



January 3, 2007

SHAW-MC-CK35-0005
Project No. 122336

Mr. Lee Coker
U.S. Army Corps of Engineers, Mobile District
Attn: EN-GE/Lee Coker
109 St. Joseph Street
Mobile, Alabama 36602

**Contract: DACA21-96-D-0018, Task Order CK35
 Fort McClellan, Alabama**

**Subject: Final SLERA for Ranges Near Training Area T-24A, Parcels 187(7), 88(6),
 108(7)/82Q-X, 112Q, 113Q-X, 213Q, and 214Q**

Dear Mr. Coker:

The subject document is enclosed for your review. The SLERA, which is Chapter 7.0 of the T-24A RI report, was revised based on EPA comments on the Draft RI report. The purpose of submitting this SLERA independently of the revised RI report is to finalize the list of COPECs so that the Problem Formulation/Study Design can be expeditiously prepared for a Baseline Ecological Risk Assessment. Upon completion of your review, please provide either a letter of concurrence for the COPECs identified in the SLERA that will be addressed in the BERA or written comments with suggested changes.

At your request, I have distributed copies of this submittal as indicated below. If you have questions, or need further information, please contact me at (865) 694-7361.

Sincerely,


Stephen G. Moran, P.G.
Project Manager

Attachment

Distribution: Lisa Holstein, FTMC (6 copies; 1 CD)
Brandi Little, ADEM (2 copies; 1 CD)
Doyle Brittain, EPA Region 4 (1 copy; 1 CD)
Miki Schneider, JPA (1 copy)
Michelle Beekman, Matrix Environmental (1 copy)
Richard Henry, USFWS (1 copy)
Peter Tuttle, USFWS (2 copies)
Steve Miller, USFWS (2 copies)
Ricky Ingram, USFWS (2 copies)



November 13, 2007

SHAW-MC-CK35-0019
Project No. 122336

Mr. Lee Coker
U.S. Army Corps of Engineers, Mobile District
Attn: EN-GE/Lee Coker
109 St. Joseph Street
Mobile, Alabama 36602

**Contract: DACA21-96-D-0018, Task Order CK35
Fort McClellan, Alabama**

**Subject: Draft Baseline Ecological Risk Assessment Problem Formulation and Study
Design for the Ranges Near Training Area T-24A**

Dear Mr. Coker:

The subject document is enclosed in both hardcopy and electronic format for your review. This document describes the field activities to be conducted in support of a BERA based on the recommendations presented in the draft remedial investigation report for this site. Shaw has also addressed ADEM comments on the final SLERA for this site (issued in January 2007) as discussed in the response to comments attached to this letter. Upon completion of your review, please provide either a letter of concurrence or written comments on this submittal.

At your request, I have distributed copies of this document as indicated below. If you have questions, or need further information, please contact me at (865) 694-7361.

Sincerely,

Stephen G. Moran, P.G.
Project Manager

Enclosure

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Response to ADEM Review Comments
Final SLERA for Ranges Near Training Area T-24A
Parcels 187(7), 88(6), 108(7)/82Q-X, 112Q, 113Q-X, 213Q, and 214Q
Dated January 3, 2007
Fort McClellan, Calhoun County, Alabama

Comments from Stephen A. Cobb, ADEM Chief, Governmental Hazardous Waste Branch, Land Division, in a letter to Ms. Lisa Holstein dated August 20, 2007.

I. GENERAL COMMENTS

Comment 1: The Final SLERA for the Ranges Near Training Area T-24A concludes that potential risks exist to ecological receptors from a variety of COPECs including metals, PAHs and phthalates present within surface soil, surface water, sediment and/or groundwater. Therefore, a Baseline Ecological Risk Assessment is warranted for the Ranges Near Training Area T-24A.

Response 1: No response is necessary. Please note that due to the nature of the comments provided by ADEM on the T-24A SLERA, the SLERA will not be revised. Rather, ADEM's comments will be addressed and incorporated into the Problem Formulation and Study Design for the T-24A Ranges.

Comment 2: In general, the list of COPECs provided in the Final SLERA adequately addresses the contaminants of concern although further clarification is needed on detected concentrations of cobalt and mercury within the surface water media as indicated below.

Response 2: Please see response to Page-Specific Comment 5.

Comment 3: Total PAHs should also be evaluated in the BERA when assessing risk to ecological receptors.

Response 3: Total PAHs will be evaluated in the BERA for the T-24A Ranges.

II. PAGE-SPECIFIC COMMENTS

Comment 4: Page 7-63, Paragraph 2, Section 7.5.2. The hierarchy for selecting sediment ecological screening values (ESVs) is presented in this section. NOAA SQRTs chronic freshwater ambient water quality criteria are incorrectly listed as the secondary sediment ESV. The NOAA SQRTs freshwater sediment threshold effects level (TEL) should be listed as the secondary source. Please correct. In addition, the hierarchy presented in this section appears to differ from the actual ESVs presented in Table 7-4 (source of ESVs in this table are from: *Human Health and Ecological Screening Values and PAH Background Summary Report, IT, 2000*).

Please clarify and correct Table 7-4 to reflect the actual hierarchy used to select ESVs.

Response 4: Agreed. The secondary source for sediment ESVs should be NOAA SQRs freshwater sediment threshold effects level (TEL) instead of NOAA SQRs chronic freshwater ambient water quality criteria. The ESVs used to screen for sediment COPECs in Table 7-4 of the T-24A SLERA are the same as the ESVs presented in Table 3-3, Ecological Benchmark Screening Values for Sediment in the *Human Health and Ecological Screening Values and PAH Background Summary Report* (IT, 2000). The hierarchy for selecting sediment ESVs is consistent between the T-24A SLERA and the *Human Health and Ecological Screening Values and PAH Background Summary Report* (IT, 2000). No changes are needed to address this comment.

Comment 5: **Page 7-71, Paragraph 4, Section 7.6.2. Detected concentrations of cobalt and mercury in surface water samples both exceed their respective ESV and were undetected in background samples. However, Table 7-3 indicates these constituents were eliminated as COPECs since detected cobalt and mercury concentrations are statistically similar to background concentrations (where these constituents were not detected). Please discuss and clarify how detected cobalt and mercury concentrations in surface water are similar to undetected background levels of these constituents.**

Response 5: Although cobalt and mercury in FTMC background surface water do not have Tier 1 background screening values (2-times the mean), they did pass the Tier 2 step of the FTMC site-to-background comparison process. This step consists of a hot measurement test (comparison of the site MDC to the background 95th upper tolerance limit [UTL] or 95th percentile, depending on the background distribution) and the Wilcoxon rank sum test (which is performed for elements with less than 50 percent nondetects in both the site and background data sets). During the hot measurement test, the 95th UTL is used as the background screening value for elements with normal or lognormal distributions in the background data set, and the 95th percentile is used as the background screening value for elements that are characterized as having nonparametric distributions (due either to the presence of greater than 15 percent nondetects or to failure of the Shapiro-Wilk test to indicate a normal or lognormal distribution). For those elements with high nondetect frequencies and nondetects in the upper decile of the background distribution (i.e., cobalt and mercury in background surface water), the maximum reporting limit is used as the background screening value and it represents an upper limit to the background distribution. This information is provided in the approved installation-wide work plan, which was issued in February 2002 (IT Corporation, 2002).

In accordance with the BCT-approved site-to-background comparison methodology, elements without Tier 1 screening values are carried forward

for Tier 2 screening. In the case of cobalt and mercury in the surface water samples from the Ranges Near T-24A, the maximum detected concentrations (18.1 J $\mu\text{g/L}$ and 0.066 J $\mu\text{g/L}$, respectively) are below the Tier 2 background screening values of $< 25 \mu\text{g/L}$ (cobalt) and $< 0.243 \mu\text{g/L}$ (mercury). Therefore, these two elements are not carried forward for additional evaluation in the site-to-background comparison. It is important to note that both of the site MDCs are low, estimated values below the reporting limit, which suggests that they do not represent site-related contamination.

Comment 6: Page 7-75, Paragraph 7.6.3. The report states that there is no ESV for iron in sediment. However, an iron ESV is presented in several of the hierarchy sediment ESV sources (e.g., USEPA Region III BTAG values). This ESV (20,000 mg/kg) should be added to Table 7-4 and the results of the comparison of detected iron concentrations with its ESV presented here.

Response 6: Agreed. The USEPA Region 3 BTAG value for iron in sediment (20,000 mg/kg) should be used as the ESV for iron. Using the USEPA Region 3 BTAG value for iron as the ESV, the HQ_{screen} value for iron in sediment is calculated to be 3.72. Two sediment samples exhibited iron concentrations greater than the BTV for iron. Geochemical evaluation indicated that all of the detected concentrations of iron in sediment were consistent with naturally occurring background concentrations of iron in sediment. Iron is often considered a macro-nutrient which is easily regulated by most organisms and only toxic at very high levels. Since iron was detected relatively infrequently at elevated concentrations compared to the BTV, is often considered a macro-nutrient that is easily regulated, and geochemical evaluation indicated that the detected iron was consistent with naturally occurring levels, iron was not identified as a COPEC in sediment. This evaluation of iron in sediment will be added to the Problem Formulation and Study Design report for the Ranges Near Training Area T-24A.

Comment 7: Page 7-75, Paragraph 4, Section 7.6.3. The elevated HQ_{screen} of 8571 calculated for mercury and presented in Table 7-4 and in this section appears incorrect. The mercury ESV listed in Table 7-4 is 0.0000245 mg/kg while the USEPA Region IV ESV is 0.13 mg/kg. Based on the sediment hierarchy presented earlier, it appears that the ESV of 0.13 mg/kg would be applicable. Please correct this discussion and Table 7-4 accordingly.

Response 7: The calculated HQ_{screen} value for mercury of 8,571 presented in the T-24A SLERA was based on the use of 0.0000245 mg/kg as the ESV for mercury in sediment (IT, 2000). This ESV is the sediment ESV for methyl-mercury. The ESV for "mercury" presented in Table 7-4 of the *Human Health and Ecological Screening Values and PAH Background Summary Report* (IT, 2000) is 0.13 mg/kg. If 0.13 mg/kg is used as the ESV for mercury in sediment, the HQ_{screen} value is calculated to be 1.62. Mercury was detected in

one sediment sample at a concentration that exceeded this ESV. Geochemical evaluation indicated that the single detection of mercury in sediment was consistent with naturally occurring background concentrations of mercury in sediment. Based on the infrequency of detection, low HQ_{screen} value, and the fact that geochemical evaluation indicated that the detected mercury was consistent with background levels, mercury was not identified as a COPEC in sediment. This evaluation will be added to the Problem Formulation and Study Design for the Ranges Near Training Area T-24A.

Comment 8: Page 7-76, Paragraph 5, Section 7.6.3. Given the elevated PQLs that determine sediment ESVs for PAHs, total PAHs should be also retained as COPECs and compared to their applicable ESV (1.684 mg/kg from USEPA Region IV).

Response 8: Total PAHs will be evaluated in the BERA for the T-24A Ranges.

**Response to U.S. Environmental Protection Agency Review Comments
Final SLERA for Ranges Near Training Area T-24A,
Parcels 187(7), 88(6), 108(7)/82Q-X, 112Q, 113Q-X, 213Q, and 214Q
Fort McClellan, Calhoun County, Alabama**

Comments received from Doyle Brittain, Senior Remedial Project Manager,

- Comment 1:** Table 7-3 and text on Page 7-73 discussed bis(2-ethylhexyl) phthalate as a COPEC in surface water. The maximum detected concentration of bis(2-ethylhexyl) phthalate was 16 ug/L relative to the Great Lakes Water Quality Initiative Tier II Ecotox Threshold Value of 32 ug/L (USEPA 1996). The Ecotox Threshold Value is suitable as a refinement screening value for this compound. It appears as if a weight of evidence argument could be made to eliminate bis(2-ethylhexyl) phthalate as a COPEC.
- Response 1:** Table 7-3 and the text in Section 7.6.2 will be revised to reflect the fact that consideration of additional lines of evidence indicates that bis(2-ethylhexyl)phthalate should not be considered a COPEC in surface water at the T-24A ranges.
- Comment 2:** Bis(2-ethylhexyl)phthalate could also be eliminated as a COPEC in groundwater by a similar argument.
- Response 2:** Table 7-5 and the text in Section 7.6.4 will be revised to reflect the fact that consideration of additional lines of evidence indicates that bis(2-ethylhexyl)phthalate should not be considered a COPEC in groundwater at the T-24A ranges.
- Comment 3:** Mercury in sediment on Table 7-4 was detected in one of seven samples at a concentration that is only slightly above most sediment screening benchmarks. The maximum detected concentration of mercury in sediment was 0.21 mg/kg. Some commonly used benchmarks for freshwater sediment are the MacDonald *et al.* (2000) consensus value of 0.18 mg/kg, the USEPA (1996) Ecotox Threshold value of 0.15 mg/kg, and the Persaud *et al.* (1993) Lowest Effects Level of 0.2 mg/kg. Based on the low frequency of detection and the low magnitude of the hazard quotient, a case could be made to eliminate mercury as a COPEC in sediment. Mercury was screened in as a COPEC due to the use of the very conservative screening value for methyl mercury. Although it is typical in a screening-level assessment to assume chemicals are present in their most toxic and most bioavailable form, normally it is safe to assume that the bulk of the mercury in sediment is in the form of inorganic mercury.

Response 3: Table 7-4 and the text in Section 7.6.3 will be revised to reflect the fact that consideration of additional lines of evidence indicates that mercury should not be considered a COPEC in sediment at the T-24A ranges.

Comment 4: **Di-n-butyl phthalate was identified as a COPEC in sediment in Table 7-4 based on a Region 5 screening value. An alternative screening value in the form of an Ecotox Threshold was presented by USEPA (1996) as 11 mg/kg. It is possible to argue that di-n-butyl phthalate is not a COPEC based on the maximum concentration being less than the alternative benchmark and the low frequency of detection.**

Response 4: Table 7-4 and the text in Section 7.6.3 will be revised to reflect the fact that consideration of additional lines of evidence indicates that di-n-butylphthalate should not be considered a COPEC in sediment at the T-24A ranges.

Comment 5: **The PAHs fluoranthene and pyrene, which were detected in sediment, were measured at maximum concentrations in the part-per-million range. Their concentrations were higher than the typical sediment benchmarks, such as those reported by MacDonald *et al.* (2000). EPA agrees with the recommendation in the SLERA that these compounds may indicate isolated areas of contamination and should remain COPECs.**

Response 5: Comment noted. No response is necessary.

REFERENCES:

MacDonald, D.D., C.G. Ingersoll, and T.A. Berger, 2000, Development and evaluation of consensus-based sediment quality guidelines for freshwater ecosystems. *Arch. Environ. Contam. Toxicol.* 39:20-31.

Persaud, D., R. Jaagumagi and A. Hayton, 1993, *Guidelines for the protection and management of aquatic sediment quality in Ontario*. Ontario Ministry of Environment and Energy.

USEPA, 1996, *Ecotox Threshold for 67 Chemicals Commonly Found at Superfund Sites*. Eco Update Vol. 3, No. 2. January 1996.

7.0 Screening-Level Ecological Risk Assessment

7.1 Introduction

In order to determine the potential for ecological risks posed by site-related chemicals at the Ranges Near Training Area T-24A, a screening-level ecological risk assessment (SLERA) was conducted. This SLERA consisted of a description of the habitat(s) in and around the Ranges Near Training Area T-24A, a discussion of the constituents detected in samples collected from environmental media at the ranges, 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

The Ranges Near Training Area T-24A are located in the southeastern portion of the Main Post and consist of seven individual parcels including:

- Former Machine Gun Range (Parcel 112Q)
- Former Bandholtz Machine Gun Qualification Range (Parcel 213Q)
- Former Bandholtz Field Firing Range (Parcel 214Q)
- Range 24A, Former Multi-Purpose Range (Parcel 108[7]/82Q-X)
- Range 24A, Former Chemical Munitions Disposal Area (Parcel 187[7])
- Former Demolition Area (Parcel 113Q-X)
- Range 24A, Fog Oil Drum Storage (Parcel 88[6]).

Three of the parcels, Parcel 112Q, Parcel 213Q, and Parcel 214Q are firing ranges. The area encompassed by Parcels 213Q and 214Q overlaps the area of Range 24A, Multi-Purpose Range (Parcel 108[7]/82Q-X), which in turn overlaps Parcel 133Q-X, Parcel 187(7), and Parcel 88(6). Four of the seven overlapping parcels (Parcel 108[7]/82Q-X, Parcel 88[6], Parcel 113Q-X, and Parcel 187[7]) that comprise the Ranges Near Training Area T-24A are located within a valley area just south of the edge of the Fort McClellan geologic window. The remaining three overlapping parcels (Parcel 112Q, Parcel 213Q, and Parcel 214Q) cover an extensive area extending to as much as 10,000 feet to the north, east, and south. Stanley Hill and the Skeleton Mountains arise along the southern boundary of these parcels. The elevation across these parcels ranges from a maximum of approximately 1,125 feet above mean sea level (amsl) at the northeastern corner of Parcel 108(7)/82Q-X to valley areas of less than approximately 975 feet amsl.

1 Surface water drainage at the Ranges Near Training Area T-24A consists of several intermittent
2 and perennial streams that generally flow to the north and west across these parcels and
3 constitutes the headwaters of the South Branch of Cane Creek.

4
5 The environmental setting of the Ranges Near Training Area T-24A is varied. The majority of
6 the area is forest consisting of mixed deciduous/coniferous forest (deciduous trees dominate)
7 with significant underbrush. An area encompassing approximately 1.2 acres in the western
8 portion of the study area is best described as oldfield habitat.

9
10 The forested areas of the Ranges Near Training Area T-24A are characteristic of a typical
11 mesophytic forest type. The canopy species typically found in this forest type at FTMC include
12 yellow poplar (*Liriodendron tulipifera*), sweetgum (*Liquidambar styraciflua*), black gum (*Nyssa*
13 *sylvatica*), shortleaf pine (*Pinus echinata*), loblolly pine (*Pinus taeda*), white oak (*Quercus alba*),
14 and northern red oak (*Quercus rubra*). The dominant understory species of this forest type are
15 red maple (*Acer rubrum*), flowering dogwood (*Cornus florida*), witch hazel (*Hamamelis*
16 *virginia*), sweetgum (*Liquidambar styraciflua*), wild black cherry (*Prunus serotina*), hackberry
17 (*Celtis occidentalis*), black walnut (*Juglans nigra*), and sourwood (*Oxydendrum arboreum*). The
18 shrub layer is dominated by mountain laurel (*Kalmia latifolia*), Piedmont azalea (*Rhododendron*
19 *canescens*), southern low blueberry (*Vaccinium pallidum*), southern wild raisin (*Viburnum*
20 *nudum*), and yellowroot (*Xanthorhiza simplicissima*). Muscadine grape (*Vitis rotundifolia*) is a
21 common vine found in this forest type.

22
23 The relatively small area of oldfield habitat that occurs in the western portion of the study area
24 was formerly maintained as a mowed field. Since maintenance activities have ceased in this
25 area, pioneer species are now colonizing this area. Typically, the species most likely to colonize
26 these types of areas are the “weed” species that tend to be vigorous pioneer plants that grow and
27 spread rapidly. The first of the pioneer species to invade these abandoned areas are the grasses
28 and other herbaceous species. These formerly maintained grassy areas are classified as being in
29 an early oldfield successional state. Over time, the grass and other herbaceous species will be
30 followed by shrubs and small trees. The early oldfield successional area at the Ranges Near
31 Training Area T-24A is dominated by various grasses and herbs, including dock (*Rumex spp.*),
32 clover (*Trifolium spp.*), vetch (*Astragalus spp.*), milkweed (*Asclepias spp.*), bed straw (*Galium*
33 *spp.*), ox-eye daisy (*Chrysanthemum leucanthemum*), and Johnson grass (*Sorghum halepense*).
34 Other oldfield herbaceous species occurring at the Ranges Near Training Area T-24A are black
35 raspberry (*Rubus occidentalis*), poison ivy (*Toxicodendron radicans*), smooth sumac (*Rubus*
36 *glabra*), green brier (*Smilax rotundiflora*), Japanese honeysuckle (*Lonicera japonica*), fox grape

1 (*Vitis labrusca*), and multiflora rose (*Rosa multiflora*). Scrub pine (*Pinus virginiana*) and
2 loblolly pine (*Pinus taeda*) saplings have also begun to encroach on this formerly cleared area.

3
4 Typical terrestrial species that may inhabit the Ranges Near Training Area T-24A include
5 opossum, short-tailed shrew, raccoon, white-tail deer, red fox, coyote, gray squirrel, striped
6 skunk, a number of species of mice and rats (e.g., white-footed mouse, eastern harvest mouse,
7 cotton mouse, eastern woodrat, and hispid cotton rat), and eastern cottontail. Approximately 200
8 avian species reside at FTMC at least part of the year (USACE, 1998). Common species
9 expected to occur in the vicinity of the Ranges Near Training Area T-24A include northern
10 cardinal (*Cardinalis cardinalis*), northern mockingbird (*Mimus polyglottus*), warblers
11 (*Dendroica spp.*), indigo bunting (*Passerina cyanea*), red-eyed vireo (*Vireo olivaceus*),
12 American crow (*Corvus brachyrhynchos*), bluejay (*Cyanocitta cristata*), several species of
13 woodpeckers (*Melanerpes spp.*, *Picoices spp.*), and Carolina chickadee (*Parus carolinensis*).
14 Game birds present in the vicinity of the Ranges Near Training Area T-24A may include
15 northern bobwhite (*Colinus virginianus*), mourning dove (*Zenaida macroura*), and eastern wild
16 turkey (*Meleagris gallopavo*). A variety of woodland hawks (e.g., sharp-shinned hawk) and
17 other raptors (e.g., red-tailed hawk, barred owl, and great horned owl) are expected to use this
18 area for a hunting and/or nesting area.

19
20 As stated previously, several small, ephemeral and perennial streams drain the Ranges Near
21 Training Area T-24A and conduct surface runoff to the South Branch of Cane Creek which runs
22 east-to-west across the northern portion of the study area. Much of the study area of the Ranges
23 Near Training Area T-24A comprises the headwaters of the South Branch of Cane Creek. The
24 majority of these small streams are narrow (2 to 3 feet wide) and shallow (3 to 6 inches deep).
25 The substrate is mostly cobbles and gravel with small depositional areas of sand and leaf litter,
26 interspersed throughout. A wetland/seep area is present near the northwestern corner of the
27 study area that exhibits very shallow water (less than 6 inches deep) and a substrate of organic
28 muck. The small size of these intermittent and perennial drainage features preclude the presence
29 of many fish species and other animals that might prey on fish (piscivores); however, semi-
30 aquatic species (amphibians) and some small fish species are likely to occur in these small
31 creeks.

32
33 Portions of the Ranges Near Training Area T-24A are contained within the Stanley Hill Chestnut
34 Oak Forest and South Branch of Cane Creek Special Interest Natural Areas (SINA). The Stanley
35 Hill Chestnut Oak Forest SINA is located on the northern and western slopes of Kings and
36 Stanley Hills, and represents the single largest tract of mesic woodlands on the Main Post. The
37 entire Stanley Hill Chestnut Oak Forest SINA is an inclusion within the extensive "Mountain

1 Longleaf Community Complex.” The Stanley Hill Chestnut Oak Forest SINA has been
2 identified separately because of its potential importance to breeding neotropical migratory birds
3 (Garland, 1996).

4
5 A significant portion of the Ranges Near Training Area T-24A is contained within the South
6 Branch Cane Creek SINA. The headwaters of the South Branch of Cane Creek include
7 significant stream, mountain seep, and typic mesophytic forest communities. The surrounding
8 forested mountain slopes are critical to the integrity of these aquatic and wetland communities.
9 Much of this watershed includes the forested slopes of the Stanley Hill Chestnut Oak Forest
10 SINA. A candidate 2 caddisfly, *Polycentropus carlsoni*, and an even rarer single site endemic
11 caddisfly, *Hydroptila setigera*, have been collected from this stream (Mettee and Haynes, 1979).
12 An additional thirteen caddisfly species from this stream are included on the Alabama Natural
13 Heritage Program tracking list (Garland, 1996). The primary management goal for this SINA is
14 to ensure the maintenance of water quality and minimize the influx of sediments from
15 surrounding upland areas

17 **7.3 Constituents Detected On Site**

18 The sampling and analysis programs conducted at the Ranges Near Training Area T-24A were
19 designed based on a number of factors, including:

- 21 • Site history
- 22 • Results of the environmental baseline survey (EBS)
- 23 • Results of previous sampling and analysis programs.

24
25 The sampling and analysis programs at the Ranges Near Training Area T-24A are described in
26 Chapter 2.0 of this report. Constituents detected in soil, surface water, sediment, and
27 groundwater at the Ranges Near Training Area T-24A are presented in Chapter 4.0 of this report.

28
29 Because all of the parcels included in the study area for the Ranges Near Training Area T-24A
30 are contiguous or in many cases overlapping, surface soil, surface water, sediment, and
31 groundwater are described as single data sets and are not separated by individual parcel.
32 Additionally, ecological receptors are not bound by artificial delineations of source areas, but
33 encounter contaminated media based on feeding/foraging habits and other species-specific life-
34 history parameters and habits.

35
36 Surface soil samples from the Ranges Near Training Area T-24A exhibited maximum
37 concentrations of the following constituents that exceeded ESVs:

- Aluminum
- Antimony
- Arsenic
- Barium
- Beryllium
- Cadmium
- Chromium
- Cobalt
- Copper
- Iron
- Lead
- Manganese
- Mercury
- Nickel
- Selenium
- thallium
- Vanadium
- Zinc
- Phenanthrene
- Chloroform
- Xylene
- Trichlorofluoromethane.

The following constituents exceeded their respective ESVs in surface water at the Ranges Near Training Area T-24A:

- Aluminum
- Barium
- Beryllium
- Chromium
- Cobalt
- Copper
- Iron
- Lead
- Manganese
- Mercury
- Vanadium
- Zinc
- Bis(2-ethylhexyl)phthalate

Sediment samples from the Ranges Near Training Area T-24A exhibited maximum concentrations of the following constituents that exceeded ESVs:

- Aluminum
- Arsenic
- Barium
- Beryllium
- Copper
- Iron
- Lead
- Manganese
- Mercury
- Nickel
- Selenium
- Thallium
- Vanadium
- Zinc
- Benzo(a)anthracene
- Chloromethane
- Chrysene
- Di-n-butylphthalate
- Fluoranthene
- Pyrene.

The following constituents in groundwater at the Ranges Near Training Area T-24A exceeded their corresponding surface water ESVs:

- Aluminum
- Barium
- Chromium
- Cobalt
- Iron
- Lead
- Manganese
- Mercury
- Selenium
- Silver
- Zinc
- Bis(2-ethylhexyl)phthalate
- Benzene
- Carbon tetrachloride.

1

2 **7.4 Site Conceptual Model**

3 The ecological site conceptual model (SCM) is a simplified, schematic diagram of possible
 4 exposure pathways and the means by which contaminants are transported from the primary
 5 contaminant source(s) to ecological receptors (Figure 7-1). The exposure scenarios include the
 6 sources, environmental transport, partitioning of the contaminants amongst various
 7 environmental media, potential chemical/biological transformation processes, and identification
 8 of potential routes of exposure for the ecological receptors. In this chapter the SCM will be
 9 described in relation to constituent fate and transport properties, the ecotoxicity of the various
 10 constituents, potential ecological receptors at the Ranges Near Training Area T-24A, and the
 11 complete exposure pathways expected to exist at the Ranges Near Training Area T-24A.

12

13 **7.4.1 Constituent Fate and Transport**

14 The environmental fate and transport of contaminants in surface soil, surface water, sediment,
 15 and groundwater at the Ranges Near Training Area T-24A will govern the potential for
 16 exposures to ecological receptors. In general, contaminants in environmental media may be
 17 available for direct exposure (e.g., plants exposed to surface soil), and they may also have the
 18 potential to migrate to other environmental media or areas of the site. This section discusses the
 19 mechanisms by which contaminants can be transported and the chemical properties that
 20 determine their transport.

21

22 **7.4.1.1 Fate and Transport in Soil**

23 Contaminants in surface soil at the Ranges Near Training Area T-24A have the potential to be
 24 transported from their source areas to off-site locations by a number of mechanisms, including
 25 volatilization, dust entrainment, surface runoff, and infiltration to subsurface soil/groundwater.

26

27 The inorganic constituents detected in surface soil at the Ranges Near Training Area T-24A are
 28 generally closely associated with the soil particles themselves and are not expected to be
 29 transported great distances from their source. Volatilization of inorganic constituents is expected
 30 to be insignificant, with the exception of mercury, which is expected to volatilize from exposed
 31 surface soil rapidly. Fugitive dust generation and entrainment by the wind with subsequent

1 dispersion by atmospheric mixing could transport particulate-associated contaminants to other
2 parts of the study area and to off-site locations. Fugitive dust generation could be a significant
3 transport mechanism in the cleared areas of the study area where significant vegetative cover is
4 absent. However, in portions of the study area where vegetation and thick leaf/pine needle litter
5 is present, fugitive dust generation is expected to be minimal. Several volatile organic
6 compounds (VOC) were identified in the surface soil at the Ranges Near Training Area T-24A.
7 These constituents have varying degrees of volatilization potential with subsequent off-site
8 transportation via air movement. However, VOCs were infrequently detected and at relatively
9 low concentrations; therefore, volatilization and subsequent transport of these compounds is
10 expected to be insignificant with respect to other transport mechanisms potentially active at these
11 parcels.

12
13 The transport of surface soil-associated contaminants by surface runoff is another potential
14 transport mechanism. Several perennial streams and ephemeral drainage features drain the
15 Ranges Near Training Area T-24A and eventually discharge to the South Branch of Cane Creek
16 in the northern portion of the study area. These drainage features collect surface runoff from the
17 sites and transport it off-site to the north and west. As such, surface runoff via the perennial and
18 ephemeral drainage features has the potential for significant constituent transport off-site.

19
20 Contaminants in surface soil at the Ranges Near Training Area T-24A may be transported
21 vertically to subsurface soils and groundwater via solubilization in rainwater and infiltration.
22 Migration in this manner is dependent upon contaminant solubility and frequency of rainfall.
23 The soil type (rough, stony land) in the vicinity of the Ranges Near Training Area T-24A does
24 not promote rapid infiltration, but rather is more conducive to the promotion of surface runoff.
25 Based on the constituents detected in surface soil and the soil type found at these parcels, vertical
26 migration of surface soil constituents is expected to be minimal at the Ranges Near Training
27 Area T-24A

28
29 The transfer of contaminants in surface soil to terrestrial plants through root uptake and to
30 terrestrial animals through ingestion and other pathways are potentially significant transfer
31 mechanisms. Many metals are readily absorbed from soil by plants, but they are not
32 biomagnified to a great extent through the food web, mercury being a notable exception.
33 Volatile organic compounds and semi-volatile organic compounds do not bioaccumulate to any
34 significant extent (Shugart, et al., 1990); therefore, food web transfer of these constituents is
35 expected to be minimal.

1 VOCs in the surface soil at the Ranges Near Training Area T-24A are expected to volatilize
2 and/or photolyze relatively rapidly when exposed to sunlight (half-lives of 3 hours to 5 days)
3 (Burrows et al., 1989).

4 5 **7.4.1.2 Fate and Transport in Surface Water**

6 Because a significant portion of the study area of the Ranges Near Training Area T-24A lies
7 within the headwaters of the South Branch of Cane Creek, surface water transport is potentially a
8 significant mechanism. Constituents transported to the perennial and ephemeral drainage
9 features within the study area via surface runoff could be transported off-site via surface water
10 flow. Although only one constituent was detected in surface water at an elevated concentration,
11 the surface water pathway provides a significant mechanism for the transport of constituents
12 from their source area(s) to other areas within the study area and to off-site areas, particularly
13 during periods of high precipitation. Constituents may be transported via surface water pathways
14 in either the dissolved or suspended forms.

15
16 Groundwater at the Ranges Near Training Area T-24A may infiltrate surface water bodies and
17 make up a portion of the surface water flow. The depth to groundwater at the Ranges Near
18 Training Area T-24A ranges from zero to approximately 87 feet below ground surface (bgs),
19 with an average depth of 18 feet bgs. In the areas with groundwater near the ground surface,
20 groundwater may infiltrate the surface water bodies and transfer groundwater constituents to the
21 surface water.

22 23 **7.4.1.3 Fate and Transport in Sediment**

24 As is the case with surface water, sediment transport is potentially a significant mechanism at the
25 Ranges Near Training Area T-24A. Constituents transported to the perennial and ephemeral
26 drainage features within the study area via surface runoff could be transported off-site via surface
27 water flow. The transport of sediment in the streams and drainage features at these parcels could
28 represent a transport pathway for soil-related constituents to off-site areas during periods of
29 significant precipitation. Sediment in the streams and drainage features could become entrained
30 in the runoff conducted in these drainage features and could potentially be transported off-site.

31 32 **7.4.1.4 Fate and Transport in Groundwater**

33 As stated previously, depth to groundwater at the Ranges Near Training Area T-24A ranges from
34 zero feet bgs to approximately 85 feet bgs, with an average depth to groundwater of 18 feet bgs.
35 Where the groundwater is near the ground surface, groundwater constituents could be transferred
36 to surface water bodies and transported via surface water pathways. This transport mechanism is

1 expected to be operational only in localized areas as the depth to groundwater is significantly
2 below ground surface in many portions of the study area.

3 4 **7.4.2 Ecotoxicity**

5 The ecotoxicological properties of the constituents detected at concentrations that exceeded their
6 respective ESVs and BTVs in surface soil, surface water, sediment, and groundwater at the
7 Ranges Near Training Area T-24A are discussed in the following sections.

8 9 **7.4.2.1 Aluminum**

10 Aluminum is the most abundant element in the earth's crust. Minimal evidence exists
11 concerning the essentiality of aluminum. Aluminum is generally considered to have low
12 mammalian toxicity (Hayes, 1994).

13
14 **Plants.** Aluminum appears to be essential for the growth of some plant species (Kabata-Pendias
15 and Pendias, 1992). Higher concentrations of aluminum are usually detected in older rather than
16 younger leaves (Bollard, 1983). Difference in the toxicity of aluminum to plants is closely
17 linked to the different uptake and transport of calcium (Foy, 1974). Interactions of aluminum
18 with potassium, silicon, and organic acids have also been reported (Foy, 1974). According to
19 Foy (1974), aluminum toxicity in plants usually does not occur in soils with pH values above
20 5.5. Toxicity is, however, common at soil pH values below 5.0 (Foy (1974). The addition of
21 nitrogenous fertilizers to soil increases the toxicity of aluminum to plants by displacing
22 exchangeable aluminum into soil solution and lowering soil pH (Foy, 1974).

23
24 Concentrations of aluminum in leaf tissue that are excessive or toxic to various plant species
25 range from 5 to 10 mg/kg (dry weight (Kabata-Pendias and Pendias, 1992). A soil concentration
26 of 50 mg/kg (dry weight) has been proposed by Efroymsen, et al., (1997a) as a benchmark
27 screening value for aluminum phytotoxicity. Signs of aluminum toxicity in plants include
28 overall stunting of growth, the presence of dark green leaves, purpling of stems, death of leaf
29 tips, and coraloid and damaged root systems (Kabata-Pendias and Pendias, 1992)

30
31 **Terrestrial Invertebrates.** No information was found regarding the toxicity of aluminum to
32 terrestrial invertebrates.

33
34 **Mammals.** Aluminum is not an essential element for animal growth and development. Limited
35 data exist on the concentrations and effects of aluminum on wildlife. Most absorbed aluminum
36 is eliminated through the kidney (NLM, 1996).

1
2 Data are scarce on the effects of aluminum on wild mammals. Laboratory studies have shown
3 inhalation of aluminum dust to induce infections and diseases of the lung (NLM; 1996). A
4 derived chronic no observable adverse effect level (NOAEL) of 0.043 mg/kg/day has been
5 reported for laboratory rats exposed to aluminum (EPA, 2003b). Laboratory-derived toxicity
6 data from studies conducted with mice fed aluminum (AlCl₃) in their drinking water over three
7 generations were used to derive a NOAEL value of 1.93 mg/kg/day (Ondreicka, et al., 1966).
8 Reproduction was the endpoint for these studies.

9
10 **Birds.** Dietary ingestion of aluminum at concentrations of approximately 1,400 mg/kg
11 produced declines in inorganic phosphorus levels in blood and resulted in the development of
12 severe rickets in chickens (NLM, 1996). No adverse effects were observed in black ducks (*Anas*
13 *rubripes*) fed diets containing 1,000 mg/kg aluminum as aluminum sulfate over a period of 12
14 days (Sparling, 1990). Diets with low calcium and phosphorus concentrations adversely affected
15 the response of the ducks to aluminum (Sparling, 1990). An acute LD₅₀ (lethal dose that will
16 result in 50 percent mortality in a test population) of 111 mg/kg has been reported for exposure
17 of birds to aluminum (Schafer, et al., 1983). Ringed doves fed aluminum as Al₂(SO₄)₃ for four
18 months in their diet showed no adverse effects at a dose level of 1,000 ppm. This dosage was
19 used to derive a NOAEL value of 109.7 mg/kg/day (Carreire, et al., 1986). Reproduction was
20 the critical endpoint in this study.

21
22 **Aquatic Life.** Bioconcentration of aluminum has been reported for several freshwater species.
23 A bioconcentration factor for daphnids exposed to aluminum is 574 (Cowgill and Burns, 1975).
24 Crayfish have been reported to have a bioconcentration factor for aluminum of 1,305 (Malley, et
25 al., 1987). The National Recommended Water Quality Criteria (EPA, 2002) for aluminum are
26 750 and 87 µg/L for acute and chronic exposures, respectively. The lowest chronic values for
27 aluminum toxicity reported in the literature for fish and daphnids are 3,290 and 1,900 µg/L,
28 respectively (Suter and Tsao, 1996). The test EC₂₀ (the concentration that will result in a
29 specified effect on 20 percent of the test population) for fish can be used as a benchmark
30 indicative of production within a population. The EC₂₀ value for aluminum is 4,700 µg/L (Suter
31 and Tsao, 1996). The EC₂₀ value for aluminum with respect to daphnids is 540 µg/L (Suter and
32 Tsao, 1996).

