

## 7.0 Screening-Level Ecological Risk Assessment

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### 7.1 Introduction

In order to determine the potential for ecological risks posed by site-related chemicals at the Small Weapons Repair Shop, Parcel 66(7), a screening-level ecological risk assessment (SLERA) was conducted. This SLERA consists of a description of the habitat(s) in and around Parcel 66(7), a discussion of the constituents detected in samples collected from environmental media at the site, a discussion of the conceptual site model, an estimation of the screening-level risk, the identification of the constituents of potential ecological concern, an uncertainty analysis, a discussion of the different lines of evidence, and a summary of the results and conclusions.

### 7.2 Environmental Setting

Parcel 66(7) is approximately 1.15 acres in size. The site is bounded by Waverly Road on the north, asphalt parking areas on the east and south, and Freemont Road on the west. The buildings that housed the Small Weapons Repair Shop (Building 335) and the small boiler plant (Building 336) remain on site. The site is located on a topographical divide; the northern portion of the parcel slopes slightly to the north, and the southern portion of the parcel slopes slightly to the south. The entire site and the area immediately surrounding the site are covered with asphalt or concrete paving. A 6-foot-high chain-link fence surrounds the entire study area and adjacent parking area. There are no water bodies associated with this site, other than the man-made drainage ditches that border the site along Waverly Road to the north and Freemont Road to the west.

**Terrestrial Habitat.** The site itself is entirely paved with asphalt and, as such, ecological habitat at the site is insignificant. The area surrounding the site is also entirely paved with asphalt and is used as a parking area. Therefore, ecological habitat at Parcel 66(7) and the surrounding area is virtually nonexistent.

### 7.3 Constituents Detected On-Site

The sampling and analysis programs conducted at Parcel 66(7) were designed based on a number of factors including:

- Site history
- Results of the EBS
- Results of previous sampling and analysis programs.

1 The sampling and analysis programs at Parcel 66(7) are described in Chapter 2.0 of this report.  
2 Constituents detected in surface soil at the site are summarized in Chapter 4.0 of this report.

3  
4 Surface water and sediment were not sampled at Parcel 66(7) because there are no surface water  
5 bodies associated with the site.

6  
7 In general, inorganic constituents were commonly detected in surface soils, but organic  
8 compounds (i.e., SVOCs) were less frequently detected and at relatively low concentrations.  
9 Beryllium, cobalt, copper, nickel, and zinc were detected in one surface soil sample at elevated  
10 concentrations relative to ESVs. Selenium was detected in two samples at elevated  
11 concentrations. PAHs (benzo[a]pyrene, fluoranthene, phenanthrene, and pyrene) were detected  
12 in one surface soil sample at elevated concentrations, and one pesticide (endrin) was also  
13 detected in one surface soil sample at an elevated concentration. It is important to note that the  
14 MDCs of the four PAH compounds detected in surface soil at elevated concentrations were less  
15 than the background threshold values for soil beneath pavement as presented in *Human Health  
16 and Ecological Screening Values and PAH Background Summary Report* (IT, 2000c). Thus, the  
17 SVOCs in surface soil at Parcel 66(7) are characteristic of soil beneath pavement at similar sites  
18 at FTMC and may not be indicative of site-related Army activities.

#### 19 20 **7.4 Site Conceptual Model**

21 The ecological site conceptual model (SCM) is a schematic diagram of possible exposure  
22 pathways and the means by which contaminants are transported from the primary contaminant  
23 source(s) to ecological receptors. The exposure scenarios include the sources, environmental  
24 transport, partitioning of the contaminants amongst various environmental media, potential  
25 chemical/biological transformation processes, and identification of potential routes of exposure  
26 for the ecological receptors. In this chapter, the SCM will be described in relation to constituent  
27 fate and transport properties, the ecotoxicity of the various constituents, potential ecological  
28 receptors at Parcel 66(7), and the complete exposure pathways expected to exist at Parcel 66(7).

##### 29 30 **7.4.1 Constituent Fate and Transport**

31 The environmental fate and transport of contaminants in surface soil at Parcel 66(7) will govern  
32 the potential for exposures to ecological receptors. In general, contaminants in environmental  
33 media may be available for direct exposure (e.g., plants exposed to surface soil) and they may  
34 also have the potential to migrate to other environmental media or other areas of the site. This

1 section discusses the mechanisms by which contaminants can be transported and the chemical  
2 properties that determine their transport.

#### 3 4 **7.4.1.1 Fate and Transport in Soil**

5 The fate and transport of constituents in surface soil at Parcel 66(7) is highly dependent upon the  
6 fact that the entire area of the parcel is currently covered with asphalt paving. As such, many of  
7 these fate and transport pathways are not currently applicable. Therefore, fate and transport are  
8 discussed in this section with the assumption that, while the site is currently paved, the pavement  
9 may be removed or may deteriorate significantly at some time in the future.

10  
11 Surface soil at Parcel 66(7) is currently completely paved and is not subject to erosion or dust  
12 generation, and water infiltration is significantly reduced. If the asphalt paving were to be  
13 removed, constituents in surface soil at Parcel 66(7) would have the potential to be transported  
14 from their source area to other areas within the study area and to off-site locations by a number  
15 of mechanisms, including volatilization, dust entrainment, surface runoff, and infiltration to  
16 subsurface soil/groundwater.

17  
18 Several VOCs were identified in the upper soil horizons at Parcel 66(7), albeit at very low  
19 concentrations. These volatile constituents have a high potential to volatilize to the atmosphere  
20 and be transported from their source area via air movement. The concentrations of VOCs  
21 detected in surface soil at Parcel 66(7) are low; therefore, this transport mechanism is expected to  
22 be insignificant when compared to other transport mechanisms potentially active at this site.  
23 Most of the metals and SVOCs in the surface soil at Parcel 66(7) are not expected to volatilize to  
24 any great extent, with the exception of mercury, which would be expected to volatilize relatively  
25 rapidly. Most of the metals and SVOCs in the surface soil at Parcel 66(7) are closely associated  
26 with particulate matter and could be transported from their source areas by fugitive dust  
27 generation and entrainment by the wind. Subsequent dispersion by atmospheric mixing could  
28 transport particulate-associated constituents to other parts of Parcel 66(7) and to off-site  
29 locations. The generation of fugitive dust and subsequent transport by the wind is not expected  
30 to be a significant transport mechanism at Parcel 66(7) because the entire site and surrounding  
31 area are covered with asphalt.

32  
33 The transport of surface soil-associated contaminants by surface runoff is another potential  
34 transport mechanism. Surface soil contaminants could be solubilized by rainwater and  
35 subsequently transported to the man-made drainage ditches that are present along the northern

1 and western boundaries of the site via surface runoff. The solubility of inorganics in rainwater is  
2 largely dependent upon the pH of the rainwater. Because the rainwater in this region is most  
3 likely slightly acidic, the inorganic constituents in surface soil may solubilize to some degree in  
4 the rainwater and be subject to transport via runoff. Most of the SVOCs are strongly associated  
5 with soil particles and would not solubilize to a large extent. Constituents that may be more  
6 strongly bound to particulate matter in surface soil (e.g., SVOCs and some of the inorganics)  
7 may be entrained in surface water runoff and transported to the drainage ditches via surface  
8 runoff. Many of the metals and semivolatiles are strongly sorbed to soil particles and could be  
9 transported from their source areas via this mechanism. However, the presence of asphalt over  
10 the entirety of the study area precludes the possibility of erosion of surface soil. Therefore,  
11 erosion and surface runoff of soil are expected to be insignificant at Parcel 66(7).

12  
13 Contaminants in surface soil may be transported vertically to subsurface soils and groundwater  
14 via solubilization in rainwater and infiltration. Migration in this manner is dependent upon  
15 contaminant solubility and frequency of rainfall and infiltration rate. The soil types in the  
16 vicinity of Parcel 66(7) are not expected to promote rapid infiltration of rainwater. Furthermore,  
17 the asphalt cover over the entire study area greatly reduces the infiltration rate and the subsequent  
18 potential for vertical migration of surface soil contaminants. Additionally, many of the  
19 constituents (e.g., SVOCs) detected in surface soil at Parcel 66(7) exhibit low water solubilities  
20 and are not expected to migrate vertically to any significant extent. Therefore, vertical migration  
21 of soil contaminants is expected to be insignificant at Parcel 66(7).

22  
23 The transfer of contaminants in surface soil to terrestrial plants through root uptake and transfer  
24 to terrestrial animals through ingestion and other pathways are potential transfer mechanisms.  
25 However, the presence of asphalt over the entire study area precludes the direct contact of surface  
26 soil by plants and animals. Therefore, under current conditions, root uptake by plants and  
27 ingestion by animals are expected to be insignificant transfer mechanisms at Parcel 66(7).

28  
29 VOCs in the surface soil at Parcel 66(7) would volatilize and/or photolyze rapidly (half-lives of 3  
30 hours to 5 days) when exposed to sunlight (Burrows et al., 1989). However, the asphalt paving  
31 will greatly reduce these fate processes. The other surface soil contaminants (metals, SVOCs,  
32 and one pesticide) are expected to remain in the soil relatively unchanged by physical and/or  
33 chemical processes for much longer periods of time.

34

1 **7.4.1.2 Fate and Transport in Surface Water**

2 Due to the lack of a surface water body in the immediate vicinity of Parcel 66(7), surface water  
3 fate and transport properties are not considered significant with respect to constituents detected at  
4 the site.

5  
6 **7.4.1.3 Fate and Transport in Sediment**

7 Due to the lack of a surface water body in the immediate vicinity of Parcel 66(7), sediment fate  
8 and transport properties are not considered significant with respect to constituents detected at the  
9 site.

10  
11 **7.4.2 Ecotoxicity**

12 The ecotoxicological properties of the constituents detected in surface soil at Parcel 66(7) are  
13 discussed in the following sections.

14  
15 **7.4.2.1 Beryllium**

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

18  
19 **Plants.** Beryllium uptake by plants occurs when beryllium is present in the soluble form. The  
20 highest levels of beryllium are found in the roots, with lower levels in the stems and foliage  
21 (EPA, 1985a).

22  
23 Soluble forms of beryllium are easily taken up by plants, probably in a manner similar to calcium  
24 and magnesium, but it is not readily translocated from roots to shoots (Peterson and Girling,  
25 1981). Beryllium has been reported to inhibit seed germination, enzyme activation, and uptake  
26 of calcium and magnesium by roots. Common symptoms of beryllium toxicity to plants are  
27 brown, retarded roots and stunted foliage (Romney and Childress, 1965). The phytotoxicity  
28 benchmark value for beryllium (10 mg/kg) is based on unspecified toxic effects on plants grown  
29 in surface soil amended with 10 mg/kg beryllium (Kabata-Pendias and Pendias, 1992).

