

4.0 Site Conceptual Model

The ecological site conceptual model (SCM) is a simplified, schematic diagram of possible exposure pathways and the means by which contaminants are transported from the primary contaminant source(s) to ecological receptors. The exposure scenarios include the sources, environmental transport, partitioning of the contaminants amongst various environmental media, potential chemical/biological transformation processes, and identification of potential routes of exposure for the ecological receptors. In this chapter, the SCM will be described in relation to constituent fate and transport properties, the ecotoxicity of the various constituents, potential ecological receptors at the ranges, and the complete exposure pathways expected to exist at the ranges.

4.1 Constituent Fate and Transport

The environmental fate and transport of contaminants in the various media at the IMR ranges will govern the potential for exposures to ecological receptors. In general, contaminants in environmental media may be available for direct exposure (e.g., plants exposed to surface soil), and they may also have the potential to migrate to other environmental media or other areas of the site. This section discusses the mechanisms by which contaminants can be transported and the chemical properties that determine their transport.

4.1.1 Fate and Transport in Soil

Contaminants in surface soil at the IMR ranges have the potential to be transported from their source area to other areas within the respective ranges and to off-site locations by a number of mechanisms, including volatilization, dust entrainment, surface runoff, and infiltration to subsurface soil/groundwater.

Several volatile organic compounds (VOC) were identified in the upper soil horizons at the IMR ranges. These volatile constituents have a high potential to volatilize to the atmosphere and be transported from their source area via air movement. The concentrations of VOCs detected in surface soil at the IMR ranges are low; therefore, this transport mechanism is expected to be insignificant with respect to other transport mechanisms active at this site. Most of the metals and semivolatile organic compounds (SVOC) in the surface soil at the IMR ranges are not expected to volatilize to any great extent, with the exception of mercury, which would be expected to volatilize relatively rapidly. Most of the metals and SVOCs in the surface soil at the IMR ranges are generally closely associated with particulate matter and would be transported from their source areas by fugitive dust generation and entrainment by the wind. Subsequent

dispersion by atmospheric mixing could transport particulate-associated contaminants to other parts of the IMR ranges and to off-site locations. The generation of fugitive dust and subsequent transport by the wind is potentially a significant transport mechanism at the IMR ranges, based on the presence of unvegetated areas and areas of sparse vegetation within certain areas of these ranges (e.g., impact areas and soil berms).

The transport of surface soil-associated contaminants by surface runoff is another potentially significant transport mechanism. Surface soil contaminants may be solubilized by rainwater and subsequently transported to drainage ditches, low-lying areas, and Remount Creek via surface runoff. The solubility of inorganics in rainwater is largely dependent upon the pH of the rainwater. Because the rainwater in this region is most likely slightly acidic, the inorganic constituents in surface soil are likely to solubilize to some degree in the rainwater and be subject to transport via runoff. Most of the semivolatile compounds are strongly associated with soil particles and would not solubilize to a large extent. Contaminants that may be more strongly bound to particulate matter in surface soil (e.g., SVOCs and some of the inorganics) may be entrained in surface water runoff and transported to drainage ditches, low-lying areas, and Remount Creek via surface runoff. Many of the metals and semivolatiles are strongly sorbed to soil particles and could be transported from their source areas via this mechanism.

Contaminants in surface soil may be transported vertically to subsurface soils and groundwater via solubilization in rainwater and infiltration. Subsequent groundwater transport to surface water in Remount Creek could result in exposure of aquatic receptors to soil contaminants. Migration in this manner is dependent upon contaminant solubility and frequency of rainfall. Although the soil types in the vicinity of the IMR ranges (sand, stone, and gravel) are expected to promote relatively rapid infiltration of rainwater, the less soluble constituents (e.g., SVOCs) found at the IMR ranges are not likely to migrate to any great extent vertically, due to their relatively low solubilities. Inorganics in soil at the IMR ranges may migrate vertically due to the acidic nature of the rainwater in this area and the increased solubility of metals that it produces. However, surface water and groundwater monitoring data indicate that this transport mechanism is insignificant at the IMR ranges, as only lead was detected in surface water at elevated concentrations and lead was not detected in any groundwater samples at elevated concentrations. Furthermore, other constituents detected in groundwater were not detected in surface water at the IMR ranges.

The transfer of contaminants in surface soil to terrestrial plants through root uptake and transfer to terrestrial animals through ingestion and other pathways are potentially significant transfer mechanisms. Many metals are readily absorbed from soil by plants, but they are not

biomagnified to a great extent through the food web. There are several exceptions to this, namely, arsenic and nickel, which may bioconcentrate and/or biomagnify (Agency for Toxic Substances and Disease Registry [ATSDR], 1989 and 1995). Many of the SVOCs have the potential to bioaccumulate in lower trophic level organisms (e.g., terrestrial invertebrates), but most higher trophic level animals have the ability to metabolize these compounds rapidly, precluding the potential for bioconcentration (Eisler, 1987).

VOCs in the surface soil at the IMR ranges are expected to volatilize and/or photolyze rapidly (half-lives of 3 hours to 5 days) when exposed to sunlight (Burrows, et al., 1989). The other surface soil contaminants (metals and semivolatiles) are expected to remain in the soil relatively unchanged by physical and/or chemical processes for much longer periods of time.

4.1.2 Fate and Transport in Surface Water

In general, contaminants present in the surface water associated with the IMR ranges (Remount Creek and tributaries) are the result of erosion and runoff from the ranges. Contaminants in surface water at the IMR ranges may be transported from their sources to other areas at the ranges or to off-site locations by the following mechanisms: 1) volatilization; 2) transfer to groundwater; 3) transfer to sediment; and 4) flow downstream. Volatile organic contaminants in surface water would be expected to rapidly volatilize from the water-air interface and be dispersed in the atmosphere. Therefore, transport of volatile constituents in surface water is not expected to occur for any significant distance.

Water in Remount Creek originates mainly from discharge from Yahou Lake and overland flow from the surrounding watershed. There also appears to be localized contributions to creek flow from groundwater where the potentiometric surface exceeds the creek bed surface. The flow contribution in Remount Creek from groundwater varies according to the amount of precipitation, with an increase of groundwater contribution when precipitation raises the potentiometric surface.

Thus constituents in groundwater could migrate to surface water in Remount Creek and its tributaries. This transport mechanism appears to be relatively insignificant based on the fact that only lead has been detected in surface water at concentrations that are elevated (with respect to ecological screening values). Other constituents detected in groundwater have not been detected in surface water at elevated concentrations. Additionally, elevated lead concentrations in surface water are restricted to a drainage ditch and a small tributary to Remount Creek at the Skeet Range. Contaminant transfer to sediments represents another significant transfer mechanism, especially where contaminants are in the form of suspended solids, or are hydrophobic

substances (e.g., PAH) that can become adsorbed to organic matter in the sediments. The metals detected in surface water have the potential to associate with suspended particulate matter.

Contaminants in surface water can be transported to other ranges along Iron Mountain Road or off site via Remount Creek. Transfer of contaminants in surface water to aquatic organisms is also a potentially significant transfer pathway. Some of the inorganic constituents detected in surface water may bioaccumulate in lower trophic level organisms. Most of the inorganics detected in surface water are not highly bioconcentratable; therefore, transfer through the food web is expected to be minimal for these compounds.

4.1.3 Fate and Transport in Sediment

Contaminant transfer between sediment and surface water potentially represents a significant transfer mechanism, especially when contaminants are in the form of suspended solids. Sediment/surface water transfer is reversible; sediments often act as temporary repositories for contaminants and gradually release contaminants to surface waters. This is especially true in surface water systems that are acidic, as is the case with Remount Creek in the vicinity of the IMR ranges. Sorbed or settled contaminants can be transported with the sediment to downstream locations. Much of the substrate of Remount Creek and its tributaries in the vicinity of the IMR ranges is best characterized as gravel or cobbles. Very few areas of high organic content sediment or muck are present. The very low organic content of gravel and cobble create a substrate with very low binding capacity; therefore, constituents released to Remount Creek and its tributaries via surface runoff or other transport mechanisms would most likely remain suspended in the surface water, be transported downstream, and would not be sequestered in the stream substrate directly adjacent to the IMR ranges.

Although transfer of sediment-associated contaminants to bottom-dwelling biota also represents a potentially significant transfer mechanism, it is not expected to be a major mechanism at the IMR ranges. Lower trophic level organisms may accumulate metals and PAHs; however, higher trophic level organisms have the ability to metabolize PAHs and, therefore, reduce their accumulative properties. Most of the inorganics detected in sediment are not bioaccumulative. Mercury and copper may bioaccumulate to some extent due to exposures to sediment.

4.2 Ecotoxicity

The ecotoxicological properties of the constituents detected in the various environmental media at the IMR ranges are discussed in the following sections.

4.2.1 Antimony

Antimony binds to soil and particulates (especially those containing iron, manganese, or aluminum) and is oxidized by bacteria in soil. Exposure routes for aquatic organisms include ingestion and gill uptake. Antimony bioconcentrates in aquatic organisms to a small degree. Exposure routes for mammals include ingestion and inhalation. It does not biomagnify in terrestrial food chains (Ainsworth, 1988). Antimony is not significantly metabolized and is excreted in the urine and feces. Antimony causes reproductive, pulmonary, and hepatic effects in mammals (U.S. Environmental Protection Agency [EPA], 1999a).

Plants. Antimony is considered a nonessential element and is easily taken up by plants if available in the soil in soluble forms (Kabata-Pendias and Pendias, 1992). A screening level of 5.0 milligrams per kilogram (mg/kg) has been proposed by Kabata-Pendias and Pendias (1992), based on a report of unspecified phytotoxic responses by plants grown in soil amended with antimony.

Mammals. Female mice exposed to 5.0 milligrams per liter (mg/L) antimony as antimony potassium tartrate in their drinking water showed a reduction in their lifespan. This dose was equivalent to a lowest-observed-adverse-effects-level (LOAEL) of 1.25 milligrams per kilogram per day (mg/kg/day), which can be converted to a no-observed-adverse-effects-level (NOAEL) of 0.125 mg/kg/day (Integrated Risk Information Service [IRIS], 2001).

Laboratory data on antimony toxicity (as antimony potassium tartrate) in laboratory mice through drinking water ingestion were used to estimate a chronic NOAEL value of 0.125 mg/kg/day (Schroeder, et al., 1968). Lifespan and longevity were the endpoints tested.

Aquatic Life. The available data for antimony indicate that acute and chronic toxicity to freshwater aquatic life occur at concentrations as low as 9.0 and 1.6 mg/L, respectively, and would occur at lower concentrations among species that are more sensitive than those tested. Toxicity to algae can occur at concentrations as low as 0.61 mg/L.

Effects from antimony exposure on benthic community composition have been detected at levels between 3.2 and 150 mg/kg (Long and Morgan, 1990). Data on antimony suggest an effects range-low (ER-L) of 2 mg/kg and an effects range-medium (ER-M) of 25 mg/kg.

4.2.2 Arsenic

Arsenic occurs naturally as sulfides and as complex sulfides of iron, nickel, and cobalt (Woolson, 1975). In one form or another, arsenic is present in rocks, soils, water, and living

organisms at concentrations of parts per billion (ppb) to parts per million (ppm) (National Academy of Sciences [NAS], 1977a). Arsenic is ubiquitous in living tissue and is constantly being oxidized, reduced, or otherwise metabolized. In soils, insoluble or slightly soluble arsenic compounds are constantly being resolubilized, and the arsenic is being presented for plant uptake or reduction by organisms and chemical processes (NAS, 1977a). Among elements, arsenic ranks 20th in abundance in the Earth's crust (1.5 to 2 mg/kg), 14th in sea water, and 12th in the human body (Woolson, 1975). It occurs in various forms, including inorganic and organic compounds, and in trivalent and pentavalent states (Pershagen and Vahter, 1979).

