumor induction is generally the point of first contact with the PAHs (i.e., stomach tumors after ingestion, lung tumors after inhalation, etc.) (ATSDR, 1993). The following PAHs are classified as B2 carcinogens by the USEPA: B[a]A, D[ah]A, B[a]P, B[b]F, B[k]F, chrysene and I[123cd]P; these PAHs are probable human carcinogens. Anthracene, B[ghi]P, pyrene, fluorene, naphthalene, fluoranthene, acenaphthene and phenanthrene are class D carcinogens (not classifiable as to human carcinogenicity). No data exist on the carcinogenicity of acenaphthalene (IRIS, 1995).

Some of the transport and partitioning characteristics (Henry's law constant, $K_{oc}$ values, $K_{ow}$ values) of the 17 PAHs are roughly correlated to their molecular weights. PAH compounds in water tend to be removed by volatilization, binding to particulates or sediments or being bioaccumulated. The low molecular weight PAHs have Henry's Law constants in the range of $10^{-3}$ to $10^{-5}$ atm-m$^3$/mol and are therefore associated with significant volatilization. The other PAHs volatilize to a lesser extent. In the atmosphere they are associated with particulate matter, especially soot, and can travel long distances. PAHs suspended in the air are thought to undergo direct photolysis very quickly (ATSDR, 1993).

PAHs have low water solubilities and high propensity for binding to particulate or organic matter. In general PAHs do not easily dissolve in water although they have been detected in groundwater in some distances. Medium and high molecular weight PAHs are primarily removed from the water column by deposition and some volatilization (they have $K_{oc}$ values of $10^4$ to $10^6$ indicating strong tendencies to adsorb). Low molecular weight PAHs are removed by volatilization and biodegradation (they have $K_{oc}$ values $10^5$ to $10^4$ indicating moderate potential to bind). In general sorption of PAHs to soil and sediment increases with increasing organic carbon content and is also directly dependent on particle size. The ultimate fate of PAHs in the sediment is believed to be biodegradation and biotransformation by microbes. In soils and water PAH breakdown generally takes weeks to months and is due primarily to the actions of microorganisms. Photodegradation also plays a role in PAH breakdown in water (ATSDR, 1993).
PAHs can be accumulated in organisms: the higher molecular weight compounds accumulating more easily than the lower molecular weight ones. Most organisms, however, metabolize and excrete PAHs rapidly, resulting in short lived bioaccumulation (ATSDR, 1993).

1,1,2,2-TETRACHLOROETHANE

1,1,2,2-Tetrachloroethane was commonly used to produce other chemicals such as paints and pesticides or as an industrial solvent and degreasing agent. Now, it is introduced into the environment as a minor impurity or chemical intermediate of other chlorinated solvents. Exposure may occur through inhalation of contaminated air, dermal contact or incidental ingestion in water or food (ATSDR, 1994).

Inhalation of airborne fumes at greater than 360 ppm can cause fatigue, vomiting, dizziness, and possibly unconsciousness. Breathing, drinking, or skin contact with lower concentrations may cause liver damage, stomach upsets, or dizziness. An oral acute LOAEL value of 75 mg/kg/day was established for liver damage in rats (ATSDR, 1994).

Long-term effects of human exposures are not established. Reproductive, systemic, or carcinogenic effects have not been noted in human populations. Chronic oral studies with rats noted reduced body weights (108 mg/kg/day) and renal effects (284 mg/kg/day). Based on a NCI mouse study, it is suspected to be a promoter of hepatic tumors. This compound has been classified as a group C carcinogen (limited evidence of carcinogenicity in animals and inadequate data for humans) (ATSDR, 1994).

1,1,2,2-Tetrachloroethane when released to the atmosphere is fairly stable with an estimated half-life in the troposphere of two years. When released to water, the compound will be lost by volatilization in a period of days. The compound is not expected to adsorb to soil, suspended solids, and sediment unless in high clay, dry soils. The bioconcentration factor in fathead minnows has been reported as 49 which indicates little tendency for the compound to bioaccumulate in fish and aquatic organisms (ATSDR, 1994).

TETRACHLOROETHENE (PCE)

Tetrachloroethene or tetrachloroethylene is a clear, colorless, nonflammable liquid that has a characteristic odor. It is a widely used solvent, particularly as a dry cleaning agent, a degreaser, a chemical intermediate, and a fumigant, and was given orally as a medical treatment for hookworms. The most significant exposure probably occurs in the industrial environment by inhalation. It is readily absorbed after ingestion or inhalation, but dermal absorption is poor. However, skin irritation may result from direct contact with the undiluted liquid. The main excretion pathway is through exhalation of the unmetabolized compound (ATSDR, 1992).

In confined, poorly ventilated areas, single exposures to high concentrations of tetrachloroethene can result in dizziness, headache, sleepiness, confusion, nausea, difficulty in speaking and walking, and possibly unconsciousness and death. The consequences of chronic exposure to
Tetrachloroethene by breathing or ingesting low levels of the chemical are not known. In laboratory animals, studies were conducted using higher concentrations than normally found in an environmental setting. These studies suggested the potential for tetrachloroethene to result in liver and kidney damage, birth defects, toxicity to pregnant animals, liver cancer and leukemia. Based on evidence from animal studies, tetrachloroethene is considered to be carcinogenic. The USEPA classifies tetrachloroethene as a B2 carcinogen (probable human carcinogen based on animal studies, but inadequate or no evidence in humans).

Tetrachloroethene found in surface waters or on soil surfaces will predominantly evaporate into the atmosphere. However, tetrachloroethene is moderately to highly mobile in soil and susceptible to leaching. In subsurface soils where volatilization cannot occur, tetrachloroethene is only slowly degraded and may be relatively persistent. Studies have shown that tetrachloroethene has a low bioaccumulation potential (ATSDR, 1992).

**TETRACHLOROMETHANE (Carbon Tetrachloride)**

Tetrachloromethane, more commonly referred to as carbon tetrachloride, is a clear, heavy liquid with a sweet aromatic odor. It is a synthetic chemical with no natural sources. Because it evaporates very easily, it is not usually encountered in its liquid state in the environment. Carbon tetrachloride is readily absorbed from the gastrointestinal tract and more slowly absorbed through the lungs and skin. Most carbon tetrachloride leaves the body by being exhaled through the lungs within a few hours after exposure.

Acute exposures of carbon tetrachloride to humans have shown a wide range of effects. Prior exposure to alcohol, phenobarbital, and some pesticides have been shown to increase the effects of carbon tetrachloride. Single exposures to low concentrations may cause symptoms such as irritation of the eyes, moderate dizziness and headache which disappear once exposure is discontinued. Exposure to higher concentrations will cause the same symptoms as above, but additional symptoms of nausea, loss of appetite, mental confusion, agitation and the feeling of suffocation may be seen. Chronic exposure to carbon tetrachloride produces symptoms of fatigue, lassitude, giddiness, anxiety, headache and muscle twitching. Organ damage is usually restricted to the liver, although there are some reported cases of kidney damage. After chronic exposure there is usually regeneration in these organs. Carbon tetrachloride is carcinogenic in animals producing mainly liver tumors. The USEPA has classified carbon tetrachloride as a group B2 carcinogen indicating that, based on animal studies, it is probably a human carcinogen, although there are no adequate studies of cancer in humans.

Most carbon tetrachloride is released to the environment in the atmosphere. Although it is moderately soluble in water, its high rate of volatilization results in only about 1% of the total carbon tetrachloride in the environment being in surface waters and oceans. Likewise, carbon tetrachloride tends to volatilize from tap water used for showering, bathing and cooking inside a home (ATSDR, 1989).
1,1,1-TRICHLORETHANE

1,1,1-Trichloroethane (1,1,1-TCA) is a colorless liquid with a sweet characteristic odor. It is used as a solvent for adhesives, in metal degreasing, in textile processing, as an aerosol propellant and in spot cleaners. 1,1,1-TCA can enter the body through the lungs by breathing contaminated air or through the digestive system by eating or drinking contaminated food or water. Most 1,1,1-TCA is exhaled regardless of how it entered the body, but small metabolized amounts leave in the urine (ATSDR, 1990).

Acute inhalation exposure to concentrations greater than 500 ppm of 1,1,1-TCA in humans may result in dizziness, lightheadedness and loss of balance and coordination. These effects are reversible when the exposure is discontinued. Continued breathing of higher concentrations of 1,1,1-TCA could lead to unconsciousness, a decrease in blood pressure and cardiac arrest. Although the health effects of long-term low dose exposure in humans is unknown, studies in experimental animals have shown that damage occurs to the breathing passages, lungs and liver following inhalation of high levels. Studies in experimental animals have shown that exposure to high concentrations of 1,1,1-TCA during pregnancy could result in birth defects. If 1,1,1-TCA comes into direct contact with skin for more than five minutes, a mild irritation may occur, but would disappear in a few hours after removal of the 1,1,1-TCA. The USEPA classifies 1,1,1-TCA in group D (not classifiable as to human carcinogenicity).