30  
31 **Mammals.** The major exposure route for mammals is inhalation, although absorption by this  
32 route does not appear to be extensive. Beryllium is poorly absorbed from the gastrointestinal  
33 tract, and is not absorbed through intact skin to any significant degree. Mammals exposed via  
34 inhalation exhibit pulmonary effects, which may last long after exposure ceases. Once beryllium  
35 is absorbed, it is circulated in the blood as an orthophosphate colloid and is then distributed

1 primarily to the bone, liver, and kidneys in both humans and animals. Beryllium and its  
2 compounds are not biotransformed, but soluble beryllium compounds are partially converted to  
3 more insoluble forms in the lungs (Reeves and Vorwald, 1967).

4  
5 Following inhalation of soluble beryllium compounds in both humans and animals, the lung  
6 appears to be the main target organ for toxicity. Acute exposure may cause chemical  
7 pneumonitis; chronic exposure to insoluble forms may lead to chronic beryllium disease  
8 (berylliosis), a fibrotic lung disease (Agency for Toxic Substances and Disease Registry  
9 [ATSDR], 1993).

10  
11 A variety of beryllium compounds have been demonstrated to cause pulmonary tumors following  
12 inhalation in animals. However, it is thought that oral administration does not lead to  
13 carcinogenesis, due to poor absorption of the constituent from the gastrointestinal tract. The no-  
14 observed-adverse-effects-level (NOAEL) for a rat lifetime chronic exposure to beryllium in  
15 drinking water was 0.54 milligrams per kilogram of bodyweight per day (mg/kg/day) (Health  
16 Effects Assessment Summary Tables, 1997).

17  
18 Based on laboratory toxicity data for beryllium sulfate administered to rats in their drinking  
19 water, a NOAEL of 0.66 mg/kg/day for chronic oral exposure was derived (Schroeder and  
20 Mitchener, 1975).

21  
22 **Aquatic Life.** Exposure routes for aquatic organisms include ingestion and gill uptake.  
23 Beryllium does not bioconcentrate in aquatic organisms. Beryllium uptake from water is low,  
24 resulting in low bioconcentration rates. Biomagnification of beryllium in aquatic food chains  
25 does not occur (Fishbein, 1981). Beryllium can be toxic to warm water fish, especially in soft  
26 water.

27  
28 The Tier II secondary acute water quality value and secondary chronic water quality value for  
29 beryllium, as calculated by the method described in the EPA's *Final Water Quality Guidance for*  
30 *the Great Lakes System* (EPA, 1995b), are 35 and 0.66 micrograms per liter ( $\mu\text{g/L}$ ), respectively.

31  
32 The  $\text{EC}_{20}$  for fish can be used as a benchmark indicative of production within a population. It is  
33 the highest tested concentration causing less than 20 percent reduction in either weight of young  
34 fish per initial female fish in a life-cycle or partial life-cycle test, or the weight of young per egg  
35 in an early life-stage test (Suter and Tsao, 1996). The  $\text{EC}_{20}$  value for beryllium is 148  $\mu\text{g/L}$ . A

1 similar value can be determined for daphnids which reflects the highest tested concentration  
2 causing less than 20 percent reduction in the product of growth, fecundity, and survivorship in a  
3 chronic test with a daphnid species. The EC<sub>20</sub> for daphnids is 3.8 µg/L (Suter and Tsao, 1996).

#### 4 5 **7.4.2.2 Cobalt**

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

13  
14 **Plants.** Although cobalt is essential to some blue-green algae, fungi, and microorganisms, it  
15 apparently is not essential for the growth of higher plants (Kabata-Pendias and Pendias, 1992).  
16 Several abiotic factors govern the availability of cobalt to plants. Soil factors include organic  
17 matter and clay content, pH, leachability, and concentration of manganese and iron oxides.  
18 Uptake of cobalt can occur via the roots or leaves of a plant (Kabata-Pendias and Pendias, 1992).

19  
20 Concentrations of cobalt in leaf tissue that are excessive or toxic to various plant species range  
21 from 15 to 50 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). A soil concentration of  
22 20 mg/kg (dry weight) has been proposed by Efroymson et al. (1997) as a benchmark screening  
23 value for cobalt phytotoxicity. General symptoms of cobalt toxicity in plants include interveinal  
24 chlorosis in new leaves followed by induced iron chlorosis and white leaf margins and damaged  
25 root tips (Kabata-Pendias and Pendias, 1992).

26  
27 **Mammals.** Cobalt is a component of vitamin B<sub>12</sub> and, therefore, is an essential micronutrient  
28 for animal growth. No information has been located at this time on chronic toxic effects of  
29 cobalt to terrestrial wildlife; however, some acute studies have been completed. Additionally,  
30 there is little biomagnification of cobalt in animals of higher trophic levels (Jenkins, 1980).

31  
32 Young rats are unable to survive repeated 30 mg doses of cobalt metal powder in their diet for a  
33 month (total dosage about 900 mg), whereas they can tolerate 1,250 mg of the metal in a single  
34 dose (Venugopal and Luckey, 1978). Cobalt was embryotoxic to rat fetuses when it was  
35 administered during the entire gestation (dose of 0.05 mg/kg). A dose of 0.005 mg/kg was non-

1 toxic to the females; however, the progeny of treated females had a reduced survival rate  
2 (Shepard, 1986). At doses under 2 mg/kg/day, no adverse effects to sheep were noted. However,  
3 at 6 mg/kg/day, sheep exhibited loss of appetite, loss of weight, and debilitation (National  
4 Research Council, 1977).

5  
6 **Birds.** No information has been located at this time on chronic toxic effects of cobalt to birds;  
7 however, some acute studies have been completed. Additionally, there is little biomagnification  
8 of cobalt in animals of higher trophic levels (Jenkins, 1980).

9  
10 Chickens were administered cobalt in their diets at a dose of 50 mg/kg/day with acute effects of  
11 loss of appetite, loss of weight, and debilitation. At doses under 2 mg/kg/day, no adverse effects  
12 to chickens were noted (National Research Council, 1977).

13  
14 **Aquatic Life.** In most surface water bodies, cobalt is primarily associated with the sediment.  
15 However, some mobilization may occur in acidic water and in the presence of chloride ions or  
16 chelating agents. Bioaccumulation factors for freshwater fish range from 40 to 1,000 (Smith and  
17 Carson, 1981).

18  
19 Research by Evans et al. (1988) indicates that cobalt does not significantly bioaccumulate in  
20 benthic bottom feeders.

### 21 22 **7.4.2.3 Copper**

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

28  
29 Copper has been known to sorb rapidly to sediment. The rate of sorption is, of course, dependent  
30 upon factors such as the sediment grain size, organic fraction, pH, competing cations, and the  
31 presence of ligands. In industrialized freshwater environments around the world, total copper  
32 levels within sediments can range from 7 to 2,350 parts per million (ppm) (Moore and  
33 Ramamoorthy, 1984).

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

6  
7 According to Rhodes et al. (1989), copper concentrations in plant tissues do not serve as  
8 conclusive evidence of copper toxicity in species of plants such as tomatoes, because some  
9 species are able to tolerate higher concentrations of copper than others. The pH of soil may also  
10 influence the availability and toxicity of copper in soils to plants (Rhodes et al., 1989). In a  
11 study with tomato plants, Rhodes et al. (1989) found a reduction in plant growth when plants  
12 were grown in soils containing greater than 150 mg/kg of copper at a pH of less than 6.5. At pH  
13 values greater than 6.5, soil copper concentrations greater than 330 mg/kg were required to  
14 reduce plant growth.

15  
16 Concentrations of copper in leaf tissue that are excessive or toxic to various plant species range  
17 from 20 to 100 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). A soil concentration of  
18 100 mg/kg has been proposed by Efroymson et al, (1997) as a benchmark screening value for  
19 copper phytotoxicity in soil. General symptoms of copper toxicity in plants include the presence  
20 of dark green leaves followed by induced iron chlorosis; thick, short, or barbed-wire roots; and  
21 depressed tillering (Kabata-Pendias and Pendias, 1992).

22  
23 **Mammals.** Copper is an essential trace element to plants and animals (Callahan et al., 1979)  
24 but becomes toxic at concentrations only slightly higher than essential levels (EPA, 1985b).  
25 Copper is an essential element for hemoglobin synthesis and oxidative enzymes in animals.  
26 Copper is absorbed by mammals following ingestion, inhalation, or dermal exposure. Once  
27 absorbed, copper is distributed to the liver. Copper is not metabolized (Marceau et al., 1970).  
28 No evidence of bioaccumulation was obtained in a study of pollutant concentrations in the  
29 muscles and livers of 10 species of herbivorous, omnivorous, and carnivorous animals in Donana  
30 National Park in Spain (Hernandez et al., 1985). Copper concentrations in small mammals  
31 collected from various uncontaminated sites ranged from 8.3 to 13.4 mg/kg (whole-body  
32 concentrations) (Talmage and Walton, 1991). Highest concentrations of copper tend to be in  
33 hair, followed in decreasing concentration by liver, kidney, and whole body (Hunter and  
34 Johnson, 1982). Among the small mammals collected, Hunter and Johnson (1982) found shrews  
35 (*Sorex araneus*) to contain the highest concentrations of copper. Mice were found to contain the

1 lowest copper concentrations. Increased fetal mortality was observed in fetuses of mice fed more  
2 than 104 mg/kg-day of copper as copper sulfate (Lecyk, 1980). Increased mortality rates in mink  
3 offspring have been observed at levels above 3.21 mg/kg-day (Aulerich et al., 1982).

4  
5 Laboratory toxicity data from oral exposures of mink to copper sulfate in their diet were used to  
6 calculate a NOAEL value of 11.7 mg/kg/day. Symptoms of acute copper poisoning in mammals  
7 include vomiting, hypotension, melena, coma, jaundice, and death (Klaassen et al., 1991).  
8 Selenium can act as an antidote for copper poisoning.

9  
10 **Birds.** Based on toxicity test data specific to one-day old chickens fed copper oxide in their  
11 diets, a NOAEL value of 47 mg/kg/day was calculated (Mehring et al., 1960). The endpoints for  
12 this 10-week study were growth and mortality.

13  
14 **Aquatic Life.** Invertebrates inhabiting "polluted" freshwaters worldwide have been known to  
15 have tissue residues of copper ranging from 5 to 200 ppm (Moore and Ramamoorthy, 1984).  
16 Field studies have shown that there is virtually no accumulation of this metal through the food  
17 chain (Fuller and Averett, 1975). Studies by Kosalwat and Knight (1987) indicated that copper  
18 present in the substrate or sediment was significantly less toxic to chironomid species than  
19 overlying water column levels. The substrate copper concentration at which chironomid larval  
20 growth was reduced 50 percent ( $EC_{50}$ ) was 1,602 mg/kg. These researchers found that  
21 deformities in larval mouth parts were observed in elevated concentrations, and adult emergence  
22 was inhibited when the sediment concentration exceeded 1,800 mg/kg. Carins et al. (1984)  
23 reported copper toxicity in sediment for several chironomus midges and cladocerans with  $LC_{50}$   
24 values ranging from 681 to 2,296 mg/kg.