Plants. The NAS (1977a) reports background arsenic concentrations in terrestrial plants as ranging from 0.01 to 5 mg/kg (dry weight). Generally, the roots of a plant contain higher concentrations of arsenic than leaves. The toxicity of arsenic to plants may differ due to different soil conditions. Various chemical forms of arsenic have different phytotoxicities. In general, arsenates are less toxic to plants than arsenites. Concentrations of arsenic in leaf tissue that are excessive or toxic to various plant species range from 5 to 20 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). A soil concentration of 10 mg/kg has been proposed by Efrogmson, et al., (1997) as a benchmark screening value for phytotoxicity in soils. General symptoms of arsenic toxicity in plants include the presence of red-brown necrotic spots on old leaves, yellowing or browning roots, depressed tillering, wilting of new leaves, and root discoloration (Kabata-Pendias and Pendias, 1992).

Mammals. Arsenic is more toxic to wildlife in its trivalent form as compared to either pentavalent arsenic or organic arsenicals. Arsenic poisoning has been reported in acute episodes, but chronic arsenosis is rarely encountered (NAS, 1977a). The probability of chronic arsenic poisoning from continuous ingestion of small doses is rare, because detoxification and excretion are rapid (Woolson, 1975). General signs of arsenic toxicosis include intense abdominal pain, staggering gait, extreme weakness, trembling, fast and feeble pulse, collapse, and death (Eisler, 1988a). Adverse effects in mammals were noted in single oral doses of 2.5 to 33 mg/kg body weight and at chronic oral doses of 1 to 10 mg/kg body weight. As little as 1.7 mg/kg has been shown to produce fetal death and malformations during critical stages of hamster embryogenesis. Various species of rodents exposed to cacodylic acid through various routes exhibited lethal dose for 50 percent population tested (LD₅₀) ranging from 470 to 830 mg/kg body weight (Hood, 1985).

Laboratory data for arsenic toxicity (as arsenite) through drinking water in laboratory mice were used to derive a NOAEL value of 0.126 mg/kg/day (Schroeder and Mitchener, 1971).

Reproduction was the endpoint for these laboratory tests. Arsenic concentrations of greater than

10 mg/kg (wet weight) in tissue are usually indicative of arsenic poisoning (Goede, 1985). Detoxification and excretion of arsenic are relatively rapid processes, making the probability of chronic arsenic poisoning from the continuous ingestion of small amounts of arsenic a rare event (Eisler, 1988a).

Birds. As with mammals, arsenic poisoning in birds has been reported in acute episodes, but chronic arsenosis is rarely encountered. Signs of inorganic trivalent arsenite poisoning in birds include muscular incoordination, debility, slowness, jerkiness, falling hyperactivity, immobility, and seizures (Eisler, 1988a). Studies reported by Hudson, et al., (1984), using mallard ducks (*Anas platyrhynchos*) fed sodium arsenite, determined an LC₅₀ of 323 mg/kg body weight. Copper acetoarsenite fed to the northern bobwhite (*Colinus virginianus*) at 480 mg/kg proved fatal to 50 percent of the test organisms in 11 days (NAS, 1977a). The grey partridge (*Perdix perdix*) succumbed to 300 mg/kg body weight of lead arsenate in 52 hours (NAS, 1977a).

Aquatic Life. Toxic and other effects of arsenicals to aquatic life are significantly modified by numerous biological and abiotic factors (Woolson, 1975; NAS, 1977a; National Research Council of Canada (NRCC), 1978; EPA, 1980a; Howard, et al., 1984; Michnowicz and Weaks, 1984; Bryant et al., 1985; EPA, 1985a; Sanders, 1986). The LC₅₀ values, for example, are markedly affected by water temperature, pH, Eh, organic content, phosphate concentration, suspended solids, and the presence of other substances and toxicants, as well as arsenic speciation and duration of exposure. In general, inorganic arsenicals are more toxic to aquatic biota than organoarsenicals, and trivalent species are more toxic than pentavalent species. Early life stages are most sensitive, and large interspecies differences have been recorded, even among those closely related taxonomically. Juvenile bluegills (*Lepomis macrochirus*) exhibited reduced survival after sixteen weeks when exposed to a single treatment of trivalent arsenic at 0.69 mg/L (EPA, 1980a, 1985a). An adult bluegill population was reduced 42 percent after several monthly applications of 4 mg/L trivalent arsenic (NAS, 1977a).

As with fish, toxic and other effects of arsenicals to aquatic invertebrates are significantly modified by numerous biological and abiotic factors (Woolson, 1975; NAS, 1977a; NRCC, 1978; EPA, 1980a; Howard, et al., 1984; Michnowicz and Weaks, 1984; Bryant, et al., 1985; EPA, 1985a; Sanders, 1986). The cladoceran *Daphnia magna* population exposed to 4.3 mg/L trivalent arsenic exhibited a 50 percent immobilization after 96 hours, and the amphipod *Gammarus pseudolimnaeus* experienced 50 percent immobilization following 96 hours of 0.96 mg/L trivalent arsenic exposure (Lima, et al., 1984).

4.2.3 Barium

Barium, a silvery-white metal, is used in various alloys, in paints, soap, paper, rubber, and in the manufacture of ceramics and glass. Two forms of barium, barium sulfate and barium carbonate, are often found in nature as underground ore deposits. Barium is relatively abundant in nature and is found in plants and animal tissue. Plants can accumulate barium from the soil.

Most of the barium that enters an animal's body is removed within a few days, and almost all of it is gone within 1 to 2 weeks. Most of the barium that stays in the body goes into the bones and teeth. Rats exposed to barium in their diet at lower doses, but for longer time periods, showed increased blood pressure and changes in the function and chemistry of the heart (ATSDR, 1992a).

Plants. Background concentrations of barium in various food and feed plants are reported to range from 1 to 198 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). Concentrations are often highest in the leaves of cereals and legumes and lowest in grains and fruits (Kabata-Pendias and Pendias, 1992). The availability of barium to plants is greatly influenced by the pH of the soil, with barium more available under acidic soil conditions (Kabata-Pendias and Pendias, 1992). The concentration of barium in leaf tissue that has been reported as excessive or toxic to various plant species is 500 mg/kg (Kabata-Pendias and Pendias, 1992). A soil concentration of 500 mg/kg has been proposed by Efrogmson, et al. (1997) as a benchmark screening value for barium phytotoxicity.

Mammals. Barium administered to rats via their drinking water at doses of 1, 10, and 100 mg/L had no effect on food or water consumption, or growth. Because the highest dose tested (100 mg/L) did not elicit any adverse effects, it was considered the NOAEL (5.1 mg/kg/day) (IRIS, 2001). Laboratory rat toxicity data for barium chloride in drinking water was used to calculate a NOAEL value of 5.1 mg/kg/day. Growth and hypertension were the test endpoints.

Aquatic Life. The chronic value for daphnids is from a 21-day test on *Daphnia magna* by Biesinger and Christensen (1972) which resulted in 16 percent reproductive impairment. The Tier II secondary acute water quality value and secondary chronic water quality value for barium, as calculated by the method described in the EPA's *Proposed Water Quality Guidance for the Great Lakes System* (EPA, 1985b), are 110 and 4.0 micrograms per liter ($\mu\text{g/L}$), respectively.

4.2.4 Beryllium

In environmental media, beryllium usually exists as beryllium oxide. Beryllium has limited solubility and mobility in sediment and soil.

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

Soluble forms of beryllium are easily taken up by plants, probably in a manner similar to calcium and magnesium, but it is not readily translocated from roots to shoots (Peterson and Girling, 1981). Beryllium has been reported to inhibit seed germination, enzyme activation, and uptake of calcium and magnesium by roots. Common symptoms of beryllium toxicity to plants are brown, retarded roots and stunted foliage (Romney and Childress, 1965). The phytotoxicity benchmark value for beryllium (10 mg/kg) is based on unspecified toxic effects on plants grown in surface soil amended with 10 mg/kg beryllium (Kabata-Pendias and Pendias, 1992).

Mammals. The major exposure route for mammals is inhalation. Based on animal studies, beryllium is poorly absorbed from the gastrointestinal tract, and is not absorbed through intact skin to any significant degree. The most important route of exposure for beryllium is inhalation, although absorption by this route does not appear to be extensive. Mammals exposed via inhalation exhibit pulmonary effects that may last long after exposure ceases. Once beryllium is absorbed, it is circulated in the blood as an orthophosphate colloid and is then distributed primarily to the bone, liver, and kidneys in both humans and animals. Beryllium and its compounds are not biotransformed, but soluble beryllium compounds are partially converted to more insoluble forms in the lungs (Reeves and Vorwald, 1967).

Following inhalation of soluble beryllium compounds in both humans and animals, the lung appears to be the main target organ for toxicity. Acute exposure may cause chemical pneumonitis; chronic exposure to insoluble forms may lead to chronic beryllium disease (berylliosis), a fibrotic lung disease (ATSDR, 1993). Laboratory data based on beryllium sulfate exposures to rats through their drinking water were used to estimate a NOAEL value of 0.66 mg/kg/day (Schroeder and Mitchener, 1975). Longevity and weight loss were the endpoints in this study.

A variety of beryllium compounds have been demonstrated to cause pulmonary tumors following inhalation in animals. However, it is thought that oral administration does not lead to carcinogenesis, due to poor absorption of the constituent from the gastrointestinal tract. The

NOAEL for a rat lifetime chronic exposure to beryllium in drinking water was 0.54 mg/kg/day (Health Effects Assessment Summary Tables [HEAST], 1997).

Aquatic Life. Exposure routes for aquatic organisms include ingestion and gill uptake. Beryllium does not bioconcentrate in aquatic organisms. Beryllium uptake from water is low, resulting in low bioconcentration rates. Biomagnification of beryllium in aquatic food chains does not occur (Fishbein, 1981). Beryllium can be toxic to warm-water fish, especially in soft water.

The Tier II secondary acute water quality value and secondary chronic water quality value for beryllium, as calculated by the method described in the EPA's *Proposed Water Quality Guidance for the Great Lakes System* (EPA, 1985b), are 35 and 0.66 µg/L, respectively.

The effects concentration for 20 percent of a population (EC₂₀) for fish can be used as a benchmark indicative of production within a population. It is the highest tested concentration causing less than 20 percent reduction in either the weight of young fish per initial female fish in a life-cycle or partial life-cycle test or the weight of young per egg in an early life-stage test (Suter and Tsao, 1996). The EC₂₀ value for beryllium is 148 µg/L. A similar value can be determined for daphnids, which reflects the highest tested concentration causing less than 20 percent reduction in the product of growth, fecundity, and survivorship in a chronic test with a daphnid species. The EC₂₀ for daphnids is 3.8 µg/L (Suter and Tsao, 1996).

4.2.5 Cobalt

Cobalt is a natural element that is widely distributed in the Earth's crust at 0.001 to 0.002 percent (Merck Index, 1983). Small amounts of cobalt are found in rocks, soil, and surface and groundwater. Natural cobalt can stay airborne for a few days but will stay for years in the soil. In most soils, the transfer of cobalt from soils to plants is not significant, although higher transfer rates have been observed in some higher plants and in acidic soils (Boikat, et al., 1985; Francis, et al., 1980). Some cobalt may seep from acid soil into groundwater. It is present in trace quantities in most foods and is readily absorbed by the gut in humans (International Commission on Radiological Protection [ICRP], 1979).

Plants. Although cobalt is essential to some blue-green algae, fungi, and microorganisms, it apparently is not essential for the growth of higher plants (Kabata-Pendias and Pendias, 1992). Several abiotic factors govern the availability of cobalt to plants. Soil factors include organic

matter and clay content, pH, leachability, and concentrations of manganese and iron oxides. Uptake of cobalt can occur via the roots or leaves of a plant (Kabata-Pendias and Pendias, 1992).