1,1,1-TCA evaporates easily and is moderately water soluble. It volatilizes from soil, surface water, and from unconfined ground water to the soil. If released to soil as a liquid, 1,1,1-TCA does not sorb to soil and may leach to groundwater. 1,1,1-TCA is not believed to bioconcentrate in fish and aquatic organisms (ATSDR, 1990).

TRICHLOROETHENE (TCE)

Trichloroethene (TCE) or trichloroethylene is a colorless liquid at room temperature with an odor similar to ether. The major use of this chemical is as a solvent for degreasing metal parts. Exposure to TCE can occur via inhalation and by ingestion of contaminated food and water. Absorption of TCE following inhalation exposure is high with approximately 50% of the inhaled dose absorbed and 50% exhaled. Dermal absorption is poor in humans. Once absorbed the majority of the TCE is metabolized and then excreted in the urine, only a relatively small amount of absorbed TCE is exhaled via the lungs (ATSDR, 1991).

Trichloroethene is not acutely toxic by the inhalation or oral routes. Oral and inhalation exposures affect the central nervous system, liver and kidney. Trichloroethene was once used as an anesthetic; inhalation of high doses (5000 ppm) produces anesthetic effects. Human epidemiology studies have not shown a clear connection between exposure to trichloroethene and increased cancer risk (ATSDR, 1991). Laboratory animals exposed by inhalation developed cancers in the lung and liver, while animals exposed orally had increased incidence of liver and kidney carcinomas. The USEPA classifies TCE as a group B2 carcinogen (probable human carcinogen but inadequate or no
Evidence in humans (HEAST, 1994). The USEPA is currently re-evaluating the toxicity and carcinogenicity of trichloroethene (IRIS, 1994).

Environmentally, trichloroethene volatilizes rapidly from water. It is highly mobile in soil and quickly leaches to the groundwater. It exists predominantly in the vapor phase with some removal from the atmosphere via wet deposition. TCE is believed to have a low bioaccumulation potential in fish and other aquatic creatures (ATSDR, 1991).

**TRICHLOROMETHANE (Chloroform)**

Chloroform is a colorless or water-white liquid with a pleasant non-irritating odor. It can enter the body by breathing air, drinking water or eating food that contains chloroform. Because chloroform can penetrate the skin, it may enter the body by bathing or showering in water containing chloroform. Water that has been chlorinated for disinfectant purposes may contain chloroform as a by-product. In general chloroform is rapidly eliminated from the body (ATSDR, 1991).

Short-term inhalation exposure to high concentrations (900 ppm) results in CNS effects such as tiredness, dizziness and headache while higher concentrations (8,000 to 10,000 ppm) may result in unconsciousness and death. Longer-term exposure to chloroform can affect liver and kidney function. Dermal exposures may cause sores on the skin. Chloroform was used as a surgical anesthetic for many years before its harmful effects on the liver and kidneys were recognized. Chronic oral doses of chloroform at concentrations greater than 60 mg/kg/day have been found to result in liver and kidney cancer in laboratory animals. Epidemiological studies found a correlation between chlorinated drinking water and cancer of the bladder, large intestine and rectum in humans. However, chloroform is only one of many chlorinated compounds found in chlorinated drinking water that are potentially carcinogenic. The USEPA classifies chloroform as a group B2 carcinogen (probable human carcinogen based on animal studies, but inadequate or no evidence in humans) (IRIS, 1994).

Chloroform is released to the environment as a result of its manufacture and use in the chlorination of water and from other water treatment processes. Most of the chloroform released to the environment will eventually volatilize from water and soil to the atmosphere. Chloroform in the atmosphere may be degraded by photochemical reactions. Because of its limited ability to sorb to soil and its high water solubility, chloroform will leach from soil to groundwater where it may persist for a long time (ATSDR, 1991).

**VINYL CHLORIDE**

Vinyl chloride is a colorless gas with a mild, sweet odor. It is used to make polyvinyl chloride (PVC), as a refrigerant gas, and in the manufacture chlorinated compounds.

Acute exposure in humans to approximately 10,000 ppm vinyl chloride for five minutes results in central nervous system effects including dizziness, disorientation, nausea, and headaches. Death
has resulted when humans were acutely exposed to high levels of vinyl chloride. Acute inhalation of 100,000 to 400,000 ppm vinyl chloride has resulted in death in laboratory animals. Inhalation of vinyl chloride has been reported to result in impaired liver function, liver damage, and central nervous system effects at doses as low as 10 ppm in laboratory animals. Chronic inhalation exposure has also resulted in a syndrome known as vinyl chloride disease. Symptoms include circulatory disturbances in the extremities, and blood, lung, and liver effects. In animals, chronic exposure to oral and inhaled vinyl chloride resulted in decreased longevity, vinyl chloride syndrome, toxic hepatitis, kidney effects, and cancer (ATSDR, 1991). Vinyl chloride has been classified as a group A carcinogen in humans (HEAST, 1994). Increases in the occurrence of tumors in the liver (angiosarcomas), brain, lung, and blood making tissues have been associated with occupational exposure to vinyl chloride in humans (ATSDR, 1991).

Vinyl chloride is a highly mobile compound and may leach into ground water. It does not adsorb to soil. Vinyl chloride in surface water will volatilize to the atmosphere. In the atmosphere, vinyl chloride exists as a vapor and is rapidly degraded. It does not bioconcentrate significantly in aquatic organisms (ATSDR, 1991; Howard, 1990).
Appendix A
Toxicology Profiles for Contaminants

A.1 Inorganic Arsenic

Toxicity Classification:
EPA: Group A (known human carcinogen)

Toxicity Criteria:

Reference Dose (RfD)—Oral: 0.0003 milligrams per kilogram (mg/kg)/day
Uncertainty/Modifying Factor: 3
Principal Studies: Tseng et al., 1968; Tseng, 1977

Cancer Slope Factor—Oral: 1.5 (mg/kg/day)^{-1}
Principal Studies: Tseng et al., 1968; Tseng, 1977

Cancer Slope Factor—Inhalation: 4.3 E^{-3} (mg/m^3)^{-1}
Principal Studies: Brown and Chu, 1983a-c; Lee-Feldstein, 1983; Higgins, 1982; Enterline and Marsh, 1982

Target Organs (Primary):

- skin (hyperpigmentation and hyperkeratosis)
- nervous system (peripheral neuropathy)
- vascular system
- hematopoietic system
- gastrointestinal
- lungs
- liver
- kidney

A.1.1 General Information

The toxicity of inorganic arsenic (As) depends on its valence state (-3, +3, or +5), and also on the physical and chemical properties of the compound in which it occurs. Trivalent (As^{+3}) compounds are generally more toxic than pentavalent (As^{+5}) compounds, and the more water soluble compounds are usually more toxic and more likely to have systemic effects than the less soluble compounds, which are more likely to cause chronic pulmonary effects if inhaled.

One of the most toxic inorganic arsenic compounds is arsine gas (AsH_3). It should be noted that laboratory animals are generally less sensitive than humans to the toxic effects of inorganic arsenic. In addition, in rodents, the critical effects appear to be immunosuppression and hepato-
renal dysfunction, whereas in humans the skin, vascular system, and peripheral nervous system are the primary target organs.

Water soluble inorganic arsenic compounds are absorbed through the G.I. tract (>90 percent) and lungs; distributed primarily to the liver, kidney, lung, spleen, aorta, and skin; and excreted mainly in the urine at rates as high as 80 percent in 61 hrs following oral dosing (EPA, 1984). Pentavalent arsenic is reduced to the trivalent form and then methylated in the liver to less toxic methyarsinic acids (ATSDR, 1989).

A.1.2 Basis for Toxicity Criteria

The Reference Dose (RfD) for chronic oral exposures, 0.0003 mg/kg/day, is based on a No Observed Adverse Effects Level (NOAEL) of 0.0008 mg/kg/day and a Lowest Observed Adverse Effects Level (LOAEL) of 0.014 mg/kg/day for hyperpigmentation, keratosis, and possible vascular complications in a human population consuming arsenic-contaminated drinking water (EPA, 1991a). Because of uncertainties in the data, the U.S. Environmental Protection Agency (EPA) (1991a) states that "strong scientific arguments can be made for various values within a factor of 2 or 3 of the currently recommended RfD value." The subchronic Reference Dose is the same as the chronic RfD, 0.0003 mg/kg/day (EPA, 1992).