#### 25 26 **7.4.2.4 Nickel**

27 Nickel is a naturally occurring silvery metal that is found in the Earth's crust in the form of  
28 various nickel minerals. Exposure of organisms to nickel and its compounds results from  
29 breathing air, ingesting water and food that contain nickel and compounds, and skin contact with  
30 a medium contaminated with nickel (ATSDR, 1995).

31  
32 **Plants.** Nickel is not believed to be an essential element for plant growth; however, beneficial  
33 effects of nickel have been reported on the growth of legumes. Background concentrations of  
34 nickel in grasses and clovers collected in the United States averaged 0.13 and 1.5 mg/kg,

1 respectively (Kabata-Pendias and Pendias, 1992). The concentration of nickel in plants is  
2 positively correlated with nickel concentrations in soil.

3  
4 Concentrations of nickel in leaf tissue that are excessive or toxic to plant species range from 10  
5 to 100 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). A soil concentration of 30  
6 mg/kg has been proposed by Efraymson et al. (1997) as a benchmark screening value for nickel  
7 phytotoxicity. General symptoms of nickel toxicity in plants include the presence of interveinal  
8 chlorosis in new leaves, gray-green leaves, and brown and stunted root and plant growth. The  
9 uptake of nutrients and minerals, especially iron, can be substantially reduced as a consequence  
10 of nickel toxicity in plants (Kabata-Pendias and Pendias, 1992).

11  
12 **Mammals.** Nickel is a non-essential element for animal life. Nickel concentrations within the  
13 whole bodies of small mammals from uncontaminated sites were reported to range from 2.2 to  
14 6.2 mg/kg (dry weight) (Talmage and Walton, 1991). Highest concentrations were measured in  
15 the deer mouse (*Peromyscus maniculatus*). Highest tissue concentrations of nickel are usually  
16 found in the liver of mammals (Schroeder et al., 1964). Because nickel is poorly absorbed by the  
17 gastrointestinal tract, ingested nickel is generally not of great toxicological concern. Inhaled  
18 nickel, however, is relatively toxic. Rats fed nickel in their diet as nickel sulfate hexahydrate  
19 over three generations were studied for effects on reproduction. They were fed three dose levels  
20 (250, 500, and 1,000 ppm nickel) in their diet, and only the highest dose level caused reduced  
21 offspring body weights. No adverse effects were observed in the other dose levels. Because this  
22 study considered exposures over multiple generations, the 500 ppm dose was considered to be  
23 the chronic NOAEL and the 1,000 ppm dose was considered to be the chronic lowest-observed-  
24 adverse-effects-level (LOAEL) (EPA, 1999a). Based on toxicity data for rats exposed to nickel  
25 sulfate hexahydrate in their diets, a NOAEL of 40 mg/kg/day was derived (Ambrose et al.,  
26 1976). The endpoint for this study was reproduction.

27  
28 **Birds.** Mallard ducklings were fed nickel as nickel sulfate in their diet for a duration of 90 days  
29 to study the effects on mortality, growth, and behavior. They were fed three dose levels (176,  
30 774, and 1,069 ppm nickel), and only the highest dose reduced growth and resulted in 70 percent  
31 mortality. Because the study considered exposure over 90 days, the 774 ppm dose was  
32 considered to be the chronic NOAEL and the 1,069 dose was considered to be the chronic  
33 LOAEL (Cain and Pafford, 1981).

1 **Aquatic Life.** The bioavailability and toxicity of nickel to aquatic biota is influenced by the pH  
2 of the water (Schubauer-Berigan et al., 1993). The National Recommended Water Quality  
3 Criteria for the protection of aquatic life for acute and chronic exposures to nickel are 470 and 52  
4  $\mu\text{g/L}$ , respectively (EPA, 1999b). Background concentrations of nickel in adult anurans ranged  
5 between 0.9 and 2.9 mg/kg (dry weight) (Hall and Mulhern, 1984).

6  
7 The test  $\text{EC}_{20}$  for fish can be used as a benchmark indicative of production within a population.  
8 It is the highest tested concentration causing less than 20 percent reduction in either the weight of  
9 young fish per initial female fish in a life-cycle or partial life-cycle test, or the weight of young  
10 per egg in an early life-stage test. The  $\text{EC}_{20}$  value for nickel is 62  $\mu\text{g/L}$  (Suter and Tsao, 1996).  
11 A similar value can be determined for daphnids which represents the highest tested concentration  
12 causing less than 20 percent reduction in the product of growth, fecundity, and survivorship in a  
13 chronic test with a daphnid species. The  $\text{EC}_{20}$  benchmark for daphnids has been determined to be  
14 45  $\mu\text{g/L}$  (Suter and Tsao, 1996).

#### 15 16 **7.4.2.5 Selenium**

17 Selenium is distributed widely in nature and is found in most rocks and soils at concentrations  
18 between 0.1 and 2.0 mg/kg (Fishbein, 1981). The primary factor determining the fate of  
19 selenium in the environment is its oxidation state. Selenium is stable in four valence states (-2,  
20 0, +4, and +6) and forms chemical compounds similar to those of sulfur. The selenides (-2) are  
21 insoluble in water, as is elemental selenium. The inorganic alkali selenites (+4) and the selenates  
22 (+6) are soluble in water and are, therefore, more bioavailable.

23  
24 Conditions such as pH, Eh, and the presence of metal oxides affect the partitioning of the various  
25 compounds of selenium in the environment. In general, elemental selenium is stable in soils and  
26 is found at low levels in water because of its ability to co-precipitate with sediments. The  
27 soluble selenates are readily taken up by plants and converted to organic compounds such as  
28 selenomethionine, selenocysteine, dimethyl selenide, and dimethyl diselenide. Selenium is  
29 bioaccumulated by aquatic organisms and may also biomagnify in aquatic organisms.

30  
31 **Plants.** The role of selenium in plant growth is not fully understood. It is generally not  
32 considered essential in plant nutrition (Kabata-Pendias and Pendias, 1992). The concentration of  
33 selenium in plants has been shown to be positively correlated with the concentration of selenium  
34 in soil. Soil parameters such as pH, oxidation-reduction potential, and moisture content  
35 determine the amount of selenium available for plant uptake. Concentrations of selenium in leaf

1 tissues that have been shown to be toxic to various plant species range from 5 to 30 mg/kg  
2 (Kabata-Pendias and Pendias, 1992). General symptoms of selenium toxicity in plants include  
3 signs of interveinal chlorosis or black spots in plants containing approximately 4 mg/kg  
4 selenium, complete bleaching or yellowing of younger leaves at higher concentrations, and the  
5 presence of pinkish spots on roots (Kabata-Pendias and Pendias, 1992).

6  
7 **Mammals.** Selenium is an essential trace element for animal life. Concentrations that are  
8 essential to animals are in the range of 0.05 to 0.1 mg/kg in the diet (Arthur et al., 1992).  
9 According to Ganther (1974), selenium concentrations in healthy, unexposed laboratory animals  
10 and livestock range between 0.1 and 1 mg/kg. Selenium offers a protective effect against some  
11 carcinogens such as benzo(a)pyrene and benzo(a)anthracene (Hammond and Beliles, 1980).  
12 Selenium also functions as an antidote to the toxic effects of mercury, thallium, copper, arsenic  
13 and cadmium (Frost and Lish, 1975).

14  
15 Acute poisoning has been reported in livestock that consumed plant material containing 400 to  
16 800 mg/kg selenium (Eisler, 1985). Signs of acute poisoning in livestock include abnormal  
17 movements, lowered head, drooped ears, diarrhea, elevated temperature, rapid pulse, labored  
18 breathing, bloating with abdominal pain, increased urination, and dilated pupils (Eisler, 1985).  
19 Chronic poisoning may occur in animals exposed to dietary selenium concentrations between 1  
20 and 44 mg/kg (Eisler, 1985). Based on laboratory data for rats fed potassium selenate in their  
21 drinking water, a NOAEL of 0.2 mg/kg/day was derived (Rosenfeld and Beath, 1954). The  
22 endpoint for this study was reproduction.

23  
24 **Birds.** Toxicity from selenium has also been documented in birds. The major toxic effect of  
25 selenium on avian species is on reproductive success. Both sodium selenite and  
26 selenomethionine have been reported to be embryotoxic and teratogenic (Heinz et al., 1987).  
27 Reproductive impairment is likely to occur as concentrations of selenium approach 5 mg/kg.  
28 Mortality in mallard ducklings does not occur until selenium concentrations in the diet reach 40  
29 mg/kg. Based on toxicity data for mallard ducks fed sodium selenate in their diets over a period  
30 of 78 days, a NOAEL of 0.5 mg/kg/day was derived (Heinz et al., 1987).

31  
32 **Aquatic Life.** Selenium is an essential micro-nutrient for fish. Dietary requirements of  
33 selenium for fish range from 0.07 to 0.25 mg/kg, depending on the fish species (Gatlin and  
34 Wilson, 1984). The bioconcentration of selenium from water is highly dependent on the species  
35 of selenium present. Laboratory studies have shown bioconcentration factors for

1 selenomethionine to be greater than those for selenite and selenate. Bioconcentration factors for  
2 aquatic biota exposed to 1 µg/L selenomethionine were approximately 16,000 for algae, 200,000  
3 for daphnids, and 5,000 for bluegills (Besser et al., 1993).

4  
5 The EPA's National Recommended Water Quality Criteria for Priority Toxic Pollutants for  
6 selenium in freshwater is 5 µg/L for chronic exposure (EPA, 1999b). The toxicity of selenium to  
7 freshwater fish appears to be correlated more closely with dietary than waterborne exposure  
8 (Coyle et al., 1993). Sulfate concentrations in water may also influence the toxicity of selenium  
9 to aquatic invertebrates (Maier et al., 1993).

10  
11 The test EC<sub>20</sub> for fish can be used as a benchmark indicative of production within a population.  
12 It is the highest tested concentration causing less than 20 percent reduction in the weight of  
13 young fish per initial female fish in a life-cycle or partial life-cycle test, or the weight of young  
14 per egg in an early life-stage test. The EC<sub>20</sub> for selenium is 40 µg/L (Suter and Tsao, 1996). A  
15 similar value can be determined for daphnids, which reflects the highest tested concentration  
16 causing less than 20 percent reduction in the product of growth, fecundity, and survivorship in a  
17 chronic test with a daphnid species. The EC<sub>20</sub> benchmark for daphnids is 25 µg/L selenium  
18 (Suter and Tsao, 1996).

#### 19 20 **7.4.2.6 Zinc**

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

27  
28 **Plants.** Background concentrations of zinc in terrestrial plants range from 25 to 150 mg/kg (dry  
29 weight) (NAS, 1979). The deficiency content of zinc in plants is between 10 and 20 ppm (dry  
30 weight). Roots often contain the highest concentrations of zinc (Kabata-Pendias and Pendias,  
31 1992).