33 34 **7.4.2.2 Antimony**

35 Antimony binds to soil and particulates (especially those containing iron, manganese, or
36 aluminum) and is oxidized by bacteria in soil. Exposure routes for aquatic organisms include

1 ingestion and gill uptake. Antimony bioconcentrates in aquatic organisms to a small degree.
2 Exposure routes for mammals include ingestion and inhalation. It does not biomagnify in
3 terrestrial food chains (Ainsworth, 1988). Antimony is not significantly metabolized and is
4 excreted in the urine and feces. Antimony causes reproductive, pulmonary, and hepatic effects
5 in mammals (EPA, 2005a).

6
7 **Plants.** Antimony is considered a non-essential element and is easily taken up by plants if
8 available in the soil in soluble forms (Kabata-Pendias and Pendias, 1992). A screening level of
9 5.0 mg/kg has been proposed by Kabata-Pendias and Pendias (1992) based on a report of
10 unspecified phytotoxic responses by plants grown in soil amended with antimony.

11
12 **Terrestrial Invertebrates.** The EPA (2005b) has developed an ecological soil screening level
13 (eco-SSL) for antimony of 78 mg/kg. The eco-SSL for antimony is the geometric mean of three
14 EC₂₀ values reported in the literature. Kuperman, et al. (2002) reported an EC₂₀ value using
15 enchytraeids (*Enchytraeus crypticus*) of 194 mg/kg; Phillips, et al. (2002) reported an EC₂₀ value
16 using springtails (*Folsomia candida*) of 81 mg/kg; and Simini, et al. (2002) reported an EC₂₀
17 value using earthworms (*Eisenia fetida*) of 30 mg/kg.

18
19 **Mammals.** Female mice exposed to 5.0 milligrams per liter (mg/L) antimony as antimony
20 potassium tartrate in their drinking water showed a reduction in their lifespan. This dose was
21 equivalent to a lowest-observed-adverse-effects-level (LOAEL) of 1.25 milligrams per kilogram
22 per day (mg/kg/day), which can be converted to a no-observed-adverse-effects-level (NOAEL)
23 of 0.125 mg/kg/day (EPA, 2005a).

24
25 Laboratory data on antimony toxicity (as antimony potassium tartrate) in laboratory mice
26 through drinking water ingestion were used to estimate a chronic NOAEL value of 0.125
27 mg/kg/day (Schroeder et al., 1968). Lifespan and longevity were the endpoints tested.

28
29 **Birds.** No information was found regarding the potential toxicity of antimony to birds.

30
31 **Aquatic Life.** The available data for antimony indicate that acute and chronic toxicity to
32 freshwater aquatic life occur at concentrations as low as 9.0 and 1.6 mg/L, respectively, and
33 would occur at lower concentrations among species that are more sensitive than those tested.
34 Toxicity to algae can occur at concentrations as low as 0.61 mg/L.

35
36 Effects from antimony exposure on benthic community composition have been detected at levels
37 between 3.2 and 150 mg/kg (Long and Morgan, 1990). Data on antimony suggest an effects

1 range-low (ER-L) of 2 mg/kg, and an effects range-median (ER-M) of 25 mg/kg.

3 **7.4.2.3 Barium**

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

8
9 Most of the barium that enters an animal's body is removed within a few days and almost all of it
10 is gone within 1 to 2 weeks. Most of the barium that stays in the body goes into the bones and
11 teeth. Rats exposed to barium in their diet at lower doses, but for longer time periods, showed
12 increased blood pressure and changes in the function and chemistry of the heart (ATSDR,
13 1992a).

14
15 **Plants.** Background concentrations of barium in various food and feed plants are reported to
16 range from 1 to 198 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). Concentrations are
17 often highest in the leaves of cereals and legumes and lowest in grains and fruits (Kabata-
18 Pendias and Pendias, 1992). The availability of barium to plants is greatly influenced by the pH
19 of the soil, with barium more available under acidic soil conditions (Kabata-Pendias and Pendias,
20 1992). The concentration of barium in leaf tissue that has been reported as excessive or toxic to
21 various plant species is 500 mg/kg (Kabata-Pendias and Pendias, 1992). A soil concentration of
22 500 mg/kg has been proposed by Efrogymson, et al., (1997a) as a benchmark screening value for
23 barium phytotoxicity.

24
25 **Terrestrial Invertebrates.** No information was found regarding the toxicity of barium to
26 terrestrial invertebrates.

27
28 **Mammals.** Barium administered to rats via their drinking water at doses of 1, 10, and 100
29 mg/L had no effect on food or water consumption, or growth. Because the highest dose tested
30 (100 mg/L) did not elicit any adverse effects, it was considered the NOAEL (5.1 mg/kg/day)
31 (EPA, 2005a). Laboratory rat toxicity data for barium chloride in drinking water was used to
32 calculate a NOAEL value of 5.1 mg/kg/day (Perry, et al., 1983). Growth and hypertension were
33 the test endpoints. Rats administered barium through oral gavage showed no adverse effects at
34 dosages up to 300 mg/kg/day. These data were used to derive a NOAEL value of 19.8
35 mg/kg/day (Borzelleca, et al., 1988). Mortality was the endpoint for this study.

1 **Birds.** A study of one-day old chicks fed barium in their diet was used to determine the amount
2 of barium to cause mortality. While barium exposures up to 2,000 ppm produced no mortality,
3 chicks in the 4,000 to 32,000 ppm exposure groups experienced 5 to 100 percent mortality.
4 Because 2,000 ppm was the highest non-lethal dose, this dose was considered to be the
5 subchronic NOAEL (Johnson, et al., 1960). A NOAEL value of 20.8 mg/kg/day was derived
6 from these data.

7
8 **Aquatic Life.** The chronic value for daphnids is from a 21-day test on *Daphnia magna* by
9 Biesinger and Christensen (1972) which resulted in 16 percent reproductive impairment. The
10 Tier II secondary acute water quality value and secondary chronic water quality value for
11 barium, as calculated by the method described in the EPA's *Final Water Quality Guidance for*
12 *the Great Lakes System* (EPA, 1995), are 110 and 4.0 µg/L, respectively.

13 14 **7.4.2.4 Beryllium**

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

17
18 **Plants.** Beryllium uptake by plants occurs when beryllium is present in the soluble form. The
19 highest levels of beryllium are found in the roots, with lower levels in the stems and foliage
20 (EPA, 1985a).

21
22 Soluble forms of beryllium are easily taken up by plants, probably in a manner similar to calcium
23 and magnesium, but it is not readily translocated from roots to shoots (Peterson and Girling,
24 1981). Beryllium has been reported to inhibit seed germination, enzyme activation, and uptake
25 of calcium and magnesium by roots. Common symptoms of beryllium toxicity to plants are
26 brown, retarded roots, and stunted foliage (Romney and Childress, 1965). The phytotoxicity
27 benchmark value for beryllium (10 mg/kg) is based on unspecified toxic effects on plants grown
28 in surface soil amended with 10 mg/kg beryllium (Kabata-Pendias and Pendias, 1992).

29
30 **Terrestrial Invertebrates.** No information was found regarding the toxicity of beryllium to
31 terrestrial invertebrates.

32
33 **Mammals.** The major exposure route for mammals is inhalation. Beryllium is poorly absorbed
34 from the gastrointestinal tract, and is not absorbed through intact skin to any significant degree.
35 Mammals exposed via inhalation exhibit pulmonary effects which may last long after exposure
36 ceases. Based on animal studies, beryllium is poorly absorbed through both the gastrointestinal
37 tract and the skin. The most important route of exposure for beryllium is inhalation, although

1 absorption by this route does not appear to be extensive. Once beryllium is absorbed, it is
2 circulated in the blood as an orthophosphate colloid and is then distributed primarily to the bone,
3 liver, and kidneys in both humans and animals. Beryllium and its compounds are not
4 biotransformed, but soluble beryllium compounds are partially converted to more insoluble
5 forms in the lungs (Reeves and Vorwald, 1967).

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

11
12 A variety of beryllium compounds have been demonstrated to cause pulmonary tumors following
13 inhalation in animals. However, it is thought that oral administration does not lead to
14 carcinogenesis due to poor absorption of the constituent from the gastrointestinal tract. The
15 NOAEL for a rat lifetime chronic exposure to beryllium in drinking water was 0.54 mg/kg-day
16 (HEAST, 1997).

17
18 **Aquatic Life.** Exposure routes for aquatic organisms include ingestion and gill uptake.
19 Beryllium does not bioconcentrate in aquatic organisms. Beryllium uptake from water is low,
20 resulting in low bioconcentration rates. Biomagnification of beryllium in aquatic food chains
21 does not occur (Fishbein, 1981). Beryllium can be toxic to warm water fish, especially in soft
22 water.

23
24 The Tier II secondary acute water quality value and secondary chronic water quality value for
25 beryllium, as calculated by the method described in the EPA's *Proposed Water Quality*
26 *Guidance for the Great Lakes System* (EPA, 1995), are 35 and 0.66 µg/L, respectively.

27
28 The EC₂₀ for fish can be used as a benchmark indicative of production within a population. It is
29 the highest tested concentration causing less than 20 percent reduction in either weight of young
30 fish per initial female fish in a life-cycle or partial life-cycle test, or the weight of young per egg
31 in an early life-stage test (Suter and Tsao, 1996). The EC₂₀ value for beryllium is 148 µg/L. A
32 similar value can be determined for daphnids, which reflects the highest tested concentration
33 causing less than 20 percent reduction in the product of growth, fecundity, and survivorship in a
34 chronic test with a daphnid species. The EC₂₀ for daphnids is 3.8 µg/L (Suter and Tsao, 1996).

1 **7.4.2.5 Cadmium**

2 Cadmium is a silver-white, blue-tinged, lustrous metal. It is insoluble in water, although it's
3 chloride and sulphate salts are freely soluble (Windholz, et al., 1976). Cadmium compounds in
4 soil are stable and are not subject to degradation (ATSDR, 1993b).

5
6 **Plants.** Cadmium is a non-essential element for plant growth that can be absorbed from air or
7 soil. The availability of cadmium in soil is influenced by organic content, clay content, pH, and
8 redox potential (Sharma, 1980). The distribution of accumulated cadmium within plants is
9 generally roots>stem and leaves>fruits, grains, seeds, or nutrient storage organs (Eisler, 1985).
10 Concentrations from 10 to 20 mg/kg in plant tissue are expected to result in 10 percent loss in
11 crop yield. Concentrations in leaf tissue that are excessive or toxic to various plant species range
12 from 5 to 30 mg/kg. General symptoms of cadmium poisoning in plants include growth
13 retardation, root damage, chlorosis of leaves, and red-brown coloration of leaf margins or veins
14 (Kabata-Pendias and Pendias, 1992).

15
16 **Terrestrial Invertebrates.** A number of studies have been conducted to assess the potential
17 adverse effects on terrestrial invertebrates. Van Gestel, et al (1992) evaluated the effects of
18 cadmium, added to the soil as CdCl₂, on growth and reproduction of *Eisenia andrei* after 21
19 days. A concentration of 18 mg/kg cadmium was required to reduce the number of cocoons
20 produced per week and the number of juveniles per worm. Growth and reproduction were not
21 affected at 10 mg/kg cadmium.

22
23 The effects of cadmium added to horse manure (as Cd acetate) on *Eisenia fetida* was investigated
24 by Malecki, et al. (1982). Two growth periods were studied, 8 and 20 weeks, and survival,
25 weight gain, and cocoon production were measured. The most sensitive parameter was cocoon
26 production. In the 8-week test, the lowest concentration tested, 25 mg/kg Cd, caused a 52
27 percent decrease in cocoon production. In the 20-week test, the lowest concentration tested, 50
28 mg/kg Cd, caused a 24 percent decrease in cocoon production.

29
30 Using the results of the above tests and a number of other studies, Efroymson, et al. (1997b)
31 computed an earthworm benchmark concentration of 20 mg/kg.

32
33 **Mammals.** Cadmium uptake by most animals is relatively low. Absorption and retention of
34 cadmium decreases with prolonged exposure. Cadmium tends to accumulate in the viscera,
35 especially in liver and kidney tissues. The biological half-life of cadmium in muscle, kidney,
36 and liver tissues ranges from 10 to 30 years. About 5 percent of ingested cadmium is absorbed,
37 whereas between 10 and 50 percent of inhaled cadmium is absorbed (Friberg et al., 1986).

1
2 Mammals are relatively resistant to cadmium toxicity. The production of metallothioneins helps
3 to protect mammals from cadmium toxicosis. The lowest published toxic dose of cadmium for
4 mammals is 21.5 mg/kg, which was reported to induce adverse effects on fertility of rats
5 (RTECS, 1996). Sutou, et al. (1980) studied the effects of cadmium on rats exposed through oral
6 gavage. While no adverse effects were observed at the 1 mg/kg/day dose level, fetal
7 implantations were reduced by 28 percent, fetal survivorship was reduced by 50 percent, and
8 fetal resorptions increased by 400 percent amongst the 10 mg/kg/day exposure group. Because
9 this study considered oral exposures during reproduction, the 1 and 10 mg/kg/day doses were
10 considered to be chronic NOAELs and LOAELs, respectively.

11
12 Exposure to cadmium has been shown to induce teratogenic effects in laboratory animals. The
13 teratogenic effects of cadmium can be synergized by lead and mercury salts and antagonized by
14 selenium (Ferm and Layton, 1981).

15
16 **Birds.** Birds, like mammals, are relatively resistant to cadmium toxicity. Sublethal effects in
17 birds are similar to those in other species and include growth retardation, anemia, and testicular
18 damage (Hammons, et al., 1978). In one experiment, drake mallards were fed up to 200 mg/kg
19 cadmium chloride in their diet for 90 days, and all survived with no loss in body weight. Egg
20 production was, however, suppressed in mallard hens fed 200 mg/kg cadmium chloride in their
21 diet (White and Finley, 1978). As in mammals, metallothionein binds to cadmium and thus aids
22 in the reduction of a toxic response. Japanese quail that were fed 75 mg/kg cadmium in their
23 diets developed signs of bone marrow hyperplasia, anemia, and hypertrophy of both heart
24 ventricles after six weeks of exposure (Richardson et al., 1974). White and Finley (1978) studied
25 the effects on reproduction of mallard ducks fed cadmium in their diets. Based on the results of
26 this study, a NOAEL value of 1.45 mg/kg/day has been derived.

27
28 **Aquatic Life.** Freshwater organisms are more sensitive to elevated cadmium concentrations
29 than are wildlife or plant species (Eisler, 1985). Among all species of freshwater biota
30 examined, cadmium concentrations of 0.47 to 5.0 µg/L were associated with decreases in
31 standing crop, decreases in growth, inhibition of reproduction, immobilization, and population
32 alterations (Eisler, 1985). The EPA's National Recommended Water Quality Criteria are 2.0
33 µg/L for acute exposure and 0.25 µg/L for chronic exposure of freshwater aquatic life to
34 cadmium (based on a water hardness of 100 mg/L) (EPA, 2002). The test EC₂₀ (the
35 concentration that will result in a specified effect on 20 percent of the test population) for fish
36 can be used as a benchmark indicative of production within a population. The EC₂₀ value for
37 cadmium is 1.8 µg/L (Suter and Tsao, 1996). The EC₂₀ value for cadmium with respect to

1 daphnids is 0.75 µg/L (Suter and Tsao, 1996).

2
3 Concentrations of cadmium in freshwater biota from uncontaminated areas are usually less than
4 1.0 mg/kg in fish and less than 5 mg/kg in molluscs, annelids, and macrophytes (Eisler, 1985).
5 Bioconcentration factors of 2,000 have been reported in insects, fish, and algae.

6 7 **7.4.2.6 Chromium**

8 Chromium exists in soil primarily in the form of insoluble oxides with very limited mobility. In
9 soil, chromium (+3) is readily hydrolyzed and precipitated as chromium hydroxide. In the
10 aquatic phase, chromium may be in the soluble state or attached to clay-like or organic
11 suspended solids.

12
13 **Plants.** Chromium does not play an essential role in plant metabolism. The concentration of
14 chromium in terrestrial plants is controlled primarily by soluble chromium in the soil (Kabata-
15 Pendias and Pendias, 1992). Chromium concentrations in plants are usually higher in roots than
16 in leaves or shoots. Concentrations of chromium in leaf tissue that are excessive or toxic to
17 various plant species range from 5 to 30 mg/kg soil (Kabata-Pendias and Pendias, 1992).
18 General symptoms of chromium toxicity in plants include chlorosis of new leaves, necrotic spots
19 and purpling tissues, and injured root growth (Kabata-Pendias and Pendias, 1992).

20
21 **Terrestrial Invertebrates.** Abbasi and Soni (1983) exposed the earthworm *Octochaetus*
22 *pattoni* to chromium (as $K_2Cr_2O_7$) in soil for 60 days to assess the effects on survival and
23 reproduction. Survival was the most sensitive endpoint with a 75 percent decrease resulting
24 from exposure to 2.0 ppm chromium, the lowest concentration tested. The number of cocoons
25 produced was not reduced until the concentration reached 20 ppm chromium (the highest
26 concentration tested). The number of juveniles produced was not affected.

27
28 It is difficult to establish a benchmark concentration for chromium based on earthworm toxicity
29 because the relative toxicity of Cr^{+3} and Cr^{+6} is not clear from the available data. Cr^{+6} ions can
30 pass through cell membranes with much greater ease than Cr^{+3} ions. However, it is thought that
31 Cr^{+6} is reduced to Cr^{+3} inside the cell (Molnar, et al., 1989). Without a better understanding of
32 chromium transformations in the soil, transport across earthworm cell membranes, and reactions
33 within the cell, it is difficult to separate the effects of the two different forms of chromium.
34 These difficulties notwithstanding, a soil benchmark value of 0.4 mg/kg has been suggested by
35 Efroymson, et al. (1997b), based on the work of Abbasi and Soni (1983).

1 **Mammals.** Chromium is a required element in animal nutrition. In general, hexavalent
2 chromium compounds are more toxic than the trivalent chromium compounds. Adverse effects
3 on blood and serum chemistry and morphological changes in liver have been reported in rabbits
4 and rats exposed to chromium concentrations of 1.7 mg/kg/day for six weeks. Rats exposed to
5 hexavalent chromium concentrations of 134 mg/L in drinking water over a two to three month
6 period were found to develop lesions in kidney and liver tissues (Eisler, 1986).

7
8 Laboratory data based on rats exposed to chromium (as Cr₂O₃) in their diets were used to derive
9 a NOAEL value for trivalent chromium of 2,737 mg/kg/day (Ivankovic and Preussmann, 1975).
10 Reproduction and longevity were the endpoints in this study. Laboratory data based on rats
11 exposed to chromium (as K₂Cr₂O₄) in their drinking water were used to derive a NOAEL value
12 for hexavalent chromium of 3.28 mg/kg/day (MacKenzie, et al., 1958). Body weight and food
13 consumption were the endpoints in this study. Mammalian laboratory studies have shown
14 chromium to be mutagenic, carcinogenic and teratogenic (Eisler, 1986).

15
16 **Birds.** Data on the effects of chromium to avian species is limited. No adverse effects were
17 found in chickens exposed to 100 mg/kg dietary hexavalent chromium in a 32-day study
18 (Rosomer, et al., 1961). Haseltine, et al. (1985) did not observe changes in survival,
19 reproduction or blood chemistry following exposure of adult black ducks (*Anas rubripes*) to diets
20 containing between 10 and 50 mg/kg chromium III (as CrK(SO₄)₂). Ducklings from this group
21 that were fed the same chromium diet as the parent ducks experienced alterations in growth
22 patterns and a reduction in survival. Based on these data, a NOAEL value of 1 mg/kg/day has
23 been derived.

24
25 **Aquatic Life.** In freshwater systems, hexavalent chromium appears to be more toxic than the
26 trivalent form. The National Recommended Water Quality Criteria for trivalent chromium are
27 570 µg/L for acute exposures and 74 µg/L for chronic exposures. The National Recommended
28 Water Quality Criteria for hexavalent chromium are 16 µg/L for acute exposures and 11 µg/L for
29 chronic exposures (EPA, 2002). These values are based on a water hardness of 100 mg/L.

30
31 The test EC₂₀ for fish can be used as a benchmark indicative of production within a population.
32 It is the highest tested concentration causing less than 20 percent reduction in either the weight of
33 young fish per initial female fish in a life-cycle or partial life-cycle test, or the weight of young
34 per egg in an early life-stage test. The EC₂₀ value for trivalent chromium is 89 µg/L and for
35 hexavalent chromium is 51 µg/L (Suter and Tsao, 1996). A similar value can be determined for
36 daphnids, which represents the highest tested concentration causing less than 20 percent

1 reduction in the product of growth, fecundity, and survivorship in a chronic test with a daphnid
2 species. The EC₂₀ benchmark for daphnids has been determined to be 0.5 µg/L for hexavalent
3 chromium (Suter and Tsao, 1996).

4 5 **7.4.2.7 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 and groundwater.
8 Natural cobalt can stay airborne for a few days, but will stay for years in the soil. In most soils,
9 the transfer of cobalt from soils to plants is not significant, although higher transfer rates have
10 been observed in some higher plants and in acidic soils (Boikat et al, 1985; Francis et al., 1980).
11 Some cobalt may seep from acid soil into groundwater. It is present in trace quantities in most
12 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 **Terrestrial Invertebrates.** No information was found regarding the toxicity of cobalt to
28 terrestrial invertebrates.

29
30 **Mammals.** Cobalt is a component of vitamin B₁₂ and, therefore, is an essential micro-nutrient
31 for animal growth. No information has been located at this time on chronic toxic effects of
32 cobalt to terrestrial wildlife; however, some acute studies have been completed. Additionally,
33 there is little biomagnification of cobalt in animals of higher trophic levels (Jenkins, 1980).

34
35 Young rats are unable to survive repeated 30 mg doses of cobalt metal powder in their diet for a
36 month (total dosage about 900 mg), whereas they can tolerate 1,250 mg of the metal in a single
37 dose (Venugopal and Luckey, 1978). Cobalt was embryotoxic to rat fetuses when it was

1 administered during the entire gestation (dose of 0.05 mg/kg). A dose of 0.005 mg/kg was non-
2 toxic to the females; however, the progeny of treated females had a reduced rate (Shepard, 1986).
3 At doses under 2 milligrams per kilogram of body weight per day (mg/kg-bw/day), no adverse
4 effects to sheep were noted. However, at 6 mg/kg-bw/day, sheep exhibited loss of appetite, loss
5 of weight, and debilitation were noted (NRC, 1977).

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

10
11 Chickens were administered 50 mg/kg of diet/day with acute effects of loss of appetite, loss of
12 weight, and debilitation. At doses under 2 mg/kg-bw/day, no adverse effects to chickens were
13 noted (NRC, 1977).

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

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

22 23 **7.4.2.8 Copper**

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

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

35
36 **Plants.** Copper is an essential nutrient for the growth of plants. Background concentrations of
37 copper in grasses and clovers collected in the United States averaged 9.6 mg/kg and 16.2 mg/kg

1 (dry weight) (Kabata-Pendias and Pendias, 1992). Copper is one of the least mobile heavy
2 metals in soil, and its availability to plants is highly dependent on the molecular weight of
3 soluble copper complexes (Kabata-Pendias and Pendias, 1992).

4
5 According to Rhodes, et al. (1989), copper concentrations in plant tissues do not serve as
6 conclusive evidence of copper toxicity in species of plants such as tomatoes, because some
7 species are able to tolerate higher concentrations of copper than others. The pH of soil may also
8 influence the availability and toxicity of copper in soils to plants (Rhodes et al., 1989). In a
9 study with tomato plants, Rhodes, et al. (1989) found a reduction in plant growth when plants
10 were grown in soils containing greater than 150 mg/kg of copper at a pH of less than 6.5. At pH
11 values greater than 6.5, soil copper concentrations of greater than 330 mg/kg were required to
12 reduce plant growth.

13
14 Concentrations of copper in leaf tissue that are excessive or toxic to various plant species range
15 from 20 to 100 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). A soil concentration of
16 100 mg/kg has been proposed by Efroymson et al., (1997a) as a benchmark screening value for
17 copper phytotoxicity in soil. General symptoms of copper toxicity in plants include the presence
18 of dark green leaves followed by induced iron chlorosis; thick, short, or barbed-wire roots; and
19 depressed tillering (Kabata-Pendias and Pendias, 1992).

20
21 **Terrestrial Invertebrates.** A number of studies have been conducted to study earthworm
22 toxicity with exposure to copper. The work of Streit and Jaggy (1983) and others shows that the
23 organic carbon content of the soil is a strong determinant of the bioavailability and toxicity of
24 copper. It appears that low pH has a compounding effect, with an increase in copper
25 bioavailability resulting from more acid conditions. Overall, reproduction is more sensitive than
26 mortality, and there is no consistent evidence that one genus of earthworms is any less tolerant to
27 copper under a given set of conditions than another genus.

28
29 Neuhauser, et al. (1984) evaluated the effects of soluble forms of copper on growth and
30 reproduction in *Eisenia fetida*. After 6 weeks, both growth (weight) and cocoon production were
31 decreased (75 and 85 percent) by 2,000 ppm copper, while 1,000 ppm had no effect.
32 Using OECD artificial soil (pH = 6) and a 21-day test procedure, van Gestel, et al. (1989) looked
33 at the effects of copper (as CuCl_2) on reproductive parameters of adult *Eisenia andrei*. After 21
34 days, cocoon production was decreased by 36 percent by the addition of 180 ppm copper to the
35 substrate, while 120 ppm had no effect.

1 The sublethal effects of copper on *L. rubellus* were investigated with respect to mortality,
2 growth, cocoon production, and litter breakdown activity (Ma, 1984). Loamy sand field soil (5.7
3 percent organic matter, pH = 4.8) with copper added as CuCl₂, was placed in bags with leaf litter
4 added to the top. In an experiment lasting 6 weeks, the number of cocoons produced was
5 decreased 42 percent by 131 ppm copper, while 54 ppm copper had no effect.

6
7 Efrogmson, et al. (1997b) have proposed a soil screening level of 60 ppm based on earthworm
8 toxicity.

9
10 **Mammals.** Copper is an essential trace element to plants and animals (Callahan et al., 1979),
11 but becomes toxic at concentrations only slightly higher than essential levels (EPA, 1985).
12 Copper is an essential element for hemoglobin synthesis and oxidative enzymes in animals.
13 Copper is absorbed by mammals following ingestion, inhalation, and dermal exposure. Once
14 absorbed, copper is distributed to the liver. Copper is not metabolized (Marceau et al., 1970).
15 No evidence of bioaccumulation was obtained in a study of pollutant concentrations in the
16 muscles and livers of 10 species of herbivorous, omnivorous, and carnivorous animals in Donana
17 National Park in Spain (Hernandez et al., 1985). Copper concentrations in small mammals
18 collected from various uncontaminated sites ranged from 8.3 to 13.4 mg/kg (whole-body
19 concentrations) (Talmage and Walton, 1991). Highest concentrations of copper tend to be in
20 hair, followed in decreasing concentration by liver, kidney, and whole body (Hunter and
21 Johnson, 1982). Among the small mammals collected, Hunter and Johnson (1982) found shrews
22 (*Sorex araneus*) to contain the highest concentrations of copper. Mice were found to contain the
23 lowest copper concentrations. Increased fetal mortality was observed in fetuses of mice fed
24 more than 104 mg/kg-day of copper as copper sulfate (Lecyk, 1980). Increased mortality rates in
25 mink offspring have been observed at levels above 3.21 mg/kg-day (Aulerich et al., 1982).

26
27 Laboratory toxicity data for mink exposed to copper sulfate in their diet were used to estimate a
28 NOAEL value of 11.7 mg/kg/day (Aulerich et al., 1982). Reproduction was the endpoint
29 studied. Symptoms of acute copper poisoning in mammals include vomiting, hypotension,
30 melena, coma, jaundice, and death (Klaassen et al., 1991). Selenium can act as an antidote for
31 copper poisoning.

32
33 **Birds.** Laboratory toxicity data for one-day old chicks exposed to copper oxide in their diets
34 were used to estimate a NOAEL value of 47 mg/kg/day (Mehring et al., 1960). Growth and
35 mortality were the endpoints studied. Reduced growth and mortality were seen at copper
36 concentrations as low as 749 ppm over the 10-week exposure period.

1 **Aquatic Life.** Invertebrates inhabiting [polluted] freshwaters worldwide have been known to
2 have tissue residues of copper ranging from 5 to 200 ppm (Moore and Ramamoorthy, 1984).
3 Field studies have shown that there is virtually no accumulation of this metal through the food
4 chain (Fuller and Averett, 1975). Studies by Kosalwat and Knight (1987) indicated that copper
5 present in the substrate or sediment was significantly less toxic to chironomid species than
6 overlying water column levels. The substrate copper concentration that chironomid larval
7 growth was reduced 50 percent (EC_{50}) was 1,602 mg/kg. These researchers found that
8 deformities in larval mouth parts were observed in elevated concentrations, and adult emergence
9 was inhibited when the sediment concentration exceeded 1,800 mg/kg. Carins, et al. (1984)
10 reported copper toxicity in sediment for several chironomus midges and cladocerans with LC_{50} s
11 ranging from 681 to 2296 mg/kg.

13 **7.4.2.9 Iron**

14 Iron is an essential trace element, required as a constituent of oxygen-carrying and oxidative-
15 reductive macro-molecules such as hemoglobin, myoglobin, and cytochrome P-450. As such,
16 most iron-related health concerns are induced by insufficient iron intake, rather than excess iron
17 intake (National Research Council [NRC], 1989).

18
19 **Plants.** Wallihan (1966) reported unspecified reductions in plant growth in a solution culture
20 with the addition of 10 ppm iron. Wallace et al., (1977) evaluated the effects of iron (as $FeSO_4$)
21 on leaf, stem, and root weights of bush bean seedlings grown for fifteen days in nutrient solution.
22 Iron at 28 ppm reduced all three measures 67, 52, and 67 percent, respectively, while 11.2 ppm
23 iron had no effect. After 55 days cabbage seedling plant weight was reduced 45 percent by 50
24 ppm iron added as $FeSO_4$ to nutrient solution, while 10 ppm had no effect on growth (Hara et al.,
25 1976).

26
27 Iron is the key metal required for energy transformations needed for cellular function. It occurs
28 in heme and non-heme proteins and is concentrated in chloroplasts. Organic iron complexes are
29 involved in photosynthetic electron transfer. Plant symptoms of toxicity are not specific and
30 differ among plant species and growth stages (Foy et al., 1978).

31
32 **Terrestrial Invertebrates.** No information was found regarding the toxicity of iron to
33 terrestrial invertebrates.

34
35 **Mammals.** Iron is an essential nutrient for most wildlife species and is necessary to maintain
36 homeostasis; therefore, it is only toxic at very high concentrations. Bioaccumulation factors
37 have been calculated for several small mammal species. Small herbivorous mammals were

1 estimated to have an iron bioaccumulation factor of 0.0127, and small omnivorous mammals
2 were estimated to have an iron bioaccumulation factor of 0.01209. These bioaccumulation
3 factors indicate that iron is not accumulated in small mammal tissues (Sample et al., 1998).
4 Additionally, the bioaccumulation factor for earthworms has been estimated to be 0.038,
5 indicating that iron is not accumulated in earthworm tissues (Sample et al., 1998).

6
7 **Aquatic Life.** The National Recommended Water Quality Criteria for iron (1,000 µg/L) is
8 based on field study at a site receiving acid mine drainage (EPA, 2002). The lowest chronic
9 value for daphnids (158 µg/L) is a threshold for reproductive effects from a 21-day test using
10 iron chloride with *Daphnia magna* (Dave, 1984). It is considerably lower than the 4,380 µg/L
11 concentration causing 16 percent reproductive decrement in another test using iron chloride with
12 *Daphnia magna* (Biesinger and Christensen, 1972). The lowest chronic value for fish (1,300
13 µg/L) is a concentration that caused 100 percent mortality in an embryo-larval test with rainbow
14 trout exposed to dissolved iron salts (Amelung, 1981).

15
16 The Ontario Ministry of the Environment has prepared provincial sediment quality guidelines
17 using the screening-level concentration approach, which estimates the highest concentration of a
18 particular contaminant in sediment that can be tolerated by approximately 95 percent of benthic
19 fauna (Neff et al., 1988). These values are based on Ontario sediments and benthic species from
20 a wide range of geographical areas within the province (Persaud et al., 1993). The lowest effect
21 level (Low) is the level at which actual ecotoxic effects become apparent. The severe effect
22 level (Severe) represents contaminant levels that could potentially eliminate most of the benthic
23 organisms (Persaud et al., 1993). The “Low” and “Severe” levels for iron in sediment are 2
24 percent (20,000 ppm) and 4 percent (40,000 ppm), respectively.

25 26 **7.4.2.10 Lead**

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

32
33 Lead is strongly sorbed in sediments and the rate is strongly correlated with grain size and
34 organic content. In the absence of soluble complexing species, lead is almost totally adsorbed to
35 clay particles at pHs greater than 6 (Moore and Ramamoorthy, 1984).

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

9
10 Mean background concentrations of lead in grasses and clovers have been reported to range from
11 2.1 to 2.5 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). Adverse effects of lead on
12 terrestrial plants occur only at total concentrations of several hundred mg/kg of soil (Eisler,
13 1988). This is explained by the fact that, in most cases, lead is tightly bound to soils and
14 substantial amounts must accumulate before it can affect the growth of higher plants (Bogges,
15 1977).

16
17 **Terrestrial Invertebrates.** A number of studies have been conducted to assess the potential
18 toxicity of lead to terrestrial invertebrates. Bengtsson, et al. (1986) examined the effects of lead
19 on *Dendrobaena rubida* at different soil acidities. After 4 months at pH 4.5, the number of
20 cocoons produced per worm, hatchlings per cocoon, and percent hatched cocoons were reduced
21 75, 100 and 100 percent, respectively, by 500 mg/kg lead, while 100 mg/kg lead had no effect.
22 At pH 5.5 and 6.5, lead had no adverse effects at any of the lead concentrations.

23
24 Spurgeon, et al. (1994) kept adult *Eisenia fetida* in contaminated soil for 8 weeks to examine the
25 effects of lead [as $Pb(NO_3)_2$] on survival and growth of the earthworms. After 56 days, the
26 calculated LC_{50} was 3,760 mg/kg and the EC_{50} for cocoon production was 1,940 mg/kg.

27
28 Efroymsen, et al. (1997) have established a benchmark value of 500 mg/kg for earthworms based
29 on the work of Bengtsson, et al. (1986) which showed inhibition of reproduction at this
30 concentration.

31
32 **Mammals.** As with plants, lead is not considered an essential nutrient for mammalian life.
33 Ingestion is the major route of exposure for wildlife. Lead tends to accumulate in bone, hair, and
34 teeth. Biomagnification of lead is negligible (Eisler, 1988). Reduced survival was reported at
35 acute oral doses as low as 5 mg/kg body weight in rats, at a chronic dose of 0.3 mg/kg body
36 weight in dogs, and at a dietary level of 1.7 mg/kg body weight in horses (Eisler, 1988).
37 Laboratory data from studies of rats fed lead acetate in their diets were used to estimate a

1 NOAEL value of 8.0 mg/kg/day (Azar, et al., 1973). Reproduction was the endpoint for this
2 study. Symptoms of lead poisoning in mammals are diverse and depend on the form of lead
3 ingested, the concentration, and the species and its age. These symptoms may include
4 reproductive impairment, decreased body weight, vomiting, uncoordinated body movements,
5 visual impairment, reduced life span, renal disorders, and abnormal social behavior (Eisler,
6 1988).

7
8 In laboratory studies, breeding mice exposed to low doses of lead in drinking water (25 ppm)
9 resulted in loss of the strain in two generations with many abnormalities (Schroeder and
10 Mitchner, 1971). Exposure of rats in this same experiment resulted in many early deaths and
11 runts. Blood δ -aminolevulinic acid dehydratase (ALAD) activity associated with exposure to
12 lead was reduced in white-footed mice living near a metal smelter (Beyer, et al., 1985).

13 Amounts of whole-body lead content and feeding habits of roadside rodents have been correlated
14 with highest body burdens in insectivores such as shrews, intermediate in herbivores, and lowest
15 in granivores (Boggess, 1977; Getz, et al., 1977).

16
17 **Birds.** Most of the information on the effects of lead to terrestrial vertebrates is concerned with
18 the poisoning of waterfowl by lead shot. Apparent symptoms include loss of appetite and
19 mobility, avoidance of other birds, lethargy, weakness, emaciation, tremors, dropped wings,
20 green feces, impaired locomotion, loss of balance and depth perception, nervous system damage,
21 inhibition of heme synthesis, damage to kidneys and liver, and death (Eisler, 1988; Mudge,
22 1983). Anemia, kidney disease, testicular and liver lesions, and neurological disorders have been
23 associated with high brain lead concentrations in mourning doves (*Zenaida macroura*) (Kendall,
24 1992). Hatchlings of chickens, Japanese quail, mallards and pheasants are relatively tolerant to
25 moderate lead exposure, including no effect on growth at dietary levels of 500 ppm and no effect
26 on survival at 2,000 ppm (Hoffman, et al., 1985).

27
28 Toxicity of lead to birds is dependent upon the form of lead, the route of exposure and exposure
29 duration, and the species and age of the bird. Laboratory toxicity data for American kestrels fed
30 metallic lead in their diet were used to estimate a NOAEL value of 3.85 mg/kg/day (Pattee,
31 1984). Reproduction was the endpoint for this study.

32
33 **Aquatic Life.** All life stages are sensitive to the toxic effects of lead; however, embryos are
34 more sensitive to lead than are later juvenile stages (Davies et al., 1976). Lead uptake depends
35 on exposure time, aqueous concentration, pH, temperature, salinity, diet, and other factors. For
36 example, gill, liver, kidney, and erythrocytes accumulate lead from aqueous sources in
37 proportion to exposure time and concentration (Holcombe et al., 1976). Direct erythrocyte injury

1 is considered the first and most important sign of lead poisoning in catfish (Dawson, 1935).
2 Respiratory distress occurs in fish living in rivers receiving lead mining wastes in England
3 (Carpenter, 1924; 1925; 1926). Fish are thought to be asphyxiated as a result of a mucous
4 coating over the gills (National Academy of Sciences, 1972).