Concentrations of cobalt in leaf tissue that are excessive or toxic to various plant species range from 15 to 50 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). A soil concentration of 20 mg/kg (dry weight) has been proposed by Efroymsen, et al., (1997) as a benchmark screening value for cobalt phytotoxicity. General symptoms of cobalt toxicity in plants include interveinal chlorosis in new leaves, followed by induced iron chlorosis, white leaf margins, and damaged root tips (Kabata-Pendias and Pendias, 1992).

Mammals. Cobalt is a component of vitamin B₁₂ and, therefore, is an essential micronutrient for animal growth. No information has been located at this time on chronic toxic effects of cobalt to terrestrial wildlife; however, some acute studies have been completed. Additionally, there is little biomagnification of cobalt in animals of higher trophic levels (Jenkins, 1980).

Young rats are unable to survive repeated 30 mg doses of cobalt metal powder in their diet for a month (total dosage about 900 mg), whereas they can tolerate 1,250 mg of the metal in a single dose (Venugopal and Luckey, 1978). Cobalt was embryotoxic to rat fetuses when it was administered during the entire gestation (dose of 0.05 mg/kg). A dose of 0.005 mg/kg was non-toxic to the females; however, the progeny of treated females had a reduced survival rate (Shepard, 1986). At doses under 2 milligrams per kilogram of body weight per day (mg/kg-bw/day), no adverse effects to sheep were noted. However, at 6 mg/kg-bw/day, sheep exhibited loss of appetite, loss of weight, and debilitation were noted (National Research Council [NRC], 1977).

Birds. No information has been located at this time on chronic toxic effects of cobalt to birds; however, some acute studies have been completed. Additionally, there is little biomagnification of cobalt in animals of higher trophic levels (Jenkins, 1980).

Chickens were administered 50 mg/kg of diet/day with acute effects of loss of appetite, loss of weight, and debilitation. At doses under 2 mg/kg-bw/day, no adverse effects to chickens were noted (NRC, 1977).

Aquatic Life. In most surface water bodies, cobalt is primarily associated with the sediment. However, some mobilization may occur in acidic water and in the presence of chloride ions or chelating agents. Bioaccumulation factors for freshwater fish range from 40 to 1,000 (Smith and

Carson, 1981). Research by Evans, et al., (1988) indicates that cobalt does not significantly bioaccumulate in benthic bottom feeders.

4.2.6 Copper

Copper is ubiquitously distributed in nature in its free state and in sulfides, arsenides, chlorides, and carbonates. Several copper-containing proteins have been identified in biological systems as oxygen-binding hemocyanin, cytochrome oxidase, tyrosinase, and laccase. Copper has also been identified with the development of metalloproteins employed in the sequestering and cellular detoxification of metals.

Copper has been known to sorb rapidly to sediment. The rate of sorption is, of course, dependent upon factors such as the sediment grain size, organic fraction, pH, competing cations, and the presence of ligands. In industrialized freshwater environments around the world, total copper levels within sediments can range from 7 to 2,350 ppm (Moore and Ramamoorthy, 1984).

Plants. Copper is an essential nutrient for the growth of plants. Background concentrations of copper in grasses and clovers collected in the United States averaged 9.6 mg/kg and 16.2 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). Copper is one of the least mobile heavy metals in soil, and its availability to plants is highly dependent on the molecular weight of soluble copper complexes (Kabata-Pendias and Pendias, 1992).

According to Rhodes, et al., (1989), copper concentrations in plant tissues do not serve as conclusive evidence of copper toxicity in species of plants such as tomatoes, because some species are able to tolerate higher concentrations of copper than others. The pH of soil may also influence the availability and toxicity of copper in soils to plants (Rhodes, et al., 1989). In a study with tomato plants, Rhodes, et al., (1989) found a reduction in plant growth when plants were grown in soils containing greater than 150 mg/kg of copper at a pH less than 6.5. At pH values greater than 6.5, soil copper concentrations of greater than 330 mg/kg were required to reduce plant growth.

Concentrations of copper in leaf tissue that are excessive or toxic to various plant species range from 20 to 100 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). A soil concentration of 100 mg/kg has been proposed by Efroymson, et al., (1997) as a benchmark screening value for copper phytotoxicity in soil. General symptoms of copper toxicity in plants include the presence of dark green leaves followed by induced iron chlorosis; thick, short, or barbed-wire roots; and depressed tillering (Kabata-Pendias and Pendias, 1992).

Mammals. Copper is an essential trace element to plants and animals (Callahan, et al., 1979), but becomes toxic at concentrations only slightly higher than essential levels (EPA, 1985d). Copper is an essential element for hemoglobin synthesis and oxidative enzymes in animals. Copper is absorbed by mammals following ingestion, inhalation, and dermal exposure. Once absorbed, copper is distributed to the liver. Copper is not metabolized (Marceau, et al., 1970). No evidence of bioaccumulation was obtained in a study of pollutant concentrations in the muscles and livers of ten species of herbivorous, omnivorous, and carnivorous animals in Donana National Park in Spain (Hernandez, et al., 1985). Copper concentrations in small mammals collected from various uncontaminated sites ranged from 8.3 to 13.4 mg/kg (whole-body concentrations) (Talmage and Walton, 1991). Highest concentrations of copper tend to be in hair, followed in decreasing concentration by liver, kidney, and whole body (Hunter and Johnson, 1982). Among the small mammals collected, Hunter and Johnson (1982) found shrews (*Sorex araneus*) to contain the highest concentrations of copper. Mice were found to contain the lowest copper concentrations. Increased fetal mortality was observed in fetuses of mice fed more than 104 mg/kg/day of copper as copper sulfate (Lecyk, 1980). Increased mortality rates in mink offspring have been observed at levels above 3.21 mg/kg/day (Aulerich, et al., 1985).

Laboratory toxicity data for mink exposed to copper sulfate in their diet were used to estimate a NOAEL value of 11.7 mg/kg/day (Auerlich, et al., 1982). Reproduction was the endpoint studied. Symptoms of acute copper poisoning in mammals include vomiting, hypotension, melena, coma, jaundice, and death (Klaassen, et al., 1991). Selenium can act as an antidote for copper poisoning.

Birds. Laboratory toxicity data for one-day old chicks exposed to copper oxide in their diets were used to estimate a NOAEL value of 47 mg/kg/day (Mehring, et al., 1960). Growth and mortality were the endpoints studied.

Aquatic Life. Invertebrates inhabiting “polluted” freshwaters worldwide have been known to have tissue residues of copper ranging from 5 to 200 ppm (Moore and Ramamoorthy, 1984). Field studies have shown that there is virtually no accumulation of this metal through the food chain (Fuller and Averett, 1975). Studies by Kosalwat and Knight (1987) indicated that copper present in the substrate or sediment was significantly less toxic to chironomid species than overlying water column levels. The substrate copper concentration at which chironomid larval growth was reduced 50 percent (EC₅₀) was 1,602 mg/kg. These researchers found that deformities in larval mouth parts were observed at elevated concentrations, and adult emergence was inhibited when the sediment concentration exceeded 1,800 mg/kg. Carins, et al., (1984)

reported copper toxicity in sediment for several chironomus midges and cladocerans with LC₅₀s ranging from 681 to 2,296 mg/kg.

4.2.7 4,4'-DDT and Metabolites

DDT is a chlorinated pesticide that has been banned in the United States since 1972. Dichlorodiphenyldichloroethene (DDE) and Dichlorodiphenyl-dichloroethane (DDD) are metabolites of DDT. DDT and its metabolites will adsorb very strongly to soil and are subject to evaporation and photodegradation at the soil surface. DDT and related compounds are very persistent in soils (National Library of Medicine [NLM], 1996).

Plants. DDT can be taken up by plant roots and translocated to the aboveground plant parts. Concentrations of DDT and its metabolites are usually greatest in the roots of the plants (Voerman and Besemer, 1975). The effects of DDT, DDD, and DDE on plant growth and reproduction are not well documented.

Mammals. As in plants, DDT and its major metabolites DDE and DDD are ubiquitous in wild mammals and birds. Because DDT can be biomagnified through food chains, insectivorous shrews generally contain higher concentrations than herbivorous small mammals collected from the same site. DDT residues were found to be higher in juveniles than adults and increased with increased body fat content (Talmage and Walton, 1991).

The most significant exposure route for vertebrates to DDT and its metabolites is oral exposure. Dermal exposure is believed to be very limited, and inhaled DDT and associated particulates are believed to be deposited in the upper respiratory tract and eventually swallowed. The toxicity of DDT and its metabolites to mammalian and avian wildlife is dependent on the fat content within the animal. In both mammals and birds, the storage of DDT and DDE in fat is protective, because it decreases the amount of chemical in circulation that may reach the brain, which is the site of toxic action (NLM, 1996).

Laboratory toxicity data for rats exposed to DDT in their diet were used to estimate a NOAEL value of 0.8 mg/kg/day (Fitzhugh, 1948). Reproduction was the endpoint for these studies. Signs of acute DDT poisoning in animals include paresthesia of the tongue, lips and face; apprehension; dizziness; tremor; disturbed equilibrium; and convulsions (Klaassen, et al., 1991).

Birds. DDE concentrations in eggs have been negatively correlated with eggshell thickness in bald eagles. Studies have shown reduced reproductive success at DDE egg residue concentrations of greater than 3 mg/kg for the white-faced ibis, 5 mg/kg for the snowy egret, and

8 mg/kg for the black-crowned night heron (Henny, et al., 1985). Laboratory toxicity data for brown pelicans exposed to DDT in their diet were used to estimate a NOAEL value of 0.0028 mg/kg/day (Anderson, et al., 1975). Reproduction was the endpoint for these studies. Signs of DDT poisoning in birds include ataxia, wing-drop, jerkiness in gait, continuous whole-body tremors, falling, and convulsions (Hudson, et al., 1984).

Aquatic Life. DDT in freshwater environments partitions primarily into sediment. Biodegradation of DDT in sediment may be significant. Bioconcentration factors between 51,000 and 100,000 have been reported for fathead minnows exposed to DDT (NLM, 1996). The federal water quality criteria for the protection of aquatic life for acute and chronic exposures to DDT and its metabolites in freshwater systems are 1.1 and 0.001 µg/L, respectively (EPA, 1986).

4.2.8 Iron

Iron is an essential trace element, required as a constituent of oxygen-carrying and oxidative-reductive macro-molecules such as hemoglobin, myoglobin, and cytochrome P-450. As such, most iron-related health concerns are induced by insufficient iron intake, rather than excess iron intake (Hayes, 1994).

Plants. Wallihan (1966) reported unspecified reductions in plant growth in a solution culture with the addition of 10 ppm iron. Wallace, et al., (1977) evaluated the effects of iron (as FeSO₄) on leaf, stem, and root weights of bush bean seedlings grown for 15 days in nutrient solution. Iron at 28 ppm reduced all three measures 67, 52, and 67 percent, respectively, while 11.2 ppm iron had no effect. After 55 days, cabbage seedling plant weight was reduced 45 percent by 50 ppm iron added as FeSO₄ to nutrient solution, while 10 ppm had no effect on growth (Hara, et al., 1976).

Iron is the key metal required for energy transformations needed for cellular function. It occurs in heme and non-heme proteins and is concentrated in chloroplasts. Organic iron complexes are involved in photosynthetic electron transfer. Plant symptoms of toxicity are not specific and differ among plant species and growth stages (Foy, et al., 1978).

Mammals. Iron is an essential nutrient for most wildlife species and is necessary to maintain homeostasis; therefore, it is only toxic at very high concentrations. Bioaccumulation factors have been calculated for several small mammal species. Small herbivorous mammals were estimated to have an iron bioaccumulation factor of 0.0127, and small omnivorous mammals

were estimated to have an iron bioaccumulation factor of 0.01209. These bioaccumulation factors indicate that iron is not accumulated in small mammal tissues (Sample, et al., 1998a). Additionally, the bioaccumulation factor for earthworms has been estimated to be 0.038, indicating that iron is not accumulated in earthworm tissues (Sample, et al., 1998b).