32  
33 Certain species of plants, particularly those from the families *Caryophyllaceae*, *Cyperaceae*, and  
34 *Plumbaginaceae*, and some tree species are extremely tolerant to elevated zinc concentrations  
35 (Kabata-Pendias and Pendias, 1992). Concentrations of zinc in these plants may reach 1 percent

1 (dry weight) in the plant. Concentrations in leaf tissue that are excessive or toxic to various plant  
2 species range from 100 to 400 mg/kg. Concentrations of 100 to 500 mg/kg are expected to result  
3 in a 10 percent loss in crop yield (Kabata-Pendias and Pendias, 1992). General symptoms of zinc  
4 toxicity in plants include the presence of chlorotic and necrotic leaf tips, interveinal chlorosis in  
5 new leaves, retarded growth of the entire plant, and injured roots that resemble barbed wire  
6 (Kabata-Pendias and Pendias, 1992).

7  
8 **Mammals.** Zinc is an essential trace element for normal fetal growth and development.  
9 However, exposure to high levels of zinc in the diet has been associated with reduced fetal  
10 weights, altered concentrations of fetal iron and copper, and reduced growth in offspring (Cox et  
11 al., 1969). Poisoning has been observed in ferrets and mink from chewing corroded galvanized  
12 cages (Clark et al., 1981). Symptoms of zinc toxicity are lassitude, slower tendon reflexes,  
13 bloody enteritis, diarrhea, lowered leukocyte count, depression of the central nervous system, and  
14 paralysis of the extremities (Venugopal and Luckey, 1978). A study by Kinnamon (1963)  
15 showed a NOAEL for oral exposure to a zinc compound over a period of 73 days to be 250  
16 mg/kg body weight; and mice given 500 mg/L of zinc, as zinc sulfate, in drinking water have  
17 shown hypertrophy of the adrenal cortex and pancreas. Young animals are much more  
18 susceptible to poisoning by zinc than are mature animals (Clark, et al., 1981).

19  
20 Animals are quite tolerant to high concentrations of zinc in the diet. Levels 100 times that  
21 required in the diet usually do not cause detectable symptoms of toxicosis (NAS, 1979).  
22 Laboratory data for rats fed zinc oxide in their diets during days one through sixteen of gestation  
23 were used to calculate a NOAEL of 160 mg/kg/day (Schlicker and Cox, 1968). The endpoint for  
24 this study was reproduction. Symptoms of zinc poisoning in mammals include lameness, acute  
25 diarrhea, and vomiting (Eisler, 1993).

26  
27 **Birds.** Dietary zinc concentrations of greater than 2,000 mg/kg diet are known to result in  
28 reduced growth of domestic poultry and wild birds (Eisler, 1993). Reduced survival has been  
29 documented at zinc concentrations greater than 3,000 mg/kg diet or at a single dose of greater  
30 than 742 mg/kg body weight (Eisler, 1993). Laboratory data from studies using zinc sulfate in  
31 the diets of white leghorn hens were used to calculate a NOAEL of 14.5 mg/kg/day (Stahl et al.,  
32 1990). This study was conducted over a 44-week duration, and the endpoint was reproduction.  
33 A value of 51 mg/L has been calculated as the NOAEL for chronic exposure of birds to zinc  
34 carbonate in drinking water (Sample et al., 1996).

35

1 **Aquatic Life.** Zinc residues in freshwater and marine fish are generally much lower than those  
2 found in algae and invertebrates. Thus, there is little evidence for accumulation (Moore and  
3 Ramamoorthy, 1984). Rainbow trout (*Oncorhynchus mykiss*) have the ability to detect and avoid  
4 areas of water containing 5.6 ppb zinc (Sprague, 1968). Cairns and Scheier (1968) reported 96-  
5 hour LC<sub>50</sub>s ranging from 10.13 to 12.5 ppm in hard water for bluegills (*Lepomis macrochirus*)  
6 and 96-hour LC<sub>50</sub>s ranging from 2.86 to 3.78 ppm in soft water. These results demonstrate that  
7 water hardness affects the toxicity of zinc to fish. Chronic toxicity tests have been conducted  
8 with five species of freshwater fish. Chronic values ranged from 47 µg/L for flagfish (*Jordanella*  
9 *floridae*) to 852 µg/L for brook trout (*Salvenius fontinalis*) (EPA, 1980).

10  
11 Acute toxicity to freshwater invertebrates is relatively low and, as with other metals, increasing  
12 water hardness decreases the toxicity of zinc (Moore and Ramamoorthy, 1984). As reported by  
13 Baudouin and Scoppa (1974), the 48-hour LC<sub>50</sub> for the cladoceran *Daphnia hyalina* was 0.055  
14 mg/L, and 5.5 mg/L for the copepod *Cyclops abyssorum*. Four chronic toxicity tests are reported  
15 for *Daphnia magna*, with chronic values ranging from 47 µg/L to 136 µg/L (EPA, 1980).

#### 16 17 **7.4.2.7 Polynuclear Aromatic Hydrocarbons**

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

26  
27 **Plants.** Some PAHs are synthesized by plants at very low concentrations (Sims and Overcash,  
28 1983). Background concentrations of specific PAH compounds usually range from 22 to 88  
29 micrograms per kilogram (µg/kg) in tree leaves, 48 to 66 µg/kg in cereal crop plants, 0.05 to 50  
30 µg/kg in leafy vegetables, 0.01 to 6 µg/kg in underground vegetables, and 0.02 to 0.04 µg/kg in  
31 fruits (Sims and Overcash, 1983). In general, PAH concentrations are usually greater in  
32 aboveground plant parts than in belowground parts and are greater on plant surfaces than within  
33 internal tissues (Eisler, 1987).

34

1 Lower-molecular-weight PAHs are taken up from soil by plants more readily than higher-  
2 molecular-weight PAHs (Eisler, 1987). Soil-to-plant concentration ratios for total PAHs have  
3 been reported to range from 0.001 to 0.183 (Talmage and Walton, 1991). Atmospheric  
4 deposition is believed to be the usual source of PAHs in plants, not uptake from soil (Sims and  
5 Overcash, 1983).

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

12  
13 **Mammals.** Most of the PAHs taken into the body are not accumulated but are oxidized, and the  
14 metabolites excreted (National Library of Medicine, 1996). In fact, most PAH compounds are  
15 detoxified and excreted from the body (Klaassen et al., 1991). PAHs are metabolized in  
16 vertebrates by a group of enzymes known as mixed-function oxidases in the liver. A few  
17 laboratory studies on rodents have revealed that acute oral toxicities of PAHs are greatest for  
18 benzo(a)pyrene, followed in decreasing order of toxicity by phenanthrene, naphthalene, and  
19 fluoranthene (Sims and Overcash, 1983). Based on laboratory data for mice exposed to  
20 benzo(a)pyrene through oral intubation, a NOAEL of 1.0 mg/kg/day was derived (MacKenzie  
21 and Angevine, 1981). The duration of this test was days 7 through 16 of gestation, and the  
22 endpoint was reproduction.

23  
24 Sims and Overcash (1983) have reported LC<sub>50</sub> values for rodents (*Rattus* spp. and *Mus* spp.) as  
25 50 mg/kg-day benzo(a)pyrene, 700 mg/kg-day phenanthrene, and 2,000 mg/kg-day fluoranthene.  
26 Sublethal effects manifested as decreased pup weight in mice have been reported at 10 mg/kg-  
27 day benzo(a)pyrene (MacKenzie and Angevine, 1981). Subchronic and chronic effects of  
28 exposure to PAHs in rats include liver and kidney damage, unspecified changes in peripheral  
29 blood pattern, body weight loss, genetic aberrations, and increased serum aminotransferase  
30 activity (Knobloch et al., 1969).

31  
32 **Birds.** Hoffman and Gay (1981) measured embryotoxicity of various PAHs applied externally  
33 to the surface of mallard duck eggs. Approximately 0.002 µg/egg of 7,12-  
34 dimethylbenz(a)anthracene (DMBA) caused 26 percent mortality in 18 days and, among the  
35 survivors, produced significant reduction in embryonic growth and a significant increase in the

1 percent of abnormalities, e.g., incomplete skeletal ossification, defects in eye, brain liver,  
2 feathers, and bill. At 0.1 µg DMBA/egg, only 10 percent survived to day 18.

3  
4 **Aquatic Life.** In general, PAHs as a group are not appreciably acutely toxic (Eisler, 1987;  
5 Neff, 1985). The toxicity of PAH compounds to fish is related to the solubility of the compound  
6 in water. The toxicity of PAHs to aquatic organisms is very species-specific and related to the  
7 organisms' ability to metabolize and excrete the compound (Eisler, 1987). For aquatic  
8 organisms, only PAHs in the molecular weight range from naphthalene to pyrene are considered  
9 acutely toxic. Toxicity in this group increases with increasing molecular weight. There is some  
10 evidence to suggest that PAHs are responsible for reproductive and teratogenic effects in eggs of  
11 the sand sole (*Psettichthys melanostictus*) exposed to 0.1 µg benzo(a)pyrene/L for five days. The  
12 eggs showed reduced and delayed hatch and, when compared to controls, produced larvae with  
13 high accumulations (2.1 mg/kg fresh weight) and gross abnormalities, such as tissue  
14 overgrowths, in 50 percent of the test larvae (Hose et al., 1982).

15  
16 Inhibited reproduction of daphnids and the delayed emergence of larval midges by fluorene was  
17 reported by Finger et al. (1985). When sediment PAH levels are elevated, benthic organisms  
18 obtain a majority of their PAHs from sediments through their ability to mobilize PAHs from the  
19 sediment/pore water matrix. The elevated levels in the tissues of these organisms could provide  
20 a significant source of PAHs to predatory fish. However, fish do have the ability to efficiently  
21 metabolize and degrade PAHs.

#### 22 23 **7.4.2.8 Endrin**

24 Endrin was first used as an insecticide, rodenticide, and avicide beginning in 1951 to control  
25 cutworms, voles, grasshoppers, borers, and other pests on cotton, sugarcane, tobacco, apple  
26 orchards, and grain (ATSDR, 1996). Endrin tends to persist in the environment mainly in forms  
27 sorbed to sediments and soil particles. Endrin is extremely persistent when released to soil. A  
28 conservative estimate of its half-life in sandy loam soils is approximately 14 years (Nash and  
29 Woolson, 1967).

30  
31 **Mammals.** Studies have demonstrated that the nervous system is the primary target for endrin  
32 toxicity (ATSDR, 1996). Exposure of animals to endrin causes central nervous system effects,  
33 particularly convulsions (Deichmann et al., 1970). Nonspecific degeneration of the liver, kidney,  
34 and brain was observed in animals exposed to lethal doses of endrin (Treon et al., 1955). Endrin  
35 can cause abnormal bone formation, hyperactivity, and death in fetuses of dams exposed during

1 gestation (Chernoff et al., 1979). Endrin appears to be well absorbed orally, and distribution is  
2 primarily to fat and skin (ATSDR, 1996).

