

Appendix 5

Toxicological Profiles of Selected WTC Hazardous Substances

Individual Toxicity Profiles of Selected WTC Hazardous Substances

1. Asbestos

General: The term asbestos generally refers to a group of six different fibrous minerals that occur naturally in the environment. Asbestos minerals consist of thin, separable fibers that have a parallel arrangement. There are two types of asbestos fibers: serpentine and amphibole. The amphibole types are amosite, crocidolite, tremolite, actinolite, and anthophyllite. Chrysotile is the only known form of serpentine asbestos. Chrysotile, also termed white asbestos, is the predominant commercial form of asbestos and is responsible for 99% of asbestos use in the United States (ATSDR, 2001). It is also the predominant form of asbestos found in WTC Dust.

Asbestos fibers do not have any detectable odor or taste. Asbestos fibers are resistant to heat, fire, chemical and biological degradation and do not evaporate or dissolve in water. Because of these physical and chemical properties, asbestos has been used in a wide variety of commercial products such as building materials (including insulation and ceiling tiles), friction products (including brakes and clutches), and heat-resistant fabrics. Because of the potential health effects of asbestos exposure, EPA banned all new uses of asbestos within the United States in 1989 (ATSDR, 2001).

Exposure: Although asbestos is very stable, fibers can enter the air, soil, and water from the weathering of natural deposits, the wearing down of manufactured asbestos products, or the demolition and destruction of asbestos-containing structures. Small diameter fibers and fiber-containing particles may remain suspended in the air for extended periods of time and be carried long distances by wind or water currents before settling. Larger diameter fibers and particles tend to settle more quickly. Asbestos fibers generally do not break down into other compounds in the environment, although chrysotile asbestos may have some minor mineral loss in acidic environments. When asbestos fibers are inhaled they may become trapped in the lungs. The number of fibers in lung tissue can accumulate over time, although some asbestos fibers, particularly chrysotile, can be broken down and removed from the lung (ATSDR, 2001).

Inhalation of suspended fibers is the most likely route of exposure to asbestos. Some fibers may be removed by mucociliary clearance or macrophages while others may be retained in the lungs for extended periods. Inhalation exposure, therefore, is generally regarded as cumulative, and exposures have been expressed in terms of concentration of fibers over time or fiber-years/mL (f-yr/mL).

Noncarcinogenic Health Effects: Information on the health effects of asbestos in humans comes mostly from studies of people with past exposures to asbestos fibers [measured as greater than or equal to 5 microns (μm) in length] in workplace air. Workers who repeatedly breathe in sufficient quantities of asbestos fibers with lengths greater than or equal to 5 μm may develop a disease called asbestosis. Asbestosis is characterized by the slow accumulation of scar-like tissue in the lungs and in the membrane surrounding the lungs, thus hindering normal breathing. Blood flow to the lung may also be decreased, causing the heart to enlarge. Asbestosis is a serious disease associated with occupational exposures, but is not usually of concern to people exposed to low levels of asbestos. Changes in the membrane surrounding the lung, called pleural plaques, are also seen in people occupationally exposed to asbestos and are sometimes found in people living in areas with high environmental levels of asbestos. Pleural plaques are areas of thickening that occur on the parietal pleura, most commonly on the lower chest walls and the diaphragm. Pleural plaques may be smooth or nodular in outline and can measure up to 1 centimeter (cm) in thickness. Pleural plaques and thickening are usually bilateral and fairly symmetric. Breathing effects from pleural plaques alone are usually not serious. In most cases, a person is not even aware of the condition. However, pleural plaques are visible on chest x-ray (ATSDR, 2001).

Carcinogenicity: Asbestos exposure is associated with an increased risk of two principal types of cancer: cancer of the lung tissue itself and mesothelioma, a cancer of the thin membrane that surrounds the lung and other internal organs. A latency period of many years following asbestos exposure is required for the development of these diseases. Lung cancer is usually fatal. Mesothelioma is almost always fatal (often within a few months of diagnosis), although early identification and intervention may increase survival. The levels of asbestos in air that lead to lung disease depend on level and duration of exposure, length of time since start of last exposure, the age of the exposed individual, whether the exposed individual is or has been a tobacco smoker, and the type and size distribution of the asbestos fibers (ATSDR, 2001).

The differences in the extent of disease caused by different fiber types and sizes are under debate. Several studies suggest that amphibole asbestos types (tremolite, amosite, and especially crocidolite) may be more harmful than chrysotile, particularly for mesothelioma. Other data indicate that fiber size dimensions (length and diameter) are important factors for cancer-causing potential (e.g., Berman et al., 1995; ATSDR, 2003).