Aquatic Life. The national recommended water quality criteria for iron (1,000 µg/L) is based on field study at a site receiving acid mine drainage (EPA, 1999b). The lowest chronic value for daphnids (158 µg/L) is a threshold for reproductive effects from a 21-day test using iron chloride with *Daphnia magna* (Dave, 1984). It is considerably lower than the 4,380 µg/L concentration causing 16 percent reproductive decrement in another test using iron chloride with *Daphnia magna* (Biesinger and Christensen, 1972). The lowest chronic value for fish (1,300 µg/L) is a concentration that caused 100 percent mortality in an embryo-larval test with rainbow trout exposed to dissolved iron salts (Amelung, 1981).

The Ontario Ministry of the Environment has prepared provincial sediment quality guidelines using the screening-level concentration approach. This approach estimates the highest concentration of a particular contaminant in sediment that can be tolerated by approximately 95 percent of benthic fauna (Neff, et al., 1988). These values are based on Ontario sediments and benthic species from a wide range of geographical areas within the province (Persaud, et al., 1993). The lowest effect level (Low) is the level at which actual ecotoxic effects become apparent. The severe effect level (Severe) represents contaminant levels that could potentially eliminate most of the benthic organisms (Persaud, et al., 1993). The “Low” and “Severe” levels for iron in sediment are 2 percent (20,000 ppm) and 4 percent (40,000 ppm), respectively.

4.2.9 Lead

Global production of lead from both smelter and mining operations has been high throughout this century. Lead is commonly used in storage batteries as well as in ammunition, solder, and casting materials. In addition, tetraethyl lead was a principal additive to gasolines as an anti-knock agent and was commonly used as an additive in paints. In short, lead is one of the most ubiquitous pollutants in the civilized world.

Lead is strongly sorbed in sediments, and the rate is strongly correlated with grain size and organic content. In the absence of soluble complexing species, lead is almost totally adsorbed to clay particles at pHs greater than 6 (Moore and Ramamoorthy, 1984).

Plants. Although lead is not an essential nutrient for plant growth, it is detected in plant tissues due to the prevalence of lead in the environment. The bioavailability to plants of lead in soil is limited. Bioavailability may be enhanced by a reduction in soil pH, a reduction in the content of organic matter and inorganic colloids in soil, a reduction in iron oxide and phosphorous content, and increased amounts of lead in soil (NRCC, 1973). Plants can absorb lead from soil and air. Aerial deposition of lead can also contribute significantly to the concentration of lead in above-ground plant parts. Lead is believed to be the metal of least bioavailability and the most highly accumulated metal in root tissue (Kabata-Pendias and Pendias, 1992).

Mean background concentrations of lead in grasses and clovers have been reported to range from 2.1 to 2.5 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). Adverse effects of lead on terrestrial plants occur only at total concentrations of several hundred mg/kg of soil (Eisler, 1988b). This is explained by the fact that, in most cases, lead is tightly bound to soils, and substantial amounts must accumulate before it can affect the growth of higher plants (Bogges, 1977).

Mammals. As with plants, lead is not considered an essential nutrient for mammalian life. Ingestion is the major route of exposure for wildlife. Lead tends to accumulate in bone, hair, and teeth. Biomagnification of lead is negligible (Eisler, 1988b). Reduced survival was reported at acute oral doses as low as 5 mg/kg body weight in rats, at a chronic dose of 0.3 mg/kg body weight in dogs, and at a dietary level of 1.7 mg/kg body weight in horses (Eisler, 1988b). Laboratory data from studies of rats fed lead acetate in their diets were used to estimate a NOAEL value of 8.0 mg/kg/day (Azar, et al., 1973). Reproduction was the endpoint for this study. Symptoms of lead poisoning in mammals are diverse and depend on the form of lead ingested, the concentration, and the species and its age. These symptoms may include reproductive impairment, decreased body weight, vomiting, uncoordinated body movements, visual impairment, reduced life span, renal disorders, and abnormal social behavior (Eisler, 1988b).

In laboratory studies, breeding mice exposed to low doses of lead in drinking water (25 ppm) resulted in loss of the strain in two generations with many abnormalities (Schroeder and Mitchner, 1971). Exposure of rats in this same experiment resulted in many early deaths and runts. Blood δ -aminolevulinic acid dehydratase (ALAD) activity associated with exposure to lead was reduced in white-footed mice living near a metal smelter (Beyer, et al., 1985). Amounts of whole-body lead content and feeding habits of roadside rodents have been correlated

with highest body burdens in insectivores such as shrews, intermediate in herbivores, and lowest in granivores (Boggess, 1977; Getz, et al., 1977).

Birds. Most of the information on the effects of lead to terrestrial vertebrates is concerned with the poisoning of waterfowl by lead shot. Apparent symptoms include loss of appetite and mobility, avoidance of other birds, lethargy, weakness, emaciation, tremors, dropped wings, green feces, impaired locomotion, loss of balance and depth perception, nervous system damage, inhibition of heme synthesis, damage to kidneys and liver, and death (Eisler, 1988b; Mudge, 1983). Anemia, kidney disease, testicular and liver lesions, and neurological disorders have been associated with high brain lead concentrations in mourning doves (*Zenaida macroura*) (Kendall, 1992). Hatchlings of chickens, Japanese quail, mallards, and pheasants are relatively more tolerant to moderate lead exposure, including no effect on growth at dietary levels of 500 ppm and no effect on survival at 2,000 ppm (Hoffman, et al., 1985).

Toxicity of lead to birds is dependent upon the form of lead, the route of exposure and exposure duration, and the species and age of the bird. Hatchlings of chickens, Japanese quail, mallards, and pheasants are relatively tolerant to moderate lead exposure (Eisler, 1988b). Laboratory toxicity data for American kestrels fed metallic lead in their diet were used to estimate a NOAEL value of 3.85 mg/kg/day (Pattee, 1984). Reproduction was the endpoint for this study.

Aquatic Life. All life stages are sensitive to the toxic effects of lead; however, embryos are more sensitive to lead than are later juvenile stages (Davies, et al., 1976). Lead uptake depends on exposure time, aqueous concentration, pH, temperature, salinity, diet, and other factors. For example, gill, liver, kidney, and erythrocytes accumulate lead from aqueous sources in proportion to exposure time and concentration (Holcombe, et al., 1976). Direct erythrocyte injury is considered the first and most important sign of lead poisoning in catfish (Dawson, 1935). Respiratory distress occurs in fish living in rivers receiving lead mining wastes in England (Carpenter, 1924, 1925, 1926). Fish are thought to be asphyxiated as a result of a mucous coating over the gills (NAS, 1972).

No significant biomagnification of lead occurs in aquatic ecosystems (Boggess, 1977). Background concentrations of lead in fish tend to be less than 1 mg/kg (dry weight) (Eisler, 1988b). The EPA's National Recommended Water Quality Criteria for lead in freshwater are 65 µg/L for acute exposure and 2.5 µg/L for chronic exposure (EPA, 1999b). In general, dissolved lead is more toxic than total lead, and organic forms of lead are more toxic than inorganic forms. Soluble lead in the water column becomes less bioavailable as water hardness increases.

Chronic exposure of fish to lead may result in signs of lead poisoning such as spinal curvature, anemia, darkening of the dorsal tail region, destruction of spinal neurons, difficulties in swimming, growth inhibition, changes in blood chemistry, retarded sexual development, and death (Eisler, 1988b).

The majority of benthic invertebrates do not bioconcentrate lead from water or abiotic sediment particles. There is some evidence of bioaccumulation through the food web of organic forms of lead, such as tetraethyl lead. Anderson, et al., (1980) reported lead LC₅₀s of 258 ppm for the chironomid and that growth of this amphipod was not reduced above this level in freshwater sediments. In addition, Suter and Tsao (1996) reported effect levels in the water flea (*Daphnia magna*) to be in the 12.26 ppb range, while Khangrot and Ray (1989) reported a *D. magna* LC₅₀ of 4.89 ppm.

4.2.10 Manganese

Manganese, a silver-colored metal with chemical properties similar to iron, is a naturally occurring substance found in many minerals. Manganese is usually combined with oxygen, sulfur, and/or chlorine. Manganese is present in all living organisms and manganese is an essential element for adequate nutritional needs in mammals and many other organisms. Manganese is poorly absorbed from the intestinal tract; about 3 to 5 percent of the oral dose of manganese is absorbed. Absorption efficiency is also related to dietary intake of iron and calcium. Sufficient body stores of iron decrease absorption of manganese (ATSDR, 1992b).

Plants. Manganese is an essential element for plant growth. Uptake of manganese may occur via root or leaves (Kabata-Pendias and Pendias, 1992). The concentration of manganese in plants is dependent upon plant and soil characteristics. Plants grown on flooded or acid soils tend to contain higher concentrations of manganese than plants grown in other, uncontaminated soils. In addition, concentrations of manganese in plants are positively correlated with soil organic matter (Kabata-Pendias and Pendias, 1992). Concentrations of manganese in leaf tissue that are excessive or toxic to various plant species range from 400 to 1,000 mg/kg dry weight (Kabata-Pendias and Pendias, 1992). A soil concentration of 500 mg/kg (dry weight) has been proposed by Efroymson, et al., (1997) as a benchmark screening value for manganese phytotoxicity. General symptoms of manganese toxicity in plants include the presence of chlorosis and necrotic lesions on old leaves, blackish-brown or red necrotic spots, dried leaf tips, and stunted root and plant growth (Kabata-Pendias and Pendias, 1992).

Mammals. Manganese is an essential nutrient that is homeostatically regulated in vertebrates (Vanderploeg, et al., 1975). Liver and kidney tissues generally contain the highest

concentrations of manganese in the body. Manganese in the body is primarily excreted in the feces (Gregus and Klaassen, 1986).

Divalent manganese is more toxic than the trivalent form. Exposure to manganese dust via inhalation is usually of greater toxicological concern than ingestion (Klaassen, et al., 1991). Laboratory data for rats fed manganese oxide in their diet were used to estimate a NOAEL value of 88 mg/kg/day (Laskey, et al., 1982). Reproduction was the endpoint for this study. Laboratory studies with rats have found no hematologic, behavioral, or histologic effects in animals exposed to manganese dioxide at concentrations of 47 mg/m³ for five hours per day, five days a week, for 100 days (Klaassen, et al., 1991).

Aquatic Life. As discussed previously, manganese is a required nutrient for plant and animal life. Manganese concentrations in most vertebrates are homeostatically controlled (Vanderploeg, et al., 1975). Bioconcentration factors for freshwater macrophytes have been reported to range from 190 to approximately 25,000 (Vanderploeg, et al., 1975). With regard to freshwater fish, concentrations of manganese in fish muscle are generally less than 0.5 mg/kg and range from 3 to 10 mg/kg in whole fish (Vanderploeg, et al., 1975). Bioconcentration factors from water to whole fish range from 40 to 2,300. A bioconcentration factor of 10,000 was also suggested for crustaceans (Vanderploeg, et al., 1975).

No federal water quality criteria exist for the protection of freshwater biota from elevated manganese concentrations. Suter and Tsao (1996) have estimated acute and chronic advisory levels for manganese to be 1,470 and 80.3 µg/L, respectively. The EC₂₀ for fish can be used as a benchmark indicative of production within a population. It is the highest tested concentration causing less than 20 percent reduction in either the weight of young fish per initial female fish in a life-cycle or partial life-cycle test, or the weight of young per egg in an early life-stage test (Suter and Tsao, 1996). The EC₂₀ value for manganese is 1,270 µg/L. A similar value can be determined for daphnids which reflects the highest tested concentration causing less than 20 percent reduction in the product of growth, fecundity, and survivorship in a chronic test with a daphnid species. The EC₂₀ for daphnids is less than 1,100 µg/L (Suter and Tsao, 1996).