3  
4 Rats, mice, and guinea pigs administered 4 mg/kg endrin and sacrificed 24 hours later exhibited  
5 moderate hepatic necrosis, fatty degeneration, and inflammation (Hassan et al., 1991).  
6 Neurological effects are commonly observed in animals exposed to endrin. Hyperirritability to  
7 stimuli, tremors, convulsions, and ataxia occurred in three species of animals (dog, rat, and  
8 rabbit) administered endrin for acute, intermediate, and chronic durations (Treon et al., 1955).

9  
10 Laboratory data from a study using mice fed endrin in their diets were used to calculate a  
11 NOAEL value of 0.092 mg/kg/day (Good and Ware, 1969). The duration of this study was 120  
12 days, and the endpoint was reproduction.

13  
14 **Birds.** Laboratory data for mallard ducks and screech owls fed endrin in their diets were used to  
15 estimate NOAELs of 0.3 and 0.01 mg/kg/day, respectively (Spann et al., 1986; Fleming et al.,  
16 1982). Both studies were chronic studies (greater than 200 days and greater than 83 days,  
17 respectively) during a critical lifestage, and the endpoint studied was reproduction.

18  
19 **Aquatic Life.** When released to water, endrin strongly adsorbs to sediment and bioconcentrates  
20 significantly in aquatic organisms (ATSDR, 1996). Endrin appears to be biomagnified only  
21 slightly through various levels of the food chain (Metcalf et al., 1973). Bioconcentration factors  
22 for endrin in aquatic organisms range from 80 for fathead minnows (*Pimephales promelas*) to  
23 49,000 for snails (*Physa sp.*) (ATSDR, 1996). The estimated half-life of endrin in water is more  
24 than 4 years (ATSDR, 1996). The National Recommended Water Quality Criteria for endrin in  
25 freshwater are 0.086 and 0.036 µg/L for acute and chronic exposures, respectively (EPA, 1999b).  
26 The Ontario Ministry of the Environment (1992) has identified a lowest effect level in sediment  
27 of 3 µg/kg and a severe effect level in sediment of 1,300 µg/kg (Persaud et al., 1993).

28  
29 Acute toxicity (LC<sub>50</sub>) for various freshwater fish species ranged from 0.5 µg/L for *Oncorhynchus*  
30 *kisutch* to 314 µg/L for mosquito fish (Verschuere, 1983). Acute toxicity (LC<sub>50</sub>) for various  
31 aquatic insects ranged from 0.03 µg/L for *Acroneturia pacifica* to 2.4 µg/L for *Pteronarcys*  
32 *californica*.

33  
34 Tissue residues as low as 0.0115 µg/g in largemouth bass (*Micopterus salmoides*) have been  
35 shown to reduce survival by 40 percent (Jarvinen and Ankley, 1999).

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### **7.4.3 Potential Receptors**

Potential ecological receptors at Parcel 66(7) are limited to terrestrial species, since there are no water bodies associated with Parcel 66(7). There are several major feeding guilds that could occur at Parcel 66(7) if the asphalt paving were to be removed at some time in the future, including herbivores, invertivores, omnivores, and carnivores. All of these feeding guilds have the potential to be directly exposed to surface soil at or near Parcel 66(7) via various activities (e.g., feeding, grooming, bathing). These feeding guilds may also be exposed to site-related chemicals via food web transfers. It is important to note that the entire area of Parcel 66(7) and the surrounding area are covered with asphalt paving. This asphalt paving effectively eliminates all of the surface soil exposure pathways for ecological receptors. Therefore, ecological exposure pathways are discussed in the following sections under the assumption that the asphalt paving will be removed or will deteriorate significantly in the future such that surface soil will be exposed.

Dermal absorption of PAHs from soil would be a potential pathway for all feeding guilds at Parcel 66(7) if the asphalt paving were removed; 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. Although VOCs were detected in surface soil at Parcel 66(7), they were found at very low concentrations (below ESVs); therefore, inhalation of volatiles is not a significant exposure pathway at Parcel 66(7). Inhalation of constituents sorbed to soil particles and inhaled as dust would be a potential pathway for all of the feeding guilds at Parcel 66(7) if the asphalt paving were removed.

#### **7.4.3.1 Herbivorous Feeding Guild**

The major route of exposure for herbivores is through ingestion of plants that may have accumulated contaminants from soil. Because the entire study area at Parcel 66(7) is covered with asphalt, very little vegetation is present at the site itself. The limited vegetation that does exist at the site consists of weed species that have grown through cracks and other areas where the asphalt cover has been compromised. Since terrestrial herbivores by definition are grazers and browsers, they could be exposed to chemicals that have accumulated in the vegetative tissues of the weeds at the site, although these exposure are expected to be minimal due to the asphalt cover over the entire site. Terrestrial herbivores may also be exposed to site-related chemicals in

1 soil through incidental ingestion of soil while grazing, grooming, or other activities. However,  
2 the asphalt cover will minimize these potential exposure routes. If the asphalt were removed, the  
3 potential for these exposures would increase.

4  
5 Typical herbivorous species that could occur at Parcel 66(7) if the paving and perimeter fencing  
6 were removed, and ones that are commonly used as sentinel species in ecological risk  
7 assessment, include eastern cottontail (*Sylvilagus floridanus*), eastern gray squirrel (*Sciurus*  
8 *carolinensis*), pine vole (*Pitymys pinetorum*), whitetail deer (*Odocoileus virginianus*), and wild  
9 turkey (*Meleagris gallopavo*).

#### 10 11 **7.4.3.2 Invertivorous Feeding Guild**

12 Invertivores specialize in eating insects and other invertebrates. As such, they may be exposed to  
13 site-related chemicals that have accumulated in insects and other invertebrates. Invertivores may  
14 also be exposed to site-related chemicals in soil through incidental ingestion of soil while  
15 probing for insects, grooming, or other activities. Ingestion of soil while feeding is a potential  
16 exposure pathway for invertivores, since much of their food (i.e., earthworms and other  
17 invertebrates) lives on or below the soil surface. These exposure pathways are expected to be  
18 minimal because the site is currently covered with asphalt. If the asphalt were to be removed at  
19 some time in the future, these exposures would be more significant.

20  
21 Typical invertivorous species that could occur at Parcel 66(7) if the paving and perimeter fencing  
22 were removed, and ones that are commonly used as sentinel species in ecological risk  
23 assessment, include American woodcock (*Philohela minor*), carolina wren (*Thryothorus*  
24 *ludovicianus*), shorttail shrew (*Blarina brevicauda*), and eastern mole (*Scalopus aquaticus*).

#### 25 26 **7.4.3.3 Omnivorous Feeding Guild**

27 Omnivores consume both plant and animal material in their diet, depending upon availability.  
28 Therefore, they could be exposed to chemicals that have accumulated in the vegetative tissues of  
29 plants at the site and also chemicals that may have accumulated in smaller animal tissues that the  
30 omnivores prey upon. These soil-related exposure pathways are expected to be minimal at Parcel  
31 66(7) because the entire site is covered with asphalt. If the asphalt were to be removed, the  
32 potential for these exposure pathways to be complete would increase.

33  
34 Typical omnivorous species that could occur at Parcel 66(7) if the paving and perimeter fencing  
35 were removed, and ones that are commonly used as sentinel species in ecological risk

1 assessment, include red fox (*Vulpes vulpes*), white-footed mouse (*Peromyscus leucopus*), and  
2 American robin (*Turdus migratorius*).

#### 3 4 **7.4.3.4 Carnivorous Feeding Guild**

5 Carnivores are meat-eating animals and are, therefore, potentially exposed to site-related  
6 chemicals through consumption of prey animals that may have accumulated contaminants in  
7 their tissues. Carnivores are quite often top predators in a local food web and are often subject to  
8 exposure to contaminants that have bioaccumulated in lower trophic-level organisms or  
9 biomagnified through the food web. Food web exposures for carnivores are based on the  
10 consumption of prey animals that have accumulated constituents of potential ecological concern  
11 (COPEC) from various means. Smaller herbivores, omnivores, invertivores, and other  
12 carnivores may consume soil, plant, and animal material as food and accumulate COPECs in  
13 their tissues. Subsequent ingestion of these prey animals by carnivorous animals could expose  
14 them to COPECs. These soil-related exposure pathways are expected to be minimal at Parcel  
15 66(7) because the entire site is covered with asphalt. If the asphalt were to be removed, the  
16 potential for these exposure pathways to be complete would increase.

17  
18 Most inorganic compounds are not accumulated in animal tissues to any great extent (Shugart,  
19 1991; U.S. Army Environmental Hygiene Agency, 1994). Therefore, food web exposures to  
20 these chemicals are expected to be minimal. PAHs have the potential to accumulate in lower  
21 trophic level organisms but not in higher trophic level organisms because they have mechanisms  
22 for metabolizing and excreting this class of compounds.

23  
24 Typical carnivorous species that could occur at Parcel 66(7) if the paving and perimeter fencing  
25 were removed, and ones that are commonly used as sentinel species in ecological risk  
26 assessment, include red-tailed hawk (*Buteo jamaicensis*), black vulture (*Coragyps atratus*), and  
27 bobcat (*Lynx rufus*).

#### 28 29 **7.4.3.5 Threatened and Endangered Species**

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

- 32
- 33 • Gray Bat (*Myotis grisescens*)
- 34 • Blue Shiner (*Cyprinella caerulea*)
- 35 • Mohr's Barbara Buttons (*Marshallia mohrii*)
- 36 • Tennessee Yellow-Eyed Grass (*Xyris tennesseensis*).

1  
2 None of these species occur at Parcel 66(7) or in the immediate vicinity.

3  
4 **7.4.4 Complete Exposure Pathways**

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

- 7
- 8 • A source mechanism for contaminant release
  - 9 • A transport mechanism
  - 10 • A point of environmental contact
  - 11 • A route of uptake at the exposure point (EPA, 1989).
- 12

13 If any of these four components is absent, then a pathway is generally considered incomplete.  
14 Potentially complete exposure pathways are depicted in the SCM, as shown on Figure 7-1. It is  
15 important to note that the entire study area of Parcel 66(7) is currently covered by asphalt. The  
16 potential exposure pathways depicted in the SCM and discussed in this risk assessment assume  
17 that the asphalt will be removed or will degrade at some time in the future to a point where it no  
18 longer provides complete cover over the site.