In a proposed protocol for assessing risks from exposure to asbestos, 99.7% of the risk of lung cancer is attributable to fibers longer than 10 μm in length (Berman et al., 1995; Berman and Crump, 1999). In October, 2002, the Agency for Toxic Substances and Disease Registry (ATSDR) convened a panel of experts to discuss the issue of asbestos fiber length and disease. The meeting summary noted that,

“Given findings from epidemiologic studies, laboratory animal studies, and in vitro genotoxicity studies, combined with the lung’s ability to clear short fibers, the panelists agreed that there is a strong weight of evidence that asbestos and SVFs [synthetic vitreous fibers] shorter than 5 μm are unlikely to cause cancer in humans.” (ATSDR, 2003)

Fibers thicker than 3.0 μm are of lesser concern, because they have little chance of penetrating to the lower regions of the lung.

The EPA, International Agency for Research on Cancer (IARC), National Toxicology Program (NTP), and the American Conference of Governmental Industrial Hygienists (ACGIH) have all classified asbestos as a known human carcinogen (ATSDR, 2001).

2. Lead

General: Lead is a naturally occurring bluish-gray metal found in small amounts in the earth’s crust. Lead reacts with other chemicals to form what are known as lead compounds or lead salts. Some of the chemicals that contain lead are broken down by sunlight, air, and water into other forms of lead. Lead compounds in water may combine with different chemicals depending on the acidity and temperature of the water. Lead itself cannot be broken down. Levels of lead may build up in plants and animals from areas where air, water, or soil are contaminated with lead (ATSDR, 1999).

The most important use for lead is in the production of batteries. It is also used in the production of ammunition, metal products (such as sheet lead, solder, some brass and bronze products, and pipes), and in ceramic glazes. Chemicals containing lead, such as tetraethyl lead and tetramethyl lead, were once used as gasoline additives to increase octane rating. However, their use was phased out in the 1980s, and lead was banned for use in gasoline for transportation beginning in January 1996. Over the years, lead content has also decreased in other products including paint, ceramics, caulk, and solder. Lead is still used in a large variety of medical equipment (radiation shields for protection against X-rays, electronic ceramic parts of ultrasound machines, intravenous

pumps, fetal monitors, surgical equipment, scientific equipment, circuit boards for computers and other electronic circuitry, and military equipment including jet turbine engine blades, and military tracking systems (ATSDR, 1999). An estimated 200,000 pounds of lead were released into the environment during the WTC Event (Claudio, 2001; Nordgrén et al., 2002).

Exposure: Exposure to lead may occur through: (1) ingestion of foods or water that contain lead, (2) inhalation or ingestion of lead-containing dusts, (3) occupational contact, (4) the use of lead-containing health-care products, and (5) sculpturing (lead solder) and staining glass artwork. Foods such as fruits, vegetables, meats, grains, seafood, soft drinks, and wine may also have lead in them. Cigarette smoke also contains small amounts of lead. Sources of lead in drinking water include lead leaching from lead pipes and faucets, and leaded solder used in plumbing. Lead may be found in public drinking water systems, houses, apartment buildings, and public buildings that are more than twenty years old (ATSDR, 1999).

Between 0.5 and 1.5 million workers are exposed to lead in the workplace. Families of workers may be exposed to lead when workers bring home lead dust on their clothes (ATSDR, 1999).

Noncarcinogenic Health Effects: Once absorbed into the body, lead is widely distributed and interacts with a number of enzyme systems. As a result, lead can affect almost every organ and system in the body. The most sensitive systems are the central nervous system (particularly in children), the kidneys, the cardiovascular system, and the reproductive system. The manifestations of lead toxicity are similar whether lead is inhaled or ingested. High lead exposures may decrease reaction times, cause weakness in fingers, wrists, or ankles, and affect memory and cognitive function (Ellenhorn and Barceloux, 1988; Hazardous Substances DB, 2003). Lead exposure may also cause anemia, high blood pressure, lead nephropathy, and damage to the male reproductive system (ATSDR, 1999). Since lead absorbed into the body can cross the placental barrier, lead exposure to the unborn child is also of concern for pregnant women exposed to this chemical.

In adults, approximately 50-70% of lead is absorbed into the blood following inhalation. Following ingestion of lead in adults, only 5-10% is absorbed into the blood; however, children may absorb up to 40%. Lead salts are not absorbed through the skin, but organic lead, such as tetraethyl lead, can be absorbed through the skin in sufficient

quantities to produce systemic toxicity after a high exposure (Ellenhorn and Barceloux, 1988).