4.2.11 Nickel

Nickel is a naturally-occurring silvery metal that is found in the Earth's crust in the form of various nickel minerals. Exposure of organisms to nickel and its compounds results from breathing air, ingesting water and food that contain nickel and compounds, and skin contact with a media contaminated with nickel.

Plants. Nickel is not believed to be an essential element for plant growth; however, beneficial effects of nickel have been reported on the growth of legumes. Background concentrations of nickel in grasses and clovers collected in the United States averaged 0.13 and 1.5 mg/kg, respectively (Kabata-Pendias and Pendias, 1992). The concentration of nickel in plants is positively correlated with nickel concentrations in soil.

Concentrations of nickel in leaf tissue that are excessive or toxic to plant species range from 10 to 100 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). A soil concentration of 30 mg/kg has been proposed by Efraymson, et al. (1997) as a benchmark screening value for nickel phytotoxicity. General symptoms of nickel toxicity in plants include the presence of interveinal chlorosis in new leaves, gray-green leaves, and brown and stunted root and plant growth. The uptake of nutrients and minerals, especially iron, can be substantially reduced as a consequence of nickel toxicity in plants (Kabata-Pendias and Pendias, 1992).

Mammals. Nickel is a nonessential element for animal life. Nickel concentrations within the whole bodies of small mammals from uncontaminated sites were reported to range from 2.2 to 6.2 mg/kg (dry weight) (Talmage and Walton, 1991). Highest concentrations were measured in the deer mouse (*Peromyscus maniculatus*). Highest tissue concentrations of nickel are usually found in the liver of mammals (Schroeder, et al., 1964). Because nickel is poorly absorbed by the gastrointestinal tract, ingested nickel is generally not of great toxicological concern. Inhaled nickel, however, is relatively toxic. Rats fed nickel in their diet as nickel sulfate hexahydrate over three generations were studied for effects on reproduction. They were fed three dose levels (250, 500, and 1,000 ppm Ni) in their diet, and only the highest dose level caused reduced offspring body weights. No adverse effects were observed at the other dose levels. Because this study considered exposures over multiple generations, the 500 ppm dose was considered to be the chronic NOAEL, and the 1,000 ppm dose was considered to be the chronic LOAEL (EPA, 1999a).

Laboratory toxicity data for rats fed nickel sulfate hexahydrate in their diet were used to estimate a NOAEL value of 40 mg/kg/day (Ambrose, et al, 1976). Reproduction was the endpoint studied.

Birds. Mallard ducklings were fed nickel as nickel sulfate in their diet for a duration of 90 days to study the effects on mortality, growth, and behavior. They were fed three dose levels (176, 774, and 1,069 ppm Ni), and only the highest dose reduced growth and resulted in 70 percent mortality. Because the study considered exposure over 90 days, the 774 ppm dose was

considered to be the chronic NOAEL, and the 1,069 dose was considered to be the chronic LOAEL (Cain and Pafford, 1981). Cain and Pafford (1981) studied mallard ducklings fed nickel sulfate in their diet. A NOAEL value of 77.4 mg/kg/day was derived from this study based on mortality, growth, and behavior as endpoints.

Aquatic Life. The bioavailability and toxicity of nickel to aquatic biota is influenced by the pH of the water (Schubauer-Berigan, et al., 1993). The national recommended water quality criteria for the protection of aquatic life for acute and chronic exposure are 470 and 52 µg/L, respectively (EPA, 1999b). Background concentrations of nickel in adult anurans ranged between 0.9 and 2.9 mg/kg (dry weight) (Hall and Mulhern, 1984).

The test EC₂₀ for fish can be used as a benchmark indicative of production within a population. It is the highest tested concentration causing less than 20 percent reduction in either the weight of young fish per initial female fish in a life-cycle or partial life-cycle test, or the weight of young per egg in an early life-stage test. The EC₂₀ value for nickel is 62 µg/L (Suter and Tsao, 1996). A similar value can be determined for daphnids which represents the highest tested concentration causing less than 20 percent reduction in the product of growth, fecundity, and survivorship in a chronic test with a daphnid species. The EC₂₀ benchmark for daphnids has been determined to be 45 µg/L (Suter and Tsao, 1996).

4.2.12 Silver

Silver is a rare but naturally-occurring element. It is often found deposited as a mineral ore in association with other elements. Silver occurs primarily as sulfides, in association with iron (pyrite), lead (galena), and tellurides, and with gold. Silver is found in surface water in various forms: 1) as the monovalent ion (e.g., sulfide, bicarbonate, or sulfate salts); 2) as part of more complex ions with chlorides and sulfates; and 3) adsorbed onto particulate matter (ATSDR, 1990).

Plants. Silver is not considered essential for plant growth. Silver concentrations in plants generally range between 0.03 and 0.5 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). The availability of silver in soil to plants is dependent on soil pH, the organic matter content of the soil, and the concentration of manganese oxides in the soil (Kabata-Pendias and Pendias, 1992). Concentrations of silver in leaf tissue that are excessive or toxic to various plant species range from 5 to 10 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). A soil concentration of 2 mg/kg has been proposed by Efroymson, et al. (1997) as a benchmark screening value for silver phytotoxicity.

Mammals. Silver is not an essential element for animal life. The highest concentrations of silver in soft tissues of wildlife occur in the liver and spleen (NLM, 1996). The biological half-life of silver in animals is only a few days (NLM, 1996).

The toxicity of silver is dependent on the form of silver and the route of exposure. Ingestion, inhalation, and dermal exposure to silver can induce a toxic response in mammals. Internal antagonistic interactions have been noted between silver and selenium (NLM, 1996). Repeated exposure of animals to silver may produce anemia, enlargement of the heart, growth retardation, and degenerative changes in the liver (NLM, 1996). A value of 89 mg/kg/day has been estimated as the LOAEL (chronic) for rats (IRIS, 2001).

Aquatic Life. Accumulation of silver has been reported in algae, daphnia, freshwater mussels, and fathead minnows (NLM, 1996). Biomagnification of silver, however, has not been observed in freshwater systems. The deposition of silver into sediment is dependent on the concentrations of magnesium dioxides, ferric compounds, and clay minerals (NLM, 1996).

The national recommended water quality criteria for the protection of freshwater aquatic life for acute exposure to silver is 3.4 µg/L. The lowest chronic values of silver reported in the literature for fish and daphnia are 0.12 and 2.6 µg/L, respectively (Suter and Tsao, 1996). The test EC₂₀ for fish can be used as a benchmark indicative of production within a population. It is the highest tested concentration causing less than 20 percent reduction in either the weight of young fish per initial female fish in a life-cycle or partial life-cycle test, or the weight of young per egg in an early life-stage test. The EC₂₀ value for silver is 0.2 µg/L (Suter and Tsao, 1996). A similar value can be determined for daphnids that represents the highest tested concentration causing less than 20 percent reduction in the product of growth, fecundity, and survivorship in a chronic test with a daphnid species. The EC₂₀ benchmark for daphnids has been determined to be less than 0.56 µg/L (Suter and Tsao, 1996).

4.2.13 Thallium

Thallium is widely distributed in trace amounts in the Earth's crust and is one of the more toxic metals. In the environment, thallium exists in either the monovalent (thallous) or trivalent (thallic) form. Thallium is chemically reactive with air and moisture, undergoing oxidation. Thallium is relatively insoluble in water. Thallium adsorbs to soil and sediment and is not transformed or biodegraded (Callahan, et al., 1979).

Plants. Thallium is not essential for plant growth. When soluble forms are available, thallium is readily taken up by plants and translocated to aerial parts, probably because of its similarity to potassium. Toxic effects on plants include impairment of chlorophyll synthesis and seed germination, reduced transpiration due to interference in stomatal processes, growth reduction, stunting of roots, and leaf chlorosis (Adriano, 1986). The phytotoxicity benchmark value of 1.0 mg/kg is based on unspecified toxic effects on plants grown in surface soil amended with 1.0 mg/kg thallium (Kabata-Pendias and Pendias, 1992).

Mammals. Birds and mammals are exposed to thallium via ingestion of soil, water, and plant material. In mammals, thallium is absorbed primarily from ingestion and is distributed to several organs and tissues, with the highest levels reported in the kidneys (Manzo, et al., 1982). Thallium exposure in mammals causes cardiac, neurologic, reproductive, and dermatological effects. Various effects and toxic responses have been reported, including paralysis and pathological changes in the liver, kidneys, and stomach mucosa in rabbits exposed to thallium (Tikhonova, 1967). Testicular toxicity in rats has also been reported (Formigli, et al., 1986).

Laboratory toxicity data for rats exposed to thallium sulfate in their drinking water were used to estimate a NOAEL value of 0.0074 mg/kg/day (Formigli, et al., 1986). Reproduction was the endpoint for this study.

Aquatic Life. In aquatic organisms, thallium is absorbed primarily from ingestion and thereafter bioconcentrates in the organism. Toxic effects have been observed in numerous aquatic organisms, including daphnia, fat-head minnow, bluegill sunfish, and others (EPA, 1980b). The Tier II secondary acute water quality value and secondary chronic water quality value for beryllium, as calculated by the method described in the EPA's *Proposed Water Quality Guidance for the Great Lakes System* (EPA, 1985b), are 110 and 12 µg/L, respectively.

The test EC₂₀ for fish can be used as a benchmark indicative of production within a population. It is the highest tested concentration causing less than 20 percent reduction in either the weight of young fish per initial female fish in a life-cycle or partial life-cycle test, or the weight of young per egg in an early life-stage test. The EC₂₀ value for thallium is 81 µg/L (Suter and Tsao, 1996). A similar value can be determined for daphnids that represents the highest tested concentration causing less than 20 percent reduction in the product of growth, fecundity, and survivorship in a chronic test with a daphnid species. The EC₂₀ benchmark for daphnids has been determined to be less than 64 µg/L (Suter and Tsao, 1996).

4.2.14 Vanadium

Vanadium, a white to gray metal, occurs naturally in fuel oils and coal. It is used as a catalyst in the production of various chemicals including sulfuric acid. It is also used in the hardening of steel, the manufacture of pigments, and photography. The general population and many ecological receptors are exposed to background levels of vanadium primarily through ingestion of food.

Plants. There is some controversy over whether vanadium is an essential element for plants (Kabata-Pendias and Pendias, 1992). It appears to be required by some algal species and may be required by nitrogen-fixing bacteria. Mean background concentrations of vanadium in plants are 1.6 mg/kg for angiosperms, 0.69 mg/kg for gymnosperms, and 0.67 mg/kg for fungi (Waters, 1977). The availability of vanadium to plants is highly dependent on soil pH. Elevated levels of vanadium in soil can reduce the uptake of manganese, copper, calcium, and phosphorus (NRCC, 1980).

Concentrations of vanadium in leaf tissue that are excessive or toxic to various plant species range from 5 to 10 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). A soil concentration of 2 mg/kg has been proposed by Efroymson, et al. (1997) as a benchmark screening value for vanadium phytotoxicity.

Mammals. Vanadium has been shown to be essential in the diets of rats (Waters, 1977). Background concentrations of vanadium in the kidneys and livers of wild mammals have been reported to range from 0 to 2.07 mg/kg, and from 0 to 0.94 mg/kg, respectively (Waters, 1977). Liver and skeletal tissues usually contain the highest concentrations of vanadium (Waters, 1977). Experimental animal investigations have suggested that the liver, adrenal, and bone marrow may be adversely affected by subacute exposure to high levels of vanadium (ATSDR, 1992c; Klaassen, et al., 1991). Vanadium fed to rats prior to gestation, during gestation, and through delivery and lactation were studied for effects on reproduction. The rats were fed three dose levels (5, 10, and 20 mg NaVO₃/kg/day or 2.1 mg V/kg/day). Significant differences in reproductive parameters (e.g., number of dead young per litter, size and weight of offspring) were observed at all dose levels. Therefore, the lowest dose was considered to be a chronic LOAEL. A chronic NOAEL was estimated by applying an uncertainty factor of 0.1 (chronic NOAEL = 0.21 mg V/kg/day) (EPA, 1999a).