5
6 No significant biomagnification of lead occurs in aquatic ecosystems (Boggess, 1977).
7 Background concentrations of lead in fish tend to be less than 1 mg/kg (dry weight) (Eisler,
8 1988). The EPA's National Recommended Water Quality Criteria for lead in freshwater is 65
9 µg/L for acute exposure and 2.5 µg/L for chronic exposure (EPA, 2002). In general, dissolved
10 lead is more toxic than total lead, and organic forms of lead are more toxic than inorganic forms.
11 Soluble lead in the water column becomes less bioavailable as water hardness increases.
12 Chronic exposure of fish to lead may result in signs of lead poisoning such as spinal curvature,
13 anemia, darkening of the dorsal tail region, destruction of spinal neurons, difficulties in
14 swimming, growth inhibition, changes in blood chemistry, retarded sexual development, and
15 death (Eisler, 1988).

16
17 The majority of benthic invertebrates do not bioconcentrate lead from water or abiotic sediment
18 particles. There is some evidence of bioaccumulation through the food web of organic forms of
19 lead, such as tetraethyl lead. Anderson, et al., (1980) reported lead LC₅₀s of 258 ppm for the
20 chironomid and that growth of this amphipod was not reduced above this level in freshwater
21 sediments. In addition, Suter and Tsao (1996) reported effect levels in the water flea (*Daphnia*
22 *magna*) to be in the 12.26 ppb range, while Khangrot and Ray (1989) reported a *D. magna* LC₅₀
23 of 4.89 ppm.

24 25 **7.4.2.11 Manganese**

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

33
34 **Plants.** Manganese is an essential element for plant growth. Uptake of manganese may occur
35 via root or foliar uptake (Kabata-Pendias and Pendias, 1992). The concentration of manganese
36 in plants is dependent upon plant and soil characteristics. Plants grown on flooded or acid soils
37 tend to contain higher concentrations of manganese than plants grown in other, uncontaminated

1 soils. In addition, concentrations of manganese in plants are positively correlated with soil
2 organic matter (Kabata-Pendias and Pendias, 1992). Concentrations of manganese in leaf tissue
3 that are excessive or toxic to various plant species range from 400 to 1,000 mg/kg dry weight
4 (Kabata-Pendias and Pendias, 1992). A soil concentration of 500 mg/kg (dry weight) has been
5 proposed by Efroymson, et al., (1997) as a benchmark screening value for manganese
6 phytotoxicity. General symptoms of manganese toxicity in plants include the presence of
7 chlorosis and necrotic lesions on old leaves, blackish-brown or red necrotic spots, dried leaf tips,
8 and stunted root and plant growth (Kabata-Pendias and Pendias, 1992).

9
10 **Terrestrial Invertebrates.** No information was found regarding the potential toxicity to
11 terrestrial invertebrates from exposure to manganese.

12
13 **Mammals.** Manganese is an essential nutrient that is homeostatically regulated in vertebrates
14 (Vanderploeg, et al., 1975). Liver and kidney tissues generally contain the highest
15 concentrations of manganese in the body. Manganese in the body is primarily excreted in the
16 feces (Gregus and Klaassen, 1986).

17
18 Divalent manganese is more toxic than the trivalent form. Exposure to manganese dust via
19 inhalation is usually of greater toxicological concern than ingestion (Klaassen, et al., 1991).
20 Laboratory data for rats fed manganese oxide in their diet were used to estimate a NOAEL value
21 of 88 mg/kg/day (Laskey, et al., 1982). Reproduction was the endpoint for this study.
22 Laboratory studies with rats have found no hematologic, behavioral, or histologic effects in
23 animals exposed to manganese dioxide at concentrations of 47 mg/m³ for five hours per day, five
24 days a week, for 100 days (Klaassen, et al., 1991).

25
26 **Birds.** Japanese quail were studied by Laskey and Edens (1985) to determine the toxic effects
27 of 5,000 ppm manganese fed to one-day old chicks. Growth and aggressive behavior were
28 studied throughout the 75-day exposure period. While no reduction in growth was observed,
29 aggressive behavior was reduced in 25 to 50 percent of the chicks relative to controls. However,
30 reduced aggressive behavior was not considered a significant adverse affect. A NOAEL value of
31 977 mg/kg/day was derived from this study.

32
33 **Aquatic Life.** As discussed previously, manganese is a required nutrient for plant and animal
34 life. Manganese concentrations in most vertebrates are homeostatically controlled (Vanderploeg,
35 et al., 1975). Bioconcentration factors for freshwater macrophytes have been reported to range
36 from 190 to approximately 25,000 (Vanderploeg, et al., 1975). With regard to freshwater fish,
37 concentrations of manganese in fish muscle are generally less than 0.5 mg/kg and range from 3

1 to 10 mg/kg in whole fish (Vanderploeg, et al., 1975). Bioconcentration factors from water to
2 whole fish range from 40 to 2,300. A bioconcentration factor of 10,000 was also suggested for
3 crustaceans (Vanderploeg, et al., 1975).

4
5 No Federal water quality criteria exist for the protection of freshwater biota from elevated
6 manganese concentrations. Suter and Tsao (1996) have estimated acute and chronic advisory
7 levels for manganese to be 1,470 and 80.3 $\mu\text{g/L}$, respectively. The EC_{20} for fish can be used as a
8 benchmark indicative of production within a population. It is the highest tested concentration
9 causing less than 20 percent reduction in either weight of young fish per initial female fish in a
10 life-cycle or partial life-cycle test, or the weight of young per egg in an early life-stage test (Suter
11 and Tsao, 1996). The EC_{20} value for manganese is 1,270 $\mu\text{g/L}$. A similar value can be
12 determined for daphnids, which reflects the highest tested concentration causing less than 20
13 percent reduction in the product of growth, fecundity, and survivorship in a chronic test with a
14 daphnid species. The EC_{20} for daphnids is less than 1,100 $\mu\text{g/L}$ (Suter and Tsao, 1996).

15 16 **7.4.2.12 Mercury**

17 Mercury is a toxic compound with no known natural biological function. Mercury exists in three
18 valence states: mercuric (Hg^{2+}), mercurous (Hg^{1+}), and elemental (Hg^{0+}) mercury. It is present in
19 the environment in inorganic and organic forms. Inorganic mercury compounds are less toxic
20 than organomercury compounds; however, the inorganic forms are readily converted to organic
21 forms by bacteria commonly present in the environment. The organomercury compound of
22 greatest concern is methylmercury (EPA, 1999).

23
24 Mercury sorbs strongly to soil and sediment. Elemental mercury is highly volatile. In aquatic
25 and terrestrial receptors, some forms of mercury, especially organomercury compounds,
26 bioaccumulate significantly and biomagnify in the food chain. In all receptors, the target organs
27 are the kidney, and central nervous system. However, mercury causes numerous other effects
28 including teratogenicity and mutagenicity (EPA, 1999).

29
30 **Plants.** Mercury is not required for plant growth. Background concentrations of mercury in
31 plants usually range from 0.0026 to 0.086 mg/kg (dry weight) (Kabata-Pendias and Pendias,
32 1992). Pine needles have been reported to be good biomonitors of mercury-contaminated
33 environments (Kabata-Pendias and Pendias, 1992). In general, the concentration of mercury in
34 plants will be elevated when mercury concentrations in soils are high. Mercury concentrations in
35 plants, however, generally do not exceed those in associated soils (Lisk, 1972). Methyl mercury
36 is more available to plants than either phenyl- or sulfide-mercury. In addition to mercury uptake

1 from the soil, plants can also absorb mercury vapor (Browne and Fang, 1978).

2
3 Concentrations of mercury in leaf tissue that are excessive or toxic to various plant species range
4 from 1 to 3 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). A soil concentration of 0.3
5 mg/kg has been proposed by Efroymsen, et al., (1997a) as a benchmark screening value for
6 mercury phytotoxicity. General symptoms of mercury toxicity in plants include severe stunting
7 of seedlings and roots and leaf chlorosis and browning of leaf points (Kabata-Pendias and
8 Pendias, 1992).

9
10 **Terrestrial Invertebrates.** Abbasi and Soni (1983) exposed earthworms (*Octochaetus*
11 *pattoni*) to mercury (as HgCl) to assess the effect on reproduction and growth. Survival and
12 cocoon production were reduced at 0.5 ppm mercury, the lowest concentration tested. The
13 number of juveniles produced was not affected. Based on these test data, a benchmark value for
14 mercury in soil of 0.1 mg/kg has been proposed by Efroymsen, et al., (1997b).

15
16 **Mammals.** Mercury is not an essential element for animal life. Background mercury
17 concentrations in wildlife tend to be less than 1.0 mg/kg (wet weight) (Eisler, 1987a).
18 Biomonitoring studies have shown that mercury concentrations in mammals are highest in hair,
19 followed by kidney and liver tissues (Bull, et al., 1977; Klaassen, 1991; Wren, 1986). Mercury
20 is bioaccumulated and biomagnified in terrestrial food chains (Eisler, 1987a; Talmage and
21 Walton, 1993). Talmage (1989) has shown the insectivorous shorttail shrew (*Blarina*
22 *brevicauda*) to be a better monitor of environmental mercury contamination than the granivorous
23 white-footed mouse (*Peromyscus leucopus*). Mink (*Mustela vison*) and river otter (*Lutra*
24 *canadensis*) have been shown to be good monitors of mercury contamination within river
25 environments due to their consumption of contaminated fish (Kucera, 1983).

26
27 Organic mercury compounds, especially methyl mercury, are more toxic to mammals than
28 inorganic forms of mercury. Selenium has been shown to have a protective effect against
29 mercury poisoning (Ganther, et al., 1972). Based on laboratory data for methylmercury fed to
30 rats and mink in their diets, a NOAEL value of 0.015 has been derived. This NOAEL is based
31 on mortality, weight loss, reproduction, and ataxia as endpoints (Wobeser, et al., 1976).

32
33 Mercury has been shown to be teratogenic, mutagenic, and carcinogenic in animal studies
34 (Eisler, 1987a). Signs of mercury poisoning that have been observed in mink include anorexia,
35 weight loss, ataxia and splaying of hind legs, irregular vocalization, salivation, and convulsions
36 (Wren, 1986).

1 **Birds.** Concentrations of mercury that are acutely toxic to birds following oral exposure range
2 from 2.2 to 31 mg/kg body weight (Eisler, 1987a). Mercury concentrations in the livers of
3 methylmercury-poisoned birds ranged from 17 to 70 mg/kg (dry weight) (Solonen and Lodenius,
4 1984). Methylmercury is more toxic to avian species than inorganic mercury (Hill, 1981). In
5 addition to the form of mercury to which the bird is exposed, the species, gender, age, and health
6 of the individual may also influence the toxic response (Fimreite, 1979). Physical signs of
7 mercury poisoning in birds include muscular incoordination, falling, slowness, fluffed feathers,
8 calmness, withdrawal, hyporeactivity, and eyelid drooping (Eisler, 1987a).

9
10 Japanese quail were fed mercury in their diet for one year to study the effects on reproduction.
11 Egg production increased with increasing mercury dose, while fertility and hatchability
12 decreased. Adverse effects of mercury exposure were evident at the 8 mg/kg dose level. Based
13 on the results of this study a NOAEL value of 0.45 mg/kg/day has been derived (Hill and
14 Schaffner, 1976). Mallard ducks fed methyl mercury dicyandiamide in their diets produced
15 fewer eggs and fewer ducklings at exposure levels as low as 0.5 mg/kg. A NOAEL value of
16 0.0064 mg/kg/day was derived from these data, with reproduction the endpoint studied (Heinz,
17 1979).

18
19 **Aquatic Life.** Concentrations of mercury in freshwater fish collected from 12 monitoring
20 stations in the United States from 1978 to 1981 ranged from 0.1 to 1.1 mg/kg (wet weight), with
21 an average of 0.11 mg/kg (Lowe, et al., 1985). Elevated concentrations of mercury in fish have
22 often been associated with low pH, low calcium concentrations in the water, and low water
23 hardness (Eisler, 1987a). Methylating bacteria in sediments actively convert inorganic mercury
24 into methylmercury. This results in an increase in the bioavailability of mercury. Fish absorb
25 methylmercury more easily than inorganic mercury from the water column (Huckabee, et al.,
26 1979). Because exposure of fish to methylmercury can occur via ingestion of contaminated prey,
27 methylmercury concentrations are usually highest in organisms near the top of the food chain,
28 such as carnivorous fish (Huckabee, et al., 1979).

29
30 Exposure of aquatic organisms to elevated mercury concentrations can result in reduced growth
31 and reproduction (Eisler, 1987a). The National Recommended Water Quality Criteria for acute
32 and chronic exposure to mercury in freshwater systems are 1.4 and 0.77 µg/L, respectively
33 (EPA, 2002). The test EC₂₀ for fish can be used as a benchmark indicative of production within
34 a population. It is the highest tested concentration causing less than 20 percent reduction in
35 either the weight of young fish per initial female fish in a life-cycle or partial life-cycle test, or
36 the weight of young per egg in an early life-stage test. The EC₂₀ value for methylmercury is less
37 than 0.03 µg/L (Suter and Tsao, 1996). A similar value can be determined for daphnids, which

1 represents the highest tested concentration causing less than 20 percent reduction in the product
2 of growth, fecundity, and survivorship in a chronic test with a daphnid species. The EC₂₀
3 benchmark for daphnids has been determined to be 0.87 µg/L (Suter and Tsao, 1996).

4
5 Physical signs of acute mercury poisoning in fish include the flaring of gills, an increase in the
6 frequency of respiratory movements, loss of equilibrium, and sluggishness (Armstrong, 1979).

7 8 **7.4.2.13 Nickel**

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

13
14 **Plants.** Nickel is not believed to be an essential element for plant growth; however, beneficial
15 effects of nickel have been reported on the growth of legumes. Background concentrations of
16 nickel in grasses and clovers collected in the United States averaged 0.13 and 1.5 mg/kg,
17 respectively (Kabata-Pendias and Pendias, 1992). The concentration of nickel in plants is
18 positively correlated with nickel concentrations in soil.

19
20 Concentrations of nickel in leaf tissue that are excessive or toxic to plant species range from 10
21 to 100 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). A soil concentration of 30 mg/kg
22 has been proposed by Efrogmson, et al. (1997a) as a benchmark screening value for nickel
23 phytotoxicity. General symptoms of nickel toxicity in plants include the presence of interveinal
24 chlorosis in new leaves, gray-green leaves, and brown and stunted root and plant growth. The
25 uptake of nutrients and minerals, especially iron, can be substantially reduced as a consequence
26 of nickel toxicity in plants (Kabata-Pendias and Pendias, 1992).

27
28 **Terrestrial Invertebrates.** The effects of nickel on *Eisenia fetida* were investigated by
29 Malecki, et al. (1982). The most sensitive endpoint was cocoon production. In the 8-week test,
30 300 mg/kg nickel caused a 41 percent decrease in cocoon production, while 200 mg/kg had no
31 adverse effect. In the 20-week test, 200 mg/kg nickel caused a 23 percent decrease in cocoon
32 production, while 100 mg/kg had no effect.

33
34 Neuhauser, et al. (1984) evaluated the effects of soluble forms of nickel on growth and
35 reproduction in *Eisenia fetida*. After 6 weeks cocoon production was decreased 33 percent by
36 250 mg/kg nickel, the lowest concentration tested. Growth was not affected until 500 mg/kg
37 nickel was added to the substrate.

1
2 Efroymsen, et al. (1997b) have established a soil benchmark value for nickel of 200 mg/kg based
3 on the work of Malecki, et al. (1982), which showed inhibition of reproduction at this
4 concentration.

5
6 **Mammals.** Nickel is a nonessential element for animal life. Nickel concentrations within the
7 whole bodies of small mammals from uncontaminated sites were reported to range from 2.2 to
8 6.2 mg/kg (dry weight) (Talmage and Walton, 1991). Highest concentrations were measured in
9 the deer mouse (*Peromyscus maniculatus*). Highest tissue concentrations of nickel are usually
10 found in the liver of mammals (Schroeder, et al., 1964). Because nickel is poorly absorbed by
11 the gastrointestinal tract, ingested nickel is generally not of great toxicological concern. Inhaled
12 nickel, however, is relatively toxic. Rats fed nickel in their diet as nickel sulfate hexahydrate
13 over three generations were studied for effects on reproduction. They were fed three dose levels
14 (250, 500, and 1,000 ppm Ni) in their diet, and only the highest dose level caused reduced
15 offspring body weights. No adverse effects were observed at the other dose levels. Because this
16 study considered exposures over multiple generations, the 500 ppm dose was considered to be
17 the chronic NOAEL, and the 1,000 ppm dose was considered to be the chronic LOAEL (EPA,
18 2002). These data were used to derive a NOAEL value of 40 mg/kg/day (Ambrose, et al, 1976).
19 Reproduction was the endpoint studied.

20
21 **Birds.** Mallard ducklings were fed nickel as nickel sulfate in their diet for a duration of 90 days
22 to study the effects on mortality, growth, and behavior. They were fed three dose levels (176,
23 774, and 1,069 ppm Ni), and only the highest dose reduced growth and resulted in 70 percent
24 mortality. Because the study considered exposure over 90 days, the 774 ppm dose was
25 considered to be the chronic NOAEL, and the 1,069 dose was considered to be the chronic
26 LOAEL (Cain and Pafford, 1981). A NOAEL value of 77.4 mg/kg/day was derived from this
27 study based on mortality, growth, and behavior as endpoints.

28
29 **Aquatic Life.** The bioavailability and toxicity of nickel to aquatic biota is influenced by the pH
30 of the water (Schubauer-Berigan, et al., 1993). The national recommended water quality criteria
31 for the protection of aquatic life for acute and chronic exposure are 470 and 52 µg/L,
32 respectively (EPA, 2002). Background concentrations of nickel in adult anurans ranged between
33 0.9 and 2.9 mg/kg (dry weight) (Hall and Mulhern, 1984).

34
35 The test EC₂₀ for fish can be used as a benchmark indicative of production within a population.
36 It is the highest tested concentration causing less than 20 percent reduction in either the weight of
37 young fish per initial female fish in a life-cycle or partial life-cycle test, or the weight of young

1 per egg in an early life-stage test. The EC₂₀ value for nickel is 62 µg/L (Suter and Tsao, 1996).
2 A similar value can be determined for daphnids which represents the highest tested concentration
3 causing less than 20 percent reduction in the product of growth, fecundity, and survivorship in a
4 chronic test with a daphnid species. The EC₂₀ benchmark for daphnids has been determined to
5 be 45 µg/L (Suter and Tsao, 1996).

7 **7.4.2.14 Selenium**

8 Selenium is distributed widely in nature and is found in most rocks and soils at concentrations
9 between 0.1 and 2.0 mg/kg (Fishbein, 1981). The primary factor determining the fate of
10 selenium in the environment is its oxidation state. Selenium is stable in four valence states (-2,
11 0, +4, and +6) and forms chemical compounds similar to those of sulfur. The selenides (-2) are
12 insoluble in water, as is elemental selenium. The inorganic alkali selenites (+4) and the selenates
13 (+6) are soluble in water and are, therefore, more bioavailable.

14
15 Conditions such as pH, Eh, and the presence of metal oxides affect the partitioning of the various
16 compounds of selenium in the environment. In general, elemental selenium is stable in soils and
17 is found at low levels in water because of its ability to co-precipitate with sediments. The
18 soluble selenates are readily taken up by plants and converted to organic compounds such as
19 selenomethionine, selenocysteine, dimethyl selenide, and dimethyl diselenide. Selenium is
20 bioaccumulated by aquatic organisms and may also biomagnify in aquatic organisms.

21
22 **Plants.** The role of selenium in plant growth is not fully understood. It is generally not
23 considered essential in plant nutrition (Kabata-Pendias and Pendias, 1992). The concentration of
24 selenium in plants has been shown to be positively correlated with the concentration of selenium
25 in soil. Soil parameters such as pH, oxidation-reduction potential, and moisture content
26 determine the amount of selenium available for plant uptake. Concentrations of selenium in leaf
27 tissues that have been shown to be toxic to various plant species range from 5 to 30 mg/kg
28 (Kabata-Pendias and Pendias, 1992). General symptoms of selenium toxicity in plants include
29 the signs of interveinal chlorosis or black spots in plants containing approximately 4 mg/kg
30 selenium, complete bleaching or yellowing of younger leaves at higher concentrations, and the
31 presence of pinkish spots on roots (Kabata-Pendias and Pendias, 1992).

32
33 **Terrestrial Invertebrates.** Fischer and Koszorus (1992) tested the effects of 77 ppm of
34 selenium on growth and reproduction of *Eisenia fetida* when added to a combination of peaty
35 marshland soil and horse manure (1:1). The number of survivors and their live mass and number
36 of cocoons produced were measured. The number of cocoons produced per worm showed the
37 highest sensitivity to selenium with a 69 percent reduction at a selenium concentration of 77

1 ppm. Efroymson, et al. (1997) have proposed a soil benchmark value of 70 ppm based on this
2 study.

3
4 **Mammals.** Selenium is an essential trace element for animal life. Concentrations that are
5 essential to animals are in the range of 0.05 to 0.1 mg/kg in the diet (Arthur, et al., 1992).
6 According to Ganther (1974), selenium concentrations in healthy, unexposed, laboratory animals
7 and livestock range between 0.1 and 1 mg/kg. Selenium offers a protective effect against some
8 carcinogens such as benzo(a)pyrene and benzo(a)anthracene (Hammond and Beliles, 1980).
9 Selenium also functions as an antidote to the toxic effects of mercury, thallium, copper, arsenic
10 and cadmium (Frost and Lish, 1975).

11
12 Acute poisoning has been reported in livestock that consumed plant material containing 400 to
13 800 mg/kg selenium (Eisler, 1985b). Signs of acute poisoning in livestock include abnormal
14 movements, lowered head, drooped ears, diarrhea, elevated temperature, rapid pulse, labored
15 breathing, bloating with abdominal pain, increased urination, and dilated pupils (Eisler, 1985b).
16 Chronic poisoning may occur in animals exposed to dietary selenium concentrations between 1
17 and 44 mg/kg (Eisler, 1985b). Laboratory data from studies using rats fed potassium selenate
18 (SeO_4) in their drinking water were used to derive a NOAEL value of 0.2 mg/kg/day (Rosenfeld
19 and Beath, 1954). Reproduction was the endpoint in this study.

20
21 **Birds.** Toxicity from selenium has also been documented in birds. The major toxic effect of
22 selenium on avian species is on reproductive success. Both sodium selenite and
23 selenomethionine have been reported to be embryotoxic and teratogenic (Heinz, et al., 1987).
24 Reproductive impairment is likely to occur as concentrations of selenium approach 5 mg/kg.
25 Mortality in mallard ducklings does not occur until selenium concentrations in the diet reach 40
26 mg/kg. While consumption of 1, 5, or 10 ppm selenium in the diet of mallard ducks had no
27 effect on weight or survival of adults, 100 ppm selenium reduced adult survival and 25 ppm
28 selenium reduced duckling survival. Consumption of 10 or 25 ppm selenium in the diet resulted
29 in significantly larger frequency of lethally deformed embryos compared to the 1 or 5 ppm
30 selenium exposures. These data were used to derive a NOAEL value of 0.5 mg/kg/day, with
31 reproduction as the critical endpoint (Heinz, et al., 1987).

32
33 Screech owls fed selenium in their diets were studied for the effects on reproduction. While
34 exposure of owls to 0.44 mg/kg/day of selenomethionine had no adverse effects on reproduction,
35 exposure to 1.5 mg/kg/day reduced egg production, hatchability, and nestling survival (Sample,
36 et al., 1996). A NOAEL value of 0.44 mg/kg/day was derived from these data based on
37 reproductive effects.

1
2 Black-crowned night herons fed selenium their diets for 94 days were studied for the effects on
3 reproduction. Exposure to 1.8 mg/kg/day selenium had no adverse effects on reproduction.
4 These data were used to derive a NOAEL value of 1.8 mg/kg/day based on reproduction as the
5 critical endpoint (Sample, et al., 1996).
6

7 ***Aquatic Life.*** Selenium is an essential micro-nutrient for fish. Dietary requirements of
8 selenium for fish range from 0.07 to 0.25 mg/kg, depending on the fish species (Gatlin and
9 Wilson, 1984). The bioconcentration of selenium from water is highly dependent on the species
10 of selenium present. Laboratory studies have shown bioconcentration factors for
11 selenomethionine to be greater than those for selenite and selenate. Bioconcentration factors for
12 aquatic biota exposed to 1 µg/L selenomethionine were approximately 16,000 for algae, 200,000
13 for daphnids, and 5,000 for bluegills (Besser, et al., 1993).
14

15 The EPA's National Recommended Water Quality Criteria for Priority Toxic Pollutants for
16 selenium in freshwater is 5 µg/L for chronic exposure (EPA, 2002). The toxicity of selenium to
17 freshwater fish appears to be correlated more closely with dietary than waterborne exposure
18 (Coyle, et al., 1993). Sulfate concentrations in water may also influence the toxicity of selenium
19 to aquatic invertebrates (Maier, et al., 1993).
20

21 The test EC₂₀ for fish can be used as a benchmark indicative of production within a population.
22 It is the highest tested concentration causing less than 20 percent reduction in the weight of
23 young fish per initial female fish in a life-cycle or partial life-cycle test, or the weight of young
24 per egg in an early life-stage test. The EC₂₀ for selenium is 40 µg/L (Suter and Tsao, 1996). A
25 similar value can be determined for daphnids, which reflects the highest tested concentration
26 causing less than 20 percent reduction in the product of growth, fecundity, and survivorship in a
27 chronic test with a daphnid species. The EC₂₀ benchmark for daphnids is 25 µg/L selenium
28 (Suter and Tsao, 1996).
29

30 **7.4.2.15 Thallium**

31 Thallium is widely distributed in trace amounts in the earth's crust and is one of the more toxic
32 metals. In the environment, thallium exists in either the monovalent (thallous) or trivalent
33 (thallic) form. Thallium is chemically reactive with air and moisture, undergoing oxidation.
34 Thallium is relatively insoluble in water. Thallium adsorbs to soil and sediment and is not
35 transformed or biodegraded (Callahan et al., 1979).
36

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

8
9 **Terrestrial Invertebrates.** No information was found regarding the potential toxicity of
10 thallium to terrestrial invertebrates.

11
12 **Mammals.** Birds and mammals are exposed to thallium via ingestion of soil, water, and plant
13 material. In mammals, thallium is absorbed primarily from ingestion and is distributed to several
14 organs and tissues, with the highest levels reported in the kidneys (Manzo et al., 1982). Thallium
15 exposure in mammals causes cardiac, neurologic, reproductive, and dermatological effects.
16 Various effects and toxic responses have been reported, including paralysis and pathological
17 changes in the liver, kidneys, and stomach mucosa in rabbits exposed to thallium (Tikhonova,
18 1967). Testicular toxicity in rats has also been reported (Formigli et al., 1986).

19
20 Laboratory toxicity data for rats exposed to thallium sulfate in their drinking water for 60 days
21 were used to estimate a NOAEL value of 0.0074 mg/kg/day (Formigli et al., 1986). Reduced
22 sperm motility in males was the endpoint for this study.

23
24 **Birds.** Studies on the chronic exposures of birds to thallium were not found in the literature.
25 An acute LD₅₀ of 35 mg/kg/day was derived by Schafer (1972) using European starlings. An
26 uncertainty factor of 100 was applied to this LD₅₀ value to derive a chronic toxicity reference
27 value (TRV) of 0.35 mg/kg/day (EPA, 1999).

28
29 **Aquatic Life.** In aquatic organisms, thallium is absorbed primarily from ingestion and
30 thereafter bioconcentrates in the organism. Toxic effects have been observed in numerous
31 aquatic organisms including daphnia, fat-head minnow, bluegill sunfish, and others (EPA,
32 1980a). The Tier II secondary acute water quality value and secondary chronic water quality
33 value for beryllium, as calculated by the method described in the EPA's *Water Quality Guidance*
34 *for the Great Lakes System* (EPA, 1995), are 110 and 12 µg/L, respectively.

35
36 The test EC₂₀ for fish can be used as a benchmark indicative of production within a population.
37 It is the highest tested concentration causing less than 20 percent reduction in either the weight of

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

7 8 **7.4.2.16 Vanadium**

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

14
15 **Plants.** There is some controversy over whether vanadium is an essential element for plants
16 (Kabata-Pendias and Pendias, 1992). It appears to be required by some algal species and may be
17 required by nitrogen-fixing bacteria. Mean background concentrations of vanadium in plants are
18 1.6 mg/kg for angiosperms, 0.69 mg/kg for gymnosperms, and 0.67 mg/kg for fungi (Waters,
19 1977). The availability of vanadium to plants is highly dependent on soil pH. Elevated levels of
20 vanadium in soil can reduce the uptake of manganese, copper, calcium, and phosphorus (NRCC,
21 1980).

22
23 Concentrations of vanadium in leaf tissue that are excessive or toxic to various plant species
24 range from 5 to 10 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). A soil
25 concentration of 2 mg/kg has been proposed by Efroymson, et al. (1997) as a benchmark
26 screening value for vanadium phytotoxicity.

27
28 **Terrestrial Invertebrates.** No information was found regarding the toxicity of vanadium to
29 terrestrial invertebrates.

30
31 **Mammals.** Vanadium has been shown to be essential in the diets of rats (Waters, 1977).
32 Background concentrations of vanadium in the kidneys and livers of wild mammals have been
33 reported to range from 0 to 2.07 mg/kg, and from 0 to 0.94 mg/kg, respectively (Waters, 1977).
34 Liver and skeletal tissues usually contain the highest concentrations of vanadium (Waters, 1977).
35 Experimental animal investigations have suggested that the liver, adrenal, and bone marrow may
36 be adversely affected by subacute exposure to high levels of vanadium (ATSDR, 1992c;
37 Klaassen, et al., 1991). Vanadium fed to rats prior to gestation, during gestation, and through

1 delivery and lactation was studied for effects on reproduction. The rats were fed three dose
2 levels (5, 10, and 20 mg NaVO₃/kg/day or 2.1 mg V/kg/day). Significant differences in
3 reproductive parameters (e.g., number of dead young per litter, size and weight of offspring)
4 were observed at all dose levels. Therefore, the lowest dose was considered to be a chronic
5 LOAEL. A chronic NOAEL was estimated by applying an uncertainty factor of 0.1 (chronic
6 NOAEL = 0.21 mg V/kg/day) (EPA, 2002).

7
8 Based on oral intubation exposure of rats to sodium metavanadate by Domingo, et al. (1986), an
9 estimated NOAEL value of 0.21 mg/kg/day has been derived. Reproduction was the endpoint
10 for this study. Signs of acute toxicity in animals include alterations in nervous system responses,
11 gastrointestinal distress, hemorrhaging, paralysis, convulsions, and respiratory depression
12 (Klaassen, et al., 1991).

13
14 **Birds.** Mallard ducks were fed vanadium as vanadyl sulfate in their diet for 12 weeks and
15 observed for effects on mortality, body weight, and blood chemistry. The ducks were fed three
16 different doses (2.84, 10.36, and 110 ppm V). No effects were observed at any of the dose
17 levels. Because this study was greater than ten weeks in duration and did not consider a critical
18 life stage, the maximum dose was considered to be a chronic NOAEL (White and Dieter, 1978).
19 From these data a NOAEL value of 11.4 mg/kg/day has been estimated.

20
21 **Aquatic Life.** Background concentrations of vanadium in freshwater fish are usually less than
22 2.5 mg/kg (wet weight) (Jenkins, 1980). A bioconcentration factor of 3,000 has been reported
23 for aquatic invertebrates exposed to vanadium (Neumann, 1976). No federal ambient water
24 quality criteria exist for the protection of freshwater biota (EPA, 2002). The test EC₂₀ for fish
25 can be used as a benchmark indicative of production within a population. It is the highest tested
26 concentration causing less than 20 percent reduction in either the weight of young fish per initial
27 female fish in a life-cycle or partial life-cycle test, or the weight of young per egg in an early life-
28 stage test. The EC₂₀ value for vanadium is 41 µg/L (Suter and Tsao, 1996). A similar value can
29 be determined for daphnids that represents the highest tested concentration causing less than 20
30 percent reduction in the product of growth, fecundity, and survivorship in a chronic test with a
31 daphnid species. The EC₂₀ benchmark for daphnids has been determined to be 430 µg/L (Suter
32 and Tsao, 1996).

33 34 **7.4.2.17 Zinc**

35 Zinc is a naturally occurring element which may be found in both organic and inorganic forms
36 and, as such, is commonly found in the environment. In general, zinc is concentrated in the
37 sediments of water bodies. NAS (1977) has reported that zinc will probably be detected in 75

1 percent of all water bodies examined for the compound at various locations. The fate of zinc in
2 soils appears to have a pH basis. Studies have shown that a pH of less than 7 often favors zinc
3 desorption (EPA, 1984).

4
5 **Plants.** Background concentrations of zinc in terrestrial plants range from 25 to 150 mg/kg (dry
6 weight) (NAS, 1979). The deficiency content of zinc in plants is between 10 and 20 ppm (dry
7 weight). Roots often contain the highest concentrations of zinc (Kabata-Pendias and Pendias,
8 1992).

9
10 Certain species of plants, particularly those from the families *Caryophyllaceae*, *Cyperaceae*, and
11 *Plumbaginaceae*, and some tree species are extremely tolerant to elevated zinc concentrations
12 (Kabata-Pendias and Pendias, 1992). Concentrations of zinc in these plants may reach 1 percent
13 (dry weight) in the plant. Concentrations in leaf tissue that are excessive or toxic to various plant
14 species range from 100 to 400 mg/kg. Concentrations of 100 to 500 mg/kg are expected to result
15 in a 10 percent loss in crop yield (Kabata-Pendias and Pendias, 1992). General symptoms of
16 zinc toxicity in plants include the presence of chlorotic and necrotic leaf tips, interveinal
17 chlorosis in new leaves, retarded growth of the entire plant, and injured roots that resemble
18 barbed wire (Kabata-Pendias and Pendias, 1992).

19
20 **Terrestrial Invertebrates.** Spurgeon and Hopkin (1996) exposed the earthworm *Eisenia*
21 *fetida* to zinc in soils with differing organic matter content and soil pH. The EC₅₀ concentrations
22 for cocoon production in soils with a pH of 7.0 and 5 percent, 10 percent, and 15 percent organic
23 matter were 136, 462, and 592 mg/kg, respectively. The EC₅₀ concentrations for cocoon
24 production in soils of pH 6.0 and 5 percent, 10 percent, and 15 percent organic matter were 199,
25 343, and 548 mg/kg, respectively. The EC₅₀ concentrations for cocoon production in soils of pH
26 5.0 and 5 percent, 10 percent, and 15 percent organic matter were 142, 189, and 230 mg/kg,
27 respectively. Mortality was observed at higher zinc concentrations. A decrease in pH and/or
28 organic matter content in soil led to a lower toxic concentration of zinc.

29
30 Neuhauser, et al., (1985) determined an LC₅₀ for zinc in soil using adult *Eisenia fetida* exposed
31 for 14 days. The calculated LC₅₀ was 662 mg/kg. Data from the preceding studies were used to
32 derive a soil benchmark value for zinc in soil of 100 mg/kg (Efroymson, et al., 1997b).

33
34 **Mammals.** Zinc is an essential trace element for normal fetal growth and development.
35 However, exposure to high levels of zinc in the diet has been associated with reduced fetal
36 weights, altered concentrations of fetal iron and copper, and reduced growth in offspring (Cox, et
37 al., 1969). Poisoning has been observed in ferrets and mink from chewing corroded galvanized

1 cages (Clark, et al., 1981). Symptoms of zinc toxicity are lassitude, slower tendon reflexes,
2 bloody enteritis, diarrhea, lowered leukocyte count, depression of the central nervous system,
3 and paralysis of the extremities (Venugopal and Luckey, 1978). A study by Kinnamon (1963)
4 showed a NOAEL for oral exposure to a zinc compound over a period of 73 days to be 250
5 mg/kg body weight, and mice given 500 mg/L of zinc, as zinc sulfate, in drinking water have
6 shown hypertrophy of the adrenal cortex and pancreas. Young animals are much more
7 susceptible to poisoning by zinc than are mature animals (Clark, et al., 1981).

8
9 Animals are quite tolerant to high concentrations of zinc in the diet. Levels 100-times that
10 required in the diet usually do not cause detectable symptoms of toxicosis (NAS, 1979).
11 Laboratory data for rats exposed to zinc oxide in their diet were used to estimate a NOAEL value
12 of 160 mg/kg/day (Schlicker and Cox, 1968). Reproduction was the endpoint studied.
13 Symptoms of zinc poisoning in mammals include lameness, acute diarrhea, and vomiting (Eisler,
14 1993).

15
16 **Birds.** Dietary zinc concentrations of greater than 2,000 mg/kg diet are known to result in
17 reduced growth of domestic poultry and wild birds (Eisler, 1993). Reduced survival has been
18 documented at zinc concentrations greater than 3,000 mg/kg diet or at a single dose of greater
19 than 742 mg/kg body weight (Eisler, 1993). Laboratory data for white leghorn hens exposed to
20 zinc sulfate in their diet for 44 weeks were used to estimate a NOAEL value of 14.5 mg/kg/day
21 (Stahl, et al., 1990). Reproduction was the endpoint for this study. A value of 51 mg/L has been
22 calculated as the NOAEL for chronic exposure of birds to zinc carbonate in drinking water
23 (Sample, et al., 1996).

24
25 **Aquatic Life.** Zinc residues in freshwater and marine fish are generally much lower than those
26 found in algae and invertebrates. Thus there is little evidence for accumulation (Moore and
27 Ramamoorthy, 1984). Rainbow trout (*Oncorhynchus mykiss*) have the ability to detect and avoid
28 areas of water containing 5.6 ppb zinc (Sprague, 1968). Cairns and Scheier (1968) reported 96-
29 hour LC₅₀s ranging from 10.13 to 12.5 ppm in hard water for bluegills (*Lepomis macrochirus*),
30 and 96-hour LC₅₀s ranging from 2.86 to 3.78 ppm in soft water. These results demonstrate that
31 water hardness affects the toxicity of zinc to fish. Chronic toxicity tests have been conducted
32 with five species of freshwater fish. Chronic values ranged from 47 micrograms per liter (µg/L)
33 for flagfish (*Jordanella floridae*) to 852 µg/L for brook trout (*Salvenius fontinalis*) (EPA, 1980).

34
35 Acute toxicity to freshwater invertebrates is relatively low, and as with other metals, increasing
36 water hardness decreases the toxicity of zinc (Moore and Ramamoorthy, 1984). As reported by
37 Baudouin and Scoppa (1974), the 48-hour LC₅₀ for the cladoceran *Daphnia hyalina* was 0.055

1 mg/L, and 5.5 mg/L for the copepod *Cyclops abyssorum*. Four chronic toxicity tests are reported
2 for *Daphnia magna*, with chronic values ranging from 47 µg/L to 136 µg/L (EPA, 1980).
3 Chronic testing with the saltwater species *Mysidopsis bahia* resulted in a chronic value of 166
4 µg/L (EPA, 1980).