Acute inhalation exposures to inorganic arsenic can damage mucous membranes, cause rhinitis, pharyngitis and laryngitis, and result in nasal septum perforation (EPA, 1984). Epidemiological studies have revealed an association between arsenic concentrations in drinking water and increased incidences of skin cancers (including squamous cell carcinomas and multiple basal cell carcinomas), as well as cancers of the liver, bladder, respiratory, and gastrointestinal tracts (EPA, 1987; IARC, 1987; Chen et al., 1985, 1986). Occupational exposure studies have shown a clear correlation between exposure to arsenic and lung cancer mortality (IARC, 1987; EPA, 1991a). EPA (1991a) has placed inorganic arsenic in weight-of-evidence group A, human carcinogen. A drinking water unit risk of 5E-5(mg/L)^{-1} has been proposed (EPA, 1991a); derived from drinking water unit risks for females and males that are equivalent to slope factors of 1.0E-3 (mg/kg/day)^{-1} (females) and 2.0E-3 (mg/kg/day)^{-1} (males) (EPA, 1987). For inhalation exposures, a unit risk of 4.3E-3 (mg/m^3)^{-1} (EPA, 1991a) and a slope factor of 5.0E+1 (mg/kg/day)^{-1} have been derived (EPA, 1992).

Symptoms of acute inorganic arsenic poisoning in humans are nausea, anorexia, vomiting, epigastric and abdominal pain, and diarrhea. Dermatitis (exfoliative erythroderma), muscle cramps, cardiac abnormalities, hepatotoxicity, bone marrow suppression and hematologic abnormalities (anemia), vascular lesions, and peripheral neuropathy (motor dysfunction, paresthesia) have also been reported (U.S. Air Force, 1990; ATSDR, 1989; EPA, 1984). Primary target organs are the skin (hyperpigmentation and hyperkeratosis) [Terada et al. 1960; Tseng et al., 1968;], nervous system (peripheral neuropathy), and vascular system [Tseng et al., 1968]. Anemia, leukopenia, hepatomegaly, and portal hypertension have been reported. In addition, possible reproductive effects include a high male-to-female birth ratio.

In animals, acute oral exposures can cause gastrointestinal and neurological effects. Chronic exposures have also resulted in mild hyperkeratosis and bile duct enlargement with hyperplasia, focal necrosis, and fibrosis. Reduction in litter size, high male-to-female birth ratios, and fetotoxicity without significant fetal abnormalities occur after oral dosing; however, parenteral
dosing has resulted in exencephaly, encephaloceles, skeletal defects, and urogenital system abnormalities.

It is reported that a 12 percent incidence of skin abnormalities occurred in children whose drinking water contained 0.6 to 0.8 mg As/L. The earliest cases occurred about 4 to 5 years after the initial exposure. Cardiovascular effects, including Raynaud's syndrome, acrocyanosis, angina pectoris, hypertension, myocardial infarction, mesenteric thrombosis, systemic occlusive arterial disease, bronchiectasis, and recurrent broncho-pneumonia were also observed in this group of subjects. The bronchiectasis and recurrent broncho-pneumonia were attributed to an immunosuppressive action of arsenic in the lungs. A significant decrease in the incidence of skin abnormalities was observed after a reduction in drinking water concentration to about 0.04 mg/L. After 4 years at the lower exposure, effects were rarely seen in children younger than 12 years old. Central nervous system deficits (hearing loss, eye damage, abnormal EEGs, mental retardation, and epilepsy), electrocardiographic changes (elevated ST wave and extended QT interval), and skin abnormalities (melanosis, desquamation, rashes, and hyperkeratosis) occurred in infants who had been fed arsenic-contaminated milk for 1 to 2 months. It was estimated that the daily arsenic intake was about 3 mg/day (EPA, 1984).

A.1.3 References


A.2 Polycyclic Aromatic Hydrocarbons (PAHs)

Toxicity Classification:

**EPA:**  
B2 (probable human carcinogen, includes carcinogenic PAHs; B(a)P, B(a)A, B(b)F, B(k)F, carbazole, chrysene, D(ah)A, indeno(1,2,3-cd)pyrene, EPA, 1995)

**Comment:** Classification is based on multiple studies indicating carcinogenicity of individual components of PAHs mixture through inhalation, and dermal contact exposures

Toxicity Criteria:

**Reference Dose (RfD)—Oral:** Acenaphthene = 6.0E-2, anthracene = 3.0E-1, fluoranthene, and fluorene = 4.0E-2, and pyrene = 3.0 E-2

**Cancer Slope Factor—Oral:** B(a)P - 7.3 (mg/kg/day)^{-1}

**Comment:** Mouse skin carcinogenesis. Other carcinogenic PAHs are evaluated based on their relative potency compared with B(a)P.

The relative potency factors areas follows: B(a)P = 1.0, B(a)A = 0.1, B(b)F = 0.1, B(k)F = 0.01, chrysene = 0.001, D(ah)A = 1.0, indeno(123-cd)pyrene = 0.1.

Target Organs (Primary):

- skin (cancer)
- immune system
- nervous system

A.2.1 General Information

PAHs are products of incomplete combustion of organic materials from sources such as cigarette smoke, municipal incinerators, wood stove emissions, coal conversion, and fossil
fuel burning (diesel and gasoline automobile exhausts). PAHs are a diverse class of compounds formed as a result of incomplete combustion of organic compounds with insufficient oxygen. This leads to the formation of C-H free radicals, which can polymerize to form various PAHs. Among these PAHs are compounds such as benzo(a)pyrene (B[a]P), benz(a)anthraene (B[a]A) and Dibenz(a,h)anthracene (ATSDR, 1988).

PAHs are present in the environment from both natural and anthropogenic sources. As a group, they are widely distributed in the environment, having been detected in animal and plant tissue, sediments, soils, air, and surface water. Humans may be exposed to PAHs in the environment, in tobacco smoke and cooked food, and in the work place. Typically, individuals are not exposed to a single type of PAHs, but to a mixture of related chemicals (ATSDR, 1988).

The environmental fate of PAHs is determined largely by their low water solubilities and high propensity for binding to particulate or organic matter. In the atmosphere they are associated with particulate matter, especially soot. In aquatic environments, PAHs are usually bound to suspended particles or bed sediments. PAHs suspended in the air are thought to undergo direct photolysis very quickly. The ultimate fate of PAHs in the sediment is believed to be biodegradation and biotransformation by benthic organisms (EPA, 1986).

Unsubstituted lower molecular weight PAH compounds that contain 2 or 3 rings exhibit significant acute toxicity and other adverse effects to some organisms, but are noncarcinogenic. The higher molecular weight PAHs that contain 4 to 7 rings are significantly less toxic, but many of these are demonstrably carcinogenic, mutagenic, or teratogenic to a wide variety of organisms including fish, birds, and mammals. These animals have been exposed to PAHs (particularly B[a]P) by several routes of exposure including dermal absorption, ingestion, injection, and inhalation. The metabolism is important in determining their carcinogenicity and effects and many of the metabolites are more toxic than the parent compound. PAHs in the water column also accumulate in organisms, but many organisms metabolize and excrete PAHs rapidly, resulting in short-lived bioaccumulation (EPA, 1986).

A.2.2 Basis for Toxicity Criteria

There is no direct information available for the effects of PAHs on humans. All of the information available for PAHs is from studies on experimental animals. PAHs are well-established as experimental carcinogens for all routes to which humans would normally be exposed. Non-carcinogenic effects reported for PAHs include skin lesions and non-cancer lung diseases such as bronchitis. Benzo(a)pyrene has been associated with developmental toxicity and adverse reproductive effects in experimental animals (ATSDR, 1988).

Risk is assessed separately for carcinogens and noncarcinogens. Benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, B(a)P, chrysene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene are classified as B2 carcinogens (probable human carcinogen; sufficient evidence of carcinogenicity in animals; inadequate evidence of carcinogenicity in humans). The other PAHs have a D classification (not classified; inadequate evidence of carcinogenicity in animals). Because of recent changes in the interpretation of toxicological data, the following discussion regarding the source of information used to develop the action
levels is presented. The animal data consist of dietary, gavage, inhalation, intratracheal instillation, and dermal and subcutaneous studies in numerous strains of at least four species of rodents and several primates. Repeated B(a)P administration has been associated with increased incidences of total tumors and of tumors at the site of exposure. Distant site tumors have also been observed after B(a)P administration by various routes. B(a)P is frequently used as a positive control in carcinogenicity bioassays.