Carcinogenicity: There is little conclusive evidence that lead causes cancer in humans. Kidney tumors have developed in rats and mice given unusually large doses of lead. The NTP has determined that lead acetate and lead phosphate may reasonably be expected to be capable of causing cancer, based on sufficient evidence from animal studies, but there is inadequate evidence from human studies (ATSDR, 1999) The EPA and the IARC have classified lead and lead compounds as class B2, probable human carcinogens, based on sufficient evidence from animal data despite insufficient human data (IRIS, 2003).

3. Dioxins¹

General: Polychlorinated dibenzodioxins (PCDDs) are a family of 75 compounds divided into eight groups of chemicals based on the number of chlorine atoms in the compound. The chlorine atoms can be attached to the dioxin molecule at any one of eight positions. 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) is one of the most well-recognized PCDDs and has received the most attention. 2,3,7,8-TCDD is also one of the most toxic PCDDs to mammals and serves as a prototype for the PCDDs. PCDDs with toxic properties similar to 2,3,7,8-TCDD are often called “dioxin-like” compounds (ATSDR, 1998). In the pure form, PCDDs are colorless solids or crystals. 2,3,7,8-TCDD is odorless. The odors of the other PCDDs are not known. PCDDs are known to occur both naturally and as a result of human activities. PCDDs are naturally produced from the incomplete combustion of organic material by forest fires or volcanic activity. Dioxins are formed when organic materials burn in the presence of chlorine, such as found in polyvinyl chloride (PVC) plastics. They are also formed during combustion of polychlorinated biphenyls (PCBs). During the WTC Event, large quantities of dioxin were formed from the combustion of organic materials in the WTC towers, together with chlorine released from burning plastics such as is found in electrical wire coatings. PCDDs are not intentionally manufactured by industry, except in small amounts for research purposes. PCDDs are unintentionally produced by industrial, municipal, and domestic incineration and combustion processes (ATSDR,

¹ In keeping with commonly-recognized nomenclature, the term “Dioxin” is used throughout this report to refer to the family of compounds more specifically identified as polychlorinated dibenzodioxins (PCDD) and polychlorinated dibenzofurans (PCDF).

1998). PCDDs, especially 2,3,7,8-TCDD, are formed during the chlorine bleaching process in pulp and paper mills and as contaminants in the manufacturing process of chlorinated organic chemicals including herbicides such as 2,4,5-trichlorophenoxyacetic acid (a component of Agent Orange). PCDDs have also been detected at low levels in cigarette smoke, home heating systems, and in car and diesel exhaust.

Polychlorinated dibenzofurans (PCDFs) are a family of chemicals that contain one to eight chlorine atoms attached to the carbon atoms of the parent chemical, dibenzofuran. There are at least 135 individual congeners of dibenzofuran with varying health and environmental effects. Of the 135 congeners, the congeners with the chlorine atoms at the 2, 3, 7, and 8 positions have the greatest toxicity. Other than for laboratory use in small amounts for research purposes, PCDFs are not purposefully produced by industry. Most PCDFs are produced in small amounts as unwanted impurities of certain processes and products utilizing chlorinated compounds. In fact, only a few of the PCDF compounds have been produced in large enough quantities to evaluate their chemical properties. These PCDFs are colorless solids that do not easily dissolve in water. There is no known use for PCDFs (ATSDR, 1994). Like PCDDs, PCDFs are formed when organic material burns in the presence of chlorine.

Different dioxin compounds have different toxicities, and dioxins are most often found in mixtures rather than as single compounds in the environment. The most toxic form of dioxin is 2,3,7,8-TCDD. Scientists use a shorthand method for comparing the toxicity of mixtures of dioxins to the toxicity of 2,3,7,8-TCDD. This method is called the "2,3,7,8-TCDD equivalent," or TEQ. Throughout this report, measurements of dioxin in Building dust are reported as Dioxin TEQs in the units of picogram (pg) per 100 cm², or pg/100 cm².