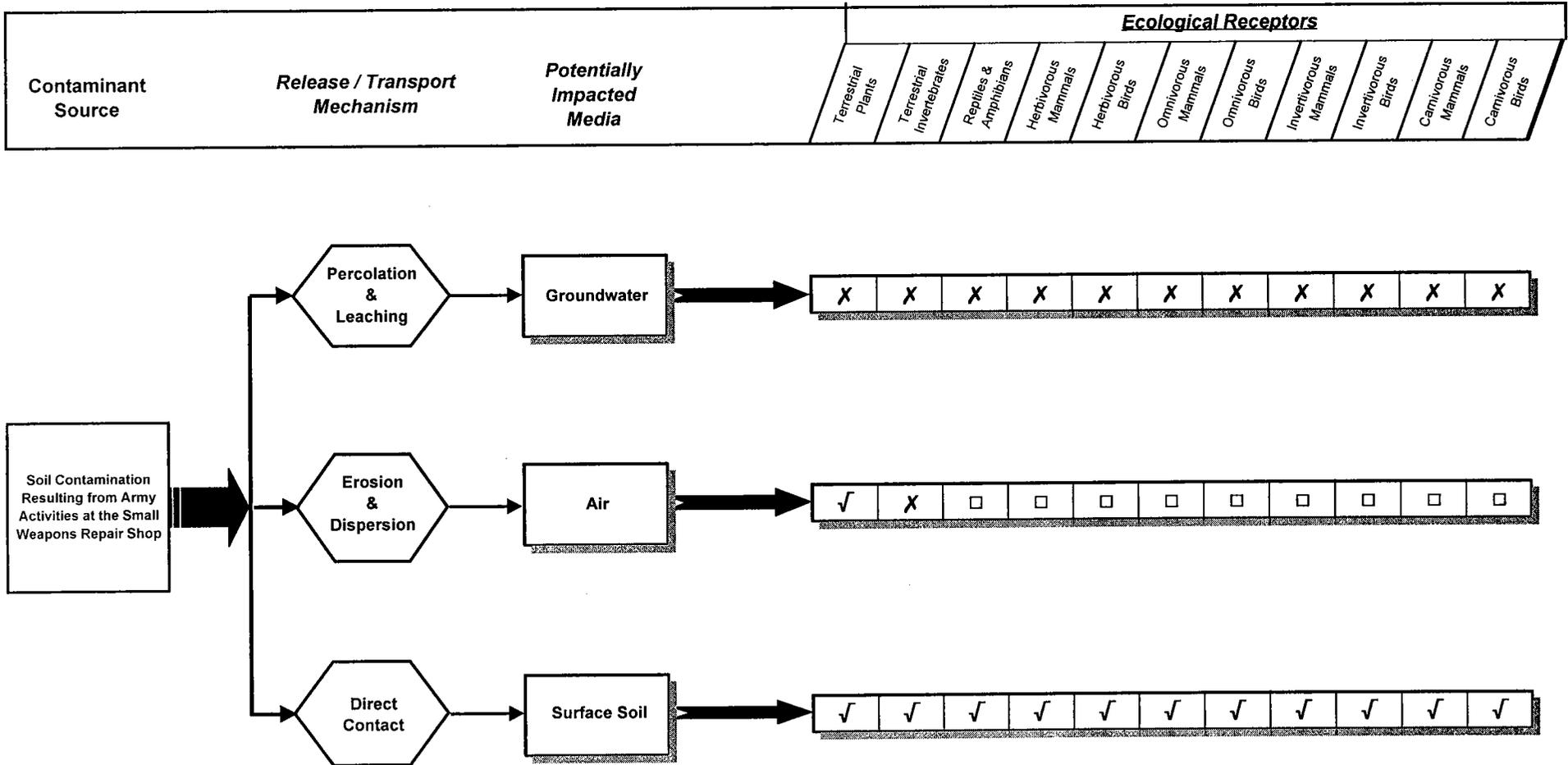
19  
20 Ecological receptors could be exposed to constituents in soils via direct and/or secondary  
21 exposure pathways. Direct exposure pathways include soil ingestion, dermal absorption, and  
22 inhalation of COPECs adsorbed to fugitive dust. Significant exposure via dermal contact is  
23 limited to organic constituents that are lipophilic and can penetrate epidermal barriers. Mammals  
24 are less susceptible to exposure via dermal contact with soils because their fur prevents skin from  
25 coming into direct contact with soil. However, soil ingestion may occur while grooming,  
26 preening, burrowing, or consuming plants, insects, or invertebrates resident in soil. Exposure via  
27 inhalation of fugitive dust is limited to contaminants present in surface soils at areas that are  
28 devoid of vegetation. The inherent moisture content of the soil and the frequency of soil  
29 disturbance also play important roles in the amount of fugitive dust generated at a particular site.

30  
31 While constituents in soils may leach into groundwater, environmental receptors will not come  
32 into direct contact with constituents in groundwater, since there is no direct exposure route. The  
33 only potential exposure pathways for ecological receptors to groundwater would be via surface  
34 water exposure routes. Since there are no surface water bodies in the vicinity of Parcel 66(7),  
35 exposure routes to groundwater are incomplete for ecological receptors.

36

Figure 7-1

Site Conceptual Model  
Former Small Weapons Repair Shop, Parcel 66(7)  
Fort McClellan, Calhoun County, Alabama



Key To Potential Exposure Routes

- √ - Potentially complete exposure pathway
- X - Incomplete exposure pathway
- - Potentially complete exposure pathway but insignificant
- NA - Not applicable

1 Secondary exposure pathways involve constituents that are transferred through different trophic  
2 levels of the food chain and may be bioaccumulated and/or bioconcentrated. This may include  
3 constituents bioaccumulated from soil into plant tissues or into terrestrial species ingesting soils.  
4 These plants or animals may, in turn, be consumed by animals at higher trophic levels. In  
5 general, the constituents detected in surface soil at Parcel 66(7) may bioaccumulate in lower  
6 trophic level organisms (e.g., terrestrial invertebrates may bioaccumulate inorganic compounds  
7 and PAHs detected in soil); however, they will not bioconcentrate through the food chain.  
8 Inorganic compounds generally do not bioconcentrate to any great extent, and PAHs are readily  
9 metabolized by higher trophic level organisms. The only compound detected in surface soil that  
10 has a propensity to bioconcentrate is endrin. These surface soil exposure pathways are  
11 potentially complete only if the asphalt paving were to be removed from the site. If the asphalt  
12 paving remains in place, these surface soil exposure pathways would be incomplete.

13  
14 Summaries of the potentially complete exposure pathways for terrestrial ecosystems at Parcel  
15 66(7) are presented in Table 7-1.

## 16 17 **7.5 Screening-Level Risk Estimation**

18 A screening-level estimation of potential risk can be accomplished by comparing the exposure  
19 point concentration of each detected constituent in each environmental medium to a  
20 corresponding screening-level ecological toxicity value. In order to conduct the SLERA, the  
21 following steps must be followed:

- 22
- 23 • Determine appropriate screening assessment endpoints
  - 24
  - 25 • Determine the ecological toxicity values that are protective of the selected
  - 26 assessment endpoints
  - 27
  - 28 • Determine the exposure point concentrations of constituents detected at the site
  - 29
  - 30 • Calculate screening-level hazard quotients.
  - 31

32 These steps are summarized below.

### 33 34 **7.5.1 Ecological Screening Assessment Endpoints**

35 Most ecological risk assessments focus on population measures as endpoints, since population  
36 responses are more well defined and predictable than are community or ecosystem responses.  
37 For screening-level assessments such as this SLERA, an assessment endpoint is any adverse

**Table 7-1**

**Feeding Guilds and Potential Exposure Pathways for Terrestrial Ecosystems  
Small Weapons Repair Shop, Parcel 66(7)  
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 terrestrial plants
	Herbivorous Mammals	Ingestion of soil Ingestion of terrestrial plants
3	Omnivorous Birds	Ingestion of soil Ingestion of terrestrial plants Ingestion of terrestrial invertebrates
	Omnivorous Mammals	Ingestion of soil Ingestion of terrestrial plants Ingestion of terrestrial invertebrates Ingestion of prey
	Invertivorous Birds	Ingestion of soil Ingestion of terrestrial invertebrates
	Invertivorous Mammals	Ingestion of soil Ingestion of terrestrial invertebrates
4	Carnivorous Birds (raptors)	Ingestion of soil Ingestion of prey
	Carnivorous Mammals	Ingestion of soil Ingestion of prey

1 effect on ecological receptors, where receptors are plant and animal populations and  
2 communities, habitats, and sensitive environments.

3  
4 Adverse effects on populations can be inferred from measures related to impaired reproduction,  
5 growth, and survival. Adverse effects on communities can be inferred from changes in  
6 community structure or function. Adverse effects on habitats can be inferred from changes in  
7 composition and characteristics that reduce the ability of the habitat to support plant and animal  
8 populations and communities.

9  
10 Due to the nature of the SLERA process, most of the screening assessment endpoints are generic  
11 in nature (e.g., protection of sediment benthic communities from adverse changes in structure or  
12 function).

13  
14 The assessment endpoints for this SLERA were identified for soil and are summarized below:

15  
16 • **Soil**

- 17 - Protection of the terrestrial invertebrate community from adverse changes in  
18 structure and function
- 19  
20 - Protection of the terrestrial plant community from adverse changes in structure  
21 and function.

22  
23 **7.5.2 Ecological Screening Values**

24 The ecological screening values (ESV) used in this assessment represent the most conservative  
25 values available from various literature sources and have been selected to be protective of the  
26 assessment endpoints described above. These ESVs have been developed specifically for FTMC  
27 in conjunction with EPA Region IV and are presented in the *Final Human Health and Ecological*  
28 *Screening Values and PAH Background Summary Report* (IT, 2000c). The ESVs used in this  
29 assessment are based on NOAELs when available. If a NOAEL-based ESV was not available  
30 for a certain COPEC, then the most health-protective value available from the scientific literature  
31 was used in this assessment.

32  
33 A hierarchy has been developed which presents an orderly method for selection of ESVs for  
34 surface soil at Parcel 66(7). This hierarchy is as follows:

- EPA Region IV constituent-specific ecological screening values
- EPA Region IV ecological screening values for general class of constituents
- EPA Region V ecological data quality levels
- EPA Region III Biological Technical Advisory Group values
- Ecological screening values from Talmage et al., 1999.

### **7.5.3 Determination of Exposure Point Concentrations**

Exposure point concentrations represent the chemical concentrations in environmental media that a receptor may contact. Since the exposure point concentration is a value that represents the most likely concentration to which receptors could be exposed, a value that reflects the central tendency of the data set is most appropriate to use. However, at the screening-level stage, the data sets are generally not robust enough for statistical analysis and the level of conservatism in the exposure estimates is high to account for uncertainties. Therefore, in the screening-level stage, the maximum detected constituent concentration in each environmental medium is used as the exposure point concentration. The use of the maximum detected constituent concentration as the exposure point concentration ensures that the exposures will not be underestimated, and therefore, constituents will not be inadvertently eliminated from further assessment.

The statistical summary (including the exposure point concentrations) for surface soil at Parcel 66(7) is presented in Table 7-2.