B(a)P administered in the diet or by gavage to mice, rats, and hamsters has produced increased incidences of stomach tumors. Neal and Rigdon (1967) fed B(a)P (purity not reported) at concentrations of zero, 1, 10, 20, 30, 40, 45, 50, 100 and 250 parts per million (ppm) in the diets of male and female CFW-Swiss mice. The age of the mice ranged from 17 to 180 days old, the treatment time from 1 to 197 days, and the size of the treated groups from 9 to 73. There were 289 mice (number of mice/sex not stated) in the control group. No forestomach tumors were reported in the zero-, 1- and 10-ppm dose groups. The incidence of forestomach tumors in the 20-, 30-, 40-, 45-, 50-, 100- and 250-ppm dose groups were 1/23, 0/37, 1/40, 4/40, 23/34, 19/23 and 66/73, respectively. The authors felt that the increasing tumor incidences were related to both the concentration and the number of doses administered. Historical control forestomach tumor data are not available for CFW-Swiss strain mice. In historical control data from a related mouse strain, SWR/J Swill, the forestomach tumor incidence rate was 2/268 and 1/402 for males and females, respectively (Rabstein et al., 1973).

Brune et al. (1981) fed 0.15 mg/kg B(a)P (reported to be highly pure) in the diet of 32 Sprague-Dawley rats/sex/group either every 9th day or 5 times per week. These treatments resulted in annual average doses of 6 or 39 mg/kg, respectively. An untreated group of 32 rats/sex served as the control. Rats were treated until moribund or dead; survival was similar in all groups. Histologic examinations were performed on each rat. The combined incidence of tumors of the forestomach, esophagus, and larynx was 3/64, 3/64 and 10/64 in the control group, the group fed B(a)P every 9th day and the group fed B(a)P 5 times/week, respectively. A trend analysis showed a statistically significant tendency for the proportion of animals with tumors of the forestomach, esophagus, or larynx to increase steadily with dose (Knauf and Rice, 1992).

As part of the same study, Brune et al. (1981) administered B(a)P (highly pure) orally to Sprague-Dawley rats by caffeine gavage. The rats were treated until moribund or dead; all rats were subjected to terminal histopathologic examination. Gavaged rats were divided into three dose groups of 32 rats/sex/group; the groups received 0.15 mg/kg per gavage either every 9th day (Group A), every 3rd day (Group B), or 5 times per week (Group C). These treatments resulted in annual average doses of 6, 18 or 39 mg/kg, respectively. Untreated and gavage (5 times/week) controls (32 rats/sex/group) were included. The median survival times for the untreated control group; the gavage control group; and groups A, B, and C were 129, 102, 112, 113, and 87 weeks, respectively. The survival time of Group C was short compared with controls, and may have precluded tumor formation (Knauf and Rice, 1992). The combined tumor incidence in the forestomach, esophagus, and larynx was 3/64, 6/64, 13/64, 26/64, and 14/64 for the untreated control group, gavage control group, group A, group B, and group C, respectively. There was a statistically significant association between the dose and the proportions of rats with tumors of the forestomach, esophagus, or larynx.
This association is not characterized by a linear trend. The linearity was affected by the apparently reduced tumor incidence that is seen in the high-dose group (Knauf and Rice, 1992).

Intratracheal instillation and inhalation studies in guinea pigs, hamsters, and rats have resulted in elevated incidences of respiratory tract and upper digestive tract tumors (EPA, 1991a). Intraperitoneal B(a)P injections have caused increases in the number of injection site tumors in mice and rats (reviewed in EPA, 1991a). B(a)P has also been reported to be carcinogenic in animals when administered by the following routes—i.v.; transplacentally; implantation in the stomach wall, lung, renal parenchyma, and brain; injection into the renal pelvis; and vaginal painting (EPA, 1991a).

At the June 1992 CRAVE Work Group meeting, it was noted that an error had been made in the 1991 document Dose-Response Analysis of Ingested Benzo[a]pyrene which is quoted in the Drinking Water Criteria Document for PAH. In the calculation of the doses in the Brune et al. (1981) study, it was erroneously concluded that doses were given in units of mg/year, whereas it was in fact mg/kg/year. When the doses are corrected, the slope factor is correctly calculated as 11.7 per (mg/kg)/day, as opposed to 4.7 per (mg/kg)/day as reported in the Drinking Water Criteria Document. The correct range of slope factors is 4.5 to 11.7 per (mg/kg)/day, with a geometric mean of 7.3 per (mg/kg)/day. A drinking water unit risk based on the revised slope factor is 2.1E-4 per (µg/L). Therefore, these values have been changed on IRIS, and an Erratum to the Drinking Water Criteria Document is being prepared.

Risk estimates were calculated from two different studies in two species of outbred rodents (Neal and Rigdon, 1967; Brune et al., 1981). These studies have several commonalities, including mode of administration, tumor sites, tumor types, and the presumed mechanisms of action. The data sets were not combined before modeling (the preferred approach) because they used significantly dissimilar protocols.

The geometric mean from several slope factors, each considered to be of equal merit, was used to calculate a single unit risk. These four slope factor estimates span less than a factor of three, and each is based on an acceptable, but less-than-optimal, data set. Each estimate is based on a low-dose extrapolation procedure that entails the use of multiple assumptions and default procedures.

Clement Associates (1990) fit the Neal and Rigdon (1967) data to a two-stage dose response model. In this model, the transition rates and the growth rate of preneoplastic cells were both considered to be exposure-dependent. A term to permit the modeling of B(a)P as its own promoter was also included. Historical control stomach tumor data from a related, but not identical, mouse strain, SWR/J Swill (Rabstein et al., 1973) and the CFW Texas colony (Neal and Rigdon, 1967) were used in the modeling. In calculating the lifetime unit risk for humans, several standard assumptions were made—mouse food consumption was 13 percent of its body weight/day; human body weight was assumed to be 70 kg; and the assumed body weight of the mouse was 0.034 kg. The standard assumption of surface area equivalence between mice an humans was the cube root of 70/0.034. A conditional upper bound estimate was calculated to be 5.9 per (mg/kg)/day (EPA, 1991a).
An EPA report (1991b) argued that the upper-bound estimate calculated in Clement Associates (1990) involved the use of unrealistic conditions placed on certain parameters of the equation. Other objections to this slope factor were also raised. The authors of this report used the Neal and Rigdon (1967) data to generate an upper-bound estimate extrapolated linearly from the 10 percent response point to the background of an empirically fitted dose-response curve (Clement Associates, 1990). Other results, from similar concepts and approaches used for other compounds, suggest that the potency slopes calculated in this manner are comparable to those obtained from a linearized multistage procedure for the majority of the other compounds. The upper bound estimate calculated in EPA (1991b) is 9.0 per (mg/kg)/day. The authors of EPA (1991b) selected a model to reflect the partial lifetime exposure pattern over different parts of the animals’ lifetimes. The authors thought that this approach more closely reflected the Neal and Rigdon (1967) regimen. A Weibull-type dose-response model was selected to accommodate the partial lifetime exposure; the upper-bound slope factor calculated from this method was 4.5 per (mg/kg)/day.

EPA selected a slope factor for B(a)P of 7.3, which is a geometric mean of 4.5 to 11.7. EPA currently proposes to regulate carcinogenic PAHs based on their relative potency in producing skin tumors in mouse skin painting studies. The toxicity values (RfDs), critical effects, and uncertainties for five of the noncancerogenic PAH compounds are verified and currently available on IRIS as listed above.

A.2.3 Standards and Criteria

Occupational Exposures: OSHA PEL (B[a]P) 0.2 mg/m³

The proposed maximum contaminant level (MCL) value for B(a)P is 0.0002 mg/L (proposed, 1990). The World Health Organization European standards for drinking water recommend a concentration of PAHs not to exceed 0.2 µg/L (EPA, 1988).

For ambient water quality criteria (AWQC) for protection of humans to water and fish, the consumption is 2.8E-3 µg/L; fish consumption alone is 3.11E-2 µg/L. The AWQC for protection of aquatic organisms is not available for fresh water organisms or for marine organisms: the acute LEC is 3.0E+2 µg/L and no chronic LEC is available. The values that are indicated as LEC are not criteria, but are the lowest effect levels found in the literature. LECs are given when the minimum data required to derive water quality criteria are not available. The values given represent PAHs as a class (45 FR 79318 (11/28/80).