Based on oral intubation exposure of rats to sodium metavanadate by Domingo, et al. (1986), an estimated NOAEL value of 0.21 mg/kg/day has been derived. Reproduction was the endpoint for this study. Signs of acute toxicity in animals include alterations in nervous system responses,

gastrointestinal distress, hemorrhaging, paralysis, convulsions, and respiratory depression (Klaasen, et al., 1991).

Birds. Mallard ducks were fed vanadium as vanadyl sulfate in their diet for 12 weeks and observed for effects on mortality, body weight, and blood chemistry. The ducks were fed three different doses (2.84, 10.36, and 110 ppm V). No effects were observed at any of the dose levels. Because this study was greater than ten weeks in duration and did not consider a critical lifestage, the maximum dose was considered to be a chronic NOAEL (White and Dieter, 1978). From these data a NOAEL value of 11.4 mg/kg/day has been estimated.

Aquatic Life. Background concentrations of vanadium in freshwater fish are usually less than 2.5 mg/kg (wet weight) (Jenkins, 1980). A bioconcentration factor of 3,000 has been reported for aquatic invertebrates exposed to vanadium (Neumann, 1976). No federal ambient water quality criteria exist for the protection of freshwater biota (EPA, 1999b). The test EC₂₀ for fish can be used as a benchmark indicative of production within a population. It is the highest tested concentration causing less than 20 percent reduction in either the weight of young fish per initial female fish in a life-cycle or partial life-cycle test, or the weight of young per egg in an early life-stage test. The EC₂₀ value for vanadium is 41 µg/L (Suter and Tsao, 1996). A similar value can be determined for daphnids that represents the highest tested concentration causing less than 20 percent reduction in the product of growth, fecundity, and survivorship in a chronic test with a daphnid species. The EC₂₀ benchmark for daphnids has been determined to be 430 µg/L (Suter and Tsao, 1996).

4.2.15 Zinc

Zinc is a naturally occurring element that may be found in both organic and inorganic forms and, as such, is commonly found in the environment. In general, zinc is concentrated in the sediments of water bodies. The NAS (1977b) has reported that zinc will probably be detected in 75 percent of all water bodies examined for the compound at various locations. The fate of zinc in soils appears to have a pH basis. Studies have shown that a pH of less than 7 often favors zinc desorption (EPA, 1984).

Plants. Background concentrations of zinc in terrestrial plants range from 25 to 150 mg/kg (dry weight) (NAS, 1979). The deficiency content of zinc in plants is between 10 and 20 ppm (dry weight). Roots often contain the highest concentrations of zinc (Kabata-Pendias and Pendias, 1992).

Certain species of plants, particularly those from the families Caryophyllaceae, Cyperaceae, and Plumbaginaceae, and some tree species are extremely tolerant to elevated zinc concentrations (Kabata-Pendias and Pendias, 1992). Concentrations of zinc in these plants may reach 1 percent (dry weight) in the plant. Concentrations in leaf tissue that are excessive or toxic to various plant species range from 100 to 400 mg/kg. Concentrations of 100 to 500 mg/kg are expected to result in a 10 percent loss in crop yield (Kabata-Pendias and Pendias, 1992). General symptoms of zinc toxicity in plants include the presence of chlorotic and necrotic leaf tips, interveinal chlorosis in new leaves, retarded growth of the entire plant, and injured roots that resemble barbed wire (Kabata-Pendias and Pendias, 1992).

Mammals. Zinc is an essential trace element for normal fetal growth and development. However, exposure to high levels of zinc in the diet has been associated with reduced fetal weights, altered concentrations of fetal iron and copper, and reduced growth in offspring (Cox, et al., 1969). Poisoning has been observed in ferrets and mink from chewing corroded galvanized cages (Clark, et al., 1981). Symptoms of zinc toxicity are lassitude, slower tendon reflexes, bloody enteritis, diarrhea, lowered leukocyte count, depression of the central nervous system, and paralysis of the extremities (Venugopal and Luckey, 1978). A study by Kinnamon (1963) showed a NOAEL for oral exposure to a zinc compound over a period of 73 days to be 250 mg/kg body weight, and mice given 500 mg/L of zinc as zinc sulfate in drinking-water have shown hypertrophy of the adrenal cortex and pancreas. Young animals are much more susceptible to poisoning by zinc than are mature animals (Clark, et al., 1981).

Animals are quite tolerant of high concentrations of zinc in the diet. Levels 100 times that required in the diet usually do not cause detectable symptoms of toxicosis (NAS, 1979). Laboratory data for rats exposed to zinc oxide in their diet were used to estimate a NOAEL value of 160 mg/kg/day (Schlicker and Cox, 1968). Reproduction was the endpoint studied. Symptoms of zinc poisoning in mammals include lameness, acute diarrhea, and vomiting (Eisler, 1993).

Birds. Dietary zinc concentrations of greater than 2,000 mg/kg diet are known to result in reduced growth of domestic poultry and wild birds (Eisler, 1993). Reduced survival has been documented at zinc concentrations greater than 3,000 mg/kg diet or at a single dose of greater than 742 mg/kg body weight (Eisler, 1993). Laboratory data for white leghorn hens exposed to zinc sulfate in their diet were used to estimate a NOAEL value of 14.5 mg/kg/day (Stahl, et al., 1990). Reproduction was the endpoint for this study. A value of 51 mg/L has been calculated as the NOAEL for chronic exposure of birds to zinc carbonate in drinking water (Sample, et al., 1996).

Aquatic Life. Zinc residues in freshwater and marine fish are generally much lower than those found in algae and invertebrates. Thus there is little evidence for bioaccumulation (Moore and Ramamoorthy, 1984). Rainbow trout (*Oncorhynchus mykiss*) have the ability to detect and avoid areas of water containing 5.6 ppb zinc (Sprague, 1968). Cairns and Scheier (1968) reported 96-hour LC₅₀s ranging from 10.13 to 12.5 ppm in hard water for bluegills (*Lepomis macrochirus*), and 96-hour LC₅₀s ranging from 2.86 to 3.78 ppm in soft water. These results demonstrate that water hardness affects the toxicity of zinc to fish. Chronic toxicity tests have been conducted with five species of freshwater fish. Chronic values ranged from 47 µg/L for flagfish (*Jordanella floridae*) to 852 µg/L for brook trout (*Salvenius fontinalis*) (EPA, 1980c).

Acute toxicity to freshwater invertebrates is relatively low and, as with other metals, increasing water hardness decreases the toxicity of zinc (Moore and Ramamoorthy, 1984). As reported by Baudouin and Scoppa (1974), the 48-hour LC₅₀ for the cladoceran *Daphnia hyalina* was 0.055 mg/L, and 5.5 mg/L for the copepod *Cyclops abyssorum*. Four chronic toxicity tests are reported for *Daphnia magna*, with chronic values ranging from 47 to 136 µg/L (EPA, 1980c). Chronic testing with the saltwater species *Mysidopsis bahia* resulted in a chronic value of 166 µg/L (EPA, 1980c).

4.2.16 Polycyclic Aromatic Hydrocarbons

PAHs are a diverse group of organic chemicals consisting of substituted and unsubstituted polycyclic and heterocyclic aromatic rings in which interlinked rings have at least two carbon atoms in common (Zander, 1983). They are formed as a result of incomplete combustion of organic materials such as wood, coal, and oil and exist in the environment in quantity, both from anthropogenic and natural sources. Activities associated with large releases of PAHs include coke production; petroleum refining; the manufacture of carbon black, coal tar pitch and asphalt; heating and power generation; and emissions from internal combustion engines. It is estimated that approximately 270,000 metric tons of PAHs reach the environment yearly (Eisler, 1987).

Plants. Some PAHs are synthesized by plants at very low concentrations (Sims and Overcash, 1983). Background concentrations of specific PAH compounds usually range from 22 to 88 µg/kg in tree leaves, 48 to 66 µg/kg in cereal crop plants, 0.05 to 50 µg/kg in leafy vegetables, 0.01 to 6 µg/kg in underground vegetables, and 0.02 to 0.04 µg/kg in fruits (Sims and Overcash, 1983). In general, PAH concentrations are usually greater in above-ground plant parts than in below-ground parts and are greater on plant surfaces than within internal tissues (Eisler, 1987).

Lower-molecular-weight PAHs are taken up from soil by plants more readily than higher-molecular-weight PAHs (Eisler, 1987). Soil-to-plant concentration ratios for total PAHs have been reported to range from 0.001 to 0.183 (Talmage and Walton, 1990). Atmospheric deposition is believed to be the usual source of PAHs in plants, not uptake from soil (Sims and Overcash, 1983).

Limited data exist on the phytotoxicity of PAHs to plants. Benzo(b)fluoranthene concentrations of 6,254 $\mu\text{g}/\text{kg}$ in soil were reported to reduce stem growth in wheat but did not affect rye plants. Benzo(a)pyrene and benzo(b)fluoranthene soil concentrations of up to 18,000 $\mu\text{g}/\text{kg}$ do not appear to be severely toxic to higher plants. There is some evidence that low concentrations of some PAHs may actually stimulate plant growth (Sims and Overcash, 1983).

Mammals. Most of the PAHs taken into the body are not accumulated but are oxidized and the metabolites excreted (NLM, 1996). In fact, most PAH compounds are detoxified and excreted from the body (Klaassen et al., 1991). PAHs are metabolized in vertebrates by a group of enzymes in the liver known as mixed-function oxidases. A few laboratory studies on rodents have revealed that acute oral toxicities of PAHs are greatest for benzo(a)pyrene, followed in decreasing order of toxicity by phenanthrene, naphthalene, and fluoranthene (Sims and Overcash, 1983). Laboratory toxicity data for mice fed benzo(a)pyrene through oral intubation were used to estimate a NOAEL value of 1.0 mg/kg/day (Mackenzie and Angevine, 1981). Reproduction was the endpoint for this study.

Sims and Overcash (1983) have reported LC_{50} values for rodents (*Rattus* spp. and *Mus* spp.) as 50 mg/kg/day benzo(a)pyrene, 700 mg/kg/day phenanthrene, and 2,000 mg/kg/day fluoranthene. Sublethal effects manifested as decreased pup weight in mice have been reported at 10 mg/kg/day benzo(a)pyrene (MacKenzie and Angevine, 1981). Subchronic and chronic effects of exposure to PAHs in rats include liver and kidney damage, unspecified changes in peripheral blood pattern, body weight loss, genetic aberrations, and increased serum aminotransferase activity (Knobloch et al., 1969).

Birds. Hoffman and Gay (1981) measured embryotoxicity of various PAHs applied externally to the surface of mallard duck eggs. Approximately 0.002 $\mu\text{g}/\text{egg}$ of 7,12-dimethylbenz(a)anthracene (DMBA) caused 26 percent mortality in 18 days and, among the survivors, produced significant reduction in embryonic growth and a significant increase in the percent of abnormalities, e.g., incomplete skeletal ossification, defects in eye, brain, liver, feathers, and bill. At 0.1 μg DMBA/egg, only 10 percent survived to day 18.

Aquatic Life. In general, PAHs as a group are not appreciably acutely toxic (Eisler, 1987; Neff, 1985). The toxicity of PAH compounds to fish is related to the solubility of the compound in water. The toxicity of PAHs to aquatic organisms is very species-specific and related to the organisms' ability to metabolize and excrete the compound (Eisler, 1987). For aquatic organisms, only PAHs in the molecular weight range from naphthalene to pyrene are considered acutely toxic. Toxicity in this group increases with increasing molecular weight. There is some evidence to suggest that PAHs are responsible for reproductive and teratogenic effects in eggs of the sand sole (*Psettichthys melanostictus*) exposed to 0.1 µg benzo(a)pyrene/L for 5 days. The eggs showed reduced and delayed hatch and, when compared to controls, produced larvae with high accumulations (2.1 mg/kg fresh weight) and gross abnormalities, such as tissue overgrowths, in 50 percent of the test larvae (Hose, et al., 1982).