Exposure: Both PCDDs and PCDFs are consistently found at very low levels in the environment. Both PCDDs and PCDFs enter the environment as mixtures containing a variety of individual components and impurities. In the environment, they are typically associated with ash, soil, or any surface with a high organic content, such as plant leaves. In air and water, a portion of the PCDDs may be found in the vapor or dissolved state, depending on the amount of particulate matter (PM), temperature, and other environmental factors. Most humans are exposed to background levels of PCDDs and PCDFs when they inhale, consume food or milk, or have skin contact with materials contaminated with PCDDs or PCDFs. For the general population, more than 90% of the daily intake of PCDDs and PCDFs comes from food, primarily meat, dairy products, and

fish (ATSDR, 1998). Some consumer products including paper towels, coffee filters, tampons, and milk cartons also contain extremely low levels of PCDFs. PCDFs enter the environment from a number of sources including fires or breakdowns involving capacitors, transformers, and other electrical equipment that contain polychlorinated biphenyls (PCBs); in the waste water, sludge, or solids released from industries that created PCDFs as contaminant compounds during the manufacture of other chemicals and consumer products; and from burning municipal and industrial waste in incinerators. The burning of leaded gasoline, coal, wood, and heating oil also releases small amounts of PCDFs into the environment (ATSDR, 1994).

Exposure to PCDDs and PCDFs can also occur through skin contact with chlorinated pesticides and herbicides, contaminated soils, or PCB transformer fluids. Background levels of PCDDs and PCDFs detected in uncontaminated soils in the United States are generally very low or not detectable. 2,3,7,8-TCDD is not usually found in rural soil, but may be found in industrialized areas. However, higher levels of 2,3,7,8-TCDD may be found in areas where PCDDs have contaminated the soil. PCDDs and PCDFs have been found in all samples of adipose tissue and blood (serum lipids) from individuals with no known previous exposure, indicating that all people are exposed to small amounts of PCDDs and PCDFs. Elevated levels of PCDDs and PCDFs have been reported in fish, shellfish, birds, and mammals collected in areas surrounding various chemical production facilities, various hazardous waste sites, and pulp and paper mills using the chlorine bleaching process. Occupational exposure to PCDDs and PCDFs generally occurs through breathing contaminated air, or through skin contact with materials containing PCDDs and PCDFs (ATSDR, 1998; ATSDR, 1994).

Noncarcinogenic Health Effects: Many studies have examined workers exposed during the manufacture of chemicals and pesticides contaminated with 2,3,7,8-TCDD. Other studies have looked at American Vietnam veterans and Vietnamese populations exposed to Agent Orange and populations exposed to 2,3,7,8-TCDD as a result of an accident. A common problem with most of the human studies is that the people were exposed to a number of chemicals at the same time; therefore, the effect of the exposure to 2,3,7,8-TCDD is difficult to analyze (ATSDR, 1998).

A number of effects have been observed in people exposed to 2,3,7,8-TCDD. The most obvious health effect in people with elevated exposures to 2,3,7,8-TCDD is chloracne. Chloracne is a severe skin disease characterized by acne-like lesions. Chloracne generally occurs on the face and upper body, but may occur elsewhere on

the body. Unlike common acne, severe chloracne is harder to cure and can be more disfiguring. Other effects to the skin, such as erythematous or red skin rashes, discoloration, and excessive body hair have been reported to occur in people following exposure to high concentrations of 2,3,7,8-TCDD. Alterations in the ability of the liver to metabolize hemoglobin, lipids, sugar, and protein have been reported in people exposed to relatively high concentrations of 2,3,7,8-TCDD. Most of the effects are considered mild and are reversible. However, in some people these effects may last for many years. Slight increases in the risk of diabetes and abnormal glucose tolerance have been observed in some studies of people exposed to 2,3,7,8-TCDD. There is not enough information available to determine if exposure to 2,3,7,8-TCDD results in reproductive or developmental effects in people, but animal studies suggest that this is a potential health concern (ATSDR, 1998).

The health effects of some PCDDs have been extensively studied in animals. Some PCDDs are much more toxic than others. 2,3,7,8-TCDD and, to a lesser extent, PCDDs with five (penta) or six (hexa) chlorine atoms substituted in the 2, 3, 7, and 8 positions, are extremely toxic to animals. Other PCDDs, which do not have chlorine atoms substituted in the 2, 3, 7, and 8 positions, are considered relatively less toxic compared to 2,3,7,8-TCDD. In general, the doses used in the animal studies result in body burdens that are 10 to 1000 times higher than human background body burdens. The results of the oral animal studies suggest that the most sensitive effects are immune, endocrine, and developmental effects. It is reasonable to assume that these will also be the most sensitive effects in humans.

The available information on the health effects of PCDFs is derived from studies of accidental poisonings in Japan and Taiwan in the 1960s and 1970s. These people ate food for several months that had been cooked in contaminated rice oil. Observed health effects included skin and eye irritations, darkened skin color, severe acne, swollen eyelids with discharge, vomiting, diarrhea, numbness and other effects on the nervous system, anemia, more frequent lung infections, and mild liver changes. Animals fed PCDFs suffered severe body weight loss, and injuries to the stomach, liver, kidney, and immune system (ATSDR, 1994).