A.2.4 References


A.3 Tetrachloroethylene

Toxicity Classification:


Toxicity Criteria:

Reference dose (RfD)—Oral: 0.01 mg/kg/day (EPA, 1991)

Uncertainty Factor: 100

NOAEL: 20 mg/kg/day (converted to 14 mg/kg/day)

Principal Study: Buben and O'Flaherty, 1985

Cancer Slope Factor—Oral: $5.1 \times 10^{-2} (\text{mg/kg/day})^{-1}$

Unit Risk: $1.5 \times 10^{-6} (\mu\text{g/L})^{-1}$

(CRAVE-EPA group verified, pending input into IRIS; quantitative estimates were not calculated by the CRAVE Workgroup [EPA, 1991].)

Cancer Slope Factor—Inhalation: $2.03E-3 (\text{mg/kg/day})^{-1}$ (provisional value)

Unit Risk: $5.8 E-7 (\mu\text{g/m}^3)^{-1}$

(CRAVE-EPA group verified, pending input into IRIS; quantitative estimates were not calculated by the CRAVE Workgroup [EPA, 1991].)

Target Organs:

- liver and kidney (oral and inhalation exposure)
- central nervous system (inhalation exposure)

A.3.1 General Information

Tetrachloroethylene (CAS No. 127-18-4) is a halogenated aliphatic hydrocarbon with a vapor pressure of 17.8 mm Hg at 25°C (EPA, 1982). The chemical is used primarily as a solvent in industry and, less frequently, in commercial dry-cleaning operations (ATSDR, 1990). Occupational exposure to tetrachloroethylene occurs via inhalation, resulting in systemic effects, and via dermal contact, resulting in local effects. Exposure to the general population can occur through contaminated air, food, and water (ATSDR, 1990).
The respiratory tract is the primary route of entry for tetrachloroethylene (NTP, 1986; EPA, 1988). The chemical is rapidly absorbed by this route and reaches an equilibrium in the blood within 3 hours after the initiation of exposure. Tetrachloroethylene is also significantly absorbed by the gastrointestinal (g.i.) tract, but not through the skin (ATSDR, 1990). The chemical accumulates in tissues with high lipid content, where the half-life is estimated to be 55 hours (ATSDR, 1990), and has been identified in perirenal fat, brain, liver, placenta/foetal tissue, and amniotic fluid. The proposed first step for the biotransformation of tetrachloroethylene is the formation of an epoxide thought to be responsible for the carcinogenic potential of the chemical. Tetrachloroethylene is excreted mainly unchanged through the lungs, regardless of route of administration (NTP, 1986). The urine and feces comprise secondary routes of excretion. The major urinary metabolite of tetrachloroethylene, trichloroacetic acid, is formed via the cytochrome P-450 system (ATSDR, 1990).

A.3.2 Basis for Toxicity Criteria

A.3.2.1 Non-carcinogenicity

Acute exposure to high concentrations of the chemical (estimated to be greater than 1,500 ppm for a 30-minute exposure) may be fatal to humans. Chronic exposure causes respiratory tract irritation, headache, nausea, sleeplessness, abdominal pains, constipation, cirrhosis of the liver, hepatitis, and nephritis in humans; and microscopic changes in renal tubular cells, squamous metaplasia of the nasal epithelium, necrosis of the liver, and congestion of the lungs in animals (NTP, 1986). Some epidemiology studies have found an association between inhalation exposure to tetrachloroethylene and an increased risk for spontaneous abortion, idiopathic infertility, and sperm abnormalities among dry-cleaning workers. The adverse effects in humans are supported in part by the results of animal studies in which tetrachloroethylene induced fetotoxicity (but did not cause malformations) in the offspring of treated dams.

A carcinogenicity bioassay in mice and rats (NCI, 1977) provided the only available chronic oral toxicity data for tetrachloroethylene. For both mice and rats, dosage adjustments were made during the study. The time-weighted average doses of the chemical, administered for 78 weeks in corn oil, were as follows: male B6C3F1 mice, 536 or 1,072 mg/kg; female mice, 386 or 772 mg/kg; Osborne-Mendel male rats, 471 or 941 mg/kg; and female rats, 474 or 949 mg/kg. Toxic nephropathy was observed at all doses in both sexes of mice and rats. The nephropathy was characterized by degenerative changes in the proximal convoluted tube at the junction of the cortex and medulla, with fatty degeneration, cloudy swelling, and necrosis of the tubular epithelium.

RfDs for chronic and subchronic oral exposure to tetrachloroethylene are 0.1 mg/kg/day and 0.01 mg/kg/day, respectively (Buben and Flaherty, 1985; EPA, 1990; 1991). These values are based on hepatotoxicity observed in mice given ≥ 100 mg tetrachloroethylene/kg body weight for 6 weeks and a NOAEL of 20 mg/kg.

A.3.2.2 Carcinogenicity

Epidemiology studies of dry cleaning and laundry workers have demonstrated excesses in mortality due to various types of cancer, including liver cancer, but the data are regarded as
inconclusive because of various confounding factors (Lynge and Thygesen, 1990; EPA, 1988). The tenuous finding of an excess of liver tumors in humans is strengthened by the results of carcinogenicity bioassays in which tetrachloroethylene, administered either orally or by inhalation, induced hepatocellular tumors in mice (NCI, 1977; NTP, 1986). The chemical also induced mononuclear cell leukemia and renal tubular cell tumors in rats. Tetrachloroethylene was negative for tumor initiation in a dermal study and for tumor induction in a pulmonary tumor assay (Van Duuren et al., 1979; Theiss et al., 1977).

On the basis of the sufficient evidence from oral and inhalation studies for carcinogenicity in animals and none or inadequate evidence for carcinogenicity to humans, tetrachloroethylene is placed in EPA's weight-of-evidence Group B2, probable human carcinogen (NCI, 1977; NTP, 1986; EPA, 1991). For oral exposure, the slope factor is $5.1 \times 10^{-2} \text{ (mg/kg/day)}^{-1}$; the unit risk is $1.5 \times 10^{6} \text{ (mg/L)}^{-1}$. For inhalation exposure, the slope factor was not calculated; the unit risk is $5.2 \times 10^{-7} \text{ (mg/m}^3\text{)}^{-1}$.

Human health effects resulting from chronic exposure to various concentrations of tetrachloroethylene include respiratory tract irritation, headache, nausea, sleeplessness, abdominal pains, constipation, cirrhosis of the liver, hepatitis, and nephritis (Coler and Rossmiller, 1953; Stewart et al., 1970; von Ottingen, 1964; Stewart, 1969). In one study, 16 of 25 workers, exposed to 59 to 442 ppm for 2 months to 27 years had significantly elevated SGOT and SGPT activity compared with controls (Chmielewski et al., 1976).

An NTP bioassay provided chronic toxicity data for animals exposed to tetrachloroethylene. Groups of 50 male and 50 female F344/N rats and B6C3F1 mice inhaled the chemical 6 hours/day, 5 days/week for 103 weeks (NTP, 1986). The exposure concentrations consisted of zero, 200, or 400 ppm for rats and zero, 100, or 200 ppm for mice. In rats, nonneoplastic effects consisted of dose-related renal tubular cell karyomegaly (males and females), renal tubular cell hyperplasia (males only), and dose-related increases in the incidences of nasal thromboses and squamous metaplasia (the thromboses were believed to have been secondary to tetrachloroethylene-induced leukemia). The incidence of renal tubular cell karyomegaly was higher in males than in females. In mice, nonneoplastic effects consisted of dose-related hepatic degeneration, hepatic necrosis, and hepatic nuclear inclusion; dose-related renal tubular cell karyomegaly; and pulmonary congestion.

Pegg et al. (1978), reported in a fate and disposition study that rats inhaling a tetrachloroethylene concentration of 600 ppm (4 g/m$^3$) 6 hours/day, 5 days/week for 12 months developed unspecified reversible liver damage.