### **7.5.4 Screening-Level Hazard Quotients**

In order to estimate whether constituents detected in environmental media at the site have the potential to pose adverse ecological risks, screening-level hazard quotients were developed. The screening-level hazard quotients were developed via a three-step process as follows:

- Comparison to ESVs
- Identification of essential macro-nutrients
- Comparison to naturally occurring background concentrations.

Constituents detected in surface soil at Parcel 66(7) were evaluated against the ESVs by calculating a screening-level hazard quotient ( $HQ_{screen}$ ) for each constituent in each environmental medium. An  $HQ_{screen}$  was calculated by dividing the maximum detected constituent concentration in each environmental medium by its corresponding ESV, as follows:

$$HQ_{screen} = \frac{MDCC}{ESV}$$

Table 7-2

**Constituents of Potential Ecological Concern in Surface Soil<sup>a</sup>**  
**Small Weapons Repair Shop, Parcel 66(7)**  
**Fort McClellan, Calhoun County, Alabama**

(Page 1 of 2)

Constituents	Background Threshold Value <sup>b</sup> (mg/kg)	Ecological Screening Value <sup>c</sup> (mg/kg)	Maximum Detected Conc. (mg/kg)	Minimum Detected Conc. (mg/kg)	Mean Detected Conc. (mg/kg)	Frequency of Detection	Maximum Hazard Quotient	Mean Hazard Quotient	Constituent Of Potential Ecological Concern <sup>d</sup>
<b>Metals</b>									
Aluminum	1.63E+04	5.00E+01	1.18E+04	6.34E+03	9.95E+03	3 / 3	236.00	198.93	3
Arsenic	1.37E+01	1.00E+01	6.30E+00	5.00E+00	5.65E+00	2 / 2	0.63	0.57	1,3
Barium	1.24E+02	1.65E+02	1.01E+02	4.65E+01	7.09E+01	3 / 3	0.61	0.43	3
Beryllium	8.00E-01	1.10E+00	1.20E+00	4.60E-01	8.23E-01	3 / 3	1.09	0.75	YES
Calcium	1.72E+03	NA	2.46E+04	3.71E+02	9.39E+03	3 / 3	ND	ND	2
Chromium	3.70E+01	4.00E-01	1.71E+01	7.60E+00	1.38E+01	3 / 3	42.75	34.50	3
Cobalt	1.52E+01	2.00E+01	2.39E+01	1.30E+00	9.67E+00	3 / 3	1.20	0.48	YES
Copper	1.27E+01	4.00E+01	4.51E+01	6.50E+00	2.41E+01	3 / 3	1.13	0.60	YES
Iron	3.42E+04	2.00E+02	3.05E+04	4.37E+03	2.08E+04	3 / 3	152.50	103.95	2,3
Lead	4.01E+01	5.00E+01	2.60E+01	1.08E+01	1.61E+01	3 / 3	0.52	0.32	1,3
Magnesium	1.03E+03	4.40E+05	7.90E+03	1.13E+03	5.02E+03	3 / 3	0.018	0.011	1,2
Manganese	1.58E+03	1.00E+02	3.63E+02	1.54E+01	1.96E+02	3 / 3	3.63	1.96	3
Mercury	8.00E-02	1.00E-01	4.10E-02	1.70E-02	2.80E-02	3 / 3	0.41	0.28	1,3
Nickel	1.03E+01	3.00E+01	4.58E+01	3.30E+00	1.90E+01	3 / 3	1.53	0.63	YES
Potassium	8.00E+02	NA	5.31E+02	4.04E+02	4.64E+02	3 / 3	ND	ND	2,3
Selenium	4.80E-01	8.10E-01	1.60E+00	1.40E+00	1.09E+00	2 / 3	1.98	1.35	YES
Vanadium	5.88E+01	2.00E+00	2.87E+01	9.40E+00	2.09E+01	3 / 3	14.35	10.43	3
Zinc	4.06E+01	5.00E+01	1.00E+02	1.61E+01	4.59E+01	3 / 3	2.00	0.92	YES
<b>Semivolatile Organic Compounds</b>									
Anthracene	NA	1.00E-01	7.90E-02	7.90E-02	1.63E-01	1 / 3	0.79	1.63	1
Benzo(a)anthracene	NA	5.21E+00	7.30E-01	4.10E-02	3.30E-01	2 / 3	0.14	0.06	1
Benzo(a)pyrene	NA	1.00E-01	1.60E+00	1.60E+00	6.70E-01	1 / 3	16.00	6.70	YES
Benzo(b)fluoranthene	NA	5.98E+01	2.10E+00	6.70E-02	7.96E-01	2 / 3	0.035	0.013	1
Benzo(ghi)perylene	NA	1.19E+02	1.10E+00	1.10E+00	5.03E-01	1 / 3	0.0092	0.0042	1
Benzo(k)fluoranthene	NA	1.48E+02	6.10E-01	6.10E-01	3.40E-01	1 / 3	0.0041	0.0023	1
Chrysene	NA	4.73E+00	9.40E-01	4.00E-02	4.00E-01	2 / 3	0.199	0.085	1
Dibenz(a,h)anthracene	NA	1.84E+01	2.70E-01	2.70E-01	2.27E-01	1 / 3	0.015	0.012	1
Fluoranthene	NA	1.00E-01	1.10E+00	8.10E-02	4.67E-01	2 / 3	11.00	4.67	1
Indeno(1,2,3-cd)pyrene	NA	1.09E+02	1.20E+00	1.20E+00	5.37E-01	1 / 3	0.011	0.005	1
Phenanthrene	NA	1.00E-01	1.80E-01	1.80E-01	1.97E-01	1 / 3	1.80	1.97	YES
Pyrene	NA	1.00E-01	1.10E+00	6.50E-02	4.62E-01	2 / 3	11.00	4.62	YES

Table 7-2

**Constituents of Potential Ecological Concern in Surface Soil<sup>a</sup>**  
**Small Weapons Repair Shop, Parcel 66(7)**  
**Fort McClellan, Calhoun County, Alabama**

(Page 2 of 2)

Constituents	Background Threshold Value <sup>b</sup> (mg/kg)	Ecological Screening Value <sup>c</sup> (mg/kg)	Maximum Detected Conc. (mg/kg)	Minimum Detected Conc. (mg/kg)	Mean Detected Conc. (mg/kg)	Frequency of Detection	Maximum Hazard Quotient	Mean Hazard Quotient	Constituent Of Potential Ecological Concern <sup>d</sup>
<b><u>Volatile Organic Compounds</u></b>									
2-Butanone	NA	8.96E+01	5.80E-03	5.80E-03	5.80E-03	1 / 1	0.000065	0.000065	1
Acetone	NA	2.50E+00	1.50E-01	1.50E-01	1.50E-01	1 / 1	0.060	0.060	1
Carbon disulfide	NA	9.40E-02	1.70E-03	1.70E-03	2.63E-03	1 / 3	0.018	0.028	1
Toluene	NA	5.00E-02	2.00E-03	2.00E-03	2.73E-03	1 / 3	0.040	0.055	1
<b><u>Chlorinated Pesticides</u></b>									
Aldrin	NA	2.50E-03	1.00E-03	1.00E-03	2.85E-03	1 / 3	0.40	1.14	1
Endrin	NA	1.00E-03	5.20E-03	5.20E-03	3.08E-03	1 / 3	5.20	3.08	<b>YES</b>
Methoxychlor	NA	1.99E-02	1.20E-02	1.20E-02	6.57E-03	1 / 3	0.60	0.33	1

<sup>a</sup> Surface soil at Parcel 66(7) is defined as the interval from 0 to 0.5 feet below ground surface.

<sup>b</sup> Background threshold value is 2-times the arithmetic mean background concentration as reported in *Final Background Metals Survey Report, Fort McClellan, Alabama* (SAIC, 1998).

<sup>c</sup> Ecological screening values are presented in *Human Health and Ecological Screening Values and PAH Background Summary Report* (IT, 2000).

<sup>d</sup> Rationale for exclusion as COPEC:

- 1 - Maximum detected concentration is less than ecological screening value (ESV).
- 2 - Essential macronutrient, only toxic at extremely high concentrations (i.e. 10-times naturally-occurring background concentration).
- 3 - Maximum detected concentration is less than background threshold value.
- 4 - No ESV available; however, maximum detected concentration is similar to concentrations of other VOAs which are below their respective ESVs.

mg/kg - Milligrams per kilogram.

NA - Not available.

ND - Not determined.

1  
2 where:

3  
4  $HQ_{screen}$  = screening-level hazard quotient  
5  $MDCC$  = maximum detected constituent concentration  
6  $ESV$  = ecological screening value.  
7

8 A calculated  $HQ_{screen}$  value of one indicated that the MDCC was equal to the chemical's  
9 conservative ESV and was interpreted in this assessment as a constituent that does not pose the  
10 potential for adverse ecological risk. An  $HQ_{screen}$  value less than one indicated that the MDCC  
11 was less than the conservative ESV and that the chemical is not likely to pose adverse ecological  
12 hazards to most receptors. Conversely, an  $HQ_{screen}$  value greater than one indicated that the  
13 MDCC was greater than the ESV and that the chemical might pose adverse ecological hazards to  
14 one or more receptors.  
15

16 In order to better understand the potential risks posed by chemical constituents at Parcel 66(7), a  
17 mean hazard quotient was also calculated by comparing the arithmetic mean constituent  
18 concentrations in surface soil to the corresponding ESVs. The calculated screening-level hazard  
19 quotients for surface soil at Parcel 66(7) are presented in Table 7-2.  
20

21 The EPA recognizes several constituents in abiotic media that are necessary to maintain normal  
22 function in many organisms. These essential macronutrients are iron, magnesium, calcium,  
23 potassium, and sodium (EPA, 1989). Most organisms have mechanisms designed to regulate  
24 nutrient fluxes within their systems; therefore, these nutrients are generally toxic only at very  
25 high concentrations. Although iron is an essential nutrient and is regulated within many  
26 organisms, it may become increasingly bioavailable at lower pH values, thus increasing its  
27 potential to elicit adverse affects. Therefore, iron was not evaluated as an essential nutrient in  
28 this SLERA. Essential macronutrients were considered COPECs only if they were present in site  
29 samples at concentrations ten times the naturally occurring background concentration.  
30

31 A study of the natural geochemical composition associated with FTMC (SAIC, 1998) determined  
32 the mean concentrations of 24 metals in surface soil, surface water, and sediment samples  
33 collected from presumably unimpacted areas. Per agreement with EPA Region IV, the  
34 background threshold value (BTV) for each metal was calculated as two times the mean  
35 background concentration for that metal. The BTV for each metal was used to represent the

1 upper boundary of the range of natural background concentrations expected at FTMC and was  
2 used as the basis for evaluating metal concentrations measured in site samples.