Carcinogenicity: Several studies of workers exposed to high levels (with body burdens more than 50 times higher than background body burden levels) of 2,3,7,8-TCDD suggest that exposure to 2,3,7,8-TCDD may increase the risk of cancer. The NTP has determined that it is reasonable to expect that 2,3,7,8-TCDD may cause

cancer. IARC has determined that 2,3,7,8-TCDD can cause cancer in people, but that it is not possible to classify other PCDDs/PCDFs as to their carcinogenicity to humans. The EPA has determined that 2,3,7,8-TCDD is a probable human carcinogen when considered alone and when considered in association with phenoxy herbicides and/or chlorophenols. The EPA has determined that a mixture of PCDDs with six chlorine atoms (4 of the 6 chlorine atoms at the 2, 3, 7, and 8 positions) is a probable human carcinogen (ATSDR, 1998).

Neither the NTP, IARC, nor the EPA have classified the carcinogenicity of PCDFs (ATSDR, 1994).

4. Other Heavy Metals

4.1. Barium

General: Barium is a silvery-white metal that occurs naturally as chemical compounds such as barium sulfate or barium carbonate. The most common use of barium is as a component in drilling muds for lubricating drill bits in the gas and oil industries. Barium sulfate is used as a contrasting agent in certain medical tests and barium carbonate is used in the manufacture of bricks and tiles (ATSDR, 1992).

Noncarcinogenic Health Effects: Humans exposed to an acute oral dose of barium experienced cardiovascular effects including hypertension, abnormal ECG, heart damage and changes in heart rhythm. Animal studies using acute, intermediate and chronic dosing reported similar effects. Acute oral exposure in humans has also been associated with death, paralysis, muscle weakness, tingling of the mouth and neck, gastrointestinal disturbances and lowered blood potassium levels. Occupational exposure to barium dust may lead to minor respiratory effects (ATSDR, 1992).

Carcinogenicity: The NTP, IARC and EPA have not classified barium as to its carcinogenicity (ATSDR, 1992).

4.2. Cadmium

General: Cadmium is a soft silvery-white metal that occurs naturally as cadmium oxide, cadmium sulfide or cadmium chloride. Cadmium is used in the manufacture of batteries, plastics, paints, glasses and pigments. Cadmium can also be found in fertilizers (ATSDR, 1999).

Noncarcinogenic Health Effects: The kidney is the primary target organ for cadmium. Cadmium exposure can lead to abnormal renal function as well as increased frequency of kidney stone formation. Cadmium can also cause bone disorders such as osteoporosis or osteomalacia. The degree of bone loss is correlated with abnormal renal function. Inhalation of high levels of cadmium may initially cause mild respiratory irritation. However, after a short latency period, this can progress to breathlessness, wheezing, and severe flu-like symptoms followed by permanent lung dysfunction or death. Chronic occupational exposures to cadmium may cause emphysema and dyspnea. Oral exposure to cadmium can cause severe gastrointestinal irritation resulting in nausea, cramps, and abdominal pain. Cadmium also reduces the gastrointestinal uptake of iron which may lead to anemia (ATSDR, 1999).

Carcinogenicity: Human and animal studies have indicated that inhalation of cadmium may result in higher incidences of lung cancer; however the data regarding the carcinogenicity of cadmium are conflicting. The IARC has determined that cadmium and cadmium compounds are known human carcinogens. The EPA has classified cadmium as a probable human carcinogen (ATSDR, 1999).

4.3. Chromium

General: Chromium is present in the environment in three forms; elemental chromium (0), trivalent chromium (III) and hexavalent chromium (VI). Chromium (III) is the most chemically stable and is subsequently the predominant form found in nature. It is used in the manufacture of metals and heat resistant brick. Chromium (III) is also an essential nutrient that enhances insulin activity. Due to its high melting point, elemental chromium is used to produce steel. Both chromium (III) and chromium (VI) are used in electroplating, leather tanning, and pigment and dye production (ATSDR, 2000).