In a Danish study, a cohort of laundry and dry-cleaning workers was studied for cancer incidence among persons exposed to tetrachloroethylene (the most commonly used solvent in Danish dry-cleaning shops) (Lynge and Thygesen, 1990). The 10-year follow-up study evaluated 8,567 women and 2,033 men employed in laundry and dry-cleaning in 1970. The study revealed a significant excess risk for primary liver cancer among the women (7 observed, 2.1 expected); but not one case of primary liver cancer was found among the men, for whom the expected value was 1.1. Although the majority of primary liver cancer cases in Denmark has been associated with excess alcohol consumption, the investigators did not believe this to be the exclusive explanation for the excess tumors among the dry-cleaning workers.
A retrospective mortality epidemiologic study of dry cleaning workers with exposure to tetrachloroethylene reported an excess of mortality from kidney and bladder cancer (8 cases versus 2.7 expected; SMR=296) and cancer of the cervix (10 observed versus 5.1 expected; SMR=296) (Brown and Kaplan, 1985). The cohort consisted of 1,690 workers with ≥ 23 years of employment. The results of this study were inconclusive because the workers had potential occupational exposure to petroleum solvents, in addition to tetrachloroethylene. However, a subcohort of the study, consisting of 615 workers with no known exposure to petroleum solvents, demonstrated no excess risk for cancer at any site (Brown and Kaplan, 1985). Other studies of dry cleaning and laundry workers have demonstrated increases in mortality due to various types of cancer (lung, cervix, kidney, skin and/or colon), but the data are also regarded as inconclusive because of various confounding factors (EPA, 1988).

In a carcinogenicity bioassay, groups of 50 male and 50 female F344/N rats and B6C3F1 mice inhaled tetrachloroethylene 6 hours/day, 5 days/week for 103 weeks (NTP, 1986). The exposure concentrations were zero, 200, or 400 ppm for rats and zero, 100, or 200 ppm for mice. Exposure to tetrachloroethylene under the conditions of the study resulted in: (a) clear evidence of carcinogenicity for male F344/N rats as shown by an increased incidence of mononuclear cell leukemia (controls, 28/50; low dose, 37/50; high dose, 37/50) and renal tubular cell adenomas or carcinomas combined (1/49, 3/49, 4/50) (the incidence of the renal tumors was not statistically significant, but these uncommon tumors had been found consistently at low incidences in male rats in other studies of chlorinated ethanes and ethylenes); (b) some evidence of carcinogenicity for female rats as shown by increased incidences of mononuclear cell leukemia (18/50, 30/50, 29/50); and (c) clear evidence of carcinogenicity for mice as shown by increased incidences of hepatocellular adenomas (11/49, 8/49, 18/50) and carcinomas (7/49, 25/49, 26/50) in males and of hepatocellular carcinomas (1/48, 13/50, 36/50) in females. There were no neoplastic changes in the respiratory tract of either species, but there was an increased incidence (non-dose-related) of squamous metaplasia in the nasal cavities of treated male rats.

Tumors were not observed in groups of 96 male and 96 female Sprague-Dawley rats exposed to tetrachloroethylene concentrations of 300 or 600 ppm, 6 hours/day, 5 days per week for 52 weeks and observed for the rest of their lives (Rampy et al., 1978).

A.3.3 References


A.4 Polychlorinated Biphenyls (PCBs)

Toxicity Classification: B2 carcinogens (EPA, 1995)
(Comment: Classification is based on hepatocellular carcinomas in rats and mice, suggestive evidence of excess risk of liver cancer in humans by ingestion, inhalation, and dermal contact.)

Toxicity Criteria:
Reference Dose (RfD)-Oral:  
- 7.0E-5 mg/kg/day (Arochlor-1016)
- 2.0E-5 mg/kg/day (Arochlor-1254)

Cancer Slope Factor-Oral:  
- 7.7 (mg/kg/day)^1

Cancer Slope Factor-Inhalation:  
To be determined

Target Organs (primary):
- skin (hyperpigmentation and hyperkeratosis)

A.4.1 General Information

There are four commercial PCB mixtures marketed in the U.S. under the name Aroclor®
(Aroclor®1016, 1242, 1254, and 1260) (USAF, 1989). Aroclor® formulations are complex
mixtures of PCBs produced by progressive chlorination of biphenyl with anhydrous chlorine,
and because they are mixtures, their physical properties and chemical behavior cannot be
precisely defined (USAF, 1989). PCBs have been used as heat transfer liquids, hydraulic
fluids, lubricants, plasticizers, surface coatings, inks, adhesives, pesticide and extenders, and

The environmental behavior of the Aroclor® mixtures is a direct function of their relative
composition with respect to the individual PCB species (USAF, 1989). Individual PCBs vary
widely in their physical and chemical properties according to the degree of chlorination and
position of the chlorines on the biphenyl structure. In general, as chlorine content increases,
adsorption increases while transport and transformation processes decrease (USAF, 1989).

Because of their very low solubility in water (~2.70 x 10^-3 mg/L at 20°C), high log octanol-
water partitioning coefficients (K_ow) of 6.1 to 9.3, and extremely high organic carbon
partition coefficients (K_oc) of 100,000 to 1,000,000,000, adsorption to soils and sediments is
the major fate process affecting PCBs in the environment, particularly in soils with high
organic carbon content (USAF, 1989). As a result, PCBs are expected to be highly immobile
in the soil, and leaching to the groundwater system is unlikely. However, in the presence of
organic solvents, PCBs are found to be highly mobile in the soil despite the high percent
retained by the organic carbon present (USAF, 1989).

Transport of PCB vapors through the air-filled pores of unsaturated soils is not expected to be
a rapid transport pathway. Volatilization (mostly from aqueous systems) followed by
atmospheric transport is expected to be slow, but may be a significant long-term transport
process and is thought to account for the widespread, almost ubiquitous, distribution of PCBs
in the environment. PCBs have been reported to be strongly resistant to chemical
degradation by oxidation or hydrolysis; however, PCBs have been shown to be susceptible to
slow-rate photolytic and biological degradation. Highly chlorinated PCBs can be
photolytically degraded, resulting in the formation of lower chlorinated species and
substituted products, as well as potential formation of biphenylenes and chlorinated
dibenzofurans. The presence of oxygen retards the photolytic degradation of PCBs (USAF,
1989).
Microbial degradation has been reported to be an important transformation process for PCBs to include both aerobic oxidative and anaerobic dechlorination biodegradation. In general, the less chlorinated PCBs were more easily degraded than the more chlorinated species. However, the presence of the lower chlorinated biphenyls has been shown to increase the rate of biodegradation of the more chlorinated PCBs through co-metabolism (USAF, 1989).

The high bioconcentration factor combined with the persistence of PCBs suggests that these compounds bioaccumulate and can be biomagnified (EPA, 1979b).
A.4.2 Basis for Toxicity Criteria

A.4.2.1 Non-carcinogenic Effects

EPA currently has not established an RfD/RfC for the noncarcinogenic effects of oral or inhalation exposures to PCBs (IRIS, 1995; HEAST, 1994). Because PCBs are slowly metabolized compounds, toxic symptoms of noncarcinogenic effects usually occur after long-term exposure and bioaccumulation. Initial symptoms of PCB poisoning are non-specific, such as loss or reduced weight gain, while more severe poisoning in rats have resulted in ataxia, diarrhea, lack of response to pain stimuli; and histopathological changes primarily in the liver and kidney (USAF, 1989). In humans exposed to PCBs in the workplace, reported adverse effects include chloracne (a long-lasting, disfiguring skin disease), impairment of liver function, neurobehavioral disorders, menstrual disorders, and minor birth abnormalities (ATSDR, 1988b; EPA, 1985b). Animals experimentally exposed to PCBs have shown most of the same symptoms as well as impaired reproduction and fetotoxicity; pathological changes in the liver, stomach, skin, spleen, lymph nodes, and thymus; and suppression of the immunological system (ATSDR, 1988b; EPA, 1985b; and USAF, 1989).

PCBs are almost completely absorbed from the digestive tract (>90 percent) with subsequent distribution to the liver and muscle tissue, followed by redistribution to body fat, skin, and other fat-containing organs (ATSDR, 1988b). Absorption via the skin is also fairly efficient, as indicated by occupational exposures where effects of PCB exposure can be detected even at doses too low to produce pathologic effects (ATSDR, 1988b).

A.4.2.2 Carcinogenicity

On the basis of the increased incidence of liver tumors following dietary exposure of rats to Aroclor® (Norback and Weltman, 1985), PCBs have been classified by EPA as B2 carcinogens (IRIS, 1995) for both the oral and inhalation routes of exposure. A classification of B2 indicates that sufficient evidence exist to show carcinogenicity in animals, but inadequate evidence of carcinogenicity in humans. Based on a statistically significant increase in the occurrence of liver tumors following oral exposure, EPA (IRIS, 1995) has developed an oral cancer potency slope factor of 7.7 (mg/kg/day) for PCBs; a CPF has not yet been determined for the inhalation route of exposure (IRIS, 1992; HEAST, 1992).

A.4.3 Standards And Criteria

EPA has promulgated the enforceable (for public water supplies) maximum contaminant level (MCL) of 0.0005 mg/L for PCBs, based on a practical quantitation limit (PQL) of 0.0005 mg/L, which is associated with a maximum lifetime individual risk of 1 x 10^-4. EPA has also proposed an MCL Goal (MCLG) of zero mg/L PCBs based on the evidence of carcinogenic potential (classification group B2) (IRIS, 1995).