Inhibited reproduction of daphnids and the delayed emergence of larval midges by fluorene was reported by Finger, et al. (1985). When sediment PAH levels are elevated, benthic organisms obtain a majority of their PAHs from sediments through their ability to mobilize PAHs from the sediment/pore water matrix. The elevated levels in the tissues of these organisms could provide a significant source of PAHs to predatory fish. However, fish do have the ability to efficiently metabolize and degrade PAHs.

4.3 Potential Receptors

Potential ecological receptors at the IMR ranges fall into two general categories: terrestrial and aquatic. Within these two general categories, there are several major feeding guilds that could be expected to occur at the IMR ranges: herbivores, invertivores, omnivores, carnivores, and piscivores. All of these feeding guilds are expected to be directly exposed to various combinations of surface soil at the IMR ranges and surface water and sediment in Remount Creek and its tributaries near the IMR ranges via various activities (e.g., feeding, drinking, grooming, bathing). These feeding guilds may also be exposed to site-related chemicals via food web transfers.

Dermal absorption of PAHs from soil is a potential exposure pathway for all feeding guilds at the IMR ranges; however, birds and mammals are less susceptible to dermal exposures because their feathers or fur prevents skin from coming into direct contact with the soil (EPA, 1993). Dermal absorption of inorganic compounds from direct contact with soil is expected to be minimal due to the low dermal permeability of these compounds. Since there are no volatile COPECs in soil at the IMR ranges, inhalation of volatiles is not a viable exposure pathway. Inhalation of

constituents sorbed to soil particles and inhaled as dust is a potential exposure pathway for all feeding guilds at the IMR ranges.

Terrestrial species may also be exposed to COPECs in surface water through ingestion of water in Remount Creek and its tributaries near the IMR ranges. Although this creek system is dry during certain periods of the year, over the course of years with “normal” levels of precipitation, it does hold standing and/or flowing water during portions of the year and could be utilized for drinking purposes.

Aquatic and semi-aquatic (i.e., amphibians) species have a greater potential for exposure to COPECs in surface water and/or sediment as they spend a majority of their lifetime in close proximity to water bodies. Aquatic and semi-aquatic species could potentially be exposed to COPECs in surface water and/or sediment via direct contact, ingestion of surface water and sediment, and ingestion of aquatic vegetation or aquatic invertebrates that may have accumulated site-related constituents. Inorganic compounds are the only constituents that have been detected in surface water or sediment at elevated concentrations with respect to ecological screening values; therefore, inorganics represent the only constituents with the potential for significant exposures to aquatic and semi-aquatic species.

4.3.1 Herbivorous Feeding Guild

The major route of exposure for herbivores is through ingestion of plants that may have accumulated contaminants from the soil, surface water, or sediment. The vegetation at the formerly maintained areas of the IMR ranges is mainly grasses and sedges, which are remnants of the maintained grass that was present when the IMR ranges were operational. Since terrestrial herbivores by definition are grazers and browsers, they could be exposed to chemicals that have accumulated in the vegetative tissues of plants at the site. Terrestrial herbivores may also be exposed to site-related chemicals in soil through incidental ingestion of soil while grazing, grooming, or other activities.

Typical herbivorous species that could be expected to occur at the IMR ranges and are commonly used as sentinel species in ecological risk assessment include eastern cottontail (*Sylvilagus floridanus*), eastern gray squirrel (*Sciurus carolinensis*), pine vole (*Pitymys pinetorum*), whitetail deer (*Odocoileus virginianus*), and wild turkey (*Meleagris gallopavo*).

Aquatic herbivores such as muskrat (*Ondatra zibethica*) and mallard (*Anas platyrhynchos*) could be exposed to site-related constituents in surface water and/or sediment in Remount Creek and its tributaries.

4.3.2 Invertivorous Feeding Guild

Invertivores specialize in eating insects and other invertebrates. As such, they may be exposed to site-related chemicals that have accumulated in insects and other invertebrates. Invertivores may also be exposed to site-related chemicals in soil through incidental ingestion of soil while probing for insects, grooming, or other activities. Ingestion of soil while feeding is potentially a major exposure pathway for invertivores, since much of their food (i.e., earthworms and other invertebrates) lives on or below the soil surface.

Typical invertivorous species that could be expected to occur at the IMR ranges and are commonly used as sentinel species in ecological risk assessment include American woodcock (*Philohela minor*), carolina wren (*Thryothorus ludovicianus*), shorttail shrew (*Blarina brevicauda*), and eastern mole (*Scalopus aquaticus*).

Aquatic invertivores could include the wood duck (*Aix sponsa*) and blacknose dace (*Rhinichthys atratulus*).

4.3.3 Omnivorous Feeding Guild

Omnivores consume both plant and animal material in their diet, depending upon availability. Therefore, they could be exposed to chemicals that have accumulated in the vegetative tissues of plants at the site and also chemicals that may have accumulated in smaller animal tissues that the omnivores prey upon. They may also be exposed to surface water through ingestion of water in Remount Creek near the IMR ranges. Omnivores may also be exposed to site-related chemicals in soil through incidental ingestion of soil while feeding, grooming, or other activities.

Typical omnivorous species expected to occur at the IMR ranges are commonly used as sentinel species in ecological risk assessment include red fox (*Vulpes vulpes*), white-footed mouse (*Peromyscus leucopus*), and American robin (*Turdus migratorius*).

Aquatic omnivores such as raccoon (*Procyon lotor*) and creek chub (*Semotilus atromaculatus*) could be exposed to COPECs in surface water and sediment in Remount Creek and its tributaries in the vicinity of the IMR ranges.

4.3.4 Carnivorous Feeding Guild

Carnivores are meat-eating animals and are, therefore, exposed to site-related chemicals through consumption of prey animals that may have accumulated contaminants in their tissues.

Carnivores are quite often top predators in a local food web and are often subject to exposure to

contaminants that have biomagnified through the food web. Food web exposures for carnivores are based on the consumption of prey animals that have accumulated COPECs through various means. Smaller herbivores, omnivores, invertivores, and other carnivores may consume soil, surface water, sediment, and plant and animal material as food and accumulate COPECs in their tissues. Subsequent ingestion of these prey animals by carnivorous animals would expose them to COPECs. Most inorganic compounds are not accumulated in animal tissues to any great extent (Shugart, et al., 1991; U.S. Army Environmental Hygiene Agency [USAEHA], 1994). Therefore, food web exposures to these chemicals are expected to be minimal. PAHs have the potential to accumulate in lower trophic level organisms, but not in higher trophic level organisms because they have mechanisms for metabolizing and excreting this class of compounds.

Carnivores may also be exposed to site-related chemicals in soil through incidental ingestion of soil while feeding, grooming, or other activities. These species may occupy the woodlands that surround the IMR ranges and the open areas of the ranges themselves.

Typical carnivorous species expected to occur at the IMR ranges and commonly used as sentinel species in ecological risk assessment include red-tailed hawk (*Buteo jamaicensis*), black vulture (*Coragyps atratus*), and bobcat (*Lynx rufus*).

Because Remount Creek and its tributaries in the vicinity of the IMR ranges are narrow and shallow, they do not have the capability to support large aquatic carnivores. Carnivorous fish such as largemouth bass (*Micropterus salmoides*) and spotted gar (*Lepisosteus oculatus*) would not be expected to occur in Remount Creek in the vicinity of the IMR ranges, due to the habitat restrictions. Additionally, carnivorous mammals such as the mink (*Mustela vison*), which depends on larger fish for food, would not be expected to occur in the vicinity of the IMR ranges.

4.3.5 Piscivorous Feeding Guild

Piscivores are specialists that feed mostly on fish. Therefore, they may be exposed to site-related chemicals that have accumulated in small fish that may inhabit Remount Creek and its tributaries in the vicinity of the IMR ranges. They may also be exposed to surface water and sediment in the creek system through ingestion of drinking water and during feeding. Although these creeks are dry during certain periods of the year, they do hold flowing and/or standing water during portions of the year and could be utilized for drinking purposes. Although piscivorous species could be expected to visit the areas around the creek system in the vicinity of the IMR ranges during periods of the year when the creeks hold water, they would not be expected to live near the IMR ranges, due to the ephemeral nature of Remount Creek.

Food web exposures for piscivores are based on the consumption of fish that have accumulated COPECs from surface water and sediment. Forage fish may consume surface water, sediment, benthic invertebrates, aquatic plants, and planktonic material as food and accumulate COPECs in their tissues. Subsequent ingestion of these forage fish by piscivorous animals would expose them to COPECs. However, most PAHs and inorganics are not accumulated in fish tissues to any great extent. Therefore, food web exposures to these chemicals are expected to be minimal. Semivolatile organic compounds are readily metabolized by most fish species and are not accumulated to any extent. Thus, the piscivorous feeding guild is not expected to be greatly exposed to COPECs at the IMR ranges through the food web.

Typical piscivorous species expected to occur near the IMR ranges and commonly used as sentinel species in ecological risk assessment include great blue heron (*Ardea herodias*) and belted kingfisher (*Megaceryle alcyon*). Larger piscivorous fish species (e.g., smallmouth bass, spotted gar) and piscivorous mammals (e.g., mink) are not expected to occur in the creek system at the IMR ranges, due to the ephemeral nature of Remount Creek in this area and its inability to support larger fish and other aquatic species.

4.3.6 Threatened and Endangered Species

Four species listed as threatened or endangered by the U.S. Fish and Wildlife Service (USFWS) have been recorded at FTMC. These threatened and endangered species are as follows:

- Gray Bat (*Myotis grisescens*)
- Blue Shiner (*Cyprinella caerulea*)
- Mohr's Barbara Buttons (*Marshallia mohrii*)
- Tennessee Yellow-Eyed Grass (*Xyris tennesseensis*).

The only federally-listed species that has the potential to occur in the vicinity of the IMR ranges is the gray bat (Garland, 1996). Remount Creek adjacent to the IMR ranges has been designated as providing "low quality" foraging habitat for the gray bat (Garland, 1996) except for the section of the creek that runs adjacent to the Skeet Range. This section of Remount Creek has been designated as providing "moderate quality" foraging habitat for the gray bat (Garland, 1996). "High quality" foraging habitat is not found on Main Post at FTMC (3DI, 1998). Additionally, no gray bat roosting sites have been identified at FTMC (3DI, 1998). The other federally-listed species occur at Pelham Range or Choccolocco Creek Corridor.

The gray bat is almost entirely restricted to cave habitats, and, with rare exceptions, roosts in caves year-round. Approximately 95 percent of the entire known population hibernates in only nine caves each winter, with more than half in a single cave. Gray bat summer foraging habitat is found primarily over open water of rivers and reservoirs. They apparently do not forage over sections of rivers or reservoirs that have lost their normal woody vegetation along the banks (USFWS, 1982). Gray bats usually follow wooded corridors from their summer caves to the open water areas used as foraging sites. Forested areas surrounding and between caves, as well as over feeding habitats, are clearly advantageous to gray bat survival as the cover provides increased protection from predators such as screech owls. In addition, surveys have demonstrated that reservoirs and rivers that have been cleared of their adjacent forest canopy are avoided as foraging areas by gray bats (USFWS, 1982).

The gray bat is entirely insectivorous, and surveys have shown that gray bats feed almost exclusively on mayflies at certain times of the year (Mount, 1986). Therefore, gray bats could be exposed to site-related constituents that have accumulated in aquatic insects from Remount Creek. Because gray bats are flying mammals and the IMR ranges do not provide roosting habitat, no other exposure pathways are potentially complete for the gray bat.