Noncarcinogenic Health Effects: Inhalation exposure to high levels of chromium (VI) can cause runny nose, nosebleeds, sneezing, and formation of holes or ulcers in the nasal septum. Long term occupational exposure to high levels may also lead to lung cancer. Ingestion of large amounts of chromium (VI) can cause gastrointestinal disturbances, liver or kidney damage, convulsions and death. Prolonged exposure to low levels of chromium (VI) does not appear to cause adverse health effects in most humans. Dermal exposure to chromium (VI) may cause skin ulcers. Some humans are allergic to chromium and may develop severe asthma or other allergic reactions after exposure to chromium (III) or chromium (VI) (ATSDR, 2000).

Carcinogenic Health Effects: The NTP, IARC and EPA have classified chromium (VI) as a human carcinogen. Chromium (III) and chromium (0) are not classifiable as to their carcinogenicity in humans (ATSDR, 2000).

4.4. Copper

General: Copper is a naturally occurring reddish metal that is an essential element for all plants and animals. Copper has several important physical properties that make it an important metal. It is durable, malleable, ductile, and conducts heat and electricity. Copper and copper compounds are used in current American coins, electrical wiring, pipe and sheet metal. Copper is also used as a fungicide (ATSDR, 2002).

Noncarcinogenic Health Effects: Occupational exposure to copper dust can cause sneezing, coughing, and runny nose. Copper toxicity has been linked to “vineyard sprayer’s lung”, a disease seen in vineyard workers applying copper sulfate as a fungicide. Exposure to copper dust can cause eye irritation, dizziness, and headache in humans. Ingestion of large amounts of copper can cause severe gastrointestinal effects. Hepatic toxicity is seen in some genetic syndromes that are associated with high liver copper levels and in animals after acute or intermediate exposure. There is also evidence of renal toxicity after ingestion of copper in both humans and animals. Acute oral exposure can cause death, due to hepatic and renal failure (ATSDR, 2002).

Carcinogenic Health Effects: The EPA has determined that copper is not classifiable as to human carcinogenicity (ATSDR, 2002).

4.5. Manganese

General: Manganese metal is a silvery metal that does not exist naturally in the environment, but as a compound with sulfur, chlorine or oxygen. Manganese is an essential element and its physiological level is tightly regulated. Manganese metal is added to steel to improve its hardness and strength, and various manganese compounds are used in fertilizers, ceramics, batteries and dietary supplements (ATSDR, 2000).

Noncarcinogenic Health Effects: Occupational inhalation exposure to high levels of manganese can cause manganism, a progressive Parkinson-like disease characterized by altered gait, stiffness, a dull wooden aspect and occasionally, psychiatric disturbances. The early symptoms are slow, halting speech accompanied by

wooden facial expressions and clumsy movements. As the disease progresses, it becomes more difficult to walk and voluntary muscle tremor develops. These symptoms have been found in miners and foundry workers. "Manganese madness" is a behavioral effect documented in manganese miners that includes bizarre impulsive fits of laughing, dancing or running as well as nervousness, irritability, and aggressive and destructive behavior. Occupational exposure to manganese can also lead to impotence. The data regarding oral exposure are less clear. Animal studies suggest that ingestion of large quantities of manganese may cause brain damage (ATSDR, 2000).

Carcinogenic Health Effects: The EPA has determined that manganese is not classifiable as a human carcinogen (ATSDR, 2000).

4.6. Zinc

General: Zinc, one of the most common elements in the earth's crust, is found in the air, soil, and water, and is present in all foods. Powdered zinc is explosive and may burst into flames if stored in damp places. Metallic zinc has many uses in industry, such as a coating material for iron or other metals, in alloys such as brass and bronze, and in dry cell batteries. Zinc forms compounds with chlorine, oxygen, and sulfur, many of which may be found at hazardous waste sites. Most zinc ore found naturally in the environment is in the form of zinc sulfide, a non-explosive, non-flammable, gray white or yellow-white solid. Zinc compounds are used in a variety of industrial settings for production of materials. Zinc is also an essential food element needed by the body in small amounts (ATSDR, 1994).

Exposure: Small exposure to zinc compounds results from food consumption. Zinc is also present in most drinking water. Drinking water or other beverages may contain high levels of zinc if they are stored in metal containers or flow through pipes that have been coated with zinc to resist rust. Drinking water may also be contaminated by zinc from industrial sources or toxic waste sites. High-level exposure to zinc may also result from taking too many zinc dietary supplements. Fetuses and nursing children may be exposed to zinc in the blood or milk of their mothers. Levels of zinc in air are relatively low and fairly constant. Average levels of zinc in the air throughout the US are less than 1 $\mu\text{g}/\text{m}^3$ of air (ATSDR, 1994).