6 **7.4.2.18 Benzene**

7 Both natural and artificial sources of benzene exist. Natural sources include volcanoes, crude
8 oil, forest fires, and volatile plant components (IARC, 1982 and Graedel, 1978). Artificial
9 sources are related to benzene's use in gasoline and as a solvent (NLM, 1996). Benzene at the
10 surface of soils is expected to rapidly volatilize. The compound will be mobile in soil and is
11 expected to leach into groundwater. Benzene in aquatic environments is expected to volatilize
12 rapidly from the water surface. Adsorption to sediment, hydrolysis, and bioconcentration are not
13 expected to be significant (NLM, 1996).

14
15 **Plants.** No information was found in the literature regarding the potential toxicity of benzene to
16 terrestrial plants.

17
18 **Terrestrial Invertebrates.** No information was found in the literature regarding the potential
19 toxicity of benzene to terrestrial invertebrates.

20
21 **Mammals.** Benzene is readily absorbed by the lung and the GI tract and accumulates mainly in
22 fat, with lower concentrations in bone marrow, brain, heart, kidney, lung, and muscle (IARC,
23 1974). Toxicity to benzene is often associated with adverse effects on the heart (NLM, 1996).
24 Toxicity to benzene is largely attributed to one or more metabolites of benzene (NLM, 1996).
25 Benzene is considered a potent bone marrow toxin in mammals. Oral LD₅₀ values for rats and
26 mice exposed to benzene are 930 and 4,700 mg/kg, respectively (RTECS, 1996). Lethal
27 concentrations that will result in death of 50 percent of the test population (LC₅₀) following
28 exposure via inhalation of benzene are 10,000 ppm over a seven hour period for rats and 9,980
29 ppm for mice (RTECS, 1996). Dermal LD₅₀ values for rabbits and guinea pigs exposed to
30 benzene are greater than 18,263 mg/kg for both species (RTECS, 1996). The lowest published
31 lethal oral dose for benzene for dogs is 2,000 mg/kg (RTECS, 1996). Benzene has been shown
32 to be genotoxic, mutagenic, and carcinogenic to rodents (RTECS, 1996).

33
34 Nawrot and Staples (1979) studied the reproductive effects of benzene in mice exposed via oral
35 gavage. Benzene exposure of 0.5 and 1.0 ml/kg/day significantly increased maternal mortality
36 and embryonic resorption. Fetal weights were significantly reduced by all three dose levels (0.3,
37 0.5, and 1.0 ml/kg/day). The 0.3 ml/kg/day dose was considered to be a chronic LOAEL and a

1 chronoc NOAEL was estimated by applying an uncertainty factor of 0.1. Therefore, a chronic
2 NOAEL of 26.36 mg/kg/day was derived from this study (Nawrot and Staples, 1979).

3
4 **Birds.** No information was found in the literature regarding the potential toxicity of benzene to
5 birds.

6
7 **Aquatic Life.** Data on the toxicity of benzene to freshwater biota are limited primarily to fish
8 studies. Ninety-six LC₅₀ values for bass (*Morone saxatilis*), crab larvae (*Cancer magister*), and
9 grass shrimp (*Palaemonetes pugio*) exposed to benzene were measured to be 5.8 to 10.9 mg/L,
10 220 mg/L, 1,108 mg/L, and 27 mg/L, respectively (Verschueren, 1983). Federal Water Quality
11 Criteria do not exist for the protection of freshwater aquatic life from exposure to benzene (EPA,
12 1996). The Tier II secondary acute and chronic values derived using the methodologies
13 suggested in the *Final Water Quality Guidance for the Great Lakes System* (EPA, 1995) are
14 2,300 and 130 µg/L, respectively. EPA Region 4 provides acute and chronic freshwater
15 screening values of 530 and 53 µg/L, respectively (EPA, 2000b). The lowest chronic benzene
16 toxicity value for daphnids is greater than 98,000 µg/L (Suter and Tsao, 1996).

17
18 The test EC₂₀ for fish can be used as a benchmark indicative of production within a population.
19 It is the highest tested concentration causing less than a 20 percent reduction in either the weight
20 of young fish per initial female fish in a life cycle or partial life cycle test or the weight of young
21 per egg in an early lifestage test. The EC₂₀ value for benzene is 21 µg/L (Suter and Tsao, 1996).

22 23 **7.4.2.19 Carbon Tetrachloride**

24 Carbon tetrachloride is a volatile organic compound whose major current use is in the production
25 of various chlorofluorocarbons that are used as refrigerants (ATSDR, 1992). Previous uses
26 include widespread application as a solvent and cleaning fluid, which probably accounts for most
27 of its occurrence in environmental media. Volatilization is the primary removal mechanism from
28 water and soil. It eventually diffuses into the stratosphere where it undergoes photolysis by
29 ultraviolet light (EPA, 1989a).

30
31 **Plants.** No information was found regarding the potential toxicity of carbon tetrachloride to
32 plants.

33
34 **Terrestrial Invertebrates.** No information was found regarding the potential toxicity of
35 carbon tetrachloride to terrestrial invertebrates.

1 **Mammals.** Carbon tetrachloride is a classic hepatotoxicant in animals exposed by any route
2 (ATSDR, 1992). High exposure levels also induced kidney effects in animals. Oral and
3 inhalation exposure to high concentrations of carbon tetrachloride results in acute CNS effects
4 including dizziness, vertigo, headache, depression, confusion, incoordination, and in severe
5 cases, respiratory failure, coma and death. Gastrointestinal problems including nausea,
6 abdominal pain, and diarrhea, often accompany these narcotic effects. Liver and kidney damage
7 can appear after the acute symptoms subside.

8
9 Subchronic and chronic exposures to doses as low as 10 ppm can result in liver and kidney
10 damage (Sax and Lewis 1989). Maternal toxicity and fetotoxic effects have been reported in rats
11 following oral or inhalation exposure to carbon tetrachloride during gestation (Wilson, 1954).
12 Repeated inhalation exposure of male rats to carbon tetrachloride concentrations of 200 ppm or
13 greater has been reported to cause degeneration of the testicular germinal epithelium as well as
14 severe liver and kidney damage (Adams, et al., 1952).

15
16 Alumot, et al. (1976) studied the effects of carbon tetrachloride on rats exposed orally through
17 their diet. Reproduction effect was the critical endpoint of their study. Because no significant
18 effects were observed at either dose level (80 and 200 ppm) and the study considered exposure
19 throughout 2 years including critical lifestages, the maximum dose of 16 mg/kg/day was
20 considered the NOAEL (Sample, et al., 1996).

21
22 **Birds.** No information was found regarding the potential toxicity of carbon tetrachloride to
23 birds.

24
25 **Aquatic Life.** There is little tendency for carbon tetrachloride to bioconcentrate in fish (NLM,
26 1996). A limited amount of data exists on the toxicity of carbon tetrachloride to freshwater
27 biota. Federal water quality criteria do not exist for the protection of freshwater aquatic life from
28 exposure to carbon tetrachloride. The Tier II secondary acute and chronic values for carbon
29 tetrachloride have been calculated to be 180 and 9.8 µg/L, respectively (Suter and Tsao, 1996).
30 The lowest chronic toxicity values for chloroform to fish and daphnids were estimated as 1,970
31 and 5,580 µg/L, respectively (Suter and Tsao, 1996). The test EC₂₀ for fish can be used as a
32 benchmark indicative of production within a population. It is the highest tested concentration
33 causing less than 20 percent reduction in either the weight of young fish per initial female fish in
34 a life-cycle or partial life-cycle test or the weight of young per egg in an early life-stage test
35 (Suter and Tsao, 1996). The EC₂₀ for carbon tetrachloride has been estimated to be 65 µg/L.
36 Sediment quality benchmark values for carbon tetrachloride were derived by Jones, et al. (1997)
37 using the equilibrium-partitioning approach. The sediment quality benchmark values calculated

1 for the protection of fish and daphnids were 9,500 and 27,000 µg/kg, respectively.

3 **7.4.2.20 Chloroform**

4 Chloroform, also known as trichloromethane, primarily enters the environment as an industrial
5 solvent. It is also released as a volatile product by plants (Howard, 1990). Chloroform adsorbs
6 poorly to soil and sediment (NLM, 1996). Near the surface of soils, chloroform is expected to
7 evaporate relatively rapidly (Howard, 1990).

8
9 **Plants.** According to IARC (1972), small amounts of chloroform have been detected in
10 tomatoes and muscat grapes. Information of the phytotoxicity of chloroform is limited.
11 Concentrations of chloroform greater than 0.25 percent have been shown to be lethal to plant
12 cells (Kayser, et al., 1982). Toxic effects and abnormal mitosis have been noted in plant cells
13 exposed to 0.025 percent chloroform (Kayser, et al., 1982).

14
15 **Terrestrial Invertebrates.** No information was found regarding the potential toxicity of
16 chloroform to terrestrial invertebrates.

17
18 **Mammals.** Information on the concentration of chloroform in wild animals is limited.
19 According to Pearson and McConnell (as cited in NLM, 1996), grey seals collected from the
20 English coast contained 7.6 to 22 µg/kg chloroform in blubber and 0 to 12 µg/kg chloroform in
21 liver.

22
23 Oral or inhalation exposure of animals to chloroform is associated with liver and kidney damage
24 (EPA, 1999). Laboratory studies have shown ingested chloroform to be eliminated in expired air
25 and in urine (NLM, 1996). The metabolism of chloroform to phosgene in the kidney can lead to
26 nephrotoxicity (NLM, 1996). Oral LD₅₀ values for rats, mice, and rabbits exposed to chloroform
27 are 908, 36, and greater than 20 mg/kg, respectively (RTECS, 1996). The dermal LD₅₀ value
28 for rabbits exposed to chloroform is greater than 20,000 mg/kg (RTECS, 1996). Adverse
29 impacts on fertility and fetotoxicity and teratogenicity have been reported in rats exposed to
30 chloroform at 30 ppm for 7 hours during the sixth to fifteenth day of pregnancy (RTECS, 1996).

31
32 Palmer, et al., (1979) conducted a study with rats over a 13 week period with dose levels of 15,
33 30, 150 and 410 mg/kg/day. Liver, kidney, and gonadal condition were the endpoints studied.
34 The 150 mg/kg/day dose level was considered the subchronic NOAEL in this study, and a
35 chronic NOAEL value of 15 mg/kg/day was derived based on the results of this study.

1 **Birds.** Marine and freshwater birds collected from England contained 0.7 to 65 µg/kg
2 chloroform (NLM, 1996). No other information was found regarding the potential toxicity of
3 chloroform to birds.

4
5 **Aquatic Life.** There is little tendency for chloroform to bioconcentrate in fish (NLM, 1996). A
6 limited amount of data exists on the toxicity of chloroform to freshwater biota. Federal water
7 quality criteria do not exist for the protection of freshwater aquatic life from exposure to
8 chloroform. The Tier II secondary acute and chronic values for chloroform have been calculated
9 to be 490 and 28 µg/L, respectively (Suter and Tsao, 1996). The lowest chronic toxicity values
10 for chloroform to fish and daphnids were estimated as 1,240 and 4,483 µg/L, respectively (Suter
11 and Tsao, 1996). The test EC₂₀ for fish can be used as a benchmark indicative of production
12 within a population. It is the highest tested concentration causing less than 20 percent reduction
13 in either the weight of young fish per initial female fish in a life-cycle or partial life-cycle test or
14 the weight of young per egg in an early life-stage test (Suter and Tsao, 1996). The EC₂₀ for
15 chloroform has been estimated to be 8,400 µg/L. Sediment quality benchmark values for
16 chloroform were derived by Jones, et al. (1997) using the equilibrium-partitioning approach.
17 The sediment quality benchmark value calculated for the protection of fish and daphnids were
18 960 and 3,500 µg/kg, respectively.

19 20 **7.4.2.21 Chloromethane**

21 Chloromethane, also known as methyl chloride, is a volatile organic compound used largely as a
22 methylating agent in the production of silicones, tetramethyl lead, methyl cellulose, methylene
23 chloride, methyl mercaptan, plastics, pesticides, pharmaceuticals, dyes, perfumes, ethers, resins,
24 agricultural chemicals, quaternary amines, and butyl rubber (ATSDR, 1998). Chloromethane is
25 a natural and ubiquitous constituent of the oceans and atmosphere. It is a product of biomass
26 combustion and is also created from biogenic emissions by wood-rotting fungi. In soil,
27 chloromethane is expected to volatilize from the surface. In water, chloromethane is expected to
28 volatilize rapidly. It is not expected to sorb to sediments or to bioconcentrate (ATSDR, 1998).

29
30 **Plants.** No information was found regarding the potential toxicity of chloromethane to plants.

31
32 **Terrestrial Invertebrates.** No information was found regarding the potential toxicity of
33 chloromethane to terrestrial invertebrates.

34
35 **Mammals.** Limited data are available regarding the effects of chloromethane following oral
36 exposure. No evidence of liver necrosis was found in rats given chloromethane at a dose of 420
37 mg/kg via gavage. This dose could be considered a NOAEL for oral exposures. A number of

1 studies have evaluated the health effects of chloromethane exposure in animals for the inhalation
2 route, although only a single comprehensive chronic study in rats and mice has been performed.
3 Health effects of acute, intermediate, and chronic inhalation exposure in animals include
4 increased mortality, liver damage, kidney damage and tumors, neurological damage, and adverse
5 reproductive, genotoxic and possibly developmental effects. A two year inhalation study in
6 animals was conducted which exposed rats and mice to several concentrations of chloromethane.
7 The liver, kidney, spleen, and brain were identified as target organs in mice, and the testes were
8 identified as target organs in rats and mice (ATSDR, 1998). No studies were found regarding
9 effects in animals after chronic oral or dermal exposure to chloromethane.

10
11 **Birds.** No information was found regarding the potential toxicity of chloromethane to birds.

12
13 **Aquatic Life.** No information was found regarding the potential toxicity of chloromethane to
14 aquatic life.

15 16 **7.4.2.22 Trichlorofluoromethane**

17 Trichlorofluoromethane is a volatile organic compound that occurs as a liquid or gas at room
18 temperature. It is used principally as a blowing agent in the production of polyurethane foams
19 (HSDB, 1999). It is also used as a refrigerant and heat exchange agent, solvent, component of
20 electrical insulation and dielectric fluid, an intermediate in organic chemical synthesis, and as a
21 propellant for pharmaceutical agents inhaled in the treatment of bronchial asthma. Its use as a
22 propellant in a wide variety of consumer products was banned in the U.S. in 1978 because of
23 concern for its accumulation and effects on the ozone ultraviolet filter in the upper atmosphere.

24
25 **Plants.** No information was found regarding the potential toxicity of trichlorofluoromethane to
26 plants.

27
28 **Terrestrial Invertebrates.** No information was found regarding the potential toxicity of
29 trichlorofluoromethane to terrestrial invertebrates.

30
31 **Mammals.** Data regarding the toxicity of acute oral exposure to trichlorofluoromethane are
32 limited to the statement that the lethal dose in rats is approximately 3,725 mg/kg (ACGIH, 1991).
33 Chronic oral toxicity data are limited to a gavage study with rats and mice in which treatment for
34 78 weeks was associated with decreased survival in both species, even at the lowest dose tested
35 (349 mg/kg/day) (EPA, 2005). The data are insufficient to identify the cause of decreased
36 survival or a target organ or system that reflects with confidence the mechanism of toxicity.

1 Inhalation LC₅₀ values include 250,000 ppm in guinea pigs and rabbits, and 100,000 ppm in rats
2 exposed for 30 minutes (HSDB, 1999).

3
4 EPA Region 5 recommends an Ecological Screening Level for trichlorofluoromethane in soil of
5 16.4 mg/kg based on exposures to masked shrew.

6
7 **Birds.** No information was found regarding the potential toxicity of trichlorofluoromethane to
8 birds.

9
10 **Aquatic Life.** No information was found regarding the potential toxicity of
11 trichlorofluoromethane to aquatic life.

12 13 **7.4.2.23 Xylene**

14 Xylenes are a widely used group of industrial solvents that include the ortho-, meta-, and para-
15 isomers (NLM, 1996). The xylene isomers have similar physical properties and are also
16 toxicologically similar. Naturally occurring sources of xylenes include coal tar, forest fires, and
17 volatile plant products (Verschueren, 1983). Xylenes are somewhat mobile in soils and may
18 leach into groundwater (NLM, 1996). The dominant removal process for xylenes in water is
19 volatilization; however, some adsorption to sediment is expected to occur (NLM, 1996).

20
21 **Plants.** Information on the phytotoxicity of xylenes is limited. Exposure of mushrooms
22 (*Agaricus bisporus*) to xylene was reported to reduce the number of sporophytes produced
23 (NLM, 1996). Allen, et al. (1961) evaluated the effect of xylene in insecticides on emergence of
24 sugar beet seedlings exposed in solution for 2 days. Root length was reduced 32 percent by 100
25 ppm xylene, the lowest concentration tested. Efrogmson, et al. (1997a) have proposed a
26 phytotoxicity benchmark value of 100 ppm based on this study.

27
28 **Terrestrial Invertebrates.** No information was found regarding the potential toxicity of
29 xylenes to terrestrial invertebrates.

30
31 **Mammals.** Exposure to xylenes occurs primarily through inhalation and ingestion. Xylenes are
32 eliminated in exhaled air with xylene metabolites excreted into urine (NLM, 1996). Oral LD₅₀
33 values for xylenes include 3,500 to 8,600 mg/kg in rats and 1,600 to 5,600 mg/kg in mice (HSDB,
34 1999). Prolonged oral exposure of animals to xylenes is associated with CNS depression and
35 increased mortality without histopathological alterations in the internal organs (EPA, 1999).

1 Adverse impacts on fertility have been reported in female mice orally exposed to xylene
2 concentrations of 31 mg/kg from the sixth to fifteenth day of pregnancy and in rabbits exposed to
3 an inhalation dose of 1.0 g/m³ during a 24-hour period from the seventh through twentieth day of
4 pregnancy (RTECS, 1996). Fetotoxic and teratogenic effects have also been observed in the
5 fetuses of pregnant rodents exposed to oral doses of 20.6 mg/kg during the sixth to fifteenth days
6 of pregnancy, or to inhalation doses of 50 mg/m³/hr during the first 21 days of pregnancy
7 (RTECS, 1996).

8
9 Marks, et al. (1982) studied the effects of xylene on mice exposed through horal gavage. Xylene
10 exposure of 2.58 mg/kg/day or greater significantly reduced fetal weights and increased the
11 incidence of fetal malformities. Although the xylene exposures evaluated in this study were of
12 relatively short duration, they occurred during a critical lifestage. Therefore, the highest dose
13 that produced no adverse effects (2.06 mg/kg/day) was considered to be a chronic NOAEL
14 (Sample, et al., 1996).

15
16 **Birds.** Data on avian toxicity to xylenes are limited. Hoffman and Eastin (as cited in NLM,
17 1996) noted no significant effects on embryonic weight and length when eggs were immersed in
18 a 10 percent solution of xylenes for 30 seconds. Teratogenic effects have been observed in chick
19 embryos exposed for 60 to 240 minutes to a xylene atmosphere at developmental periods up to
20 the 10 somite stage (NLM, 1996). Ingestion of xylene by breeding female quail can result in
21 reduced hatching rate, reduced embryonic viability, increased egg weight, and alterations in the
22 sex ratio of offspring (NLM, 1996).

23
24 **Aquatic Life.** Bioconcentration of xylene in aquatic environments is not expected to be
25 significant (NLM, 1996). Federal water quality criteria do not exist for the protection of
26 freshwater aquatic life from exposure to xylenes (EPA, 1996). The Tier II secondary acute and
27 chronic values for xylene have been calculated to be 230 and 13 µg/L, respectively (Suter and
28 Tsao, 1996). The lowest chronic toxicity values for xylene to fish was estimated as 62,308
29 µg/L (Suter and Tsao, 1996). The test EC₂₀ for fish can be used as a benchmark indicative of
30 production within a population. It is the highest tested concentration causing less than 20 percent
31 reduction in either the weight of young fish per initial female fish in a life-cycle or partial life-
32 cycle test or the weight of young per egg in an early life-stage test (Suter and Tsao, 1996). The
33 EC₂₀ for xylene has been estimated to be 2,680 µg/L. Sediment quality benchmark values for
34 xylene were derived by Jones, et al. (1997) using the equilibrium-partitioning approach. The
35 sediment quality benchmark value calculated for the protection of fish was 740,000 µg/kg.

1 **7.4.2.24 Phthalates**

2 Phthalates are a class of predominantly man-made compounds which do not naturally occur in
3 nature. They are manufactured and commonly used to produce flexible plastics, wetting agents,
4 insecticidal sprays, paints, and glues (HSDB, 1996; ATSDR, 1993). Because of their many uses,
5 phthalates are widespread in the environment and have been identified at low levels in the air,
6 water and soil. In air, phthalates may be adsorbed to particulate matter, and can be transferred to
7 water by wet or dry deposition. In water and soil phthalates are subject to microbial degradation.
8 Both aerobic and anaerobic degradation have been reported. Inman et al. (1984) demonstrated
9 that di-n-butyl phthalate in soil was completely degraded within 100 days. Di-n-butyl phthalate
10 and di-n-octyl phthalate have strong ultraviolet absorption bands at 274 nanometers extending
11 beyond 290 nanometers and are therefore strong candidates for photolysis. However, the
12 estimated photolysis half-life in natural waters is 144 days for both compounds (Callahan, et al.,
13 1979). There is some evidence that phthalate esters might be biosynthesized and occur naturally
14 in some plants and organisms (Callahan, et al., 1979).

15
16 **Plants.** No information was found regarding the toxicity of phthalates to plants.

17
18 **Terrestrial Invertebrates.** No information was found regarding the potential toxicity of
19 phthalates to terrestrial invertebrates.

20
21 **Mammals.** No studies were located on the effects of phthalate exposure to wildlife. Effects of
22 phthalate esters in laboratory animals were seen at only very high doses (one to two percent di-n-
23 butyl phthalate in the diet in oral studies). The male reproductive system appears to be the most
24 sensitive target organ for acute-duration oral exposure to di-n-butyl phthalate in animals. A
25 LOAEL of 1,000 mg/kg-day was established for decreased testis weight in rats (Oishi and
26 Hiraga, 1980). The mechanism of testicular damage by di-n-butyl phthalate may involve
27 interference with zinc metabolism (Foster et al., 1980). After oral administration, butyl benzyl
28 phthalate was rapidly excreted. Rats and mice exposed to high concentrations of butyl benzyl
29 phthalate lost weight, had testicular atrophy, hemorrhages, and hepatomegaly. LD₅₀ values for
30 these experiments were 2.3 g/kg for rats and 4.2 to 6.2 g/kg for mice (DIALOG, 1996).

31
32 Mice fed bis(2-ethylhexyl)phthalate in their diets for 105 days were studied for effects on
33 reproduction. While significant reproductive effects were observed among mice on diets
34 containing 0.1 and 0.3 percent bis(2-ethylhexyl)phthalate, no adverse effects were observed
35 among the 0.01 percent dose group. These data were used to derive a NOAEL value of 18.3
36 mg/kg/day (Lamb, et al., 1987).

1 **Birds.** Ringed doves were fed bis(2-ethylhexyl)phthalate in their diets for 4 weeks during a
2 critical lifestage and studied for reproductive effects (Peakall, 1974). No significant reproductive
3 effects were observed in the maximally exposed doves (10 ppm). These data were used to derive
4 a NOAEL value of 1.1 mg/kg/day based on reproductive effects.

5
6 **Aquatic Life.** Studies by Sasaki (1978) indicate that both di-n-butyl phthalate and di-n-octyl
7 phthalate are non- or low-bioaccumulative in fishes. Studies by Streufert et al. (1981) showed
8 the acute 48-hour LC₅₀s of di-2-ethylhexyl phthalate and di-n-butyl phthalate to the midge larvae
9 *Chironomus plumosus* to be 18 mg/L and 0.76 mg/L, respectively. Chronic life cycle toxicity
10 tests showed no effect up to 0.36 mg/L di-2-ethylhexyl phthalate on midge emergence, egg
11 production, or egg hatchability.

12 13 **7.4.2.25 Polycyclic Aromatic Hydrocarbons**

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

22
23 **Plants.** Some PAHs are synthesized by plants at very low concentrations (Sims and Overcash,
24 1983). Background concentrations of specific PAH compounds usually range from 22 to 88
25 µg/kg in tree leaves, 48 to 66 µg/kg in cereal crop plants, 0.05 to 50 µg/kg in leafy vegetables,
26 0.01 to 6 µg/kg in underground vegetables, and 0.02 to 0.04 µg/kg in fruits (Sims and Overcash,
27 1983). In general, PAH concentrations are usually greater in above-ground plant parts than in
28 below-ground parts, and are greater on plant surfaces than within internal tissues (Eisler, 1987b).

29
30 Lower molecular weight PAHs are taken up from soil by plants more readily than higher
31 molecular weight PAHs (Eisler, 1987b). Soil-to-plant concentration ratios for total PAHs have
32 been reported to range from 0.001 to 0.183 (Talmage and Walton, 1990). Atmospheric
33 deposition is believed to be the usual source of PAHs in plants, not uptake from soil (Sims and
34 Overcash, 1983).

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

6
7 **Terrestrial Invertebrates.** Earthworms (*Eisenia fetida*) exposed to benzo(a)pyrene (up to 500
8 mg/kg) in soil for 28 days did not exhibit DNA strand breaks (Honeycutt and Roberts, 1995),
9 indicating the lack of genotoxicity at the exposure concentrations. Polycyclic aromatic
10 hydrocarbon compounds were determined to be relatively non-toxic to the earthworm *Eisenia*
11 *fetida* (Roberts and Dorough, 1984).

12
13 **Mammals.** Most of the PAHs taken into the body are not accumulated but are oxidized, and the
14 metabolites excreted (NLM, 1996). In fact, most PAH compounds are detoxified and excreted
15 from the body (Klaassen et al., 1991). PAHs are metabolized in vertebrates by a group of
16 enzymes known as mixed-function oxidases in the liver. A few laboratory studies on rodents
17 have revealed acute oral toxicities of PAHs are greatest for benzo(a)pyrene, followed in
18 decreasing order of toxicity by phenanthrene, naphthalene, and fluoranthene (Sims and Overcash
19 (1983). Data from a study of mice fed benzo(a)pyrene in their diets were used to derive a
20 NOAEL value of 1.0 mg/kg/day (MacKenzie and Angevine, 1981). The critical endpoint in this
21 study was reproduction.

22
23 Sims and Overcash (1983) have reported LC₅₀ values for rodents (*Rattus* spp. and *Mus* spp.) as
24 50 mg/kg-day benzo(a)pyrene, 700 mg/kg-day phenanthrene, and 2,000 mg/kg-day fluoranthene.
25 Sublethal effects manifested as decreased pup weight in mice have been reported at 10 mg/kg-
26 day benzo(a)pyrene (MacKenzie and Angevine, 1981). Subchronic and chronic effects of
27 exposure to PAHs in rats include liver and kidney damage, unspecified changes in peripheral
28 blood pattern, body weight loss, genetic aberrations, and increased serum aminotransferase
29 activity (Knobloch et al., 1969).

30
31 **Birds.** Hoffman and Gay (1981) measured embryotoxicity of various PAHs applied externally
32 to the surface of mallard duck eggs. Approximately 0.002 µg/egg of 7,12-
33 dimethylbenz(a)anthracene (DMBA) caused 26 percent mortality in 18 days, and among the
34 survivors, produced significant reduction in embryonic growth and a significant increase in the
35 percent of abnormalities, e.g., incomplete skeletal ossification, defects in eye, brain liver,
36 feathers, and bill. At 0.1 µg DMBA/egg, only 10 percent survived to day 18.

1
2 **Aquatic Life.** In general, PAHs as a group are not appreciably acutely toxic (Eisler, 1987b;
3 Neff, 1985). The toxicity of PAH compounds to fish is related to the solubility of the compound
4 in water. The toxicity of PAHs to aquatic organisms is very species-specific and related to the
5 organisms' ability to metabolize and excrete the compound (Eisler, 1987b). For aquatic
6 organisms, only PAHs in the molecular weight range from naphthalene to pyrene are considered
7 acutely toxic. Toxicity in this group increases with increasing molecular weight. There is some
8 evidence to suggest that PAHs are responsible for the production of reproductive and teratogenic
9 effects in eggs of the sand sole (*Psettichthys melanostictus*) exposed to 0.1 µg benzo(a)pyrene/L
10 for five days showed reduced and delayed hatch and, when compared to controls, produced
11 larvae with high accumulations (2.1 mg/kg fresh weight) and gross abnormalities, such as tissue
12 overgrowths, in 50 percent of the test larvae (Hose et al., 1982).

13
14 Inhibited reproduction of daphnids and the delayed emergence of larval midges by fluorene was
15 reported by Finger, et al. (1985). When sediment PAH levels are elevated, benthic organisms
16 obtain a majority of their PAHs from sediments through their ability to mobilize PAHs from the
17 sediment/pore water matrix. The elevated levels in the tissues of these organisms could provide
18 a significant source of PAHs to predatory fish. However, fish do have the ability to efficiently
19 metabolize and degrade PAHs.

20 21 **7.4.3 Potential Receptors**

22 Potential ecological receptors in the vicinity of the Ranges Near Training Area T-24A fall into
23 two general categories: terrestrial and aquatic. Within these two general categories there are
24 several major feeding guilds that could be expected to occur in the vicinity of the Ranges Near
25 Training Area T-24A: herbivores, invertivores, omnivores, piscivores, and carnivores. All of
26 these feeding guilds have the potential to be directly exposed to soil-, surface water-, and
27 sediment-related constituents at the Ranges Near Training Area T-24A via various activities
28 (e.g., feeding, grooming, bathing). These feeding guilds may also be exposed to site-related
29 chemicals via food web interactions.

30
31 Ingestion of soil-related constituents either through direct ingestion of soil or through food web
32 interactions is expected to be the dominant exposure pathway for most organisms at the Ranges
33 Near Training Area T-24A. Dermal absorption of constituents from soil is a potential pathway
34 for all feeding guilds at the Ranges Near Training Area T-24A; however, birds and mammals are
35 less susceptible to dermal exposures because their feathers or fur prevents skin from coming into
36 direct contact with the soil (EPA, 1993). Dermal absorption of inorganic compounds from direct
37 contact with soil is expected to be minimal due to the low dermal permeability of these

1 constituents; however, dermal absorption of volatile organic compounds or semi-volatile organic
2 compounds from soil is more likely. Since volatile organic compounds were only detected in
3 very low concentrations in surface soil at the Ranges Near Training Area T-24A, inhalation of
4 volatiles is not a significant exposure pathway. Inhalation of constituents sorbed to soil particles
5 and inhaled as dust is a potential pathway for all of the feeding guilds at the Ranges Near
6 Training Area T-24A; however, in areas where the site is covered with vegetation or is forested,
7 the generation of fugitive dust is expected to be minimal. In areas devoid of vegetation, fugitive
8 dust generation and subsequent exposure via inhalation could be significant.

9
10 Terrestrial species could be exposed to constituents in surface water in the numerous ephemeral
11 drainage features at the Ranges Near Training Area T-24A and the perennial streams that run
12 through these ranges via ingestion and dermal contact. The ephemeral nature of many of these
13 drainage features in the vicinity of the Ranges Near Training Area T-24A limits the potential for
14 exposure to surface water. However, several of the streams are perennial and provide surface
15 water exposure pathways throughout most years.

16
17 Terrestrial receptors could also be exposed to constituents in groundwater through surface water
18 exposure pathways. If groundwater at the Ranges Near Training Area T-24A discharges to
19 surface water bodies, terrestrial receptors using those surface water bodies for drinking water
20 could be exposed to groundwater constituents.

21
22 Aquatic and semi-aquatic (i.e., amphibians) species have a significant potential for exposure to
23 constituents in surface water and sediment as they spend a majority of their lifetime in close
24 proximity to water bodies. Aquatic and semi-aquatic species could be exposed to constituents in
25 surface water or sediment via direct contact, ingestion of surface water and sediment, and
26 ingestion of aquatic vegetation and/or aquatic invertebrates that may have accumulated site-
27 related constituents. Since there are both perennial and ephemeral surface water features at the
28 Ranges Near Training Area T-24A, these potential exposure routes could range from continuous
29 to sporadic, depending on the individual species, water body, and precipitation received during a
30 given year.

31
32 Exposure to aquatic species (e.g. fish) and the feeding guilds (i.e. piscivores) that rely on aquatic
33 species for food have the potential to occur in the perennial streams at the Ranges Near Training
34 Area T-24A. The small ephemeral drainage features at the Ranges Near Training Area T-24A do
35 not provide suitable habitat to support most aquatic species, and by association, the species that
36 rely on them for food.

1 **7.4.3.1 Herbivorous Feeding Guild**

2 The major route of exposure for herbivores is through ingestion of plants that may have
3 accumulated contaminants from the soil. Since terrestrial herbivores by definition are grazers
4 and browsers, they could be exposed to chemicals that have accumulated in the vegetative tissues
5 of the plants at the site. Terrestrial herbivores may also be exposed to site-related chemicals in
6 soil through incidental ingestion of soil while grazing, grooming, or other activities. Herbivores
7 could also be exposed to surface water through ingestion of water in the perennial and ephemeral
8 drainage features that drain the Ranges Near Training Area T-24A; however, these exposure
9 pathways would only be operable at the ephemeral drainage features during periods of
10 significant precipitation.

11
12 Typical herbivorous species that could be expected to occur at the Ranges Near Training Area T-
13 24A and are commonly used as sentinel species in ecological risk assessment include eastern
14 cottontail (*Sylvilagus floridanus*), eastern gray squirrel (*Sciurus carolinensis*), pine vole (*Pitymys*
15 *pinetorum*), whitetail deer (*Odocoileus virginianus*), and wild turkey (*Meleagris gallopavo*).

16
17 Aquatic herbivores, such as muskrat (*Ondatra zibethica*) and mallard (*Anas platyrhynchos*) may
18 occur at the perennial creeks but not the ephemeral drainage features at the Ranges Near
19 Training Area T-24A due to habitat restrictions in the ephemeral drainage features.

21 **7.4.3.2 Invertivorous Feeding Guild**

22 Invertivores specialize in eating insects and other invertebrates. As such, they may be exposed
23 to site-related chemicals that have accumulated in insects and other invertebrates. Invertivores
24 may also be exposed to site-related chemicals in soil through incidental ingestion of soil while
25 probing for insects, grooming, or other activities. Ingestion of soil while feeding is a potential
26 exposure pathway for invertivores since much of their food (i.e., earthworms and other
27 invertebrates) lives on or below the soil surface. Invertivores could be exposed to surface water
28 through ingestion of water in the perennial creeks and ephemeral drainage features that drain the
29 Ranges Near Training Area T-24A; however, these exposure pathways would only be operable at
30 the ephemeral drainage features during periods of significant precipitation.

31
32 Typical invertivorous species that could be expected to occur at the Ranges Near Training Area
33 T-24A and are commonly used as sentinel species in ecological risk assessment include
34 American woodcock (*Philohela minor*), carolina wren (*Thryothorus ludovicianus*), shorttail
35 shrew (*Blarina brevicauda* or *Blarina carolinensis*), and eastern mole (*Scalopus aquaticus*).
36 Aquatic invertivores such as the wood duck (*Aix sponsa*) and blacknose dace (*Rhinichthys*
37 *atratulus*) may occur at the perennial creeks but not the ephemeral drainage features at the

1 Ranges Near Training Area T-24A due to habitat restrictions in the ephemeral drainage features.

3 **7.4.3.3 Omnivorous Feeding Guild**

4 Omnivores consume both plant and animal material in their diet, depending upon availability.
5 Therefore, they could be exposed to chemicals that have accumulated in the vegetative tissues of
6 plants at the site and also chemicals that may have accumulated in smaller animal tissues that the
7 omnivores prey upon. Omnivores may also be exposed to site-related chemicals in soil through
8 incidental ingestion of soil while feeding, grooming, or other activities. Omnivores could be
9 exposed to surface water through ingestion of water in the perennial creeks and ephemeral
10 drainage features that drain the Ranges Near Training Area T-24A; however, these exposure
11 pathways would only be operable at the ephemeral drainage features during periods of
12 significant precipitation.

13
14 Typical omnivorous species expected to occur at the Ranges Near Training Area T-24A and are
15 commonly used as sentinel species in ecological risk assessment include red fox (*Vulpes vulpes*),
16 white-footed mouse (*Peromyscus leucopus*), and American robin (*Turdus migratorius*). Aquatic
17 omnivores, such as raccoon (*Procyon lotor*) and creek chub (*Semotilus atromaculatus*) may
18 occur at the perennial creeks but not the ephemeral drainage features at the Ranges Near
19 Training Area T-24A due to habitat restrictions in the ephemeral drainage features.

21 **7.4.3.4 Carnivorous Feeding Guild**

22 Carnivores are meat-eating animals and are; therefore, potentially exposed to site-related
23 chemicals through consumption of prey animals that may have accumulated contaminants in
24 their tissues. Carnivores are quite often top predators in a local food web and are often subject to
25 exposure to contaminants that have bioaccumulated in lower trophic-level organisms or
26 biomagnified through the food web. Food web exposures for carnivores are based on the
27 consumption of prey animals that have accumulated COPECs from various means. Smaller,
28 herbivores, omnivores, invertivores, and other carnivores may consume soil, plant, and animal
29 material as food and accumulate COPECs in their tissues. Subsequent ingestion of these prey
30 animals by carnivorous animals would expose them to COPECs. Food web exposures to metals,
31 volatile organic compounds, and semi-volatile organic compounds in soil are expected to be
32 minimal at the Ranges Near Training Area T-24A because these compounds detected in surface
33 soil at elevated concentrations are not accumulated in animal tissues to any great extent (Shugart,
34 1991 and USAEHA, 1994). Food web exposures to pesticides and herbicides could potentially
35 be significant since many of these compounds are known to bioaccumulate and bioconcentrate
36 through food webs.

1 Carnivores may also be exposed to site-related chemicals in soil through incidental ingestion of
2 soil while feeding, grooming, or other activities. Carnivores could be exposed to surface water
3 through ingestion of water in the perennial creeks and ephemeral drainage features that drain the
4 Ranges Near Training Area T-24A; however, these exposure pathways would only be operable at
5 the ephemeral drainage features during periods of significant precipitation.

