

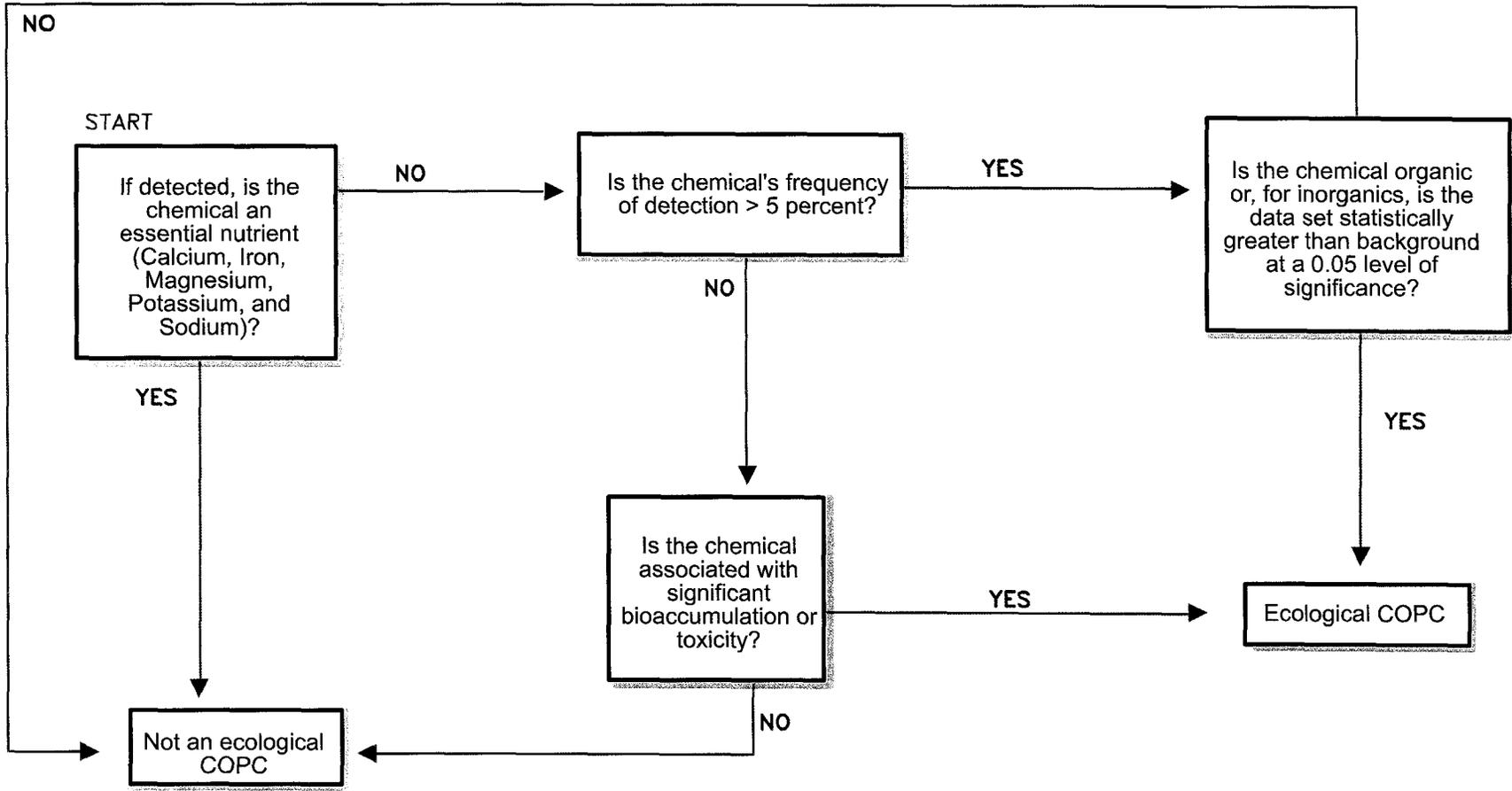
FIGURES



Alameda Point
 Department of the Navy, BRAC PMO West, San Diego, California

FIGURE G-1
OPERABLE UNITS, CERCLA SITES
AND BUFFER ZONES

Ecological Risk Assessment
 for Sites 3, 4, 11, and 21



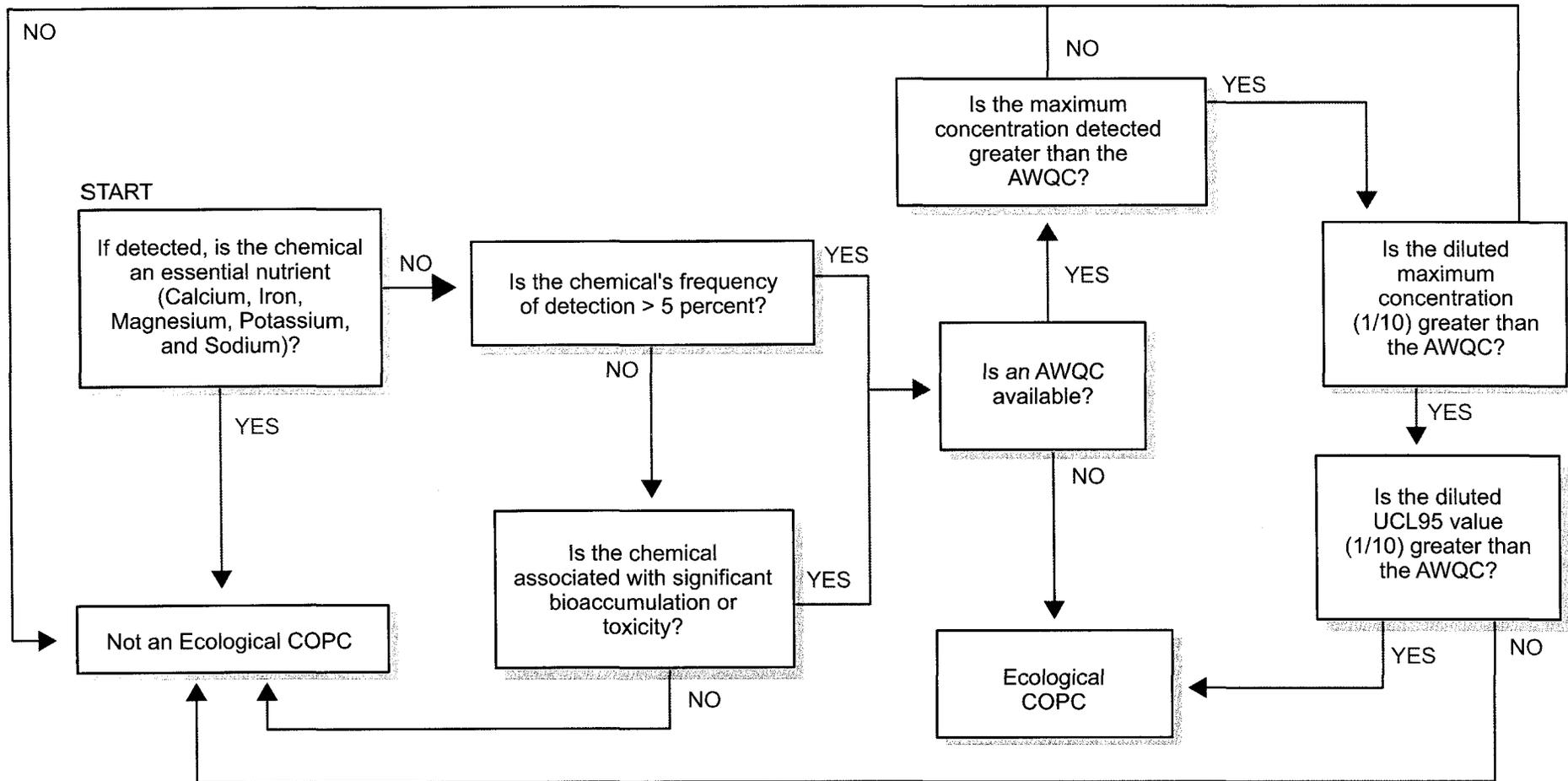
LEGEND

COPC CHEMICAL OF POTENTIAL CONCERN

SuTech

Alameda Point
U.S. Navy Southwest Division, NAVFAC, San Diego

FIGURE G-2
DECISION TREE FOR ECOLOGICAL
COPC SELECTION FOR
SURFACE SOILS AND RHIZOSPHERE
Ecological Risk Assessment for
Sites 3, 4, 11, and 21