EPA has also established ambient water quality criterion (AWQC) for human consumption of water and aquatic organisms contaminated with PCBs at 7.9 x 10^-5 µg/L (IRIS, 1995). An AWQC of 7.9 x 10^-6 µg/L has also been set for the consumption of aquatic organisms alone.
(IRIS, 1995). The proposed federal AWQC for the protection of aquatic life are 2.0 mg/L (acute) and 0.014 mg/L (chronic) (IRIS, 1992).

A.4.4 References


MNA sampling event. These TOC concentrations represent the total organic carbon for the aquifer matrix (which includes the non-dissolved or immobile carbon).

**Dunn Field**

Biodegradation rates for TCE at Dunn Field ranged from 0.093 to 0.199 per year (Table 10). Based on the biodegradation rates, the half-life for TCE ranged from 3.5 to 7.5 years. Travel time between MW70 and MW54 for TCE is estimated at 21.1 to 45.1 years. For data input, two hydraulic gradients were used in the biodegradation calculation to more completely represent the characteristics of the area of concern: MW46 to MW35 (east of the groundwater extraction system) and MW15 to MW54. Soil adsorption coefficients used in the calculation represent high, low, and average estimates for TCE from previous studies (EPA, 1998). TOC concentrations collected during January 1996 from monitoring wells MW40 (4,760 mg/kg), 42 (2,220 mg/kg), 46 (56.3 mg/kg), and October 1998 from recovery wells 1 (5,400 mg/kg), 1A (1,200 mg/kg), 1B (2,400 mg/kg), and 2 (4,000 mg/kg) soil samples were used to represent the organic matter content within Dunn Field instead of the aqueous phase TOC concentrations collected during the MNA sampling event; these concentrations from MW40, 42, and 46, and RW1, 1a, 1b, and 2 represent the total organic carbon for the aquifer matrix (which includes the non-dissolved or immobile carbon).

**Summary and Conclusions**

**Main Installation**

In accordance with biodegradation byproduct concentrations within the plume, the dissolved CAH plume, which appears to have originated as PCE, is migrating toward the northeast. Data show an increase of PCE in MW47 from 1 µg/L in October 1998 to 200 µg/L in March 2000. Chemical and geochemical evidence indicate that although dissolved CAHs at the MI are undergoing biologically facilitated reductive dechlorination the occurrence of this process is limited and localized. As a result, PCE still comprises the majority of the contamination present in groundwater throughout most of the plume.

Limited biodegradation of the PCE plume in the MI groundwater appears to be occurring, primarily by the reductive dechlorination of PCE to TCE to DCE. Available information indicates that the PCE plume originating at the southwest corner is exhibiting a Type 3 behavior. This area of the MI is characterized by low concentrations of native and/or anthropogenic carbon (in groundwater) and elevated concentrations of DO. The evidence supporting limited PCE and TCE biodegradation is summarized as follows:

- The occurrence of elevated TCE and cis-1,2-DCE concentrations in groundwater within the source area (MW21 and MW47) is a direct indication that PCE is being reductively dechlorinated.
- Source area well MW47, side-gradient wells MW20, 22, and 23, and downgradient well MW39, all have chloride concentrations of at least 20 mg/L. The elevated chloride concentrations are consistent with the PCE/TCE plume and support biodegradation of chlorinated solvents.
• Dissolved hydrogen in the source area and dissolved plume range from 1.39 to 3.13 nm/L. Dissolved hydrogen above 1 nm/L enhances the potential for reductive dechlorination.

• All monitoring wells except MW72 and MW22 had sulfate concentrations less than 20 mg/L, which supports reductive dechlorination.

• Monitoring wells MW22, MW23, and MW47 had groundwater temperatures greater than 20°C, which aids in accelerating biochemical processes.

• The groundwater pH in the area of concern falls within the optimal range for reductive dechlorination.

• Biodegradation rates at the MI for PCE ranged from 0.086 to 0.215 per year, and for TCE ranged from 0.158 to 0.359 per year. The half-life for PCE ranged from 3.2 to 8.1 years and for TCE ranged from 1.9 to 4.4 years. Travel time between MW21 and MW39 for PCE is estimated at 11.4 to 28.6 years and for TCE is estimated at 18.6 to 53.9 years.

Wiedemeier et al. (1999) created a worksheet used as an initial screening to assess whether biodegradation could be occurring at the site. This worksheet includes guidelines used to calculate a score for different groundwater parameters and an interpretation of those points awarded during the groundwater screening. Wiedemeier worksheets with detailed descriptions for all the monitoring wells are located in Table 11.

A summary of the total Wiedemeier scores are included below:

<table>
<thead>
<tr>
<th>Monitoring Well (Main Installation)</th>
<th>Total Score</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>MW24</td>
<td>-1</td>
<td>Inadequate evidence for biodegradation of chlorinated organics.</td>
</tr>
<tr>
<td>MW23</td>
<td>2</td>
<td>Inadequate evidence for biodegradation of chlorinated organics.</td>
</tr>
<tr>
<td>MW72</td>
<td>2</td>
<td>Inadequate evidence for biodegradation of chlorinated organics.</td>
</tr>
<tr>
<td>MW62</td>
<td>3</td>
<td>Inadequate evidence for biodegradation of chlorinated organics.</td>
</tr>
<tr>
<td>MW22</td>
<td>7</td>
<td>Limited evidence for biodegradation of chlorinated organics.</td>
</tr>
<tr>
<td>MW20</td>
<td>8</td>
<td>Limited evidence for biodegradation of chlorinated organics.</td>
</tr>
<tr>
<td>MW21</td>
<td>8</td>
<td>Limited evidence for biodegradation of chlorinated organics.</td>
</tr>
<tr>
<td>MW39</td>
<td>8</td>
<td>Limited evidence for biodegradation of chlorinated organics.</td>
</tr>
<tr>
<td>MW47</td>
<td>9</td>
<td>Limited evidence for biodegradation of chlorinated organics.</td>
</tr>
</tbody>
</table>

Five out of nine monitoring wells indicated limited evidence for biodegradation of chlorinated organics (score of 6 to 14). The remaining four wells showed inadequate evidence for biodegradation (score <6), which corresponds with locations outside of the PCE plume. MW20 is the only well outside of the plume that shows limited evidence for biodegradation.
In conclusion, monitoring wells within the MI area of concern have some degradation of PCE to TCE to DCE, with some wells within the plume scoring high enough on the Wiedemeier worksheet to indicate limited evidence for biodegradation.

**Dunn Field**

The dissolved CAH plume appears to have originated as PCE and/or TCE and is migrating toward the northwest. Chemical and geochemical evidence indicate that although dissolved CAHs at Dunn Field are undergoing biologically facilitated reductive dechlorination the occurrence of this process is limited and localized. As a result, TCE still comprises the majority of the contamination present in groundwater throughout most of the plume.

Limited biodegradation of the PCE/TCE plume in the Dunn Field groundwater appears to be occurring, primarily by the reductive dechlorination of PCE to TCE to DCE. Available information indicates that the PCE/TCE plume originating at Dunn Field is exhibiting a mixed Type 2 and 3 behavior. Type 2 behavior could be occurring in the area near MW70, where the DO concentrations are sufficiently low to allow reductive dechlorination to proceed. Although the DO is low in this area, TOC concentrations (in groundwater) are also low, which does not aid in reductive dechlorination. PCE, TCE, DCE, VC, and ethene/ethane concentrations in MW70 suggest a degree of reductive dechlorination. Type 3 behavior appears to be prevalent throughout the rest of the plume. This area of Dunn Field is characterized by low concentrations of native and/or anthropogenic carbon and elevated concentrations of DO. The evidence supporting limited PCE/TCE biodegradation is summarized as follows:

- The occurrence of elevated cis-1,2-DCE and VC concentrations in groundwater within the source area (MW54 and MW70) is a direct indication that PCE/TCE is being reductively dechlorinated.
- The occurrence of elevated ethene/ethane concentration in groundwater within the source area (MW70) is a direct indication that PCE/TCE is being reductively dechlorinated.
- Chloride concentrations in the downgradient wells MW54 and MW40 all have chloride concentrations of at least 19.8 mg/L. The elevated chloride concentration in MW54 is consistent with the PCE/TCE plume and supports limited biodegradation of chlorinated solvents.
- Nitrate concentrations in the immediate vicinity of MW31 (0.9 mg/L) are greater than 1 mg/L. Therefore, this area supports reductive dechlorination.
- Source area monitoring wells MW70, MW35, and MW15, and background well MW46 had sulfate concentrations less than 20 mg/L (12, 13, 18, and 17 mg/L, respectively). Sulfate concentrations in these wells support reductive dechlorination.
- Monitoring wells MW31 and MW40 had groundwater temperatures greater than 20°C, which aids in accelerating biochemical processes.
- The groundwater pH in the area of concern falls within the optimal range for reductive dechlorination.
• Biodegradation rates for TCE at Dunn Field ranged from 0.093 to 0.199 per year. The half-life for TCE ranged from 3.5 to 7.5 years, and the travel time between MW70 and MW54 for TCE is estimated at 21.1 to 45.1 years.