6
7 Typical carnivorous species expected to occur at the Ranges Near Training Area T-24A and are
8 commonly used as sentinel species in ecological risk assessment include red-tailed hawk (*Buteo*
9 *jamaicensis*), black vulture (*Coragyps atratus*), and bobcat (*Lynx rufus*).

11 **7.4.3.5 Piscivorous Feeding Guild**

12 Piscivores are specialists that feed almost exclusively on fish. Therefore, they may be exposed
13 to site-related chemicals that have accumulated in small fish that may inhabit the perennial
14 creeks and small ephemeral drainage features at the Ranges Near Training Area T-24A. They
15 may also be exposed to surface water and sediment in these water bodies through ingestion of
16 drinking water and during feeding. Because many of the drainage features at the Ranges Near
17 Training Area T-24A are ephemeral in nature, they would only provide habitat and a drinking
18 water supply during periods of significant precipitation. However, there are several small
19 perennial creeks that drain the Ranges Near Training Area T-24A that may provide sufficient
20 habitat to support year-round aquatic species and a perennial drinking water source.

21
22 Food web exposures for piscivores are based on the consumption of fish that have accumulated
23 COPECs from surface water and sediment. Forage fish may consume surface water, sediment,
24 benthic invertebrates, aquatic plants, and planktonic material as food and accumulate COPECs in
25 their tissues. Subsequent ingestion of these forage fish by piscivorous animals would expose
26 them to COPECs. However, most inorganic compounds are not accumulated in fish tissues to
27 any great extent. Therefore, food web exposures to these chemicals are expected to be minimal.
28 Semi-volatile and volatile organic compounds are readily metabolized by most fish species and
29 are not accumulated to any extent. Thus, the piscivorous feeding guild is not expected to have
30 significant exposure to semi-volatile or volatile organic compounds at the Ranges Near Training
31 Area T-24A through the food web. Chlorinated herbicides and pesticides have the potential to
32 bioaccumulate and biomagnify through the food chain; therefore, there is the potential for
33 significant exposure to these classes of chemicals by piscivores, if they are present at the Ranges
34 Near Training Area T-24A.

35
36 Typical piscivorous species that could occur at the Ranges Near Training Area T-24A and are
37 commonly used as sentinel species in ecological risk assessment include great blue heron (*Ardea*

1 *herodias*) and belted kingfisher (*Megaceryle alcyon*). Larger piscivorous fish species (e.g.,
2 smallmouth bass, spotted gar, etc.) are not expected to occur in the creeks and drainage features
3 at the Ranges Near Training Area T-24A due to the habitat limitations of these small creeks and
4 drainage features. Piscivorous birds and mammals (e.g., mink) may occur in the vicinity of these
5 creeks at the Ranges Near Training Area T-24A since they may provide a food source (small fish
6 and other aquatic species) for these piscivores.

7 8 **7.4.3.6 Threatened and Endangered Species**

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

- 11
- 12 • Gray Bat (*Myotis grisescens*)
- 13 • Blue Shiner (*Cyprinella caerulea*)
- 14 • Mohr's Barbara Buttons (*Marshallia mohrii*)
- 15 • Tennessee Yellow-Eyed Grass (*Xyris tennesseensis*)
- 16

17 An additional endangered species, the red-cockaded woodpecker (*Picoides borealis*), historically
18 has inhabited the installation.

19
20 The only Federally listed species that has the potential to occur in the vicinity of the Ranges Near
21 Training Area T-24A is the gray bat (Garland, 1996). The perennial creeks and ephemeral
22 drainage features in the vicinity of these sites have been designated as providing "low quality" or
23 "moderate quality" foraging habitat for the gray bat (Garland, 1996). The other Federally listed
24 species occur at Pelham Range or Choccolocco Creek corridor.

25
26 Gray bat summer foraging habitat is found primarily over open water of rivers and reservoirs.
27 They apparently do not forage over sections of rivers or reservoirs that have lost their normal
28 woody vegetation along the banks (USFWS, 1982). Gray bats usually follow wooded corridors
29 from their summer caves to the open water areas used as foraging sites. Forested areas
30 surrounding and between caves, as well as over feeding habitats, are clearly advantageous to
31 gray bat survival as the cover provides increased protection from predators such as screech owls.
32 In addition, surveys have demonstrated that reservoirs and rivers that have been cleared of their
33 adjacent forest canopy are avoided as foraging areas by gray bats (USFWS, 1982).

34
35 Most foraging occurs within 5 meters of the water's surface, usually near a shoreline or stream
36 bank. Mist net surveys were conducted on and adjacent to FTMC in 1995. Gray bats were
37 captured along both Choccolocco Creek (east of FTMC Main Post) and Cane Creek on Pelham
38 Range (west of FTMC Main Post) during these mist net surveys (Garland, 1996). These

1 preliminary data suggest that these major stream corridors at FTMC may provide at least a
2 minimum foraging habitat for gray bats. However, gray bat surveys have not been conducted on
3 the South Branch of Cane Creek or the small drainage features in the vicinity of the Ranges Near
4 Training Area T-24A.

5
6 Although historical records indicate the presence of red cockaded woodpeckers (RCW) at
7 FTMC, the last remaining active cluster of RCW at FTMC was recorded in 1968. Subsequent
8 surveys in 1972, 1982, and 1985 failed to find any RCW at FTMC. Thus, it can be concluded
9 that RCW no longer exist at FTMC.

10
11 A significant portion of the Ranges Near Training Area T-24A is contained within the South
12 Branch Cane Creek SINA. The headwaters of the South Branch of Cane Creek include
13 significant stream, mountain seep, and typic mesophytic forest communities. Much of this
14 watershed also includes the forested slopes of the Stanley Hill Chestnut Oak Forest SINA. A
15 candidate 2 caddisfly, *Polycentropus carlsoni*, and an even rarer single site endemic caddisfly,
16 *Hydroptila setigera*, have been collected from the South Branch of Cane Creek (Mettee and
17 Haynes, 1979). An additional thirteen caddisfly species from this stream are included on the
18 Alabama Natural Heritage Program tracking list (Garland, 1996). The primary management goal
19 for this SINA is to ensure the maintenance of water quality and minimize the influx of sediments
20 from surrounding upland areas

21 22 **7.4.4 Complete Exposure Pathways**

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

- 25
- 26 • A source mechanism for contaminant release
- 27 • A transport mechanism
- 28 • A point of environmental contact
- 29 • A route of uptake at the exposure point (EPA, 1989b).
- 30

31 If any of these four components is absent, then a pathway is generally considered incomplete.
32 Potentially complete and incomplete exposure pathways at the Ranges Near Training Area T-
33 24A are depicted in the SCM shown on Figure 7-1.

34
35 Ecological receptors may be exposed to constituents in soils via direct and/or secondary
36 exposure pathways. Direct exposure pathways include soil ingestion, dermal absorption, and
37 inhalation of volatile constituents and constituents adsorbed to fugitive dust. Significant
38 exposure via dermal contact is limited to organic constituents that are lipophilic and can

1 penetrate epidermal barriers. Mammals are less susceptible to exposure via dermal contact with
2 soils because their fur prevents skin from coming into direct contact with soil. However, soil
3 ingestion may occur while grooming, preening, burrowing, or consuming plants, insects, or
4 invertebrates resident in soil.

5
6 Ecological receptors could be exposed to constituents in surface water or sediment via direct
7 contact or through consumption of water. Receptors could also potentially be exposed to
8 constituents in groundwater via surface water pathways if groundwater discharges to surface
9 water bodies. Exposures to site-related constituents in surface water and sediment are expected
10 to be minimal in the ephemeral drainage features but may be more significant in the perennial
11 creeks (e.g. South Branch of Cane Creek).

12
13 Exposure via inhalation of fugitive dust is limited to contaminants present in surface soils at
14 areas that are devoid of vegetation or other cover material. The amount of vegetative cover, the
15 inherent moisture content of the soil, and the frequency of soil disturbance play important roles
16 in the amount of fugitive dust generated at a particular site. In forested areas and other areas of
17 the study area covered with vegetation, fugitive dust generation is expected to be minimal.
18 However, in areas devoid of vegetation, fugitive dust generation and subsequent exposure via
19 inhalation may be a significant exposure pathway.

20
21 Secondary exposure pathways involve constituents that are transferred through different trophic
22 levels of the food chain and may be bioaccumulated and/or bioconcentrated. This may include
23 constituents bioaccumulated from soil into plant tissues or into terrestrial species ingesting soils.
24 These plants or animals may, in turn, be consumed by animals at higher trophic levels.

25
26 In general, the metals and semi-volatile organic compounds detected in surface soil at the Ranges
27 Near Training Area T-24A may bioaccumulate in lower trophic level organisms (i.e. terrestrial
28 invertebrates may bioaccumulate inorganic compounds detected in soil); however, they will not
29 bioconcentrate through the food chain. The volatile organic compounds detected in soil and
30 groundwater are unlikely to bioaccumulate or bioconcentrate. Certain pesticides and herbicides
31 have a propensity to bioconcentrate through food web interactions; however, these compounds
32 were detected infrequently and at very low concentrations. Therefore, food chain exposures for
33 the constituents detected at the Ranges Near Training Area T-24A are most likely insignificant.

34
35 A summary of the feeding guilds and potentially complete exposure pathways at the Ranges
36 Near Training Area T-24A is presented in Table 7-1.

1 **7.5 Screening-Level Risk Estimation**

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

- 6
- 7 • Determine appropriate screening assessment endpoints
- 8
- 9 • Determine the ecological toxicity values that are protective of the selected
- 10 assessment endpoints
- 11
- 12 • Determine the exposure point concentrations of constituents detected at the site
- 13
- 14 • Calculate screening-level hazard quotients.
- 15

16 These steps are summarized below.

17

18 **7.5.1 Ecological Screening Assessment Endpoints**

19 Most ecological risk assessments focus on population measures as endpoints, since population
20 responses are better defined and more predictable than are community or ecosystem responses.
21 For screening-level assessments such as this SLERA, assessment endpoints are any adverse
22 effects on ecological receptors, where receptors are plant and animal populations and
23 communities, habitats, and sensitive environments.

24

25 Adverse effects on populations can be inferred from measures related to impaired reproduction,
26 growth, and survival. Adverse effects on communities can be inferred from changes in
27 community structure or function. Adverse effects on habitats can be inferred from changes in
28 composition and characteristics that reduce the ability of the habitat to support plant and animal
29 populations and communities.

30

31 Due to the nature of the SLERA process, most of the screening assessment endpoints are generic
32 in nature (i.e., protection of sediment benthic communities from adverse changes in structure or
33 function).

34

35 The assessment endpoints for this SLERA were identified for surface soil, surface water, and
36 sediment and are summarized below. It is important to note that exposure to groundwater
37 directly by ecological receptors is unlikely; however, in order to account for the potential
38 discharge of groundwater constituents to surface water, constituents detected in groundwater
39 were assessed by comparing groundwater data to surface water ESVs.

- 1
- 2 • **Soil**
- 3 - Protection of the terrestrial invertebrate community from adverse changes in
- 4 structure and function
- 5
- 6 - Protection of the terrestrial plant community from adverse changes in
- 7 structure and function.
- 8
- 9 • **Surface Water**
- 10 - Protection of the aquatic community from adverse changes in structure and
- 11 function
- 12
- 13 • **Sediment**
- 14 - Protection of the benthic community from adverse changes in structure and
- 15 function.
- 16

17 **7.5.2 Ecological Screening Values**

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

26

27 For each environmental medium sampled at the Ranges Near Training Area T-24A (surface soil,
28 surface water, sediment, and groundwater), a hierarchy has been developed which presents an
29 orderly method for selection of ESVs. The hierarchy for selecting ESVs for surface soil is as
30 follows:

- 31
- 32 • EPA Region IV constituent-specific ecological screening values
- 33 • EPA Region IV ecological screening values for general class of constituents
- 34 • EPA Region V ecological data quality levels (EDQL)
- 35 • EPA Region III Biological Technical Advisory Group (BTAG) values
- 36 • Ecological screening values from Talmage et al., 1999.
- 37

38 The hierarchy for selecting ESVs for surface water, which were also used to assess groundwater
39 data at the Ranges Near Training Area T-24A, is as follows:

- 1 • EPA Region IV constituent-specific ecological screening values
- 2
- 3 • NOAA Screening Quick Reference Tables (SQRT), chronic freshwater ambient
- 4 water quality criteria
- 5
- 6 • EPA Region V EDQLs
- 7
- 8 • Office of Solid Waste and Emergency Response (OSWER) Ecotox Threshold
- 9 values
- 10
- 11 • EPA Region III BTAG values
- 12
- 13 • Lowest chronic value from Suter and Tsao, 1996
- 14
- 15 • Ecological screening values from Talmage, et al., 1999.
- 16

17 The hierarchy for selecting ESVs for sediment is as follows:

- 18
- 19 • EPA Region IV constituent-specific ecological screening values
- 20
- 21 • NOAA SQRTs, chronic freshwater ambient water quality criteria
- 22
- 23 • EPA Region V EDQLs
- 24
- 25 • OSWER ecotox threshold values
- 26
- 27 • EPA Region III BTAG values
- 28
- 29 • Lowest effect levels from Ontario Ministry of the Environment (1992) presented in
- 30 Jones, et al., (1997)
- 31
- 32 • Ecological screening values from Talmage, et al., 1999
- 33
- 34 • Sediment quality adverse effect threshold (AET) values from the Puget Sound
- 35 Estuary Program.
- 36

37 **7.5.3 Determination of Exposure Point Concentrations**

38 Exposure point concentrations represent the chemical concentrations in environmental media that
39 a receptor may contact. Since the exposure point concentration is a value that represents the
40 most likely concentration to which receptors could be exposed, a value that reflects the central
41 tendency of the data set is most appropriate to use for free-ranging animals that would be
42 expected to use the site indiscriminately. Smaller, more sessile organisms with smaller home
43 ranges may be exposed to only a portion of a site. Additionally, habitat preferences or

1 preferential avoidance behavior may result in exposure to only portions of a given site.
2 Therefore, a subset of the data would be most appropriate to estimate an exposure point
3 concentration for these species. The most conservative approach is the use of the maximum
4 detected constituent concentrations as exposure point concentrations. At the screening-level
5 stage, the data sets are generally not robust enough for statistical analysis and the level of
6 conservatism in the exposure estimates is high to account for uncertainties. Therefore, in the
7 screening-level stage, the maximum detected constituent concentration in each environmental
8 medium is used as the exposure point concentration. The use of the maximum detected
9 constituent concentration as the exposure point concentration ensures that the exposures will not
10 be underestimated, and therefore, constituents will not be inadvertently eliminated from further
11 assessment.

12
13 The statistical summaries (including the exposure point concentrations) for surface soil, surface
14 water, sediment, and groundwater at the Ranges Near Training Area T-24A are presented in
15 Tables 7-2 through 7-5.

16 17 **7.5.4 Screening-Level Hazard Quotients**

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

- 21
22
- 23 • Comparison to ESVs
 - 24 • Identification of essential macronutrients
 - 25 • Comparison to background threshold values (BTV).

26 Constituents that were detected in environmental media at the Ranges Near Training Area T-24A
27 were evaluated against the ESVs by calculating a screening-level hazard quotient (HQ_{screen}) for
28 each constituent in each environmental medium. An HQ_{screen} was calculated by dividing the
29 maximum detected constituent concentration in each environmental medium by its
30 corresponding ESV as follows:

31

$$32 \quad HQ_{screen} = \frac{MDCC}{ESV}$$

33
34 where:

- 35 HQ_{screen} = screening-level hazard quotient;
36 $MDCC$ = maximum detected constituent concentration; and
37 ESV = ecological screening value.

1
2 A calculated HQ_{screen} value of one indicated that the MDCC was equal to the chemical's
3 conservative ESV and was interpreted in this assessment as a constituent that does not pose the
4 potential for adverse ecological risk. An HQ_{screen} value less than one indicated that the MDCC
5 was less than the conservative ESV and that the chemical is not likely to pose adverse ecological
6 hazards to most receptors. Conversely, an HQ_{screen} value greater than one indicated that the
7 MDCC was greater than the ESV and that the chemical might pose adverse ecological hazards to
8 one or more receptors and requires further assessment.

9
10 In order to better understand the potential risks posed by chemical constituents at the Ranges
11 Near Training Area T-24A, a mean hazard quotient was also calculated by comparing the
12 arithmetic mean constituent concentration in surface soil, surface water, sediment, and
13 groundwater to the corresponding ESV. The calculated screening-level hazard quotients for
14 surface soil, surface water, sediment, and groundwater at the Ranges Near Training Area T-24A
15 are presented in Tables 7-2 through 7-5.

16
17 EPA recognizes several constituents in abiotic media that are necessary to maintain normal
18 function in many organisms. These essential macronutrients are iron, magnesium, calcium,
19 potassium, and sodium (EPA, 1989). Most organisms have mechanisms designed to regulate
20 nutrient fluxes within their systems; therefore, these nutrients are generally only toxic at very
21 high concentrations. Although iron is an essential nutrient and is regulated within many
22 organisms, it may become increasingly bioavailable at lower soil pH values, thus increasing its
23 potential to elicit adverse affects. Therefore, iron was not evaluated as an essential nutrient in
24 this SLERA. Essential macronutrients were only considered COPECs if they were present in site
25 samples at concentrations ten times the naturally occurring background concentration.

26
27 A comparison of detected constituent concentrations to background constituent concentrations
28 was conducted in order to identify inorganic constituents that may be present in site media at
29 concentrations consistent with background concentrations. In the process of calculating
30 screening level hazard quotients (HQ_{screen}), the background analysis consisted of a comparison of
31 the maximum detected constituent concentrations to the BTVs. A study of the natural geochemi-
32 cal composition associated with FTMC (SAIC, 1998) determined the mean concentrations of 24
33 metals in surface soil, surface water, sediment, and groundwater samples collected from
34 presumably un-impacted areas. Per agreement with EPA Region IV, the BTV for each metal
35 was calculated as two times the mean background concentration for that metal. The BTV for
36 each metal was used to represent the upper boundary of the range of natural background
37 concentrations expected at FTMC, and was used as the basis for evaluating metal concentrations

1 measured in site samples. Site sample metal concentrations less than or equal to the
2 corresponding BTV represent the natural geochemical composition of media at FTMC, and not
3 contamination associated with site activity. Site sample metal concentrations greater than the
4 corresponding BTV require further background assessment.

5
6 Thus, the first step in determining screening-level hazard quotients was a comparison of
7 maximum detected constituent concentrations to appropriate ESVs. Constituents with HQ_{screen}
8 values less than one were considered to pose insignificant ecological risk and were eliminated
9 from further consideration. Constituents with HQ_{screen} values greater than one were eliminated
10 from further consideration if they were macronutrients and their detected concentrations were
11 less than ten-times background levels. Those constituents that had HQ_{screen} values greater one
12 and were not considered macronutrients were then compared to BTVs. If constituent
13 concentrations were determined to be less than their respective BTV concentrations, then a risk
14 management decision could result in eliminating these constituents from further assessment.

15 16 **7.6 Identification of Constituents of Potential Ecological Concern**

17 A constituent was identified as a COPEC if the following conditions were met:

- 18
- 19 • The maximum detected constituent concentration exceeded the ESV
 - 20
 - 21 • The maximum detected constituent concentration was 10 times the BTV if the
 - 22 constituent was identified as a macronutrient
 - 23
 - 24 • Constituent concentrations were determined to be greater than their respective BTVs.
 - 25

26 If a constituent in a given environmental medium did not meet these conditions, then it was not
27 considered a COPEC at the Ranges Near Training Area T-24A and was not considered for
28 further assessment. If a constituent met these conditions, then it was considered a COPEC.
29 Identification of a constituent as a COPEC indicates that further assessment of that particular
30 constituent in a given environmental medium may be appropriate. It does not imply that a
31 particular constituent poses risk to ecological receptors.

32
33 The COPECs that have been initially identified for surface soil, surface water, sediment and
34 groundwater at the Ranges Near Training Area T-24A are presented in Tables 7-2 through 7-5,
35 respectively.

36
37 In order to focus future ecological assessment efforts (if necessary) on the constituents that are
38 the most prevalent at the Ranges Near Training Area T-24A and have the greatest potential to

1 pose ecological risk, additional lines of evidence were assessed. Additional lines of evidence are
2 sometimes useful in determining whether a certain constituent is in fact site-related and a
3 COPEC. Some of the additional lines of evidence used in the process of identifying COPECs
4 include: 1) frequency of detection, 2) magnitude of the HQ_{screen} value, 3) spatial distribution, 4)
5 comparison to alternative ESVs; 5) additional background evaluation; and 6) association of a
6 chemical with known Army activities. These additional lines-of-evidence were used to further
7 define the COPECs at the Ranges Near Training Area T-24A and are discussed below.

8
9 Additional background evaluations were conducted if maximum constituent concentrations were
10 greater than the BTVs. Tier two of the background comparison consists of statistical
11 comparisons of the site data to background data using the hot measurement test and the
12 Wilcoxon Rank Sum (WRS) Test. If the site data failed either the hot measurement test or the
13 WRS Test, then the site data were subjected to a geochemical evaluation to determine whether
14 concentrations of inorganic compounds are naturally occurring or are elevated due to
15 contamination (Tier 3). The three-tier background comparison process is described in detail in
16 Appendix J of the RI report.

17

18 **7.6.1 Surface Soil**

19 The following constituents exceeded their respective ESVs and BTVs in surface soil at the
20 Ranges Near Training Area T-24A and are not essential macronutrients:

21

- | | | | |
|----|-------------|----|--------------------------|
| 22 | • Aluminum | 31 | • Lead |
| 23 | • Antimony | 32 | • Mercury |
| 24 | • Barium | 33 | • Nickel |
| 25 | • Beryllium | 34 | • Selenium |
| 26 | • Cadmium | 35 | • Zinc |
| 27 | • Chromium | 36 | • Chloroform |
| 28 | • Cobalt | 37 | • Phenanthrene |
| 29 | • Copper | 38 | • Trichlorofluoromethane |
| 30 | • Iron | 39 | • Xylene |

40

41 Antimony, copper, lead, and zinc were detected in numerous surface soil samples from the
42 Ranges Near Training Area T-24A at concentrations that exceeded ESVs and naturally occurring
43 levels. Based on the frequency of detection at elevated concentrations, these four metals have
44 been identified as COPECs in surface soil at the T-24A Ranges.

45

46 Per EPA (2003) guidance, aluminum toxicity is associated with soluble aluminum only.
47 Numeric screening values for aluminum are considered inappropriate due to the uncertainty in

1 the solubility of aluminum in any given soil type under different environmental conditions.
2 Alternatively, potential ecological risks associated with exposure to aluminum are associated
3 with soil pH. Aluminum is identified as a COPEC only if the soil pH is less than 5.5 (EPA,
4 2003). Since the pH of soils at the T-24A Ranges is greater than 5.5, aluminum is not considered
5 a COPEC in surface or depositional soil at the T-24A Ranges. Additionally, geochemical
6 evaluation indicated that all of the detected aluminum in surface and depositional soils were
7 consistent with naturally occurring aluminum concentrations in surface soil.

8
9 Eleven surface soil samples out of 110 total surface soil samples exhibited barium concentrations
10 greater than the ESV. The calculated HQ_{screen} value for barium was 2.27. If the EPA (2005a)
11 recommended Eco-SSLs for barium are used for comparison, one sample slightly exceeds the
12 Eco-SSL that is protective of soil invertebrates (330 mg/kg) and all of the detected barium
13 concentrations in surface soil are less than the Eco-SSL that is protective of mammalian
14 receptors (2,000 mg/kg). Geochemical evaluation indicated that all of the detected
15 concentrations of barium were consistent with naturally occurring background concentrations of
16 barium. Based on the relatively low HQ_{screen} value for barium, the fact that none of the detected
17 concentrations of barium exceeded the Eco-SSL derived for the protection of mammalian
18 receptors, and the fact that geochemical evaluation indicated the detected concentrations of
19 barium were consistent with naturally occurring background concentrations, barium was not
20 identified as a COPEC in surface soil at the T-24A Ranges.

21
22 Excluding “B”-flagged data, five surface soil samples out of 106 total surface soil samples
23 exhibited beryllium concentrations greater than the ESV. The calculated HQ_{screen} value for
24 beryllium was 2.1. If the EPA (2005b) recommended Eco-SSLs for beryllium are used for
25 comparison, all of the detected concentrations of beryllium were less than the Eco-SSLs for the
26 protection of terrestrial invertebrates (40 mg/kg) and mammalian receptors (21 mg/kg).
27 Geochemical evaluation indicated that all of the detected concentrations of beryllium were
28 consistent with naturally occurring background concentrations of beryllium. Therefore,
29 beryllium was not considered a COPEC in surface soil at the T-24A Ranges.

30
31 Cadmium was detected in one out of 110 surface soil samples collected at the T-24A Ranges.
32 The calculated HQ_{screen} value for cadmium was 1.06. Geochemical evaluation indicated that all
33 of the detected concentrations of cadmium were consistent with naturally occurring background
34 concentrations of cadmium. Based on the infrequency of detection, the low level of the HQ_{screen}
35 value, and the fact that geochemical evaluation indicated the detected concentrations of cadmium
36 were consistent with naturally occurring background concentrations, cadmium was not identified

1 as a COPEC in surface soil at the T-24A Ranges.

2
3 One surface soil sample out of 110 samples exhibited a chromium concentration above its ESV
4 and BTV. The EPA (2004) recommends an Eco-SSL for chromium (III) ranging from 61 mg/kg
5 (based on toxicity to avian receptors) to 160 mg/kg (based on toxicity to mammalian receptors)
6 and an Eco-SSL for chromium (VI) of 380 mg/kg (based on mammalian toxicity). Only one
7 surface soil sample (location FTA-88-GP01) exhibited a chromium concentration that slightly
8 exceeded the EPA-recommended Eco-SSLs for Cr(III) for avian receptors ($HQ_{\text{screen}} = 1.8$). All
9 other detected concentrations of chromium were less than the Eco-SSLs. Geochemical
10 evaluation indicated that all of the detected concentrations of chromium were consistent with
11 naturally occurring background concentrations. Based on the infrequency of detection at
12 elevated concentrations and the fact that geochemical evaluation indicated the detected
13 concentrations of chromium were consistent with naturally occurring background concentrations
14 of chromium, chromium was not identified as a COPEC in surface soil at the T-24A Ranges.

15
16 Excluding “B”-flagged data, six surface soil samples out of 107 samples exhibited cobalt
17 concentrations above the ESV. The calculated HQ_{screen} value for cobalt was 2.1. Although a
18 number of samples exceeded the Eco-SSL (EPA, 2005) for the protection of plants (13 mg/kg),
19 none of the surface soil samples exhibited cobalt concentrations that exceeded the Eco-SSLs for
20 the protection of mammals (240 mg/kg) or birds (190 mg/kg). Geochemical evaluation indicated
21 that all of the detected concentrations of cobalt were consistent with naturally occurring
22 background concentrations of cobalt. Due to the low frequency of detected concentrations of
23 cobalt in exceedence of the ESV and Eco-SSLs, the relatively low magnitude of the HQ_{screen}
24 value, and the fact that geochemical evaluation indicated that all of the detected cobalt
25 concentrations were consistent with naturally occurring background concentrations of cobalt,
26 cobalt was not identified as a COPEC in surface soil at the T-24A Ranges.

27
28 Iron was detected in seven out of 110 surface soil samples at concentrations greater than the
29 BTV. Iron was detected at a maximum concentration that was 2.4 times the BTV for iron.
30 Geochemical evaluation indicated that iron was detected in surface soil at concentrations
31 consistent with naturally occurring levels. Iron is an essential macronutrient; thus, most
32 organisms have mechanisms to regulate the levels of iron within their systems. As such, iron is
33 only toxic at very high concentrations. Per EPA (2003) guidance the main concern from an
34 ecological risk perspective for iron is not direct chemical toxicity per se, but the effect of iron as
35 a mediator in the geochemistry of other metals and the potential physical hazard of depositing
36 flocculent under certain soil conditions. As such, EPA (2003) does not recommend a numerical
37 Eco-SSL for iron in soil, rather it is recommended that soil geochemical conditions (e.g. pH, Eh)

1 as well as the presence of iron floc are the critical determining factors when considering the
2 relative importance of iron in soil. Since iron was infrequently detected at elevated
3 concentrations relative to background, the soil pH is within normal ranges (pH of 5.5 to 8), the
4 fact that iron is a macronutrient that is easily regulated, and geochemical evaluation determined
5 that all of the detected concentrations of iron were found to be consistent with naturally
6 occurring levels, iron was not considered a COPEC in surface soil at the T-24A Ranges.

7
8 Excluding "B"-flagged data, mercury was detected in 3 surface soil samples out of 105 total
9 samples at concentrations that exceeded the ESV and naturally occurring levels. The HQ_{screen}
10 value for mercury was calculated to be 2.9. Based on the infrequency of detection at elevated
11 concentrations and the relatively low magnitude of the HQ_{screen} value, mercury was not identified
12 as a COPEC in surface soil at the T-24A Ranges.

13
14 Excluding "B"-flagged data, nickel was only detected in one surface soil sample (location R24A-
15 187-GP54) out of 104 samples at a concentration that slightly exceeded the ESV. The calculated
16 HQ_{screen} value for nickel was 1.3. The maximum detected concentration of nickel (37.7 mg/kg)
17 is less than the Eco-SSL of 38 mg/kg (EPA, 2004). Based on the infrequency of detection, low
18 HQ_{screen} value, and the fact that the maximum detected concentration is less than the Eco-SSL,
19 nickel was not identified as a COPEC in surface soil at the T-24A Ranges.

20
21 Excluding "B"-flagged data, 18 surface soil samples out of 92 samples exhibited selenium
22 concentrations above the ESV. The calculated HQ_{screen} value for selenium was 2.96.
23 Geochemical evaluation indicated that all of the detected concentrations of selenium were
24 consistent with naturally occurring background concentrations of selenium. Due to the relatively
25 low magnitude of the HQ_{screen} value and the fact that geochemical evaluation indicated that all of
26 the detected selenium concentrations were consistent with naturally occurring background
27 concentrations, selenium was not identified as a COPEC in surface soil at the T-24A Ranges.

28
29 Phenanthrene was initially identified as a COPEC in surface soil; however, it was only detected
30 in one surface soil sample out of 81 samples analyzed for this constituent at a concentration that
31 exceeded the ESV. The HQ_{screen} value for phenanthrene was calculated to be 1.9. The maximum
32 detected concentration of phenanthrene (0.19 mg/kg) is less than the background concentration
33 for phenanthrene in soils adjacent to asphalt (1.08 mg/kg) (IT, 2000). Based on the infrequency
34 of detection at elevated concentrations, the low magnitude of the HQ_{screen} value, and the fact that
35 the maximum detected concentration is less than the background concentration for phenanthrene
36 in soils adjacent to asphalt, phenanthrene was not identified as a COPEC in surface soil at the T-
37 24A Ranges.

1
2 Xylene and trichlorofluoromethane were each detected in one surface soil sample (location
3 R24A-187-MW25) out of 44 samples analyzed for these constituents at concentrations that
4 exceeded their ESVs. The HQ_{screen} values for xylene and trichlorofluoromethane were calculated
5 to be 1.4 and 2.0, respectively. Based on the low frequency of detection at concentrations above
6 the ESVs and the low magnitude of the HQ_{screen} values, xylene and trichlorofluoromethane were
7 not identified as COPECs in surface soil at the T-24A Ranges.

8
9 Chloroform was detected in 2 surface soil samples (locations R24A-187-MW25 and R24A-187-
10 GP31) out of 44 samples analyzed for this constituent at concentrations that exceeded the ESV.
11 The HQ_{screen} value for chloroform was calculated to be 320. EPA Region 5 (2003) provides an
12 Ecological Screening Level (ESL) for chloroform of 1.19 mg/kg in soil based on exposures to
13 masked shrews. All of the detected concentrations of chloroform are less than the EPA Region 5
14 ESL. Due to the low frequency of detection and the fact that all of the detected concentrations
15 are less than the EPA Region 5 ESL, chloroform was not identified as a COPEC in surface soil at
16 the T-24A Ranges.

17
18 The constituents in surface soil at the T-24A Ranges that were identified as COPECs through
19 examination of multiple lines of evidence were the following:

- 20
21
- 22 • Antimony
 - 23 • Copper
 - 24 • Lead
 - 25 • Zinc.

26 **7.6.2 Surface Water**

27 The following constituents exceeded their respective ESVs and BTVs in surface water at the T-
28 24A Ranges, and are not essential macronutrients:

- 29
- | | | | |
|----|-------------|----|-------------------------------|
| 30 | • Aluminum | 35 | • Lead |
| 31 | • Barium | 36 | • Vanadium |
| 32 | • Beryllium | 37 | • Zinc |
| 33 | • Chromium | 38 | • Bis(2-ethylhexyl)phthalate. |
| 34 | • Copper | | |

39
40 All surface water samples were analyzed for total recoverable constituent concentrations.

41

1 One surface water sample (location R24A-187-SW/SD07) exhibited aluminum at a concentration
2 greater than the BTV. Geochemical evaluation indicated that all of the detected concentrations
3 of aluminum in surface water were consistent with naturally occurring background
4 concentrations. Aluminum is the most abundant metal in the earth's crust. Because only a single
5 sample exhibited an aluminum concentration greater than the BTV and geochemical evaluation
6 indicated all of the detected aluminum was consistent with naturally occurring levels, aluminum
7 was not identified as a COPEC in surface water at the T-24A Ranges.

8
9 Two surface water samples (locations R24A-187-SW/SD06 and R24A-187-SW/SD07) out of 11
10 total samples exhibited barium concentrations greater than the ESV and BTV. Geochemical
11 evaluation indicated that all of the detected concentrations of barium in surface water were
12 consistent with naturally occurring background concentrations. The HQ_{screen} value for barium in
13 surface water was calculated to be 126. Based on the magnitude of the HQ_{screen} value and the
14 fact that the elevated concentrations of barium were co-located with elevated concentrations of
15 other surface water COPECs, barium was identified as a COPEC in surface water at the T-24A
16 Ranges.

17
18 Beryllium was detected in one surface water sample out of 11 samples collected at a
19 concentration exceeding its ESV and BTV. Geochemical evaluation indicated that the single
20 detected concentration of beryllium in surface water was consistent with naturally occurring
21 background concentrations of beryllium in surface water. The HQ_{screen} value for beryllium in
22 surface water was calculated to be 4.2. Because beryllium was infrequently detected at an
23 elevated concentration, the HQ_{screen} value is relatively low, and geochemical evaluation indicated
24 beryllium concentrations were consistent with naturally occurring concentrations, beryllium was
25 not identified as a COPEC in surface water at the T-24A Ranges.

26
27 Chromium was detected in two samples out of 11 samples at concentrations that exceeded the
28 ESV and BTV. The calculated HQ_{screen} value for chromium is 3.7. This HQ_{screen} value was
29 calculated using the ESV which is based on hexavalent chromium toxicity. If the National
30 Recommended Water Quality Criterion (EPA, 2002) for trivalent chromium is used for
31 comparison, all of the detected concentrations of chromium are below the recommended
32 criterion. Additionally, geochemical evaluation indicated that both the detected concentrations
33 of chromium in surface water were consistent with naturally occurring background
34 concentrations of chromium in surface water. Therefore, chromium was not identified as a
35 COPEC in surface water at the T-24A Ranges.

1 Copper and lead were detected in a total of three out of 11 surface water samples at
2 concentrations that exceeded their respective ESVs and BTVs. The HQ_{screen} values for copper
3 and lead were calculated to be 16.4 and 326, respectively. Geochemical evaluation indicated that
4 all of the detected concentrations of copper and lead in surface water were consistent with
5 naturally occurring background concentrations of copper and lead in surface water. However,
6 since both copper and lead were identified as COPECs in surface soil at the T-24A Ranges and
7 are known components of munitions, both copper and lead were identified as COPECs in surface
8 water in at least a limited area at the T-24A Ranges.

9
10 One surface water sample out of 11 samples exhibited a vanadium concentration greater than the
11 ESV and BTV and one surface water sample out of six samples (excluding "B"-flagged data)
12 exhibited a zinc concentration greater than the ESV and BTV. Geochemical evaluation indicated
13 that all of the detected concentrations of vanadium and zinc in surface water were consistent with
14 naturally occurring background concentrations. The HQ_{screen} value for vanadium was calculated
15 to be 3.5. Based on the infrequency of detection at elevated concentrations, the relatively low
16 HQ_{screen} value, and the fact that geochemical evaluation indicated vanadium in surface water was
17 naturally occurring, vanadium was not identified as a COPEC in surface water at the T-24A
18 Ranges. Although zinc was only detected in one surface water sample at an elevated
19 concentration, zinc was identified as a COPEC in surface water because zinc was identified as a
20 COPEC in surface soil at the T-24A Ranges and the elevated concentration of zinc in surface
21 water was co-located with the elevated concentrations of copper and lead in surface water.

22
23 Bis(2-ethylhexyl)phthalate was detected in two surface water samples out of 11 samples at
24 concentrations that exceeded the ESV. The HQ_{screen} value was calculated to be 53.3. Although
25 infrequently detected, the HQ_{screen} value indicates the potential for concern; therefore, bis(2-
26 ethylhexy)phthalate was identified as a COPEC in surface water at the T-24A Ranges.

27
28 The constituents in surface water at the T-24A Ranges that were identified as COPECs through
29 examination of additional lines of evidence were the following:

- 30
- 31 • Barium
- 32 • Copper
- 33 • Lead
- 34 • Zinc
- 35 • Bis(2-ethylhexyl)phthalate.
- 36

1 **7.6.3 Sediment**

2 Sediment samples from the T-24A Ranges exhibited maximum concentrations of the following
3 constituents that exceeded ESVs and BTVs, and were not essential macronutrients:

- 4
- | | | | |
|----|-------------|----|-----------------------|
| 5 | • Aluminum | 13 | • Thallium |
| 6 | • Barium | 14 | • Zinc |
| 7 | • Beryllium | 15 | • Di-n-butylphthalate |
| 8 | • Copper | 16 | • Chloromethane |
| 9 | • Iron | 17 | • Benzo(a)anthracene |
| 10 | • Lead | 18 | • Chrysene |
| 11 | • Mercury | 19 | • Fluoranthene |
| 12 | • Nickel | 20 | • Pyrene. |

21

22 Aluminum was detected in five out of 11 sediment samples at concentrations that exceeded the
23 BTV. There is no ESV for aluminum in sediment. Geochemical evaluation indicated that all of
24 the detected concentrations of aluminum in sediment were consistent with naturally occurring
25 background concentrations of aluminum in sediment. Aluminum is the most abundant metal in
26 the earth's crust. Because the maximum detected aluminum concentrations only slightly
27 exceeded the BTV and geochemical evaluation indicated that all of the detected aluminum in
28 sediment was naturally occurring, aluminum was not identified as a COPEC in sediment.