About 150,000 workers are exposed to zinc at their jobs. Jobs where people are exposed to zinc include zinc mining, smelting, and welding; manufacture of brass, bronze, or other zinc-containing alloys; manufacture of galvanized metals; and

manufacture of machine parts, rubber, paint, linoleum, oil cloths, batteries, some kinds of glass and ceramics, and dyes. People at construction jobs, automobile mechanics, and painters are also exposed to zinc (ATSDR, 1994).

Noncarcinogenic Health Effects: Taking too much zinc into the body through food, water, or dietary supplements can adversely affect health. Ingesting high levels of zinc for several months may cause anemia, damage the pancreas, and decrease levels of high-density lipoprotein (HDL) cholesterol (ATSDR, 1994).

Carcinogenicity: EPA has determined that zinc is not classifiable as to its human carcinogenicity (ATSDR, 1994).

5. Mercury

General: Mercury occurs naturally in the environment and exists as metallic mercury (elemental mercury), inorganic mercury salts, and organic mercury. Metallic mercury is a shiny, silver-white metal used in thermometers and some electrical switches. At room temperature, metallic mercury is volatile, releasing colorless and odorless vapors. Inorganic mercury salts occur when mercury combines with elements such as chlorine, sulfur, or oxygen, forming white powders or crystals. Organic mercury compounds, or organomercurials, form when mercury combines with carbon. Organomercurials are abundant in the environment, the most common being methylmercury. Several forms of mercury occur naturally in the environment, the most common being metallic mercury, mercuric sulfide (cinnabar ore), mercuric chloride, and methylmercury.

Some inorganic mercury compounds are used as fungicides. Inorganic salts of mercury, including ammoniated mercuric chloride and mercuric iodide, have been used in skin-lightening creams. Mercuric chloride is a topical antiseptic or disinfectant agent. Mercuric sulfide and mercuric oxide may be used to color paint, and mercuric sulfide is one of the red coloring agents used in tattoo dyes. Until the 1970s, methylmercury and ethylmercury compounds were used to protect seed grains from fungal infections. Once the adverse health effects of methylmercury were known, the use of methylmercury and ethylmercury as fungicides was banned. Until 1991, phenylmercuric compounds were used as antifungal agents in both interior and exterior paints, but this use was also banned because mercury vapors were released from these paints (ATSDR, 1999).

Exposure: Because it occurs naturally, humans are exposed to very low levels of mercury in air, water, and food. Background levels are about 6 ng/m³ or less. Mercury

levels in surface water are generally less than 5 parts per trillion (ppt). Soil levels range from 20 to 625 parts per billion (ppb). A potential source of exposure to metallic mercury for the general population is mercury released from dental amalgam fillings (ATSDR, 1999).

Spills of metallic mercury from broken thermometers or damaged electrical switches in the home may result in exposure to mercury vapors in indoor air. Exposure to mercury vapors can also result from the use of fungicides or other products that contain mercury. Metallic mercury and its vapors are extremely difficult to remove from clothes, furniture, carpet, floors, walls, and other such items. Without proper cleaning, the mercury can remain for months or years and continue to be a source of exposure (ATSDR, 1999).

Metallic mercury vapor is easily absorbed through the lungs into the bloodstream. Mercury is retained in both the kidney and brain and remains in the body for weeks to months even after exposure stops. Inorganic mercury salts and organic mercury are absorbed mainly via ingestion and absorption from the digestive tract. Like metallic mercury, these forms are also retained in the body for months after exposure ends.

Noncarcinogenic Health Effects: The primary adverse effects of mercury involve the central nervous system. Permanent brain damage can result from exposure to high levels of metallic mercury. Exposure to metallic mercury vapors or organic mercury may cause personality changes (irritability, shyness, nervousness), tremors, changes in vision (constriction of the visual field), deafness, muscle in-coordination, loss of sensation, and difficulties with memory (ATSDR, 1999). Consumption of fish contaminated with large amounts of methylmercury or other organomercurials can cause permanent brain and kidney damage.

Very young children may be more sensitive to mercury than adults. In addition, mercury can cross the placenta and may cause brain damage, mental retardation, in-coordination, blindness, seizures, and speech impairment. Mercury can also be passed from mother to child in breast milk (ATSDR, 1999).

The kidneys are sensitive to the effects of mercury due to renal mercury accumulation causing higher exposures to these tissues. All forms of mercury are nephrotoxic at sufficient concentrations, though the kidneys can recover from mercury toxicity once the body clears itself of the contamination (ATSDR, 1999).