Most foraging occurs within 5 meters of the water's surface, usually near a shoreline or stream bank. Mist net surveys were conducted on and adjacent to FTMC in 1995. Gray bats were captured along both Choccolocco Creek (east of FTMC Main Post) and Cane Creek on Pelham Range (west of FTMC Main Post) and near the golf course on Main Post during these mist net surveys (3DI, 1998). Although no critical gray bat habitat has been identified on or near Main Post, these preliminary data suggest that these major stream corridors at FTMC may provide at least a minimum foraging habitat for gray bats. However, gray bat surveys have not been conducted on Remount Creek in the vicinity of the IMR ranges.

4.4 Complete Exposure Pathways

For exposures to occur, a complete exposure pathway must exist between the contaminant and the receptor. A complete exposure pathway requires the following four components:

- A source mechanism for contaminant release
- A transport mechanism
- A point of environmental contact
- A route of uptake at the exposure point (EPA, 1989).

If any of these four components is absent, then a pathway is generally considered incomplete. Potentially complete exposure pathways are depicted in the SCM as Figure 4-1.

Ecological receptors may be exposed to constituents in soils via direct and/or secondary exposure pathways. Direct exposure pathways include soil ingestion, dermal absorption, and inhalation of COPECs adsorbed to fugitive dust. Significant exposure via dermal contact is limited to organic constituents that are lipophilic and can penetrate epidermal barriers. Mammals are less susceptible to exposure via dermal contact with soils because their fur prevents skin from coming into direct contact with soil. However, soil ingestion may occur while grooming, preening, burrowing, or consuming plants, insects, or invertebrates resident in soil.

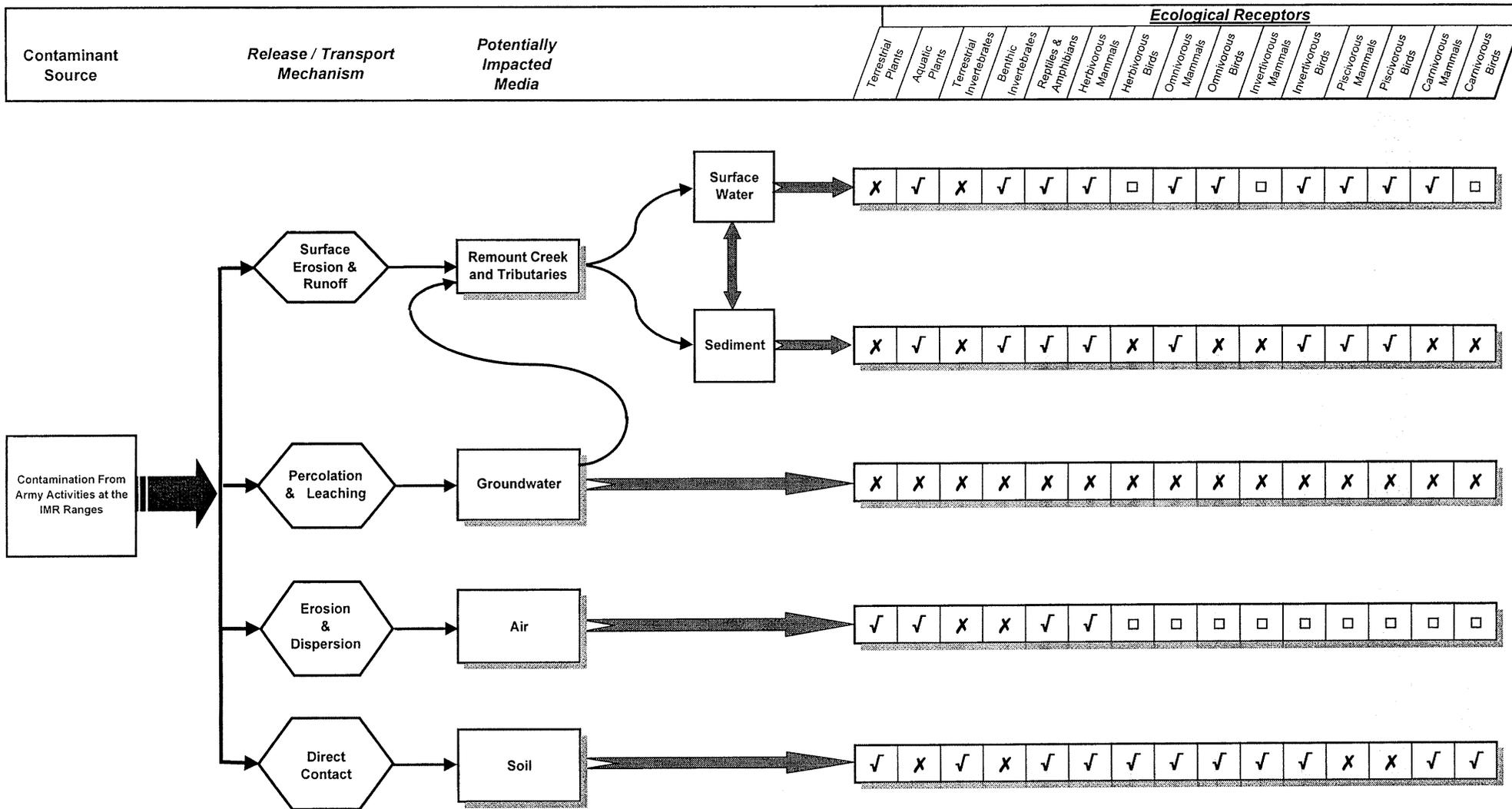
Ecological receptors may be exposed to constituents in surface water via direct contact or through consumption of water. Aquatic organisms inhabiting contaminated waters would be in constant contact with COPECs.

Exposure via inhalation of fugitive dust is limited to contaminants present in surface soils at areas that are devoid of vegetation. The moisture content of the soil and the frequency of soil disturbance also play important roles in the amount of fugitive dust generated at a particular site.

Constituents present in sediment may result from erosion or adsorption of water-borne constituents onto sediment particles. If sediments are present in an area that is periodically inundated with water, then previous exposure pathways for soils would be applicable during dry periods. Water overlying sediments prevents contaminants from being carried by wind erosion. Because all of the constituents detected in sediment are inorganic compounds that are not prone to volatilization, volatilization from sediments is not an important fate mechanism. Therefore, inhalation of constituents originating from the sediment is not a significant exposure pathway. Exposure via dermal contact may occur, especially for benthic organisms and wading birds or other animals that may use Remount Creek as a feeding area. Some aquatic organisms consume sediment and ingest organic material from the sediment. Inadvertent ingestion of sediments may also occur as the result of feeding on benthic organisms and plants.

While constituents in soils may leach into groundwater, environmental receptors will not come into direct contact with constituents in groundwater since there is no direct exposure route. The only potential exposure pathways for ecological receptors to groundwater would be via surface water exposure routes. As described in previous sections of this report, groundwater discharge to surface water at the IMR ranges is potentially a viable transport mechanism for dissolved constituents; however, exposure to these constituents by ecological receptors is only possible via

Figure 4-1
Site Conceptual Model
For Iron Mountain Road Ranges
 Fort McClellan, Calhoun County, Alabama



Key To Potential Exposure Routes

- √ - Potentially complete exposure pathway
- X - Incomplete exposure pathway
- - Potentially complete exposure pathway but insignificant

surface water exposure routes. Potential exposure to groundwater-related constituents is expected to be insignificant based on the fact that lead was the only constituent detected at elevated concentrations in surface water at the IMR ranges and lead was not detected at elevated concentrations in groundwater. Furthermore, none of the constituents detected in groundwater at elevated concentrations were detected in surface water at the IMR ranges.

Secondary exposure pathways involve constituents that are transferred through different trophic levels of the food chain and may be bioaccumulated and/or bioconcentrated. This may include constituents bioaccumulated from soil into plant tissues or into terrestrial species ingesting soils. These plants or animals may, in turn, be consumed by animals at higher trophic levels. Water-borne and sediment-borne COPECs may bioaccumulate into aquatic organisms, aquatic plants, or animals which frequent surface waters and then be passed through the food chain to impact organisms at higher trophic levels.

In general, the constituents detected in surface soil at the IMR ranges may bioaccumulate in lower trophic level organisms (i.e., terrestrial invertebrates may bioaccumulate inorganic compounds and PAHs detected in soil); however, they will not bioconcentrate through the food chain. Inorganic compounds generally do not bioconcentrate to any great extent and PAHs are readily metabolized by higher trophic level organisms. The only compound detected in surface soil that has a propensity to bioconcentrate is 4,4'-DDT. 4,4'-DDT was detected infrequently (one sample) and at very low concentrations; therefore, bioconcentration is not expected to be a significant exposure pathway at the IMR ranges.

As is the case with surface soil, the constituents detected in surface water and sediment may bioaccumulate in lower trophic level organisms (i.e., benthic invertebrates may bioaccumulate inorganic compounds detected in sediment); however, they will not bioconcentrate through the food chain. Inorganic compounds and volatile organics generally do not bioconcentrate to any great extent.

Summaries of the potentially complete exposure pathways for the terrestrial and aquatic ecosystems at the IMR ranges are presented in Tables 4-1 and 4-2, respectively.

Table 4-1

**Feeding Guilds and Exposure Pathways for Terrestrial Ecosystems at the IMR Ranges
Fort McClellan, Calhoun County, Alabama**

| Trophic Level | Feeding Guild | Exposure Pathways |
|---------------|-----------------------------|---|
| 1 | Primary Producers | Direct uptake from soil |
| 2 | Terrestrial Invertebrates | Ingestion of soil Direct contact with soil |
| | Herbivorous Birds | Ingestion of soil Ingestion of surface water Ingestion of terrestrial plants |
| | Herbivorous Mammals | Ingestion of soil Ingestion of surface water Ingestion of terrestrial plants |
| 3 | Omnivorous Birds | Ingestion of soil Ingestion of surface water Ingestion of terrestrial plants Ingestion of terrestrial invertebrates |
| | Omnivorous Mammals | Ingestion of soil Ingestion of surface water Ingestion of terrestrial plants Ingestion of terrestrial invertebrates Ingestion of prey |
| | Invertivorous Birds | Ingestion of soil Ingestion of surface water Ingestion of terrestrial invertebrates |
| | Invertivorous Mammals | Ingestion of soil Ingestion of surface water Ingestion of terrestrial invertebrates |
| 4 | Carnivorous Birds (raptors) | Ingestion of soil Ingestion of surface water Ingestion of prey |
| | Carnivorous Mammals | Ingestion of soil Ingestion of surface water Ingestion of prey |

Table 4-2

**Feeding Guilds and Exposure Pathways for Freshwater Stream Ecosystems at the IMR Ranges
Fort McClellan, Calhoun County, Alabama**

| Trophic Level | Feeding Guild | Exposure Pathways |
|---------------|--|---|
| 1 | Primary Producers | Direct uptake from surface water Direct uptake from sediment |
| 2 | Benthic Invertebrates Herbivorous Waterfowl Herbivorous Mammals Forage Fish | Ingestion of sediment Direct contact with sediment Ingestion of sediment Ingestion of surface water Ingestion of aquatic plants Ingestion of sediment Ingestion of surface water Ingestion of aquatic plants Ingestion of plankton Ingestion of aquatic plants and algae Absorption through gills |
| 3 | Omnivorous Mammals Omnivorous Waterfowl Invertivorous Waterfowl | Ingestion of sediment Ingestion of surface water Ingestion of aquatic plants Ingestion of benthic invertebrates Ingestion of sediment Ingestion of surface water Ingestion of aquatic plants Ingestion of benthic invertebrates Ingestion of sediment Ingestion of surface water Ingestion of benthic invertebrates |
| 4 | Piscivorous Mammals Piscivorous Birds | Ingestion of sediment Ingestion of surface water Ingestion of fish Ingestion of sediment Ingestion of surface water Ingestion of fish |