29

30 Three out of 11 sediment samples exhibited barium and beryllium concentrations greater than
31 their respective BTVs. There are no ESVs for barium or beryllium in sediment. Geochemical
32 evaluation indicated that all of the detected concentrations of barium and beryllium in sediment
33 were consistent with naturally occurring background concentrations. Neither barium nor
34 beryllium was identified as a COPEC in surface soil at the T-24A Ranges. Because the
35 maximum detected barium and beryllium concentrations only slightly exceeded their BTVs and
36 geochemical evaluation indicated that all of the detected barium and beryllium in sediment was
37 naturally occurring, neither barium nor beryllium were identified as COPECs in sediment.

38

39 Copper was detected in four sediment samples at concentrations that exceeded the ESV and
40 BTV. The HQ_{screen} value for copper in sediment was calculated to be 1.9. Long, et al., (1995)
41 have derived two values for sediment that describe the distribution of the toxicological effects
42 data in the scientific literature. The lower 10th percentile of the effects data for each chemical
43 was identified and referred to as the effects range-low (ERL). The median, or 50th percentile, of
44 the effects data was identified and referred to as the effects range-median (ERM). Sediment
45 constituent concentrations less than the ERL are rarely associated with adverse effects. Sediment
46 constituent concentrations between the ERL and ERM are occasionally associated with adverse

1 effects. Sediment constituent concentrations greater than the ERM are frequently associated
2 with adverse effects. Long, et al., (1995) derived the ERL and ERM values for copper as 34
3 mg/kg and 270 mg/kg, respectively. One of the sediment samples exhibited copper (R24A-187-
4 SD07) at a concentration that slightly exceeded the ERL, but was less than the ERM. Based on
5 the methodology presented by Long, et al. (1995), copper in sediment from one location could be
6 expected to induce adverse effects in sensitive benthic organisms rarely to occasionally, but not
7 frequently. All of the other sediment samples exhibited copper concentrations less than the ERL.
8 Geochemical evaluation indicated that all of the detected concentrations of copper in sediment
9 were consistent with naturally occurring background concentrations of copper in sediment.
10 Based on the relatively low HQ_{screen} value, the low frequency of detection at concentrations
11 exceeding the ERM, and the fact that geochemical evaluation indicated that the detected
12 concentrations of copper were consistent with naturally occurring background, copper was not
13 identified as a COPEC in sediment.

14
15 Two sediment samples exhibited iron concentrations greater than the BTV for iron. There is no
16 ESV for iron in sediment. Geochemical evaluation indicated that all of the detected
17 concentrations of iron in sediment were consistent with naturally occurring background
18 concentrations. Iron is often considered a macronutrient which is easily regulated by most
19 organisms and only toxic at very high levels. Since iron was detected relatively infrequently at
20 elevated concentrations compared to the BTV and geochemical evaluation indicated that the
21 detected iron was consistent with naturally occurring levels, iron was not identified as a COPEC
22 in sediment.

23
24 Lead was detected in four sediment samples at concentrations that exceeded the ESV. The
25 HQ_{screen} value for lead in sediment was calculated to be 5.2. Geochemical evaluation indicated
26 that all of the detected concentrations of lead in sediment were consistent with naturally
27 occurring background concentrations of lead in sediment. However, because lead is a known
28 component of munitions and is present in several samples at elevated concentrations, lead was
29 identified as a COPEC in sediment.

30
31 Mercury was detected in one sediment sample at a concentration that exceeded the ESV and
32 BTV. The HQ_{screen} value for mercury in sediment was calculated to be 8571. One sediment
33 sample (location FTA-88-SW/SD01) exhibited mercury at a concentration that slightly exceeded
34 the ERL, but was less than the ERM. Based on the methodology presented by Long, et al.
35 (1995), mercury in sediment from one location could be expected to induce adverse effects in
36 sensitive benthic organisms rarely to occasionally, but not frequently. Excluding "B"-flagged
37 data, none of the other sediment samples had detectable concentrations of mercury.

1 Geochemical evaluation indicated that the single detection of mercury in sediment was consistent
2 with naturally occurring background concentrations of mercury in sediment. However, based on
3 the magnitude of the HQ_{screen} value, mercury was identified as a COPEC in sediment.

4
5 Nickel was detected in one sediment sample at a concentration that exceeded the ESV and BTV.
6 The HQ_{screen} value for nickel in sediment was calculated to be 1.7. One sediment sample
7 (location FTA-108-SW/SD02) exhibited nickel at a concentration that slightly exceeded the
8 ERL, but was less than the ERM. Based on the methodology presented by Long, et al. (1995),
9 nickel in sediment from one location could be expected to induce adverse effects in sensitive
10 benthic organisms rarely to occasionally, but not frequently. All of the other sediment samples
11 exhibited nickel concentrations less than the ERL. Geochemical evaluation indicated that all of
12 the detected concentrations of nickel in sediment were consistent with naturally occurring
13 background concentrations of nickel in sediment. Based on the infrequency of detection at
14 concentrations above the ESV, the low magnitude of the HQ_{screen} value, and the fact that
15 geochemical evaluation indicated that nickel in sediment was naturally occurring, nickel was not
16 identified as a COPEC in sediment.

17
18 Thallium was detected at estimated (“J”-flagged) concentration in one out of 11 sediment
19 samples. The detected concentration exceeded the BTV. There is no ESV for thallium in
20 sediment. Although the estimated concentration of thallium is greater than the BTV, the
21 infrequency of detection results in thallium not being identified as a COPEC in sediment.

22
23 Zinc was detected in one out of 11 sediment samples at a concentration that exceeded the ESV
24 and BTV. The HQ_{screen} value for zinc in sediment was calculated to be 1.5. One sediment
25 sample (location FTA-88-SW/SD02) exhibited zinc at a concentration that slightly exceeded the
26 ERL, but was less than the ERM. Based on the methodology presented by Long, et al. (1995),
27 zinc in sediment from one location could be expected to induce adverse effects in sensitive
28 benthic organisms rarely to occasionally, but not frequently. All of the other sediment samples
29 exhibited zinc concentrations less than the ERL. Geochemical evaluation indicated that all of the
30 detected concentrations of zinc in sediment were consistent with naturally occurring background
31 concentrations. Based on the infrequency of detection at concentrations above the ESV, the low
32 magnitude of the HQ_{screen} value, and the fact that geochemical evaluation indicated that zinc in
33 sediment was naturally occurring, zinc was not identified as a COPEC in sediment.

34
35 Benzo(a)anthracene, chrysene, di-n-butylphthalate, fluoranthene, and pyrene were all detected in
36 one or two samples at concentrations that exceeded their respective ESVs. The HQ_{screen} values
37 for these SVOCs ranged from 2.88 to 6.06. Although these SVOCs were relatively infrequently

1 detected, the maximum detected concentrations may indicate isolated areas of contamination;
2 therefore, these SVOCs were identified as COPECs in sediment.

3
4 Chloromethane was detected in one out of 11 sediment samples at a concentration that exceeded
5 the ESV. The HQ_{screen} value for chloromethane in sediment was calculated to be 42. The ESV
6 for chloromethane has been rescinded by EPA Region 5 due to the lack of supporting data. The
7 ESV for chloromethane is four orders of magnitude lower than the ESVs for most other VOCs.
8 If the ESV for chloromethane was similar to the ESVs for the other VOCs, then it is likely the
9 maximum detected concentration of chloromethane in sediment would be less than the screening
10 level. For this reason, chloromethane was not identified as a COPEC in sediment.

11
12 The constituents in sediment at the T-24A Ranges that were identified as COPECs through
13 examination of additional lines of evidence were the following:

- 14
- 15 • Lead
- 16 • Mercury
- 17 • Benzo(a)anthracene
- 18 • Chrysene
- 19 • Di-n-butylphthalate
- 20 • Fluoranthene
- 21 • Pyrene.
- 22

23 **7.6.4 Groundwater**

24 Groundwater samples from the T-24A Ranges exhibited maximum concentrations of the
25 following constituents that exceeded surface water ESVs and available groundwater BTVs, and
26 were not essential macronutrients:

- 27
- 28 • Aluminum
- 29 • Barium
- 30 • Chromium
- 31 • Iron
- 32 • Manganese
- 33 • Mercury
- 34 • Bis(2-ethylhexyl)phthalate
- 35 • Benzene
- 36 • Carbon tetrachloride.
- 37

38 It is important to note that ecological receptors do not have a direct exposure pathway to
39 groundwater. Ecological receptors can only be exposed to constituents in groundwater if
40 groundwater is expressed at the ground surface as seeps or is discharged to lakes or streams via

1 springs. Exposure of ecological receptors to groundwater could then occur via surface water
2 pathways. Contaminants that may have entered groundwater in the past are likely to have been
3 mostly, if not entirely, transported to surface water bodies by now, and if ongoing groundwater
4 contamination of surface water bodies were a concern, surface water samples would indicate the
5 presence of groundwater contaminants.

6
7 Aluminum was detected in one groundwater sample out of 37 samples at a concentration that
8 exceeded the BTV and the detected concentrations routinely exceeded the ESV for aluminum in
9 surface water. Geochemical evaluation indicated that all of the detected concentrations of
10 aluminum in groundwater were consistent with naturally occurring background concentrations of
11 aluminum in groundwater. Based on the low frequency of detection at concentrations above
12 background and the fact that geochemistry evaluation indicated that the detected aluminum is
13 naturally occurring, aluminum was not identified as a COPEC in groundwater.

14
15 Barium was detected in three groundwater samples that exceeded the BTV and the detected
16 concentrations routinely exceeded the ESV for barium in surface water. The HQ_{screen} value for
17 barium was calculated to be 805. Based on the magnitude of the HQ_{screen} value, the frequency of
18 detection at concentrations exceeding the ESV, and the fact that barium was identified as a
19 COPEC in surface water, barium was also identified as a COPEC in groundwater.

20
21 Chromium was detected in one groundwater sample out of 38 samples at a concentration that
22 exceeded both the ESV and BTV for chromium in surface water. The HQ_{screen} value for
23 chromium was calculated to be 1.1. Geochemical evaluation indicated that all of the detected
24 concentrations of chromium in groundwater were consistent with naturally occurring background
25 concentrations of chromium in groundwater. Based on the infrequency of detection at elevated
26 concentrations, the low magnitude of the HQ_{screen} value, and the fact that geochemical evaluation
27 indicated that the detected chromium was naturally occurring, chromium was not identified as a
28 COPEC in groundwater.

29
30 Iron was detected in five groundwater samples at concentrations greater than the BTV and the
31 detected concentrations in several samples exceeded the ESV for iron in surface water. The
32 HQ_{screen} value for iron was calculated to be 11.7. Geochemical evaluation indicated that all of
33 the detected concentrations of iron in groundwater were consistent with naturally occurring
34 background concentrations of iron in groundwater. Iron is often considered a macronutrient
35 which is easily regulated by most organisms and only toxic at very high levels. Because the
36 maximum detected concentrations of iron in groundwater only slightly exceed the BTV,
37 geochemical evaluation indicated all of the detected iron in groundwater is naturally occurring,

1 and iron is often considered an essential macronutrient, iron was not considered a COPEC in
2 groundwater.

3
4 Manganese was detected in 13 groundwater samples at concentrations that exceeded the BTV
5 and the detected concentrations routinely exceeded the ESV for manganese in surface water.
6 Geochemical evaluation indicated that all of the detected concentrations of manganese in
7 groundwater were consistent with naturally occurring background concentrations of manganese
8 in groundwater. Although manganese was detected fairly frequently at concentrations that
9 exceeded the BTV, the detected concentrations did not exceed the BTV by significant amounts.
10 Since geochemical evaluation indicated that the detected concentrations of manganese were
11 naturally occurring and manganese was not identified as a COPEC in surface soil or surface
12 water, manganese was not identified as a COPEC in groundwater.

13
14 Mercury was detected in one groundwater sample out of 38 samples at a concentration that
15 exceeded the ESV for mercury in surface water. There are no background values for mercury in
16 groundwater or surface water at FTMC (SAIC, 1998). However, geochemical evaluation
17 indicated that all of the detected concentrations of mercury in groundwater were consistent with
18 naturally occurring levels. Based on the infrequency of detection, the fact that mercury was not
19 identified as a COPEC in surface soil or surface water, and the fact that geochemical evaluation
20 indicated that all of the detected mercury in groundwater was naturally occurring, mercury was
21 not identified as a COPEC in groundwater.

22
23 Bis(2-ethylhexyl)phthalate was detected in two out of 37 groundwater samples at concentrations
24 that exceeded the surface water ESV. Although bis(2-ethylhexyl)phthalate was infrequently
25 detected in groundwater, it was identified as a COPEC in surface water; therefore, it was also
26 identified as a COPEC in groundwater.

27
28 Benzene and carbon tetrachloride were each detected in one out of 61 groundwater samples at
29 concentrations that exceeded their respective surface water ESVs. The HQ_{screen} values for
30 benzene and carbon tetrachloride were calculated to be 18.3 and 1.1, respectively. Neither
31 benzene nor carbon tetrachloride was detected in surface soil or surface water. Based on the
32 infrequency of detection, the relatively low HQ_{screen} values, and the fact that these constituents
33 were not detected in any other environmental medium at T-24A, benzene and carbon
34 tetrachloride were not identified as COPECs in groundwater.

35
36 The constituents in groundwater at the T-24A Ranges that were identified as COPECs through
37 examination of additional lines of evidence were the following:

- Barium
- Bis(2-ethylhexyl)phthalate.

The initial list of COPECs was scrutinized using additional lines of evidence. These additional lines of evidence included 1) frequency of detection, 2) magnitude of the HQ_{screen} value, 3) spatial distribution, 4) comparison to alternative ESVs; 5) additional background evaluation; and 6) association of a chemical with known Army activities. Based on these additional lines of evidence, the COPECs that have been identified at the Ranges Near Training Area T-24A are summarized below:

- **Surface Soil** – Antimony, copper, lead, and zinc.
- **Surface Water** – Barium, copper, lead, zinc, and bis(2-ethylhexyl)phthalate.
- **Sediment** – Lead, mercury, benzo(a)anthracene, chrysene, di-n-butylphthalate, fluoranthene, and pyrene.
- **Groundwater** – Barium, and bis(2-ethylhexyl)phthalate.

7.7 Uncertainty Analysis

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

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

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

1
2 Some COPECs (e.g., bromomethane) do not have ESVs. The lack of toxicity data for certain
3 COPECs makes it impossible to determine the potential for ecological risk posed by those
4 constituents. Risks may be under- or over-estimated due to this uncertainty.

5
6 The ESVs used in this assessment are all the most conservative values from the scientific
7 literature and many are based on the most sensitive endpoint (NOAEL values) for the most
8 sensitive species tested. A less sensitive endpoint that is still protective of the ecological
9 populations or communities of interest may be the LOAEL or some other endpoint. The use of
10 NOAEL-based ESVs may overestimate potential for risks from certain COPECs. Additionally,
11 certain ESVs may not be applicable to conditions at the Ranges Near Training Area T-24A.
12 For instance, the soil ESVs do not take into account site-specific conditions at the Ranges Near
13 Training Area T-24A and thus introduce a potentially significant level of uncertainty into the
14 assessment.

15
16 It is important to note that the soil and surface water ESVs for chromium are based on toxicity
17 studies using Cr^{+6} , which is more mobile and generally more toxic than Cr^{+3} . Measured
18 chromium concentrations in surface soil and surface water are total chromium values. It is
19 unknown what portion, if any, of the measured chromium in surface soil and surface water at the
20 Ranges Near Training Area T-24A is in the hexavalent form. Because it is likely that only a
21 portion or none of the measured chromium in surface soil and surface water is in the hexavalent
22 form, the ESV and subsequent $\text{HQ}_{\text{screen}}$ values are highly conservative.

23
24 Another area of uncertainty is the inherent limitations of the hazard quotient method for
25 estimating risks. Hazard quotients (HQ) are not explicit expressions of risk (i.e. they are not
26 probabilities of toxicological effects occurring in an ecological population). Additionally,
27 because HQs are ratios, after unity has been exceeded, the magnitude of the HQ has little bearing
28 on the potential severity of adverse effects that may be anticipated. An HQ of five does not
29 indicate the potential ecological risk is greater than a HQ of three. Hazard quotients are not
30 population measures, but rather measures based on sensitive individuals from a test population.

31
32 Another area of uncertainty is the lack of consideration of synergisms and/or antagonisms
33 between COPECs. Although it is widely accepted that synergisms and antagonisms occur
34 between certain constituents under certain conditions, the SLERA process does not provide
35 methods for assessing these potential synergisms/antagonisms.

1 One source of uncertainty that may impart a non-conservative bias on the SLERA results is the
2 exclusion of certain metals determined to be present at concentrations comparable to naturally-
3 occurring background concentrations from consideration as COPECs. As noted above, the
4 exclusion of chemicals from the list of COPECs based on comparison to naturally-occurring
5 levels is performed via a three-tiered protocol (Shaw, 2005). Tier 1 – comparison of the
6 maximum detected constituent concentration to the BTV – and Tier 2 – statistical comparison of
7 detected constituent concentrations to the background data set - are generally considered to be
8 sufficiently conservative so that the uncertainty associated with chemicals eliminated in these
9 two tiers of the protocol is minimal. The greatest uncertainty in the background evaluation is
10 present in the geochemical evaluation (Tier 3). Therefore, the geochemical evaluation is only
11 used in conjunction with other lines of evidence to draw conclusions regarding the
12 inclusion/exclusion of a given constituent for consideration as a COPEC. The use of multiple
13 lines of evidence reduces the uncertainty in the COPEC identification process.

14 15 **7.8 Summary and Conclusions**

16 The potential for ecological risks at the Ranges Near Training Area T-24A was determined
17 through a SLERA. This ecological screening process consisted of a characterization of the
18 ecological setting at the Ranges Near Training Area T-24A, development of a SCM, a
19 description of the fate and transport of constituents detected in various environmental media, a
20 description of the ecotoxicity of the various constituents detected at the Ranges Near Training
21 Area T-24A, a description of the ecological receptors, a description of the complete exposure
22 pathways, calculation of screening-level hazard quotients, and a description of the uncertainties
23 within the process.

24
25 A number of constituents were detected in surface soil at the Ranges Near Training Area T-24A
26 that exhibited maximum concentrations that exceeded their respective ESVs and BTVs.

27 Consideration of additional lines of evidence resulted in the identification of the following
28 COPECs at the Ranges Near Training Area T-24A:

29 30 Surface Soil COPECs

- 31 • Antimony
- 32 • Copper
- 33 • Lead
- 34 • Zinc.

35 36 Surface Water COPECs

- 37 • Barium
- 38 • Copper

- 1 • Copper
- 2 • Lead
- 3 • Zinc
- 4 • Bis(2-ethylhexyl)phthalate.

5

6 Sediment COPECs

- 7 • Lead
- 8 • Mercury
- 9 • Benzo(a)anthracene
- 10 • Chrysene
- 11 • Di-n-butyl phthalate
- 12 • Fluoranthene
- 13 • Pyrene.

14

15 Groundwater COPECs

- 16 • Barium
- 17 • Bis(2-ethylhexyl)phthalate.

18

19 **Conclusions.** The SLERA for the Ranges Near Training Area T-24A determined that several
20 constituents in surface soil, surface water, sediment, and groundwater were COPECs. Because
21 several constituents were detected in the environmental media at the Ranges Near Training Area
22 T-24A at concentrations that exceeded their respective ESVs and naturally occurring levels, and
23 because conservative assessment techniques were used in the SLERA process, a more thorough
24 assessment (e.g. Baseline Ecological Risk Assessment) is warranted to reduce uncertainties
25 inherent in the SLERA process and to determine the potential for ecological risk from
26 constituents in surface soil, surface water, sediment, and groundwater at the Ranges Near
27 Training Area T-24A.

1 **References**

2
3 Abbasi, S.A. and R. Soni, 1983. Stress-Induced Enhancement of Reproduction in Earthworm
4 *Octochaetus pattoni* Exposed to Chromium (VI) and Mercury (II) – Implications in
5 Environmental Management. *Intern. J. Environ. Stud.*, 22: 43-47.

6
7 Adriano, D. C., 1986. Trace Elements in the Terrestrial Environment. Springer-Verlag, New
8 York, NY.

9
10 Adams, E.M., H.C. Spencer, V.K. Rowe, D.D. McCollister, and D.D. Irish, 1952. Vapor
11 Toxicity of Carbon Tetrachloride Determined by Experiments on Laboratory Animals. *Arch.*
12 *Ind. Hyg. Occup. Med.* 6: 50-66.

13
14 Agency for Toxic Substances and Disease Registry (ATSDR), 1998. *Toxicological Profile for*
15 *Chloromethane*. U.S. Public Health Service.

16
17 Agency for Toxic Substances and Disease Registry (ATSDR), 1993a. *Toxicological Profile for*
18 *Beryllium*. U.S. Public Health Service.

19
20 Agency for Toxic Substances and Disease Registry (ATSDR), 1993b. *Toxicological Profile for*
21 *Cadmium*. U.S. Public Health Service.

22
23 Agency for Toxic Substances and Disease Registry (ATSDR), 1993c. *Toxicological Profile for*
24 *Diethyl Phthalate*. Draft for Public Comment, DHHS, Public Health Service, Atlanta, Georgia.

25
26 Agency for Toxic Substances and Disease Registry (ATSDR), 1992a. *Toxicological Profile for*
27 *Barium*. U.S. Public Health Service.

28
29 Agency for Toxic Substances and Disease Registry (ATSDR), 1992b. *Toxicological Profile for*
30 *Manganese*. U.S. Public Health Service.

31
32 Agency for Toxic Substances and Disease Registry (ATSDR), 1992c. *Toxicological Profile for*
33 *Vanadium*. U.S. Public Health Service.

34
35 Agency for Toxic Substances and Disease Registry (ATSDR), 1992d. *Toxicological Profile for*
36 *Carbon Tetrachloride*. U.S. Public Health Service.

37
38 Ainsworth, N., 1988. Distribution and Biological Effects of Antimony in Contaminated
39 Grassland. Dissertation. As cited in ATSDR, 1990.

40
41 Allen, W.R., W.L. Askew, and K. Schreiber, 1961. Effect of Insecticide Fertilizer Mixtures and
42 Seed Treatments on Emergence of Sugar Beet Seedlings. *J. Econ. Entom.* 54(1): 181-187.

43
44 Alumot, E., E. Nachtomi, E. Mandel, and P. Holstein, 1976. Tolerance and Acceptable Daily
45 Intake of Chlorinated Fumigants in the Rat Diet. *Fd. Cosmet. Toxicol.* 14: 105-110.

46
47 Ambrose, A.M., P.S. Larson, J.F. Borzelleca, and G.R. Hennigar, 1976. Long-Term Toxicologic
48 Assessment of Nickel in Rats and Dogs. *J. Food Sci. Tech.* 13: 181-187.

- 1
2 Amelung, M., 1981. *Auswirken Geloster Eisenverbindungen auf die Ei- und*
3 *Larvalentwicklung von Salmo gairdneri* (Richardson). Arch. Fisch Wiss. 32:77-87.
4
5 American Conference of Governmental Industrial Hygienists (ACGIH), 1991. Documentation
6 of the Threshold Limit Values and Biological Exposure Indices, Sixth Edition. Cincinnati, OH.
7 Pp. 1619-1623.
8
9 Anderson, R. L., C. T. Walbridge, and J. T. Fiandt, 1980. Survival and Growth of *Tanytarsus*
10 *dissimilis* (Chironomidae) Exposed to Copper, Cadmium, Zinc, and Lead. *Arch. Environ.*
11 *Contam. Toxicol.*, 9:329-335.
12
13 Armstrong, F.A.J., 1979. Effects of Mercury Compounds on Fish. In: J.O. Nriagu (ed.) *The*
14 *Biogeochemistry of Mercury in the Environment*, Elsevier/North-Holland Biomedical Press,
15 New York, pp. 657-670.
16
17 Arthur, M.A., G. Rubin, P.B. Woodbury, R.E. Schneider, and L.H. Weinstein, 1992. *Uptake*
18 *and Accumulation of Selenium by Terrestrial Plants Growing on a Coal Fly Ash Landfill;*
19 *Part 2: Forage and Root Crops*. Environmental Toxicology and Chemistry, Vol. 11, pp. 1289-
20 1299.
21
22 Aulerich, R.J., R.K. Ringer, M.R. Bleavins, et al., 1982. Effects of Supplemental Dietary
23 Copper on Growth, Reproductive Performance, and Kit Survival of Standard Dark Mink and the
24 Acute Toxicity of Copper to Mink. *J. Animal Sci.* 55: 337-343.
25
26 Azar, A., H.J. Trochimowicz, and M.E. Maxwell, 1973. Review of Lead Studies in Animals
27 Carried Out at Haskell Laboratory: Two Year Feeding Study and Response to Hemorrhage
28 Study, In: Environmental Health Aspects of Lead: Proceedings, International Symposium. D.
29 Barth, et al., eds., *Commission of European Communities*, pp. 199-210.
30
31 Baudouin, M. F., and P. Scoppa, 1974. Acute Toxicity of Various Metals to Freshwater
32 Zooplankton. *Bull. Environ. Contam. Toxicol.*, 12:745-751.
33
34 Bengtsson, G., T. Gunnarsson, and S. Rundgren, 1986. Effects of Metal Pollution on the
35 Earthworm *Dendrobaena rubida* (Sav.) in Acidified Soils. *Water Soil Air Pollut.* 28: 361-383.
36
37 Besser, J.M., T.J. Canfield, and T.W. LaPoint, 1993. *Bioaccumulation of Organic and*
38 *Inorganic Selenium in a Laboratory Food Chain*. Environmental Contamination and
39 Toxicology, Vol. 12, pp. 57-72.
40
41 Beyer, W. N., O. H. Pattee, L. Sileo, D. J. Hoffman, and B. M. Mulhern, 1985. Metal
42 Contamination in Wildlife Living Near Two Zinc Smelters. *Environ. Pollut.*, 38A:63-86.
43
44 Biesinger, K.E. and G.M. Christensen, 1972. *Effects of Various Metals on Survival, Growth,*
45 *Reproduction, and Metabolism of Daphnia magna*. J. Fish. Res. Bd. Canada, 29:1691-1700.
46
47 Boggess, W. R. (Ed.), 1977. Lead in the Environment. *National Science Foundation, Rep.*
48 *NSF/RA 770214*, 272 pp.

- 1
2 Boikat, U., A. Fink, and J. Bleck-Neuhaus, 1985. *Cesium and Cobalt Transfer from Soil to*
3 *Vegetation on Permanent Pastures*. Radiation and Environmental Biophysics, 24: 287-301.
4
5 Bollard, E.G., 19893. *Involvement of Unusual Elements in Plant Growth and Nutrition*. In:
6 Inorganic Plant Nutrition. A. Lauchli and R.L. Bielecki (eds.). Springer-Verlag, Berlin,
7 Germany. Pp. 743-795.
8
9 Borzelleca, J.F., L.W. Condie, and J.L. Egle, 1988. *Short-Term Toxicity (One- and Ten-Day*
10 *Gavage) of Barium Chloride in Male and Female Rats*. J. American College of Toxicology, 7:
11 675-685.
12
13 Browne, C.L., and S.C. Fang, 1978. Uptake of Mercury Vapor by Wheat: An Assimilation
14 Model. *Plant Physiology*, Vol. 61, p. 430.
15
16 Bull, K.R., R.D. Roberts, M.J. Inskip, and G.T. Goodman, 1977. Mercury Concentrations in
17 Soil, Grass, Earthworms, and Small Mammals Near an Industrial Emission Source.
18 *Environmental Pollution*, Vol. 12, pp. 135-140.
19
20 Burrows, E.P., D.H. Rosenblatt, W.R. Mitchell, and D.L. Parmer, 1989. Organic Explosives and
21 Related Compounds: Environmental and Health Considerations. *U.S. Army Technical Report*
22 *8901*.
23
24 Cain, B.W. and E.A. Pafford, 1981. Effects of Dietary Nickel on Survival and Growth of
25 Mallard Ducklings. *Arch. Environ. Contam. Toxicol.*, 10: 737-745.
26
27 Cairns, J., and A. Scheier, 1968. A Comparison of the Toxicity of Some Common Industrial
28 Waste Components Tested Individually and Combined. *Prog. Fish-Cult.* 30:3-8.
29
30 Callahan, M. A., M. W. Slimak, and N. Gabel, 1979. *Water-Related Environmental Fate of*
31 *129 Priority Pollutants, Volume I*. Office of Water and Waste Management, U.S.
32 Environmental Protection Agency, EPA/440/4-79/092a, Washington, DC.
33
34 Carins, M.A., A.V. Nebeker, J.H. Gakstatter, and W.L. Griffis, 1984. Toxicity of Copper Spiked
35 Sediments to Freshwater Invertebrates. *Environ. Toxicol. Chem.* 3(3): 435-446.
36
37 Carpenter, K. E., 1926. The Lead Mine as an Active Agent in River Pollution. *Ann. Appl. Biol.*,
38 13:395.
39
40 Carpenter, K. E., 1925. On the Biological Factors Involved in the Destruction of River Fisheries
41 by Pollution Due to Lead Mining. *Ann. Appl. Biol.*, 12:1.
42
43 Carpenter, K. E., 1924. A Study of the Faunal Rivers Polluted by Lead Mining in the
44 Aberystwyth District of Cardiganshire. *Ann. Appl. Biol.*, 11:1.
45
46 Carriere, D., K. Fischer, D. Peakall, and P. Angehrn, 1986. *Effects of Dietary Aluminum in*
47 *Combination With Reduced Calcium and Phosphorus on the Ring Dove (*Streptopelia risoria*)*.
48 Water, Air, and Soil Poll., 30: 757-764.

- 1
2 Clark, M. L., D. G. Harvey, and D. J. Humphreys, 1981. *Veterinary Toxicology - Second*
3 *Edition*, Bailliere-Tindall, London, England.
4
5 Cowgill, U.M. and C.W. Burns, 1975. *Differences in Chemical Composition Between Two*
6 *Species of Daphnia and Some Freshwater Algae Cultured in the Laboratory*. Limnology and
7 Oceanography, Vol. 20, pp. 1005-1011.
8
9 Cox, D. H., S. A. Schlicker, and R. C. Chu, 1969. Excess Dietary Zinc for the Maternal Rat and
10 Zinc, Iron, Copper, Calcium, and Magnesium Content and Enzyme Activity in Maternal and
11 Fetal Tissues. *J. Nutr.*, 98:459-466.
12
13 Coyle, J.J., D.R. Buckler, C.G. Ingersoll, J.F. Fairchild, and T.W. May, 1993. *Effect of Dietary*
14 *Selenium on the Reproductive Success of Bluegills (Lepomis macrochirus)*. Environmental
15 Toxicology and Chemistry, Vol. 12, pp. 551-565.
16
17 Dave, G., 1984. *Effects of Waterbourne Iron on Growth, Reproduction, Survival, and*
18 *Haemoglobin in Daphnia magna*. Comp. Biochem. Physiol. 78C:433-438.
19
20 Davies, P. M., J. P. Goettl Jr., J. R. Sinley, and N. F. Smith, 1976. Acute and Chronic Toxicity
21 of Lead to Rainbow Trout (Salmo Gairdneri) in Hard and Soft Water. *Water Res.*, 10:199.
22
23 Dawson, A. B., 1935. The Hemopoietic Response in the Catfish, Ameiurus nebulosus, to
24 Chronic Lead Poisoning. *Biol. Bull.*, 68:335.
25
26 DIALOG, 1996. Institute for Science Information.
27
28 Domingo, J.L., J.L. Paternain, J.M. Llobet, and J. Corbella, 1986. *Effects of Vanadium on*
29 *Reproduction, Gestation, Parturition, and Lactation in Rats Upon Oral Administration*. Life
30 Sci., 39: 819-824.
31
32 Efroymsen, R.A., M.E. Will, G.W. Suter, and A.C. Wooten, 1997a. Toxicological Benchmarks
33 for Screening Contaminants of Potential Concern for Effects on Terrestrial Plants, 1997
34 Revision. Office of Environmental Management, USDOE, Oak Ridge, Tennessee. *ES/ER/TM-*
35 *85/R3*.
36
37 Efroymsen, R.A., M.E. Will, and G.W. Suter, 1997b. Toxicological Benchmarks for
38 Contaminants of Potential Concern for Effects on Soil and Litter Invertebrates and Heterotrophic
39 Process: 1997 Revision. Office of Environmental Management, USDOE, Oak Ridge, Tennessee.
40 *ES/ER/TM-126/R2*.
41
42 Eisler, R., 1993. Zinc Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. *U.S.*
43 *Fish and Wildlife Service, Biological Report*, 85(1.26), 123 pp.
44
45 Eisler, R., 1988. Lead Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. *U.S.*
46 *Fish and Wildlife Service, Biological Report*, 85(1.14), 134 pp.
47
48 Eisler, R., 1987a. Mercury Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review.

- 1 *U.S. Fish and Wildlife Service, Biol. Report*, 85(1.10), 75 pp.
- 2
- 3 Eisler, R., 1987b. Polycyclic Aromatic Hydrocarbon Hazards to Fish, Wildlife, and
4 Invertebrates: A Synoptic Review. *U.S. Fish and Wildlife Service, Biol. Report*. 85(1.11), 81
5 pp.
- 6
- 7 Eisler, R., 1986. *Chromium Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review*.
8 U.S. Fish and Wildlife Service Biological Report 85 (1.6). 60 pp.
- 9
- 10 Eisler, R., 1985. *Cadmium Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review*.
11 U.S. Fish and Wildlife Service, Biol. Rep. 85(1.2), 46 pp.
- 12
- 13 Eisler, R., 1985b. *Selenium Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review*.
14 U.S. Fish and Wildlife Service, Biol. Rep. 85(1.5), 57 pp.
- 15
- 16 Evans, R. D., D. Andrews, and R. J. Cornett, 1988. *Chemical Fractioning and Bioavailability*
17 *of Cobalt-60 to Benthic Deposit Feeders*. Journal of Canadian Fisheries and Aquatic Sciences,
18 45:228-236.
- 19
- 20 Ferm, V.H. and W.M. Layton, Jr., 1981. *Teratogenic and Mutagenic Effects of Cadmium*. In:
21 J.O. Nriagu (ed.), *Cadmium in the Environment, Part I: Ecological Cycling*. John Wiley & Sons,
22 New York, NY, pp. 743-756.
- 23
- 24 Fimreite, N., 1979. Accumulation and Effects of Mercury on Birds. In: J.O. Nriagu (ed.) *The*
25 *Biogeochemistry of Mercury in the Environment*, Elsevier/North-Holland Biomedical Press,
26 New York, pp. 601-627.
- 27
- 28 Finger, S. E., E. F. Little, M. G. Henry, J. F. Fairchild, and T. P. Boyle, 1985. Comparison of
29 Laboratory and Field Assessment of Fluorene -- Part I: Effects of Fluorene on the Survival,
30 Growth, Reproduction, and Behavior of Aquatic Organisms in Laboratory Tests, Validation and
31 Predictability of Laboratory Methods for Assessing the Fate and Effects of Constituents in
32 Aquatic Ecosystems. T. P. Boyle, ed., *American Society for Testing and Materials, ASTM STP*
33 **865**: 120-133.
- 34
- 35 Fischer, E. and L. Koszorus, 1992. *Sublethal Effects, Accumulation Capacities and*
36 *Elimination Rates of As, Hg, and Se in the manure worm, Eisenia fetida* (Oligochaeta,
37 Lumbricidae). *Pedobiologia*, 36: 172-178.
- 38
- 39 Fishbein, L., 1981. *Sources, Transport, and Alterations of Metal Compounds: an Overview. I.*
40 *Arsenic, Beryllium, Cadmium, Chromium, and Nickel*. Environmental Health Perspectives
41 40:43-64.
- 42
- 43 Formigli, L., R. Scelsi, P. Poggi, C. Gregotti, A. DiNucci, E. Sabbioni, and L. Manzo, 1986.
44 Thallium-Induced Testicular Toxicity in the Rat. *Env. Res.* 40: 531-539.
- 45
- 46 Foster, P., M.D., L. V. Thomas, and M. W. Cook, 1980. Testicular Effects and Changes in Zinc
47 Excretion Produced by Some n-Alkyl Phthalates in Rats. *Toxicol. Appl. Pharmacol.*, 54(3):392-
48 398.