Short-term inhalation exposure (hours) to high levels of metallic mercury vapor can damage the lining of the mouth and irritate the lungs and airways, causing airway

tightness, a burning sensation in the lungs, and coughing. Other effects from exposure to mercury vapor include nausea, vomiting, diarrhea, increases in blood pressure or heart rate, skin rashes, and eye irritation. Damage to the lining of the mouth and lungs can also occur from exposure to lower levels of mercury vapor over longer periods. Skin contact with metallic mercury has been shown to cause an allergic reaction resulting in skin rashes in some people (ATSDR, 1999).

Carcinogenicity: Studies in workers exposed to metallic mercury vapors have not shown any mercury-related increase in cancer. Animals given inorganic mercury salts by mouth for most of their lifetime had increases in some kinds of tumors at the highest dose tested. Male rats and mice that received organic mercury (methylmercury or phenylmercury) in their drinking water or feed for most of their lives had increased incidences of cancer of the kidney at the highest doses. As a result, NTP and IARC have not classified mercury as to its human carcinogenicity. In contrast, the EPA has classified mercuric chloride and methylmercury as possible human carcinogens (ATSDR, 1999).

6. Polychlorinated Biphenyls (PCBs)

General: PCBs are a group of synthetic organic chemicals with no known natural source in the environment. PCBs can be oily liquids or solids and are colorless to light yellow with no known smell or taste. Some PCBs are volatile and may exist as a vapor in air. PCBs enter the environment as mixtures containing a variety of individual chlorinated biphenyl components, or congeners, as well as impurities. Some commercial PCB mixtures are known in the United States by their industrial trade name, such as Aroclor. Because PCBs have low flammability and excellent insulating properties, PCBs were used widely as coolants and lubricants in transformers, capacitors, and other electrical equipment. The manufacture of PCBs ended in the United States in 1977 because of evidence of environmental buildup and harmful effects in exposed populations. Consumer products that may contain PCBs include old fluorescent lighting fixtures, electrical devices or appliances containing PCB capacitors made before PCB use was stopped, old microscope oil, and old hydraulic oil (ATSDR, 2000).

Exposure: PCBs adsorb strongly to soil, do not readily break down, and may persist in dust and soil for long periods of time. The greater degree of chlorination present in the PCB congener, the more slowly the compound degrades (ATSDR, 2000).

Many older transformers and capacitors still contain PCBs, as the lifetime of use for this equipment exceeds 30 years. Overheating of PCB-containing electrical devices can raise the level of PCBs in indoor air. Small amounts of PCBs can be found in almost all outdoor and indoor air, soil, sediments, surface water, and animals. However, PCB levels have generally decreased since PCB production stopped in 1977. The general population is exposed to PCBs primarily from contaminated food and breathing contaminated air. The major dietary sources of PCBs are fish, meat, and dairy products (ATSDR, 2000).

Workplace exposure to PCBs can occur during repair and maintenance of PCB-containing transformers; accidents, fires, or spills involving PCB transformers and older computers and instruments; and disposal of PCB materials. In addition to older electrical instruments and fluorescent lights that contain PCB-filled capacitors, caulking materials, elastic sealants, and heat insulation have also been known to contain PCBs. Contact with PCBs at hazardous waste sites can happen when workers breathe air and touch soil containing PCBs (ATSDR, 2000).

Noncarcinogenic Health Effects: Exposure to PCBs has been reported to cause adverse health effects in both workers and the general population. Skin conditions, such as chloracne and rashes, may occur in people exposed to high levels of PCBs. These effects on the skin are well documented, but are not likely to result from exposures in the general population. Some studies in workers suggest that exposure to PCBs may also cause irritation of the nose and lungs, gastrointestinal discomfort, changes in the blood and liver, and depression and fatigue (ATSDR, 2000).

Rats fed large amounts of PCBs for short periods of time experienced mild liver damage. Rats, mice, and monkeys fed smaller amounts of PCBs over several weeks or months developed anemia, acne-like skin conditions, and liver, stomach, and thyroid gland injuries. Other effects of PCBs included decreased immune system function, changes in behavior, and reproductive problems. Some PCBs affect endocrine function by mimicking or blocking the action of hormones from the thyroid and other glands. (ATSDR, 2000).

Carcinogenicity: Studies of workers provide evidence that PCBs are associated with cancer of the liver and biliary tract. Rats that ate commercial PCB mixtures throughout their lives developed liver cancer. Based on the evidence for cancer in animals, the NTP has stated that PCBs may reasonably be anticipated to be

carcinogens. Both the EPA and IARC have determined that PCBs are probably carcinogenic to humans (ATSDR, 2000).