3  
4 In order to determine whether metals detected in site samples were the result of site-related  
5 activities or were indicative of naturally occurring conditions, the maximum metal concentrations  
6 measured in site samples were compared to their corresponding BTVs. Site sample metal  
7 concentrations less than or equal to the corresponding BTV represent the natural geochemical  
8 composition of media at FTMC, and not contamination associated with site activity. Site sample  
9 metal concentrations greater than the corresponding BTV represent contaminants that may be the  
10 result of site-related activities and require further assessment.

11  
12 Thus, the first step in determining screening-level hazard quotients was a comparison of  
13 maximum detected constituent concentrations to appropriate ESVs. Constituents with  $HQ_{screen}$   
14 values less than one were considered to pose insignificant ecological risk and were eliminated  
15 from further consideration. Constituents with  $HQ_{screen}$  values greater than one were eliminated  
16 from further consideration if they were macronutrients. Those constituents that had  $HQ_{screen}$   
17 values greater one and were not considered macronutrients were then compared to their  
18 corresponding BTVs. If constituent concentrations were determined to be less than their  
19 naturally occurring background concentration, then a risk management decision could result in  
20 eliminating these constituents from further assessment.

## 21 22 **7.6 Identification of Constituents of Potential Ecological Concern**

23 Constituents were identified as COPECs if any of the following conditions were met:

- 24  
25 • The maximum detected constituent concentration exceeded the ESV, or
- 26  
27 • The maximum detected constituent concentration was 10-times the BTV if the  
28 constituent was identified as a macro-nutrient, or
- 29  
30 • The maximum detected constituent concentration exceeded the BTV for  
31 inorganics.

32  
33 If a constituent did not meet one of these conditions, then it was not considered a COPEC at  
34 Parcel 66(7) and was not considered for further assessment. If a constituent met one of these  
35 conditions, then it was considered a COPEC. Identification of a constituent as a COPEC  
36 indicates that further assessment of that particular constituent in a given environmental medium

1 may be appropriate. It does not imply that a particular constituent poses risk to ecological  
2 receptors.

3  
4 The COPECs that have been identified for surface soil at Parcel 66(7) are presented in Table 7-2  
5 and summarized in Table 7-3.

6  
7 Inorganic constituents that were initially identified as COPECs in surface soil at Parcel 66(7)  
8 include beryllium, cobalt, copper, nickel, selenium, and zinc. The organic compounds that were  
9 identified as COPECs include benzo(a)pyrene, fluoranthene, phenanthrene, pyrene, and endrin.  
10 In order to more closely scrutinize the relationship between site-related chemicals and naturally  
11 occurring soil constituents, an integrated statistical and geochemical evaluation was conducted.  
12 Details regarding the specific methodologies used in this evaluation are presented in Appendix  
13 K. The results of the additional statistical and geochemical evaluation indicated that beryllium,  
14 cobalt, copper, nickel, selenium, and zinc were all associated with iron oxides in the soil at  
15 relatively constant ratios. These results indicate that these inorganic constituents of soil are  
16 naturally occurring and may not be the result of site-related activities. Thus, these inorganic  
17 constituents may not be considered COPECs in soil at Parcel 66(7).

18

### 19 **7.7 Uncertainty Analysis**

20 Uncertainties are inherent in any risk assessment, and even more so in a SLERA due to the  
21 nature of the assessment process and the assumptions used in the process. A number of the  
22 major areas of uncertainty in this assessment are presented below.

23

24 A significant level of uncertainty is introduced into this SLERA due to the presence of asphalt  
25 over the entire surface area of Parcel 66(7) and adjacent parking area. The asphalt that is  
26 currently present precludes surface soil exposures for all ecological receptors. However, for this  
27 SLERA, it was assumed that the asphalt would be removed or would degrade significantly over  
28 time, such that soil exposures could occur at some time in the future. This assumption greatly  
29 overestimates the potential for ecological exposures currently occurring at the site. A chain-link  
30 fence also surrounds the perimeter of the site, precluding larger animals from entering the site. It  
31 was assumed for this SLERA that the chain-link fence would be removed at some time in the  
32 future. This assumption also overestimates the potential for ecological exposures currently  
33 occurring at the site.

34

**Table 7-3**

**Summary of COPECs in Surface Soil  
Small Weapons Repair Shop, Parcel 66(7)  
Fort McClellan, Calhoun County, Alabama**

Beryllium  
Cobalt  
Copper  
Nickel  
Selenium  
Zinc  
Benzo(a)pyrene <sup>1</sup>  
Fluoranthene <sup>1</sup>  
Phenanthrene <sup>1</sup>  
Pyrene <sup>1</sup>  
Endrin

---

<sup>1</sup> Maximum detected concentration in surface soil is less than the background threshold value for soil beneath asphalt at FTMC (IT, 2000).

1 An area of uncertainty that is inherent in a SLERA is the use of the maximum detected  
2 constituent concentration as the exposure point concentration for all receptors in a given medium.  
3 Most receptors have a home range large enough that precludes individuals from being exposed to  
4 the maximum constituent concentration for their entire lifetimes. Therefore, the actual exposure  
5 point concentration of a given constituent for many receptor species would be less than the  
6 maximum detected concentration. The use of the maximum detected constituent concentrations  
7 as the exposure point concentrations for all receptors may result in an overestimation of exposure  
8 for many receptors.

9  
10 Additionally, there is no consideration given to the bioavailability of COPECs to different  
11 organisms. In this SLERA, it is assumed that all constituents are 100 percent bioavailable to all  
12 receptor organisms. It is known that many constituents (particularly inorganic compounds) have  
13 significantly lower bioavailabilities (e.g., 1 to 10 percent for some inorganics in soil) than the  
14 100 percent that was assumed in this assessment. This assumption has the potential to greatly  
15 overestimate exposures to certain COPECs.

16  
17 The ESVs used in this assessment are all the most conservative values from the scientific  
18 literature, and many are based on the most sensitive endpoint (NOAEL values) for the most  
19 sensitive species tested. A less sensitive endpoint that is still protective of the ecological  
20 populations or communities of interest may be the LOAEL or some other endpoint. The use of  
21 NOAEL-based ESVs may overestimate potential for risks from certain COPECs.

22  
23 Another area of uncertainty is the lack of consideration of synergisms and/or antagonisms  
24 between COPECs. Although it is widely accepted that synergisms and antagonisms occur  
25 between certain constituents under certain conditions, the SLERA process does not provide  
26 methods for assessing these potential synergisms/antagonisms.

## 27 28 **7.8 Summary and Conclusions**

29 The potential for ecological risks at Parcel 66(7) was determined through a SLERA. This  
30 ecological screening process consisted of a characterization of the ecological setting at Parcel  
31 66(7), development of an SCM, a description of the fate and transport of constituents detected in  
32 various environmental media, a description of the ecotoxicity of the various constituents detected  
33 at Parcel 66(7), a description of the ecological receptors, a description of the complete exposure  
34 pathways, calculation of screening-level hazard quotients, and a description of the uncertainties  
35 within the process.

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**7.8.1 SLERA Summary**

The SLERA at Parcel 66(7) determined that the MDCs of several inorganic constituents, several SVOCs, and one chlorinated pesticide in surface soil exceeded their respective ESVs. Further review of these COPECs is important in making risk management decisions.

The inorganic constituents whose MDCs exceeded their respective ESVs in surface soil were beryllium, cobalt, copper, nickel, selenium, and zinc. These six constituents exhibited maximum hazard quotients that ranged from 1.09 to 2.0. Mean concentrations of all of the inorganic constituents were less than their respective ESVs, except for selenium, indicating that these inorganic COPECs may be indicative of naturally occurring background concentrations of inorganics in soil and may not be attributable to site activities.

The integrated statistical and geochemical evaluation (Appendix K) conducted for inorganic constituents in surface soil at Parcel 66(7) indicated that all of the inorganic constituents that exhibited MDCs greater than their respective ESVs were associated with iron oxides in Parcel 66(7) soils at a relatively constant ratio. This analysis indicated that these inorganic constituents are naturally occurring and may not be present as a result of site-related Army activities.

The SVOCs whose MDCs exceeded their respective ESVs in surface soil were benzo(a)pyrene, fluoranthene, phenanthrene, and pyrene. These three constituents exhibited maximum hazard quotients that ranged from 1.8 to 16. It is important to note that the maximum concentrations of all of the SVOCs that were detected at elevated concentrations in surface soil were less than the background concentrations for soil beneath pavement as presented in the *Human Health and Ecological Screening Values and PAH Background Summary Report* (IT, 2000c). Thus, the SVOCs in surface soil at Parcel 66(7) are characteristic of soil beneath pavement at similar sites at FTMC and may not be indicative of site-related Army activities.

Endrin was detected in a single surface soil sample at a concentration that exceeded its ESV.

The COPECs at Parcel 66(7) (Table 7-3) have been identified through a very conservative screening process that utilizes ESVs based largely on NOAEL values from the scientific literature. If additional lines of evidence are considered, it could be concluded that there are no constituents in surface soil at Parcel 66(7) that have the potential to pose adverse ecological effects to terrestrial populations at FTMC. If, based on a risk management decision, the potential

1 ecological risks at Parcel 66(7) are determined to be “unacceptable” at this screening-level stage,  
2 then a baseline ecological risk assessment is appropriate. The goal of the baseline ecological risk  
3 assessment, if deemed necessary, will be to reduce the levels of uncertainty and conservatism in  
4 the assessment process and to determine the potential for ecological risk at Parcel 66(7) through  
5 a number of lines of evidence.

### 6 7 **7.8.2 Conclusions of SLERA**

8 The SLERA at Parcel 66(7) determined that the maximum concentrations of several constituents  
9 in surface soil exceeded their respective ESVs. Further review of these COPECs is important in  
10 making risk management decisions. The integrated statistical and geochemical evaluation  
11 conducted for inorganic constituents in surface soil at Parcel 66(7) indicated that all of the  
12 inorganic constituents that exhibited MDCs greater than their respective ESVs were associated  
13 with iron oxides in surface soils at a relatively constant ratio. This analysis indicated that these  
14 inorganic constituents are naturally occurring and may not be present as a result of site-related  
15 Army activities. It is also important to note that the maximum concentrations of all of the  
16 SVOCs (all PAHs) that were detected at elevated concentrations in surface soil were less than the  
17 background concentrations for soil beneath pavement as presented in the *Human Health and*  
18 *Ecological Screening Values and PAH Background Summary Report* (IT, 2000c). Thus, the  
19 PAHs in surface soil at Parcel 66(7) are characteristic of soil beneath pavement at similar sites at  
20 FTMC and may not be indicative of site-related Army activities.

21  
22 One surface soil sample exhibited a concentration of endrin that slightly exceeded its ESV.

23  
24 Based on further review (including additional lines of evidence) of the COPECs identified in  
25 surface soil at Parcel 66(7) via the conservative assessment techniques used in the SLERA  
26 process, it could be concluded that none of the constituents in surface soil present risks to  
27 terrestrial ecosystems at FTMC. Therefore, further ecological assessment is not warranted at  
28 Parcel 66(7).