- 1
2 Foy, C.D., R.L. Chaney, and M.C. White, 1978. *The Physiology of Metal Toxicity in Plants*.
3 Ann. Review Plant Physiol. 29:511-566.
4
5 Foy, C.D., 1974. *Effects of Aluminum on Plant Growth*. In: The Plant Root and Its
6 Environment. E.W. Carson (ed.), University Press of Virginia, Charlottesville, VA. PP. 601-
7 642.
8
9 Francis, C. W., E. C. Davis, and J. C. Goyert, 1980. *Plant Uptake of Trace Elements from Coal*
10 *Gasification Ashes*. Journal of Environmental Quality, 14:561-569.
11
12 Friberg, L., G.F. Nordberg, E. Kessler, and V.B. Vouk (eds.), 1986. *Handbook of the*
13 *Toxicology of Metals, 2nd edition, Volume II*. Elsevier Science Publishers, B.V., Amsterdam,
14 Holland, 130 pp.
15
16 Frost, D.V. and P.M. Lish, 1975. *Selenium in Biology*. Annual Rev. Pharmacology, Vol. 15,
17 pp. 259-284.
18
19 Fuller, R.H. and R.C. Averett, 1975. Evaluation of Copper Accumulation in Part of the
20 California Aqueduct. *Water Res. Bull.* 11: 946-952.
21
22 Ganther, H.E., 1974. *Biochemistry of Selenium*. In: Selenium. R.A. Zingaro and W.C. Cooper,
23 eds. Van Nostrand Reinhold Co., New York, pp. 546-614.
24
25 Ganther, H.E., C. Goude, M.L. Sundi, M.J. Kopeky, P. Wagner, S. Oh, and W.G. Hoeksta, 1972.
26 Selenium: Relationship to Decreased Toxicity of Methylmercury Added to Diets Containing
27 Tuna. *Science*, Vol. 175, pp. 1122-1124.
28
29 Garland, B.W., 1996. *Endangered Species Management Plan for Fort McClellan, Alabama*.
30 Directorate of Environment.
31
32 Gatlin, D.M. and R.P. Wilson, 1984. *Dietary Selenium Requirement of Fingerling Channel*
33 *Catfish*. Journal of Nutrition, Vol. 114, pp. 627-633.
34
35 Getz, L. L., A. W. Haney, R. W. Larimore, J. W. McNurney, H. W. Leyland, P. W. Price, G. L.
36 Rolfe, R. L. Wortman, J. L. Hudson, R. L. Soloman, and K. A. Reinbold, 1977. Transport and
37 Distribution in a Watershed Ecosystem: Lead in the Environment. Boggess (ed.), *National*
38 *Science Foundation*, p. 105-134.
39
40 Graedel, T.E., 1978. Chemical Compounds in Atoms. Academic Press, New York, NY.
41
42 Gregus, Z. and C.D. Klaassen, 1986. *Disposition of Metals in Rats: A Comparative Study of*
43 *Fecal, Urinary, and Biliary Excretion and Tissue Distribution of Eighteen Metals*. Toxicology
44 and Applied Pharmacology, Vol. 85, pp. 24-38.
45
46 Hall, R.J. and B.M. Mulhern, 1984. Are Anuran Amphibians Heavy Metal Accumulators? In:
47 R.A. Seigel, L.E. Hunt, J.L. Knight, L. Malaret, and N.L. Zuschlag (eds.), *Vertebrate Ecology*
48 *and Systematics – A Tribute to Henry S. Fitch*, Museum of Natural History, University of

- 1 Kansas, Lawrence, Kansas, pp.123-133.
- 2
- 3 Hammond, P.B. and R.P. Beliles, 1980. *Metals*. In: J. Doull, C.D. Klaassen, and M.O. Amdur
4 (eds.), Casarett and Doull's Toxicology: The Basic Science of Poisons, 2nd ed. Macmillan
5 Publishing Co., Inc., New York, NY, pp. 409-4671.
- 6
- 7 Hammons, A.S., J.E. Huff, H.M. Braunstein, J.S. Drury, C.R. Shriner, E.B. Lewis, B.L.
8 Whitfield, and L.E. Towill, 1978. *Reviews of the Environmental Effects of Pollutants: IV*
9 *Cadmium*. U.S. Environmental Protection Agency Report 600/1-78-026. 251 pp.
- 10
- 11 Hara, T., Y. Sonoda, and I. Iwai, 1976. *Growth Response of Cabbage Plants to Transition*
12 *Elements Under Water Culture Conditions*. Soil Sci. Plant Nutr. 22(3):307-315.
- 13
- 14 Haseltine, S.D., L. Sileo, D.J. Hoffman, and B.D. Mulhern, 1985. *Effects of Chromium on*
15 *Reproduction and Growth in Black Ducks*. J. Wildl. Manage., 47: 1124-1129.
- 16
- 17 Hayes, A.W., 1994. *Principles and Methods of Toxicology*. Third edition, Raven Press, New
18 York, New York.
- 19
- 20 Hazardous Substances Data Bank (HSDB), 1999. *Toxicological Profile for*
21 *Trichlorofluoromethane*. National Library of Medicine, National Toxicology Information
22 Program, Bethesda, Maryland.
- 23
- 24 Hazardous Substances Data Bank (HSDB), 1996. *Toxicological Profile for Dibutyl Phthalate*.
25 National Library of Medicine, National Toxicology Information Program, Bethesda, Maryland,
26 October, 1996.
- 27
- 28 Heinz, G.H., D.J. Hoffman, A.J. Krynitsky, and D.M.G. Weller, 1987. Reproduction in Mallards
29 Fed Selenium. *Environ. Toxicol. Chem.* 6: 423-433.
- 30
- 31 Heinz, G.H., 1979. Methyl Mercury: Reproductive and Behavioral Effects on Three Generations
32 of Mallard Ducks. *J. Wildlife Mgmt.*, 43: 394-401.
- 33
- 34 Hernandez, L.M., J. Gonzalez, C. Rico, et al., 1985. Presence and Biomagnification of
35 Organochlorine Pollutants and Heavy Metals in Mammals in Donana National Park (Spain). *J.*
36 *Environ. Sci. Health*, 20: 633-650.
- 37
- 38 Hill, H.F., 1981. Inorganic and Organic Mercury Chloride Toxicity to Cortunix: Sensitivity
39 Related to Age and Quantal Assessment of Physiological Responses. *Ph.D. Thesis, University*
40 *of Maryland*, College Park.
- 41
- 42 Hill, E.F. and C.S. Schaffner, 1976. Sexual Maturation and Productivity of Japanese Quail Fed
43 Graded Concentrations of Mercuric Chloride. *Poult. Sci.*, 55: 1449-1459.
- 44
- 45 Hoffman, D. J., J. C. Franson, O. H. Pattee, C. M. Bunck, and H. C. Murray, 1985. Biochemical
46 and Hematological Effects of Lead Ingestion in Nesting American Kestrels (*Falco sparverinus*).
47 *Comp. Biochem. Physiol.*, 80C:431-439.
- 48

- 1 Hoffman, D. J., and M. L. Gay, 1981. Embryotoxic Effects of Benzo(a)pyrene, Chrysene, and
2 7,12-Dimethylbenz(a)anthracene in Petroleum Hydrocarbon Mixtures in Mallard Ducks. *J.*
3 *Toxicol. Environ. Health*, 7: 775-787.
- 4
- 5 Holcombe, G. W., D. A. Benoit, E. N. Leonard, and J. M. McKim, 1976. Long Term Effects of
6 Lead Exposure on Three Generations of Brook Trout (*Salvelinus fontinalis*). *J. Fish. Res. Bd.*
7 *Can.*, 33:1731.
- 8
- 9 Honeycutt, M.E. and B.L. Roberts, 1995. Assessment of DNA Strand Breaks in the Earthworm,
10 *Eisenia fetida*, as a Bioindicator of Soil Contamination. *Environmental Sciences*, 3,4, pp 199-
11 207.
- 12
- 13 Hose, J. E., J. B. Hannah, D. Dijulio, M. L. Landolt, B. S. Miller, W. T. Iwaoka, and S. P. Felton,
14 1982. Effects of Benzo(a)pyrene on Early Development of Flatfish. *Arch. Environ. Contam.*
15 *Toxicol.*, 11:167-171.
- 16
- 17 Howard, P.H., 1991. *Handbook of Environmental Fate and Exposure Data for Organic*
18 *Chemicals, Volume III: Pesticides*. Lewis Publishers, Chelsea, Michigan, 684 pp.
- 19
- 20 Howard, P.H. (ed.), 1990. *Handbook of Environmental Fate and Exposure Data for Organic*
21 *Chemicals, Volume II: Solvents*. Lewis Publishers, Chelsea, Michigan.
- 22
- 23 Huckabee, J.W., J.W. Elwood, and S.G. Hidebrand, 1979. Accumulation of Mercury in
24 Freshwater Biota. In: J.O. Nriagu (ed.) *The Biogeochemistry of Mercury in the Environment*.
25 Elsevier/North-Holland Biomedical Press, New York, pp. 277-302.
- 26
- 27 Hunter, B.A. and M.S. Johnson, 1982. Food Chain Relationship of Copper and Cadmium in
28 Contaminated Grassland Ecosystems. *Oikos*, Vol. 39, pp. 108-177.
- 29
- 30 ICRP, 1979. *Limits for Intakes of Radionuclides by Workers*. Publication 39, Part 1,
31 Commission on Radiological Protection, Washington, DC.
- 32
- 33 Inman, J. C., S. D. Strachan, L. W. Sommers, et al., 1984. The Decomposition of Phthalate
34 Esters in Soil. *J. Environ. Sci. Health*, B19:245-247.
- 35
- 36 IT Corporation (IT), 2000. *Final Human Health and Ecological Screening Values and PAH*
37 *Background Summary Report, Fort McClellan, Calhoun County, Alabama*. July.
- 38
- 39 International Agency for Research on Cancer (IARC), 1982. Monograph on the Evaluation of
40 the Carcinogenic Risk of Chemicals to Man. World Health Organization, Geneva, Switzerland.
- 41
- 42 International Agency for Research on Cancer (IARC), 1974. Monograph on the Evaluation of
43 the Carcinogenic Risk of Chemicals to Man. World Health Organization, Geneva, Switzerland.
- 44
- 45 Ivankovic, S. and R. Preussmann, 1975. *Absence of Toxic and Carcinogenic Effects After*
46 *Administration of High Doses of Chromic Oxide Pigment in Subacute and Long-Term*
47 *Feeding Experiments in Rats*. Ed. Cosmet. Toxicol., 13: 347-351.
- 48

- 1 Jenkins, D. W., 1980. *Biological Monitoring of Toxic Trace Metals: Volume 1*. Biological
2 Monitoring and Surveillance, NTIS PB81-103475.
- 3
- 4 Jones, D.S., G.W. Suter, and K.N. Hull, 1997. *Toxicological Benchmarks for Screening*
5 *Constituents of Potential Concern for Effects on Sediment-Associated Biota: 1997 Revision*.
6 Risk Assessment Program, USDOE, Oak Ridge, Tennessee. ES/ER/TM-95/R4.
- 7
- 8 Kabata-Pendias, A., and H. Pendias, 1992. *Trace Elements in Soils and Plants, 2nd edition*.
9 CRC Press, Boca Raton, FL, 365 pp.
- 10
- 11 Kayser, R., D. Sterling, and D. Viviani (eds.), 1982. Intermedia Priority Pollutant Guidance
12 Documents. EPA, Washington, DC.
- 13
- 14 Kendall, R., 1992. Wildlife Toxicology. *Environ. Sci. Tech.*, Vol. 16, No. 8:448A-453A.
- 15
- 16 Khangrot, B. S. and P. K. Ray, 1989. Investigation of Correlation Between Physiochemical
17 Properties of Metals and their Toxicity to the Water Flea *Daphnia magna* Straus. *Ecotoxicol.*
18 *Environ. Saf.*, 18(2):109-120.
- 19
- 20 Kinnamon, K. E., 1963. Some Independent and Combined Effects of Copper, Molybdenum, and
21 Zinc on the Placental Transfer of Zinc-65 in the Rat. *J. Nutr.*, 81:312-320.
- 22
- 23 Klaassen, C. D., M. O. Amdur, and S. Doull, 1991. *Toxicology: The Basic Science of Poisons*.
24 Pergamon Press, Inc., Elmsford, New York.
- 25
- 26 Knobloch, K., S. Szendzikowski, and A. Slusarczyk-Zalobna, 1969. Acute and Subacute
27 Toxicity of Acenaphthene and Acenaphthylene. *Med. Pracy.*, 20(3):210-222.
- 28
- 29 Kosalwat, P. and A.W. Knight, 1987. Chronic Toxicity of Copper to a Partial Life Cycle of the
30 Midge *Chironomus decorus*. *Arch. Environ. Contam. Toxicol.* 16(3): 283-290.
- 31
- 32 Kucera, E., 1983. Mink and Otter as Indicators of Mercury in Manitoba Waters. *Canadian*
33 *Journal of Zoology*, Vol. 61, pp. 2250-2256.
- 34
- 35 Kuperman, R.G., R.T Checkai, C.T. Phillips, M. Simini, J.A. Speicher, and D.J. Barclift, 2002.
36 *Toxicity Assessments of Antimony, Barium, Beryllium, and Manganese for Development of*
37 *Ecological Soil Screening Levels (Eco-SSL) Using Enchytraeid Reproduction Benchmark*
38 *Values*. Technical Report No. ECBC-TR-324. U.S. Army Edgewood Chemical Biological
39 Center, Aberdeen Proving Ground, MD.
- 40
- 41 Lamb, J.C., R.E. Chapin, J. Teague, A.D. Lawton, and J.R. Reel, 1987. Reproductive Effects of
42 Four Phthalic Acid Esters in the Mouse. *Toxicol. Appl. Pharmacol.*, 88: 255-269.
- 43
- 44 Laskey, J.W. and F.W. Edens, 1985. *Effects of Chronic High-Level Manganese Exposure on*
45 *Male Behavior in the Japanese Quail (Cortunix japonica)*. *Poult. Sci.*, 64: 579-584.
- 46
- 47 Laskey, J.W., G.L. Rehnberg, J.F. Hein, and S.D. Carter, 1982. *Effects of Chronic Manganese*
48 *(Mn₃O₄) Exposure on Selective Reproductive Parameters in Rats*. *J. Toxicol. Environ. Health*,

1 9: 677-687.

2
3 Lecyk, M., 1980. Toxicity of Cupric Sulfate in Mice Embryonic Development. *Zoo. Pollut.*
4 28(2): 101-105.

5
6 Lisk, D.J., 1972. Trace Metals in Soil, Plants, and Animals. *Advances Agronomy*, Vol. 24, pp.
7 267-325.

8
9 Long, E.R., D.D. MacDonald, S.L. Smith, F.D. Calder, 1995. Incidence of Adverse Biological
10 Effects Within Ranges of Chemical Concentrations in Marine and Estuarine Sediments.
11 *Environmental Management*, Vol. 19, No. 1, pp. 81-97.

12
13 Long, E. R. and L. G. Morgan, 1990. The Potential for Biological Effects of Sediment-sorbed
14 Contaminants Tested in the National Status and Trends Program. National Oceanic and
15 Atmospheric Administration Technical Memorandum, *NOSOMA 52*, NOAA, Seattle,
16 Washington.

17
18 Lowe, T.P., T.W. May, W.G. Brumbaugh, and D.A. Kane, 1985. National Constituent
19 Biomonitoring Program: Concentrations of Seven Elements in Freshwater Fish, 1978-1981.
20 *Archives of Environmental Contamination and Toxicology*, Vol. 14, pp. 363-388.

21
22 Ma, W.C., 1984. Sublethal Toxic Effects of Copper on Growth, Reproduction, and Litter
23 Breakdown Activity in the Earthworm *Lumbricus rubellus*, with Observations on the Influence
24 of Temperature and Soil pH. *Environ. Pollut. Ser. A* 33: 207-219.

25
26 Mackenzie, R.D., R.U. Byerrum, C.F. Decker, C.A. Hoppert, and R.F. Langham, 1958. *Chronic*
27 *Toxicity Studies, II. Hexavalent and Trivalent Chromium Administered in Drinking Water to*
28 *Rats*. Am. Med. Assoc. Arch. Ind. Health, 18: 232-234.

29
30 MacKenzie, K. M., and D. M. Angevine, 1981. Infertility in Mice Exposed *in Utero* to
31 Benzo(a)pyrene. *Biology of Reproduction*, Vol. 24, pp. 183-191.

32
33 Maier, K.J., C.G. Foe, and A.W. Knight, 1993. *Comparative Toxicity of Selenate, Selenite,*
34 *Seleno-DL-methionine, and Seleno-DL-cystine to Daphnia magna*. Environmental Toxicology
35 and Chemistry, Vol. 12, pp. 755-763.

36
37 Malecki, M.R., E.F. Neuhauser, and R.C. Loehr, 1982. The Effect of Metals on the Growth and
38 Reproduction of *Eisenia foetida* (Oligochaeta, Lumbricidae). *Pedobiologia*, 24: 129-137.

39
40 Malley, D.F., P.S. Chang, C.M. Moore, and S.G. Lawrence, 1987. *Changes in Aluminum*
41 *Content of Tissues of Crayfish Held in the Laboratory and in Experimental Field Enclosures*.
42 G.H. Green and K.L. Woodward (eds.), Canadian Technical Report on Fish and Aquatic
43 Sciences, No. 1480, 330 p.

44
45 Manzo, L., R. Scelsi, A. moglia, P. Poggi, E. Alfonsi, R. Pietra, F. Mousty, and E. Sabbioni,
46 1982. Long-Term Toxicity of Thallium in the Rat. In: *Chemical Toxicology and Clinical*
47 *Chemistry of Metals*, Academic Press, London, pp. 4-1-405.

1 Marceau, N., N. Aspin, and A. Sass-Kortsak, 1970. Absorption of Copper from Gastrointestinal
2 Tract of the Rat. *American Journal of Physiology*, 218: 377-383.

3
4 Marks, T.A., T.A. Ledoux, and J.A. Moore, 1982. Teratogenicity of a Commercial Xylene
5 Mixture in the Mouse. *J. Toxicol. Environ. Health* 9: 97-105.

6
7 Mehring, A.L., J.H. Brumbaugh, A.J. Sutherland, and H.W. Titus, 1960. The Tolerance of
8 Growing Chickens for Dietary Copper. *Poult. Sci.* 39: 713-719.

9
10 *Merck Index*, 1983. 10th edition, Rahway, New Jersey, Merck Co., Inc.

11
12 Mettee, M.F. and R.R. Haynes, 1979. *A Study of the Endangered and Threatened Plants and*
13 *Animals on Fort McClellan Military Installation and Pelham Range, Calhoun County,*
14 *Alabama*. Prepared by the Geological Survey of Alabama under contract DACA01-79-C-0075
15 to the U.S. Army Corps of Engineers, Mobile, AL.

16
17 Molnar, L., E. Fischer, and M. Kallay, 1989. *Laboratory Studies on the Effect, Uptake, and*
18 *Distribution of Chromium in Eisenia fetida (Annelida, Oligochaeta)*. *Zool. Anz.*, 223(1/2): 57-
19 66.

20
21 Moore, J. W. and S. Ramamoorthy, 1984. *Heavy Metals in Natural Waters: Applied*
22 *Monitoring and Impact Assessment*. R. S. DeSanto, ed., Springer-Verlag, New York, New
23 York.

24
25 Mudge, G. P., 1983. The Incidence and Significance of Ingested Lead Pellet Poisoning in British
26 Waterfowl. *Biol. Conserv.*, 27:333-372.

27
28 National Academy of Sciences (NAS), 1979. *Zinc*. National Academy of Sciences,
29 Washington, D.C., 471 pp.

30
31 National Academy of Sciences (NAS), 1977. *Drinking Water and Health - Inorganic Solutes*,
32 National Academy of Sciences, Washington, D.C., pp. 205-488.

33
34 National Academy of Sciences (NAS), 1972. *Lead: Airborne Lead in Perspective*,
35 Washington, DC, 188.

36
37 National Library of Medicine (NLM), 1996. *Hazardous Substance Data Bank*. Produced by
38 Micromedix, Inc.

39
40 National Research Council (NRC), 1989. *Recommended Dietary Allowances*. 10th edition,
41 National Academy Press, Washington, DC.

42
43 National Research Council (NRC), 1977. *Drinking Water and Health, Volume 1*. Washington,
44 DC, National Academy Press.

45
46 National Research Council of Canada (NRCC), 1980. *Effects of Vanadium in the Canadian*
47 *Environment*. National Research Council of Canada, Publ. No. NRCC 18132.

- 1 National Research Council of Canada (NRCC), 1973, *Lead in the Canadian Environment*.
2 Publication No. BY73-7(ES), 119p.
3
- 4 Nawrot, P.S. and R.E. Staples, 1979. Embryofetal Toxicity and Teratogenicity of Benzene and
5 Toluene in the Mouse. *Teratology*, 19: 41A.
6
- 7 Neff, J.M., B.W. Cornaby, R.M. Vaga, T.C. Gulbransen, J.A. Scanlon, and D.J. Bean, 1988. *An*
8 *Evaluation of the Screening Level Concentration Approach for Validation of Sediment*
9 *Quality Criteria for Freshwater and Saltwater Ecosystems*. pp. 115-127 in: Aquatic
10 Toxicology and Hazard Assessment: 10th Volume, ASTM STP 971, ed. W.J. Adams, G.A.
11 Chapman, and W.G. Landis, American Society for Testing and Materials, Philadelphia, PA.
12
- 13 Neff, J. M., 1985. Polycyclic Aromatic Hydrocarbons. *Fundamentals of Aquatic Toxicology*,
14 G. M. Rand and S. R. Petrocelli, eds., Hemisphere Publishing Corp., Washington, D.C.
15
- 16 Neuhauser, E.F, R.C. Loehr, D.L. Milligan, and M.R. Malecki, 1985. Toxicity of Metals to the
17 Earthworm *Eisenia fetida*. *Biol. Fertil. Soils*, 1: 149-152.
18
- 19 Neuhauser, E.F., M.R. Malecki, and R.C. Loehr, 1984. Growth and Reproduction of the
20 Earthworm *Eisenia fetida* After Exposure to Sublethal Concentrations of Metals. *Pedobiologia*,
21 27: 89-97.
22
- 23 Neumann, G., 1976. *Concentration Factors for Stable Metals and Radionuclides in Fish,*
24 *Mussels, and Crustaceans – A Literature Survey*. National Swedish Environmental Protection
25 Board, Sweden.
26
- 27 Oishi, S. and K. Hiraga, 1980. Testicular Atrophy Induced by Phthalate Acid Esters: Effect on
28 Testosterone and Zinc Concentrations. *Toxicol. Appl. Pharmacol.*, 53(1):35-41.
29
- 30 Ondreicka, R., E. Ginter, and J. Kortus, 1966. *Chronic Toxicity of Aluminum in Rats and Mice*
31 *and its Effects on Phosphorus Metabolism*. Brit. J. Indust. Med. 23: 305-313.
32
- 33 Ontario Ministry of the Environment, 1992. *Guidelines for the Protection and Management of*
34 *Aquatic Sediment Quality in Ontario*.
35
- 36 Palmer, A.K., A.E. Street, F.J.C. Roe, A.N. Worden, and N.J. VanAbbe, 1979. Safety
37 Evaluation of Toothpaste Containing Chloroform, II. Long Term Studies in Rats. *J. Environ.*
38 *Pathol. Toxicol.* 2: 821-833.
39
- 40 Pattee. O.H., 1984. Eggshell Thickness and Reproduction in American Kestrels Exposed to
41 Chronic Dietary Lead. *Arch. Environ. Contam. Toxicol.*, 13: 29-34.
42
- 43 Peakall, D.B., 1974. Effects of Di-n-butylphthalate and Di-2-ethylhexylphthalate on the Eggs of
44 Ring Doves. *Bull. Environ. Contam. Toxicol.*, 12: 698-702.
45
- 46 Perry, H.M., E.F. Perry, M.N. Erlanger, and S.J. Kopp, 1983. *Cardiovascular Effects of*
47 *Chronic Barium Ingestion*. In: Proc. 17th Annual Conference on Trace Substances in Environ.
48 Health, Vol. 17. University of Missouri Press, Columbia, MO.

- 1
2 Persaud, D., R. Jaagumagi, and A. Hayton, 1993. ***Guidelines for the Protection and***
3 ***Management of Aquatic Sediment Quality in Ontario***. Ontario Ministry of the Environment
4 and Energy.
5
6 Peterson, P.J. and C.A. Girling, 1981. ***Other Trace Metals***. In: Effect of Heavy Metal Pollution
7 on Plants, Vol. 1, Effects of Trace Metals on Plant Function. N.W. Lepp (ed.), Applied Science
8 Publishers, New Jersey, pp. 213-278.
9
10 Phillips, C.T., R.T Checkai, R.G.Kuperman, M. Simini, J.A. Speicher, and D.J. Barclift, 2002.
11 ***Toxicity Assessments of Antimony, Barium, Beryllium, and Manganese for Development of***
12 ***Ecological Soil Screening Levels (Eco-SSL) Using Folsomia Reproduction Benchmark***
13 ***Values***. Technical Report No. ECBC-TR-326. U.S. Army Edgewood Chemical Biological
14 Center, Aberdeen Proving Ground, MD.
15
16 Reeves, A. and A. Vorwald, 1967. ***Beryllium Carcinogenesis***. Pulmonary Deposition and
17 Clearance of Inhaled Beryllium Sulfate in the Rat. *Cancer Research* 27:446-451.
18
19 Registry of Toxic Effects of Chemical Substances (RTECS), 1996. Produced by Micromedex.
20
21 Rhodes, F.M., S.M. Olsen, and A. Manning, 1989. Copper Toxicity in Tomato Plants. ***Journal***
22 ***of Environmental Quality***, Vol. 18, pp. 195-197.
23
24 Richardson, M.E., M.R.S. Fox, and B.E. Fry, Jr., 1974. ***Pathological Changes Produced in***
25 ***Japanese Quail by Ingestion of Cadmium***. *J. Nutr.*, 104: 323-338.
26
27 Roberts, B.L. and H.W. Dorough, 1984. Relative Toxicities of Chemicals to the Earthworm
28 *Eisenia fetida*. ***Environmental Toxicology and Chemistry***, Vol. 3, pp. 67-78.
29
30 Romney, E.M. and J.D. Childress, 1965. ***Effects of Beryllium in Plants and Soil***. *Soil Sci.*
31 100(2):210-217.
32
33 Rosenfeld, I. and O.A. Beath, 1954. ***Effect of Selenium on Reproduction in Rats***. *Proc. Soc.*
34 *Exp. Biol. Med.*, 87: 295-297.
35
36 Rosomer, G.L., W.A. Dudley, L.J. Machlin, and L. Loveless, 1961. ***Toxicity of Vanadium and***
37 ***Chromium for the Growing Chick***. *Poultry Science*, Vol. 40, pp. 1171-1173.
38
39 Sample, B.E., J.J. Beauchamp, R.A. Efroymsen, and G.W. Suter, 1998. ***Development and***
40 ***Validation of Bioaccumulation Models for Small Mammals***. Office of Environmental
41 Management, USDOE, Oak Ridge, Tennessee. ES/ER/TM-219.
42
43 Sample, B.E., D.M. Opresko, and G.W. Suter, 1996. Toxicological Benchmarks for Wildlife:
44 1996 Revision. Risk Assessment Program, Office of Environmental Management, USDOE, Oak
45 Ridge, Tennessee. ***ES/ER/TM-86/R3***.
46
47 Sasaki, S., 1978. ***Aquatic Pollutants: Transformation and Biological Effects***. O. Huntzinger et
48 al. (eds.), Pergamon Press, Oxford.

- 1
2 Sax, N.I. and R.J. Lewis, 1989. Dangerous Properties of Industrial Materials. 7th ed. Vol. II.
3 Van Nostrand Reinhold, new York.
4
5 Schafer, E.W., W.A. Bowles, and J. Hurlbut, 1983. The *Acute Toxicity, Repellency, and*
6 *Hazard Potential of 998 Chemicals to One or More Species of Wild and Domestic Birds.*
7 Archives of Environmental Contamination and Toxicology, Vol. 12, pp. 355-382.
8
9 Schafer, E.W., 1972. The Acute Oral Toxicity of 369 Pesticidal, Pharmaceutical, and Other
10 Chemicals to Wild Birds. *Toxicological and Applied Pharmacology*, Vol. 21, pp. 315-330.
11
12 Schlicker, S.A., and D.H. Cox, 1968. Maternal Dietary Zinc and Development and Zinc, Iron,
13 and Copper Content of the Rat Fetus. *J. Nutri.* 95: 287-294.
14
15 Science Applications International Corporation (SAIC), 1998. *Background Metals Survey*
16 *Report, Fort McClellan.*
17
18 Schroeder, H. A. and M. Mitchner, 1971. Toxic Effects of Trace Elements on Reproduction of
19 Mice and Rats. *Arch. Environ. Health*, 23:102-106.
20
21 Schroeder, H.A., M. Mitchener, J.J. Baalassa, M.. Kanisawa, and A.P. Nason, 1968. Zirconium,
22 Niobium, Antimony, and Fluorine in Mice: Effects on Growth, Survival, and Tissue Levels. *J.*
23 *Nutr.*, 95: 95-101.
24
25 Schroeder, H.A., J.J Balassa, and W.H. Vinton, 1964. Chromium, Lead, Cadmium, Nickel, and
26 Titanium in Mice: Effects on Mortality , Tumors, and Tissue Levels. *Journal of Nutrition*, Vol.
27 83, pp. 239-250.
28
29 Sharma, R.P., 1980. *Soil-Plant-Animal Distribution of Cadmium in the Environment.* In: J.O.
30 Nriagu, ed., Cadmium in the Environment, Part I, Ecological Cycling. John Wiley & Sons, New
31 York, NY, pp. 587-605.
32
33 Shaw Environmental, Inc., 2005. Selecting Site-Related Chemicals for Human Health and
34 Ecological Risk Assessments for FTMC: Revision 3. *Technical Memorandum from Karen*
35 *Thorbjornsen, Jonathan Myers, and Paul Goetchius.*
36
37 Schubauer-Berigan, M.K., J.R. Dierkes, P.D. Monson, and G.T. Ankley, 1993. pH-Dependent
38 Toxicity of Cd, Cu, Ni, Pb, and Zn to *Ceriodaphnia dubia*, *Pimephales promelas*, *Hyallolella*
39 *azteca*, and *Lumbriculus variegatus*. *Environmental Contamination and Toxicology*, Vol. 12,
40 pp. 1261-1266.
41
42 Shepard, T. H., 1986. *Catalog of Teratogenic Agents, 5th edition.* Baltimore, Maryland, The
43 Johns Hopkins University Press.
44
45 Shugart, L.R., 1991. Dinitrotoluene in Deer Tissue. Oak Ridge National Laboratory, *Final*
46 *Report, ORNL/M-1765*, September.
47

- 1 Shugart, L.R., W.H. Griest, E. tan, C. Guzman, J.E. Caton, C.H. Ho, and B.A. Tomkins, 1990.
2 ***TNT Metabolites in Animal Tissues, Final Report***. U.S. Army Biomedical Research and
3 Development laboratory, Fort Detrick, MD.
4
- 5 Simini, M., R.T. Checkai, R.G.Kuperman, C.T. Phillips, J.A. Speicher, and D.J. Barclift, 2002.
6 Toxicity Assessments of Antimony, Barium, Beryllium, and Manganese for Development of
7 Ecological Soil Screening Levels (Eco-SSL) Using Earthworm (*Eisenia fetida*) Benchmark
8 Values. Technical Report No. ECBC-TR-325. U.S. Army Edgewood Chemical Biological
9 Center, Aberdeen Proving Ground, MD.
10
- 11 Sims, R. C., and M. R. Overcash, 1983. Fate of Polynuclear Aromatic Compounds (PNAs) in
12 Soil-Plant Systems. ***Resource Review***, Vol. 88, pp. 1-68.
13
- 14 Smith, I. C. and B. L. Carson, 1981. ***Trace Metals in the Environment. Volume 6: Cobalt and***
15 ***Appraisal of Environmental Exposure***. Ann Arbor, Michigan, Ann Arbor Science Publishers,
16 Inc.
17
- 18 Solonen, T., and M. Lodenius, 1984. Mercury in Finnish Sparrowhawks, *Accipter nisus*. ***Ornis***
19 ***Fennica***, Vol. 61, pp. 58-63.
20
- 21 Sparling, D.W., 1990. ***Conditioned Aversion of Aluminum Sulfate in Black Ducks***.
22 ***Environmental Toxicology and Chemistry***, Vol. 9, pp. 479-483.
23
- 24 Sprague, J. B., 1968. Avoidance Reactions of Rainbow Trout to Zinc Sulfate Solutions. ***Wat.***
25 ***Res.***, 2:367.
26
- 27 Spurgeon, D.J. and S.P. Hopkin, 1996. The Effects of Metal Contamination on Earthworm
28 Populations Around a Smelting Works: Quantifying Species Effects. ***Appl. Soil Ecol.*** 4: 147-
29 160.
30
- 31 Spurgeon, D.J., S.P. Hopkin, and D.T. Jones, 1994. Effects of Cadmium, Copper, Lead, and
32 Zinc on Growth, Reproduction, and Survival of the Earthworm *Eisenia fetida* (*Savigny*):
33 Assessing the Environmental Impact of Point-Source Metal Contamination in Terrestrial
34 Ecosystems. ***Environ. Pollut.***, 84: 123-130.
35
- 36 Stahl, J.L., J.L. Greger, and M.E. Cook, 1990. Breeding-Hen and Progeny Performance When
37 Hens are Fed Excessive Dietary Zinc. ***Poult. Sci.*** 69: 259-263.
38
- 39 Streit, B. and A. Jaggy, 1983. Effect of Soil Type on Copper Toxicity and Copper Uptake in
40 *Octolasion cyaneum* (Lumbricidae). In: ***New Trends in Soil Biology***. Ph. Lebrun, et al. (eds.)
41 pp. 569-575. Ottignies-Louvain-la-Neuve.
42
- 43 Streufert, J. M., J. R. Jones, and H. O. Sanders, 1981. Toxicity and Biological Effects of
44 Phthalate Esters on Midges *Chironomus plumosus*. ***Trans. Mo. Acad. Sci.***, 14(0):33-40.
45
- 46 Suter, G. W., and C. L. Tsao, 1996. Toxicological Benchmarks for Screening Potential
47 Contaminants of Concern for Effects on Aquatic Biota: 1996 Revision. Risk Assessment
48 Program, U. S. DOE, Oak Ridge, Tennessee, ***ES/ER/TM-96/R2***.

- 1
2 Sutou, S., K. Yamamoto, H. Sendota, K. Tomomatsu, Y. Shimizu, and M. Sugiyama, 1980.
3 ***Toxicity, Fertility, Teratogenicity, and Dominant Lethal Tests in Rats Administered Cadmium***
4 ***Subchronically, I. Toxicity Studies.*** *Ecotoxicol. Environ. Safety*, 4: 39-50.
5
6 Talmage, S.S., D.M. Opresko, C.J. Maxwell, C.J.E. Welsh, F.M. Cretella, P.H. Reno, and F.B.
7 Daniel, 1999. Nitroaromatic Munition Compounds: Environmental Effects and Screening
8 Values. ***Rev. Environ. Contam. Toxicol.*** 161: 1-156.
9
10 Talmage, S.S. and B.T. Walton, 1993. Food Chain Transfer and Potential Toxicity of Mercury
11 to Small Mammals at a Contaminated Terrestrial Field Site. ***Ecotoxicology***, Vol. 2, pp. 243-256.
12
13 Talmage, S.S. and B.T. Walton, 1991. Small Mammals as Monitors of Environmental
14 Contaminants. ***Reviews in Environmental Contamination and Toxicology***, Vol. 119, pp. 47-
15 145.
16
17 Talmage, S.S. and B.T. Walton, 1990. Comparative Evaluation of Several Small Mammal
18 Species as Monitors of heavy Metals, Radionuclides, and Selected Organic Compounds in the
19 Environment. ***ORNL/TM-11605***, Oak Ridge National Laboratory, Oak Ridge, Tennessee.
20
21 Talmage, S.S., 1989. Comparative Evaluation of Several Small Mammal Species as Monitors of
22 Heavy Metals, Radionuclides, and Selected Organic Compounds in the Environment. Ph.D.
23 Dissertation, University of Tennessee, Knoxville.
24
25 Tikhonova, T.S., 1967. Toxicity of Thallium and its Compounds in Workers. ***Nov. Dannye***
26 ***Toksikol. Redk. Metal. Ikh Soedin. Chem. Abstr.*** 71: 53248j.
27
28 U.S. Army Corps of Engineers (USACE), 1998. ***Final Environmental Impact Statement.***
29 ***Disposal and Re-Use of Fort McClellan.***
30
31 U.S. Army Environmental Hygiene Agency (USAEHA) 1994. ***Health Risk Assessment for***
32 ***Consumption of Deer Muscle and Liver from Joliet Army Ammunition Plant.*** Toxicology
33 Division.
34
35 U.S. Environmental Protection Agency (EPA), 2005. Integrated Risk Information System
36 (IRIS). National Center for Environmental Assessment, Cincinnati, OH.
37
38 U.S. Environmental Protection Agency (EPA), Region 5, 2005b. ***Ecological Screening Level***
39 ***for Antimony.*** Office of Solid Waste and Emergency Response, Washington, DC. OSWER
40 Directive 9285.7-61.
41
42 U.S. Environmental Protection Agency (EPA), 2003a. ***Guidance for Developing Ecological***
43 ***Soil Screening Levels.*** Office of Solid Waste and Emergency Response, Washington, DC.
44 OSWER Directive 9285.7-55.
45
46 U.S. Environmental Protection Agency (EPA), Region 5, 2003b. ***Ecological Screening Level***
47 ***for Aluminum.*** Office of Solid Waste and Emergency Response, Washington, DC. OSWER
48 Directive 9285.7-60.

1
2 U.S. Environmental Protection Agency (EPA), 2002. *National Recommended Water Quality*
3 *Criteria for Priority Toxic Pollutants*. Office of Water, Washington, DC. EPA/822-R-02-047.

4
5 U.S. Environmental Protection Agency (EPA), 2000a. *Ecological Soil Screening Level*
6 *Guidance, Draft*. Office of Emergency and Remedial Response, Washington, DC.

7
8 U.S. Environmental Protection Agency, Region 4 (EPA), 2000b. Region 4 Waste Management
9 Division Freshwater Surface Water Screening Values for Hazardous Waste Sites.

10
11 U.S. Environmental Protection Agency (EPA), 1999. Screening Level Ecological Risk
12 Assessment Protocol for Hazardous Waste Combustion Facilities. Office of Solid Waste and
13 Emergency Response, Washington, DC. EPA-530-D-99-001c.

14
15 U.S. Environmental Protection Agency (EPA), 1996. Eco Update. *Ecotox Thresholds*. Office
16 of Solid Waste and Emergency Response, Washington, DC. EPA 540/F-95/038.

17
18 U.S. Environmental Protection Agency (EPA), 1995. *Final Water Quality Guidance for the*
19 *Great Lakes System*. Office of Water, Washington, DC.

20
21 U.S. Environmental Protection Agency (EPA), 1993. *Wildlife Exposure Factors Handbook*.
22 Office of Research and Development, Washington, DC., EPA/600-R-93/187.

23
24 U.S. Environmental Protection Agency (EPA), 1989a. Updated Health Affects Assessment for
25 Carbon Tetrachloride. Prepared by the Office of Health and Environmental Assessment,
26 Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Emergency and
27 Remedial Response, Washington, DC.

28
29 U.S. Environmental Protection Agency (EPA), 1989b. *Risk Assessment Guidance for*
30 *Superfund, Vol. I: Human Health Evaluation Manual*. Office of Emergency and Remedial
31 Response, Washington, DC, EPA/540/1-89/002.

32
33 U.S. Environmental Protection Agency (EPA), 1985a. *Environmental Profiles and Hazard*
34 *Indices for Constituents of Municipal Sludge: Beryllium*. Office of Water Regulations and
35 Standards, Washington, DC.