A summary of the total Wiedemeier scores is included below. Wiedemeier worksheets with detailed descriptions for all of the monitoring wells are located in Table 12:

<table>
<thead>
<tr>
<th>Monitoring Well (Main Installation)</th>
<th>Total Score</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>MW46</td>
<td>4</td>
<td>Inadequate evidence for biodegradation of chlorinated organics.</td>
</tr>
<tr>
<td>MW71</td>
<td>4</td>
<td>Inadequate evidence for biodegradation of chlorinated organics.</td>
</tr>
<tr>
<td>MW15</td>
<td>8</td>
<td>Limited evidence for biodegradation of chlorinated organics.</td>
</tr>
<tr>
<td>MW31</td>
<td>9</td>
<td>Limited evidence for biodegradation of chlorinated organics.</td>
</tr>
<tr>
<td>MW35</td>
<td>9</td>
<td>Limited evidence for biodegradation of chlorinated organics.</td>
</tr>
<tr>
<td>MW54</td>
<td>10</td>
<td>Limited evidence for biodegradation of chlorinated organics.</td>
</tr>
<tr>
<td>MW70</td>
<td>10</td>
<td>Limited evidence for biodegradation of chlorinated organics.</td>
</tr>
<tr>
<td>MW40</td>
<td>14</td>
<td>Limited evidence for biodegradation of chlorinated organics.</td>
</tr>
</tbody>
</table>

Six out of eight monitoring wells indicated limited evidence for biodegradation of chlorinated organics (score of 6 to 14). The remaining two wells showed inadequate evidence for biodegradation (score <6), which includes background well MW46 and a source area well MW71. MW40 is the only well outside of the plume that shows limited evidence for biodegradation.

In conclusion, monitoring wells within the Dunn Field area of concern have some degradation of PCE to TCE to DCE to VC to ethane/ethene, with some wells within the plume scoring high enough on the Wiedemeier worksheet to indicate limited evidence for biodegradation.

References


- 642 acres
- 1942 - inventory & supply materials for army
- 1964 - distribution CTR for military command
- 1997 -

Down field - DU 1 - 68 acres - was judgment used as landfill
SwSSC storage in mixed stackage
NE yard as stores - ready & pesticide storage

Until 1970 Army supplies were buried on NW section.

Used oils & greases, paint, mostly metals, pesticides,
herbicides

Contaminated S/S Fluent Cyl

1989-96, R/E/ES study - dissolved core & VOCs

Heavy metals ? MCM

Hydrogeology

Local 20-30 ft thick
Terrain deposits 70-80 ft thick - salty sandy sand
SW clay - 5-75 ft thick

NPL - 1992
CU/MRH - 1995

Record of Decision (ROD) - RUs lead & S/S plumes

- Reduce contaminant core levels
- Permit for discharge
- Monitor discharge for discharge
- Create wetlands buffer for future impact
750' extraction wells, 1 percent drilled, ultrasound pump system was installed 1997

RW1, RW 1-A, RW 1-B, RW 2 installed 2001 - located east of RW 3

10/01 - SW samples collected
Bass filled w/distilled HD-oil, 2 great different key samples were placed for each 5 foot 7 second interval
9/11 - diff Keez installed retention 1/3/01
Tempa = 26.8f
1/60
- Effluent sampling quarterly

3.0 Monitoring Results

- SW levels collected (2) event on 11/98
- SW effluent level 11/01
- SW flow wast

{DCE - tetrachloroethene (PCE), Trichloroethene (TCE),
1,1,2,2 Tetrachloroethane (1,1,2,2 PCE), Cis 1,2 -
Pdichloroethene (Cis 1,2 DCE), 1,1 Dibromo ethene
(1,1 DCE), Chloroform, Carbontetrahydro

- VD50 collected quarterly, metals annually
for analysis
Trichloroethylene (TCE) - exceed MCL in all RW except NW 09.
Carbon Tetrachloride - exceed NCE - RW 1, 1A, 1B, 2, 3 - located in western half sector of Dunn Field.
(CIS 1,2 DCE) - NW 3, 7, 8
PCE - RW 1A, 4, 5, 6, 7, 8
(1,1,1 DCE) - NW 8, 9

Carbon tetrachloride plume is confined to the central SW and SE area of Dunn Field. Migration has occurred in a west NW direct.

4.0 Conclusion

- Carbon tetrachloride - exceeds pre-SW Dunn levels
  NW 58 - migrated W-NW directly to NW 32 & 57.

- Chloroform - SW Dunn 2W1A highest concentration,
migrated W-WW MW 71, 32, Den theory to
  MW 71, N.D. RW 6 conc.

- TCE & PCE - highest conc NW 70. SW flow appears to
  be towards high conc. in to east & south
  of NW 70. TCE & PCE appear to be migrated
  migrating WNW with NW 71, 76, 31, 32 showing
  an impact.

- SW flow during the quarter was generally west,
  returned to WSW w/slight W-NW component from the
  northern end of Dunn. Water table gradient was
  SW 12'12" N to NW 12.4'12" N.
- Cis 12, DEE - exceeded effluent discharge permit limit to P278 in 12/01. 53.6 mg/L acceptable 1 day max. When limit is 100 mg/L but exceeded the monthly avg. level 50 mg/L. AS NO. scuba samples are more closely pop. if a 1 day max level, discharge permit compliance is still achieved.

- DW system is exerting significant influence on the flow on the west edge of Farm.

- MW-44 has shown care. 32 seedling target. canopy of the 4th consecutive monitoring period. New shading well might be considered for future MW site.

- Diffusor breaks usage

\[ \text{EMSL} = 211 \text{ feet} \]
<table>
<thead>
<tr>
<th>Compound</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetone</td>
<td>NS</td>
</tr>
<tr>
<td>Benzene</td>
<td>-5</td>
</tr>
<tr>
<td>Bromo dichloromethane</td>
<td>NS</td>
</tr>
<tr>
<td>Carbon disulfide</td>
<td>-5</td>
</tr>
<tr>
<td>Carbon tetrachloride</td>
<td>100</td>
</tr>
<tr>
<td>Chloro benzene</td>
<td>NS</td>
</tr>
<tr>
<td>Chloroform</td>
<td>NS</td>
</tr>
<tr>
<td>Chloromethane</td>
<td>NS</td>
</tr>
<tr>
<td>1,1-Dichloromethane</td>
<td>-5</td>
</tr>
<tr>
<td>1,2-Dichloroethene</td>
<td>-7</td>
</tr>
<tr>
<td>1,1-Dichloroethane</td>
<td>100</td>
</tr>
<tr>
<td>Tans 1,2, Dichloroethene</td>
<td>70</td>
</tr>
<tr>
<td>Clin 1,2, Dichloroethene</td>
<td>NS</td>
</tr>
<tr>
<td>1,1,2,2-Tetrachloroethane</td>
<td>5</td>
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<tr>
<td>Tetrachloroethene</td>
<td>1000</td>
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<tr>
<td>Toluene</td>
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<tr>
<td>1,2,4-Trichloro benzene</td>
<td>-200</td>
</tr>
<tr>
<td>1,1,1-Trichloroethane</td>
<td>-5</td>
</tr>
<tr>
<td>1,1,2-Trichloroethane</td>
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</tr>
<tr>
<td>Trichloroethane</td>
<td>-2</td>
</tr>
<tr>
<td>Vinyl Chloride</td>
<td></td>
</tr>
</tbody>
</table>
ND - mw-42
  80

MW - 44-60

44-61

MW - 44-60
  CMBP 220.5 2/05 7.1
  11, 22, 7 324.7
  1.29 7.1
  6.47 FRCB 7.1
  8.24 DMB 7.1
  2.77 Tridel 7.1
  2.84 Tridel 7.1
  2.28 DMB 7.1
  1.26 Tridel 7.1

30.67 - CMBP 1.87