36
37 U.S. Environmental Protection Agency (EPA), 1985. *Drinking Water Criteria Document on*
38 *Copper*. Office of Drinking Water, Washington, DC.

39
40 U.S. Environmental Protection Agency (EPA), 1984. *Health Effects Assessment for Zinc (and*
41 *Compounds)*, Cincinnati, Ohio.

42
43 U.S. Environmental Protection Agency (EPA), 1980a. Ambient Water Quality Criteria for
44 Thallium. Office of Water Regulations and Standards, Washington, D.C., EPA 440/5-80-074.

45
46 U.S. Environmental Protection Agency (EPA), 1980. *Ambient Water Quality Criteria for Zinc*.
47 Office of Water Regulations and Standards, Washington, D.C., EPA 440/5-80-079.

- 1 U.S. Fish and Wildlife Service (USFWS), 1982. *Gray Bat Recovery Plan*. Washington, DC, 26
2 pp.
- 3
- 4 Vanderploeg, H.A., D.C. Parzyck, W.H. Wilcox, J.R. Kercher, and S.V. Kaye, 1975.
5 *Bioaccumulation Factors for Radionuclides in Freshwater Biota*. ORNL-5002, Oak Ridge
6 National Laboratory, Oak Ridge, Tennessee.
- 7
- 8 Van Gestel, C.A.M., E.M. Dirven-van Breeman, R. Baerselman, H.J.B. Emans, J.A.M. Janssen,
9 R. Postuma, and P.J.M. van Vliet, 1992. *Comparison of Sublethal and Lethal Criteria for Nine
10 Different Chemicals in Standardized Toxicity Tests Using the Earthworm Eisenia andrei*.
11 *Ecotoxicol. Environ. Saf.*, 23: 206-220.
- 12
- 13 Van Gestel, C.A.M., W.A. van Dis, E.M. van Breemen, and P.M. Sparenburg, 1989.
14 Development of a Standardized Reproduction Toxicity Test with the Earthworm Species *Eisenia
15 fetida andrei* Using Copper, Pentachlorophenol, and 2,4-Dichloroaniline. *Ecotoxicol. Environ.*
16 *Saf.* 18: 305-312.
- 17
- 18 Venugopal, B., and T. D. Luckey, 1978. *Metal Toxicity in Mammals, Volume 2*, New York,
19 New York, Plenum Press.
- 20
- 21 Verschueren, K., 1983. *Handbook of Environmental Data on Organic Chemicals, Second
22 Edition*. Van Nostrand Reinhold, New York, NY.
- 23
- 24 Wallace, A., G.V. Alexander, and F.M. Chaudhry, 1977. *Pytotoxicity and Some Interactions of
25 the Essential Trace Metals Iron, Manganese, Molybdenum, Zinc, Copper, and Boron*.
26 *Commun. Soil Sci. Plant Anal.* 8(9):741-750.
- 27
- 28 Wallihan, E.F., 1966. *Iron*. In: Diagnostic Criteria for Plants and Soils. H.D. Chapman (ed.),
29 Univ. of California, Div. Agric. Sci., Riverside, pp. 203-212.
- 30
- 31 Waters, M.D., 1977. *Toxicology of Vanadium*. In: Advances in Modern Toxicology, Volume
32 2: Toxicology of Trace Elements. R.A. Goyer and M.A. Mehlman (eds.). John Wiley and Sons,
33 New York, NY, pp. 147-189.
- 34
- 35 White, D.H. and M.T. Finley, 1978. *Uptake and retention of Dietary Cadmium in Mallard
36 Ducks*. *Environ. Res.*, 17: 53-59.
- 37
- 38 White, D.H. and M.P. Dieter, 1978. *Effects of Dietary Vanadium in Mallard Ducks*. *J.*
39 *Toxicol. Environ. Health*, 4: 43-50.
- 40
- 41 Wilson, J.T., 1954. Influence of the Offspring of Altered Physiologic States During Pregnancy
42 in the Rat. *Ann. NY Acad. Sci.* 57: 517-525.
- 43
- 44 Windholz, M., S. Budavari, L.Y. Stroumtsos, and M.N. Fertig (eds.), 1976. *The Merck Index,
45 9th edition*. Merck and Co., Rahway, New Jersey.
- 46
- 47 Wobeser, G., N.O. Nielson, and B. Schiefer, 1976. Mercury and Mink II, Experimental Methyl
48 Mercury Intoxication. *Can. J. Comp. Med.* 34-45.

- 1
2 Wren, C.D., 1986. A Review of Metal Accumulation and Toxicity in Wild Mammals: I.
3 Mercury. *Environmental Research*, Vol. 40, pp. 210-244.
4
5 Zander, M., 1983. Physical and Chemical Properties of Polycyclic Aromatic Hydrocarbons,
6 *Handbook of Polycyclic Aromatic Hydrocarbons*, A. Bjorseth, ed., Marcel Dekker, Inc., New
7 York, New York, pp. 1-25.

Table 7-1

Feeding Guilds and Exposure Pathways
 Ranges Near Training Area T-24A
 Fort McClellan, Alabama

Trophic Level	Feeding Guild	Exposure Pathways
1	Primary Producers (terrestrial plants)	Direct uptake from soil
2	Terrestrial Invertebrates	Ingestion of soil Direct contact with soil
	Benthic Invertebrates	Ingestion of sediment Direct contact with sediment
	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
	Amphibians & Reptiles	Ingestion of soil and sediment Ingestion of terrestrial plants Ingestion of terrestrial invertebrates Ingestion of benthic invertebrates
4	Carnivorous Birds (raptors)	Ingestion of soil Ingestion of prey
	Carnivorous Mammals	Ingestion of soil Ingestion of prey
	Piscivorous Birds	Ingestion of sediment Ingestion of surface water
	Piscivorous Mammals	Ingestion of sediment Ingestion of surface water

Table 7-2

**Constituents of Potential Ecological Concern in Surface Soil^a
Ranges Near Training Area T-24A
Fort McClellan, Alabama**

(Page 1 of 2)

Constituents	Background Threshold Value ^b (mg/kg)	Ecological Screening Value ^c (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 ^d
Metals									
Aluminum	1.63E+04	5.00E+01	3.85E+04	4.52E+03	1.33E+04	110 / 110	770	265.9	5,7
Antimony	1.99E+00	3.50E+00	3.19E+02	4.04E+00	5.65E+00	9 / 110	91	1.6	YES
Arsenic	1.37E+01	1.00E+01	1.02E+01	3.37E-01	3.53E+00	109 / 109	1.02	0.35	3,4
Barium	1.24E+02	1.65E+02	3.75E+02	1.60E+01	9.37E+01	109 / 110	2.27	0.57	5,7
Beryllium	8.00E-01	1.10E+00	2.35E+00	1.81E-01	6.38E-01	96 / 106	2.14	0.58	5,7
Cadmium	2.90E-01	1.60E+00	1.70E+00	1.70E+00	2.40E-01	1 / 110	1.06	0.15	5,7
Calcium	1.72E+03	NA	1.71E+04	5.30E+01	6.72E+02	106 / 109	ND	ND	2,5
Chromium	3.70E+01	4.00E-01	1.09E+02	3.89E+00	1.45E+01	110 / 110	273	36.2	5,7
Cobalt	1.52E+01	2.00E+01	4.18E+01	6.79E-01	7.51E+00	101 / 107	2.09	0.38	5,7
Copper	1.27E+01	4.00E+01	3.43E+02	3.64E+00	3.49E+01	110 / 110	8.58	0.87	YES
Iron	3.42E+04	2.00E+02	8.15E+04	4.53E+03	1.95E+04	110 / 110	408	97.7	5,7
Lead	4.01E+01	5.00E+01	1.09E+05	5.80E+00	1.21E+03	110 / 110	2180	24.2	YES
Magnesium	1.03E+03	4.40E+05	4.21E+03	1.60E+02	6.79E+02	106 / 110	0.0096	0.0015	1,2,5
Manganese	1.58E+03	1.00E+02	2.76E+03	2.41E+01	4.00E+02	110 / 110	28	4.0	4
Mercury	8.00E-02	1.00E-01	2.88E-01	2.50E-02	4.21E-02	73 / 105	2.88	0.42	7
Nickel	1.03E+01	3.00E+01	3.77E+01	1.40E+00	6.68E+00	103 / 104	1.26	0.22	7
Potassium	8.00E+02	NA	4.40E+03	1.66E+02	1.33E+03	84 / 90	ND	ND	2,5
Selenium	4.80E-01	8.10E-01	2.40E+00	4.66E-01	5.82E-01	30 / 92	2.96	0.72	5,7
Silver	3.60E-01	2.00E+00	5.87E-01	5.00E-01	5.34E-01	4 / 110	0.29	0.27	1,4
Sodium	6.34E+02	NA	1.32E+02	2.38E+01	5.17E+01	58 / 97	ND	ND	2,3
Thallium	3.43E+00	1.00E+00	2.36E+00	4.20E-01	4.59E-01	9 / 108	2.36	0.46	3
Vanadium	5.88E+01	2.00E+00	4.31E+01	6.62E+00	1.74E+01	107 / 110	22	8.7	3
Zinc	4.06E+01	5.00E+01	3.44E+02	1.03E+01	3.91E+01	106 / 106	6.88	0.78	YES
Chlorinated Pesticides									
4,4'-DDE	NA	2.50E-03	7.00E-04	7.00E-04	1.49E-03	1 / 16	0.28	0.60	1
alpha-BHC	NA	2.50E-03	2.10E-03	2.10E-03	1.80E-03	1 / 16	0.84	0.72	1
Endrin aldehyde	NA	1.05E-02	1.00E-03	1.00E-03	1.57E-03	1 / 16	0.10	0.15	1
Endrin ketone	NA	1.05E-02	1.30E-03	1.30E-03	1.60E-03	1 / 16	0.12	0.15	1
Nitroaromatics									
2,4-Dinitrotoluene	NA	1.28E+00	1.20E+00	1.20E+00	5.68E-02	1 / 62	0.94	0.044	1
Semivolatile Organic Compounds									
2,4-Dinitrotoluene	NA	1.28E+00	8.60E-01	8.60E-01	9.31E-02	1 / 62	0.67	0.073	1
2-Methylnaphthalene	NA	NA	2.10E-01	2.10E-01	1.15E-01	1 / 81	ND	ND	6
Bis(2-Ethylhexyl)phthalate	NA	9.26E-01	6.00E-02	6.00E-02	8.85E-02	1 / 73	0.065	0.10	1
Butyl benzyl phthalate	NA	2.39E-01	4.20E-02	4.20E-02	1.00E-01	1 / 81	0.18	0.42	1

Table 7-2

**Constituents of Potential Ecological Concern in Surface Soil^a
Ranges Near Training Area T-24A
Fort McClellan, Alabama**

(Page 2 of 2)

Constituents	Background Threshold Value ^b (mg/kg)	Ecological Screening Value ^c (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 ^d
N-Nitrosodiphenylamine	NA	2.00E+01	5.50E-01	5.50E-01	1.05E-01	1 / 81	0.028	0.005	1
Phenanthrene	NA	1.00E-01	1.90E-01	1.90E-01	9.96E-02	1 / 81	1.90	1.00	7
Volatile Organic Compounds									
1,2,4-Trimethylbenzene	NA	1.00E-01	3.50E-03	3.50E-03	1.34E-03	1 / 44	0.035	0.013	1
2-Butanone	NA	8.96E+01	2.30E-02	2.90E-03	4.99E-03	9 / 37	0.00026	0.00006	1
Acetone	NA	2.50E+00	1.40E+00	5.10E-02	2.40E-01	24 / 24	0.56	0.10	1
Bromomethane	NA	NA	3.40E-03	3.40E-03	1.55E-03	1 / 27	ND	ND	6
Chloroform	NA	1.00E-03	3.20E-01	1.90E-03	8.67E-03	2 / 44	320	8.67	7
Cis-1,2-Dichloroethene	NA	1.00E-01	8.30E-03	8.30E-03	1.43E-03	1 / 44	0.083	0.014	1
Ethylbenzene	NA	5.00E-02	7.00E-03	7.00E-03	1.42E-03	1 / 44	0.14	0.028	1
m,p-Xylenes	NA	5.00E-02	7.00E-02	7.00E-02	3.03E-03	1 / 44	1.40	0.061	7
Naphthalene	NA	1.00E-01	1.10E-03	1.10E-03	2.13E-03	1 / 44	0.011	0.021	1
p-Cymene	NA	NA	1.80E-02	1.20E-03	1.69E-03	6 / 44	ND	ND	6
Styrene	NA	1.00E-01	8.90E-04	8.90E-04	1.34E-03	1 / 44	0.009	0.013	1
Toluene	NA	5.00E-02	6.30E-03	6.70E-04	1.38E-03	12 / 44	0.13	0.028	1
Trichlorofluoromethane	NA	1.00E-01	2.00E-01	2.20E-03	7.09E-03	3 / 44	2.00	0.071	7
Chemical Agent Breakdown									
Methylphosphonic Acid	NA	NA	6.20E-02	6.20E-02	2.94E-02	1 / 8	ND	ND	6

^a Surface soil at the 24-A Ranges is defined as the interval from 0 to 1 foot below ground surface.

^b Background threshold value is two times (2x) the arithmetic mean of background metals (SAIC, 1998). For SVOCs, the BTV is the background screening value for soils adjacent to asphalt as given in IT Corporation (IT), 2000, *Final Human Health and Ecological Screening Values and PAH Background Summary Report, Fort McClellan, Calhoun County, Alabama*, July.

^c Ecological Screening Values (ESV) are presented in *Human Health and Ecological Screening Values and PAH Background Summary Report* (IT, 2000).

^d Rationale for exclusion as a COPEC:

- 1 - Maximum detected concentration is less than ESV
- 2 - Essential macro-nutrient, only toxic at extremely high concentrations (i.e. 10-times naturally-occurring background concentrations).
- 3 - Maximum detected concentration is less than the background threshold value (BTV).
- 4 - Slippage Test and Wilcoxon Rank Sum Test indicate the concentration of this constituent is statistically similar to background concentrations.
- 5 - Geochemical evaluation of the data indicate that this constituent is naturally occurring.
- 6 - No ESV available; however, maximum detected concentration of this constituent is less than ESV for similar compounds.
- 7 - Additional lines of evidence indicate that this constituent may not be a COPEC (see text).

NA - Not available.

ND - Not determined.

Table 7-3

**Constituents of Potential Ecological Concern in Surface Water
Ranges Near Training Area T-24A
Fort McClellan, Alabama**

Constituents	Background Threshold Value ^a (mg/L)	Ecological Screening Value ^b (mg/L)	Maximum Detected Conc. (mg/L)	Minimum Detected Conc. (mg/L)	Mean Detected Conc. (mg/L)	Frequency of Detection	Maximum Hazard Quotient	Mean Hazard Quotient	Constituent Of Potential Ecological Concern ^c
Metals									
Aluminum	5.26E+00	8.70E-02	6.43E+01	4.71E-02	8.53E+00	8 / 8	739.1	98.1	5,7
Antimony	NA	1.60E-01	3.00E-02	2.77E-02	2.39E-02	2 / 11	0.19	0.15	1,5
Arsenic	2.17E-03	1.90E-01	9.05E-03	2.41E-03	3.75E-03	2 / 11	0.048	0.020	1,5
Barium	7.54E-02	3.90E-03	4.91E-01	1.31E-02	7.89E-02	11 / 11	125.9	20.2	YES
Beryllium	3.90E-04	5.30E-04	2.24E-03	2.24E-03	1.48E-03	1 / 11	4.23	2.79	5,7
Calcium	2.52E+01	1.16E+02	8.75E+01	2.94E-01	9.15E+00	11 / 11	0.75	0.079	1,2,5
Chromium	1.11E-02	1.10E-02	4.07E-02	5.34E-03	8.65E-03	3 / 11	3.70	0.79	5,7
Cobalt	NA	3.00E-03	1.81E-02	1.81E-02	1.51E-02	1 / 11	6.03	5.03	4
Copper	1.27E-02	6.54E-03	1.07E-01	3.25E-03	1.85E-02	3 / 10	16.36	2.83	YES
Iron	1.96E+01	1.00E+00	4.56E+01	3.25E-02	5.26E+00	11 / 11	45.6	5.26	4
Lead	8.67E-03	1.32E-03	4.31E-01	1.18E-02	4.48E-02	3 / 11	326.5	33.9	YES
Magnesium	1.10E+01	8.20E+01	8.10E+00	3.41E-01	1.88E+00	11 / 11	0.10	0.023	1,2,3
Manganese	5.65E-01	8.00E-02	9.49E-01	3.35E-03	1.27E-01	11 / 11	11.9	1.58	4
Mercury	NA	3.00E-06	6.60E-05	6.30E-05	7.08E-05	3 / 11	22.0	23.6	4
Nickel	2.25E-02	8.77E-02	3.00E-02	1.04E-02	1.52E-02	3 / 11	0.34	0.17	1,4
Potassium	2.56E+00	5.30E+01	1.38E+01	1.40E+00	3.60E+00	7 / 11	0.26	0.07	1,2,5
Selenium	NA	5.00E-03	2.60E-03	2.60E-03	1.74E-03	1 / 11	0.52	0.35	1,4
Sodium	3.44E+00	6.80E+02	3.97E+00	1.23E+00	1.82E+00	10 / 10	0.0058	0.0027	1,2,5
Vanadium	1.52E-02	1.90E-02	6.62E-02	5.20E-03	1.64E-02	2 / 11	3.48	0.87	5,7
Zinc	4.04E-02	5.89E-02	3.27E-01	5.88E-03	6.62E-02	4 / 6	5.55	1.12	YES
Semivolatile Organic Compounds									
2-Methylphenol	NA	4.89E-01	9.50E-03	9.50E-03	3.57E-03	1 / 11	0.019	0.0073	1
4-Methylphenol	NA	4.89E-01	1.00E-01	1.00E-01	1.19E-02	1 / 11	0.204	0.024	1
Bis(2-Ethylhexyl)phthalate	NA	3.00E-04	1.60E-02	8.50E-03	5.00E-03	2 / 11	53.3	16.7	YES
Volatile Organic Compounds									
2-Butanone	NA	7.10E+00	3.20E-02	3.20E-02	8.13E-03	1 / 6	0.0045	0.0011	1
Acetone	NA	7.80E+01	1.70E-02	1.70E-02	1.70E-02	1 / 1	0.00022	0.00022	1
Toluene	NA	1.75E-01	1.30E-02	1.30E-02	1.63E-03	1 / 11	0.074	0.0093	1

^a Background threshold value is two times (2x) the arithmetic mean of background metals (SAIC, 1998).

^b Ecological Screening Values (ESV) are presented in *Human Health and Ecological Screening Values and PAH Background Summary Report* (IT, 2000).

^c Rationale for exclusion as a COPEC:

NA - Not available.

ND - Not determined.

1 - Maximum detected concentration is less than ESV

2 - Essential macro-nutrient, only toxic at extremely high concentrations (i.e. 10-times naturally-occurring background concentrations).

3 - Maximum detected concentration is less than the background threshold value (BTV).

4 - Slippage Test and Wilcoxon Rank Sum Test indicate the concentration of this constituent is statistically similar to background concentrations.

5 - Geochemical evaluation of the data indicate that this constituent is naturally occurring.

6 - No ESV available; however, maximum detected concentration of this constituent is less than ESV for similar compounds.

7 - Additional lines of evidence indicate that this constituent may not be a COPEC (see text).

Table 7-4

**Constituents of Potential Ecological Concern in Sediment
Ranges Near Training Area T-24A
Fort McClellan, Alabama**

(Page 1 of 2)

Constituents	Background Threshold Value ^a (mg/kg)	Ecological Screening Value ^b (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 ^c
Metals									
Aluminum	8.59E+03	NA	1.20E+04	5.56E+03	8.30E+03	11 / 11	ND	ND	5,7
Antimony	7.30E-01	1.20E+01	5.46E+00	5.46E+00	4.54E+00	1 / 11	0.46	0.38	1,5
Arsenic	1.13E+01	7.24E+00	9.20E+00	1.56E+00	4.42E+00	11 / 11	1.27	0.61	3
Barium	9.89E+01	NA	1.26E+02	4.13E+01	7.97E+01	11 / 11	ND	ND	5,7
Beryllium	9.70E-01	NA	2.40E+00	5.20E-01	9.40E-01	11 / 11	ND	ND	5,7
Calcium	1.11E+03	NA	1.03E+04	1.07E+02	1.25E+03	11 / 11	ND	ND	2,5
Chromium	3.12E+01	5.23E+01	2.89E+01	7.19E+00	1.61E+01	11 / 11	0.55	0.31	1,3
Cobalt	1.10E+01	5.00E+01	1.11E+01	2.74E+00	6.68E+00	10 / 11	0.22	0.13	1,5
Copper	1.71E+01	1.87E+01	3.57E+01	3.78E+00	1.74E+01	11 / 11	1.91	0.93	5,7
Iron	3.53E+04	NA	7.44E+04	7.14E+03	2.67E+04	11 / 11	ND	ND	5,7
Lead	3.78E+01	3.02E+01	1.56E+02	6.04E+00	4.86E+01	11 / 11	5.17	1.61	YES
Magnesium	9.06E+02	NA	5.21E+03	3.60E+02	9.04E+02	11 / 11	ND	ND	2,5
Manganese	7.12E+02	NA	6.17E+02	3.88E+01	2.83E+02	11 / 11	ND	ND	3
Mercury	1.10E-01	2.45E-05	2.10E-01	2.10E-01	4.05E-02	1 / 7	8571	1652	YES
Nickel	1.30E+01	1.59E+01	2.77E+01	5.00E+00	9.82E+00	10 / 11	1.74	0.62	5,7
Potassium	1.01E+03	NA	3.41E+03	5.75E+02	2.06E+03	11 / 11	ND	ND	2,5
Selenium	7.20E-01	NA	1.00E+00	5.20E-01	4.94E-01	3 / 11	ND	ND	4
Sodium	6.92E+02	NA	2.27E+02	2.62E+01	5.17E+01	5 / 7	ND	ND	2,3
Thallium	1.30E-01	NA	1.20E+00	1.20E+00	7.39E-01	1 / 11	ND	ND	7
Vanadium	4.09E+01	NA	2.41E+01	8.59E+00	1.64E+01	11 / 11	ND	ND	3
Zinc	5.27E+01	1.24E+02	1.86E+02	1.35E+01	4.80E+01	11 / 11	1.50	0.39	5,7
Semivolatile Organic Compounds									
Acenaphthylene	NA	3.30E-01	4.10E-02	4.10E-02	2.13E-01	1 / 11	0.12	0.65	1
Anthracene	NA	3.30E-01	6.80E-02	6.50E-02	1.93E-01	2 / 11	0.21	0.58	1
Benzo(a)anthracene	NA	3.30E-01	9.90E-01	2.20E-01	2.89E-01	2 / 11	3.00	0.87	YES
Benzo(a)pyrene	NA	3.30E-01	3.40E-01	1.70E-01	2.33E-01	2 / 11	1.03	0.71	1
Benzo(b)fluoranthene	NA	6.55E-01	6.80E-01	2.40E-01	2.83E-01	2 / 11	1.04	0.43	1
Benzo(ghi)perylene	NA	6.55E-01	1.20E-01	1.00E-01	1.95E-01	2 / 11	0.18	0.30	1
Benzo(k)fluoranthene	NA	6.55E-01	5.80E-01	2.10E-01	2.48E-01	2 / 11	0.89	0.38	1
Chrysene	NA	3.30E-01	9.80E-01	4.20E-01	3.14E-01	2 / 11	2.97	0.95	YES

Table 7-4

**Constituents of Potential Ecological Concern in Sediment
Ranges Near Training Area T-24A
Fort McClellan, Alabama**

(Page 2 of 2)

Constituents	Background Threshold Value ^a (mg/kg)	Ecological Screening Value ^b (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 ^c
Dibenz(a,h)anthracene	NA	3.30E-01	6.60E-02	5.00E-02	1.80E-01	2 / 11	0.20	0.54	1
Di-n-butyl phthalate	NA	1.11E-01	3.20E-01	1.90E-01	2.24E-01	2 / 11	2.88	2.01	YES
Fluoranthene	NA	3.30E-01	1.50E+00	3.00E-01	3.44E-01	2 / 11	4.55	1.04	YES
Indeno(1,2,3-cd)pyrene	NA	6.55E-01	1.20E-01	1.10E-01	1.89E-01	2 / 11	0.18	0.29	1
Phenanthrene	NA	3.30E-01	6.70E-02	6.70E-02	2.10E-01	1 / 11	0.20	0.64	1
Pyrene	NA	3.30E-01	2.00E+00	3.10E-01	3.97E-01	2 / 11	6.06	1.20	YES
Volatile Organic Compounds									
2-Butanone	NA	1.37E-01	3.90E-02	5.70E-03	1.64E-02	6 / 11	0.28	0.12	1
Acetone	NA	4.53E-01	3.80E-01	3.70E-02	1.62E-01	9 / 9	0.84	0.36	1
Chloromethane	NA	7.85E-05	3.30E-03	3.30E-03	6.93E-03	1 / 11	42.0	88.2	7
Methylene chloride	NA	1.26E+00	2.30E-01	2.30E-01	2.30E-01	1 / 1	0.18	0.18	1
p-Cymene	NA	NA	2.40E-02	1.40E-03	6.90E-03	4 / 11	ND	ND	6
Toluene	NA	6.70E-01	4.40E-03	1.10E-03	3.79E-03	5 / 11	0.0066	0.0057	1
Chemical Agent Breakdown									
Thiodiglycol	NA	NA	3.20E-02	7.60E-03	1.32E-02	3 / 4	ND	ND	6

^a Background threshold value is two times (2x) the arithmetic mean of background metals (SAIC, 1998).

^b Ecological Screening Values (ESV) are presented in *Human Health and Ecological Screening Values and PAH Background Summary Report* (IT, 2000).

^c Rationale for exclusion as a COPEC:

- 1 - Maximum detected concentration is less than ESV
- 2 - Essential macro-nutrient, only toxic at extremely high concentrations (i.e. 10-times naturally-occurring background concentrations).
- 3 - Maximum detected concentration is less than the background threshold value (BTV).
- 4 - Slippage Test and Wilcoxon Rank Sum Test indicate the concentration of this constituent is statistically similar to background concentrations.
- 5 - Geochemical evaluation of the data indicate that this constituent is naturally occurring.
- 6 - No ESV available; however, maximum detected concentration of this constituent is less than ESV for similar compounds.
- 7 - Additional lines of evidence indicate that this constituent may not be a COPEC (see text).

NA - Not available.

ND - Not determined.

Table 7-5

**Constituents of Potential Ecological Concern in Groundwater
Ranges Near Training Area T-24A
Fort McClellan, Alabama**

(Page 1 of 2)

Constituents	Background Threshold Value ^a (mg/L)	Ecological Screening Value ^b (mg/L)	Maximum Detected Conc. (mg/L)	Minimum Detected Conc. (mg/L)	Mean Detected Conc. (mg/L)	Frequency of Detection	Maximum Hazard Quotient	Mean Hazard Quotient	Constituent Of Potential Ecological Concern ^c
Metals									
Aluminum	2.34E+00	8.70E-02	5.38E+00	5.00E-02	4.11E-01	29 / 37	61.8	4.73	5,7
Antimony	3.19E-03	1.60E-01	3.57E-02	3.09E-02	1.55E-02	2 / 38	0.22	0.097	1
Arsenic	1.78E-02	1.90E-01	2.98E-03	2.71E-03	1.39E-03	2 / 36	0.016	0.0073	1,3
Barium	1.27E-01	3.90E-03	3.14E+00	3.53E-03	1.30E-01	37 / 37	805.1	33.2	YES
Calcium	5.65E+01	1.16E+02	1.73E+02	1.02E-01	8.95E+00	38 / 38	1.49	0.077	2,5
Chromium	1.11E-02	1.10E-02	1.23E-02	3.40E-03	2.83E-03	4 / 38	1.12	0.26	5,7
Cobalt	2.34E-02	3.00E-03	2.21E-02	1.81E-02	6.79E-03	4 / 38	7.37	2.26	3
Copper	2.55E-02	6.54E-03	6.44E-03	3.39E-03	2.33E-03	7 / 35	0.98	0.36	1,3
Iron	7.04E+00	1.00E+00	1.17E+01	2.55E-02	2.32E+00	34 / 35	11.7	2.3	5,7
Lead	8.00E-03	1.32E-03	5.63E-03	1.61E-03	8.44E-04	2 / 35	4.27	0.64	3
Magnesium	2.13E+01	8.20E+01	2.89E+01	1.58E-01	3.91E+00	38 / 38	0.35	0.048	1,2,5
Manganese	5.81E-01	8.00E-02	2.29E+00	3.25E-03	5.08E-01	37 / 37	28.6	6.4	5,7
Mercury	NA	3.00E-06	2.46E-04	2.46E-04	7.01E-05	1 / 38	82.0	23.4	5,7
Nickel	2.25E-02	8.77E-02	3.08E-02	3.10E-03	7.49E-03	10 / 36	0.35	0.085	1,5
Potassium	7.20E+00	5.30E+01	1.52E+02	1.52E+00	1.02E+01	31 / 37	2.87	0.19	2,5
Selenium	NA	5.00E-03	5.06E-03	1.96E-03	1.32E-03	7 / 36	1.01	0.26	4
Silver	4.00E-03	1.20E-05	6.91E-03	6.91E-03	2.56E-03	1 / 33	575.8	213.2	4
Sodium	1.48E+01	6.80E+02	5.10E+01	7.91E-01	5.76E+00	37 / 37	0.075	0.0085	1,2,5
Vanadium	1.70E-02	1.90E-02	8.85E-03	8.85E-03	2.71E-03	1 / 38	0.47	0.14	1,3
Zinc	2.20E-01	5.89E-02	2.97E-01	4.20E-03	1.47E-02	12 / 35	5.04	0.25	4
Chemical Agent Breakdown									
Thiodiglycol	NA	NA	1.40E-02	1.40E-02	1.21E-03	1 / 37	ND	ND	6
Chlorinated Pesticides									
beta-BHC	NA	5.00E+01	2.70E-05	1.90E-05	2.47E-05	2 / 14	0.0000005	0.0000005	1
Nitroaromatics									
4-Amino-2,6-dinitrotoluene	NA	NA	7.80E-04	7.80E-04	1.19E-04	1 / 37	ND	ND	6
Semivolatile Organic Compounds									
Bis(2-Ethylhexyl)phthalate	NA	3.00E-04	4.30E-03	3.90E-03	1.66E-03	2 / 37	14.3	5.5	YES
Diethyl phthalate	NA	5.21E-01	2.50E-03	2.50E-03	1.31E-03	1 / 37	0.0048	0.0025	1

Table 7-5

**Constituents of Potential Ecological Concern in Groundwater
Ranges Near Training Area T-24A
Fort McClellan, Alabama**

(Page 2 of 2)

Constituents	Background Threshold Value ^a (mg/L)	Ecological Screening Value ^b (mg/L)	Maximum Detected Conc. (mg/L)	Minimum Detected Conc. (mg/L)	Mean Detected Conc. (mg/L)	Frequency of Detection	Maximum Hazard Quotient	Mean Hazard Quotient	Constituent Of Potential Ecological Concern ^c
Phenol	NA	2.56E-01	1.30E-02	1.30E-02	1.24E-03	1 / 37	0.051	0.0048	1
Volatile Organic Compounds									
1,2,4-Trimethylbenzene	NA	NA	2.70E-03	1.10E-03	1.30E-04	2 / 61	ND	ND	6
1,2-Dimethylbenzene	NA	NA	3.70E-04	1.50E-04	6.80E-05	3 / 61	ND	ND	6
1,3,5-Trimethylbenzene	NA	NA	6.80E-04	6.80E-04	8.49E-05	1 / 61	ND	ND	6
2-Butanone	NA	7.10E+00	4.70E-02	1.10E-02	1.23E-02	4 / 11	0.0066	0.0017	1
2-Hexanone	NA	1.71E+00	1.90E-03	1.90E-03	5.25E-04	1 / 55	0.0011	0.0003	1
Acetone	NA	7.80E+01	5.10E-02	4.20E-02	6.01E-03	2 / 18	0.00065	0.00008	1
Benzene	NA	5.30E-02	9.70E-01	2.40E-04	1.60E-02	4 / 61	18.3	0.3	7
Carbon disulfide	NA	8.40E-02	4.30E-03	2.20E-04	1.75E-04	8 / 59	0.051	0.0021	1
Carbon tetrachloride	NA	3.52E-01	3.80E-01	3.00E-03	6.92E-03	3 / 61	1.08	0.020	7
Chloroform	NA	2.89E-01	1.70E-01	2.40E-04	3.08E-03	7 / 61	0.59	0.011	1
Chloromethane	NA	5.50E+00	1.10E-03	5.90E-04	2.34E-04	3 / 61	0.00020	0.00004	1
N-Propylbenzene	NA	NA	4.90E-04	4.90E-04	8.18E-05	1 / 61	ND	ND	6
p-Cymene	NA	NA	2.70E-04	2.70E-04	9.30E-05	1 / 61	ND	ND	6
sec-Butylbenzene	NA	NA	5.50E-04	5.50E-04	9.75E-05	1 / 61	ND	ND	6
tert-Butylbenzene	NA	NA	2.30E-04	2.30E-04	6.77E-05	1 / 61	ND	ND	6
Tetrachloroethene	NA	8.40E-02	6.40E-04	6.40E-04	9.90E-05	1 / 61	0.0076	0.0012	1
Toluene	NA	1.75E-01	1.40E-04	1.30E-04	6.28E-05	2 / 54	0.00080	0.00036	1

^a Background threshold value is two times (2x) the arithmetic mean of background metals (SAIC, 1998).

^b Ecological Screening Values (ESV) are presented in *Human Health and Ecological Screening Values and PAH Background Summary Report* (IT, 2000).

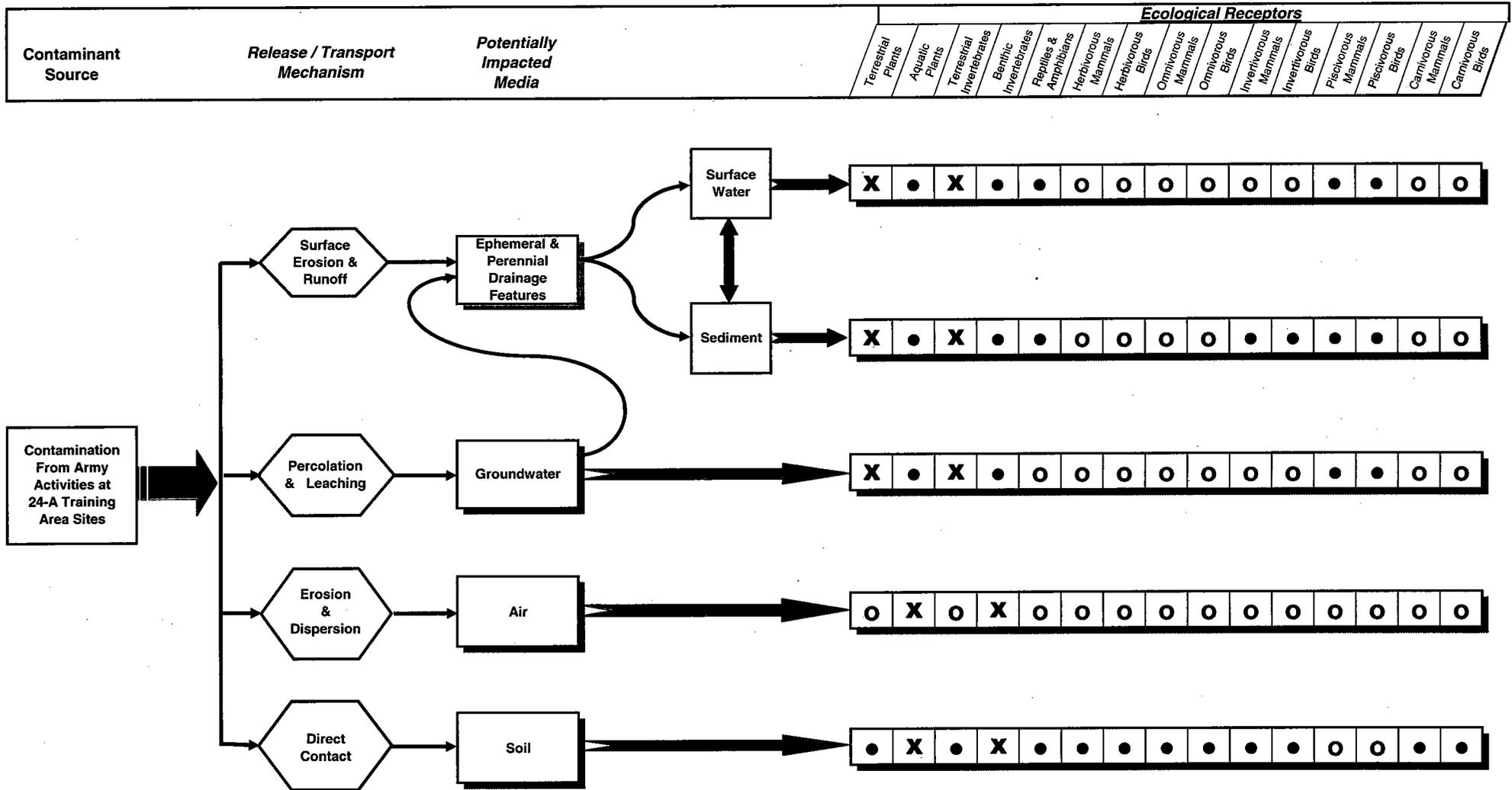
^c Rationale for exclusion as a COPEC:

- 1 - Maximum detected concentration is less than ESV
- 2 - Essential macro-nutrient, only toxic at extremely high concentrations (i.e. 10-times naturally-occurring background concentrations).
- 3 - Maximum detected concentration is less than the background threshold value (BTV).
- 4 - Slippage Test and Wilcoxon Rank Sum Test indicate the concentration of this constituent is statistically similar to background concentrations.
- 5 - Geochemical evaluation of the data indicate that this constituent is naturally occurring.
- 6 - No ESV available; however, maximum detected concentration of this constituent is less than ESV for similar compounds.
- 7 - Additional lines of evidence indicate that this constituent may not be a COPEC (see text).

NA - Not available.

ND - Not determined.

Figure 7-1
 Site Conceptual Model
 Ranges Near Training Area T-24A
 Fort McClellan, Alabama



Key To Potential Exposure Routes

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