SuTech

LEGEND

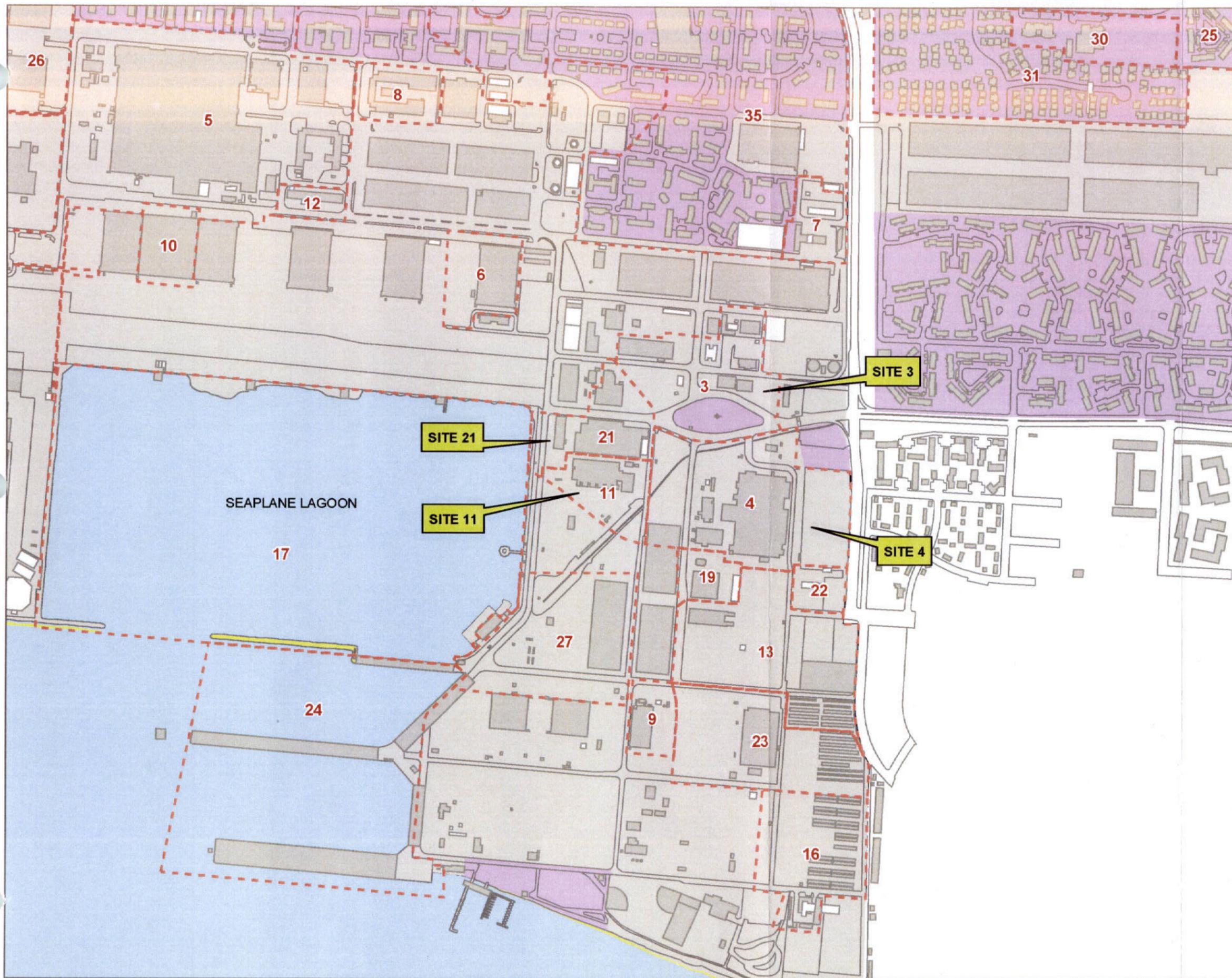
AWQC
COPC
UCL95

AMBIENT WATER QUALITY CRITERIA
CHEMICAL OF POTENTIAL CONCERN
95TH PERCENTILE UPPER CONFIDENCE LIMIT ON THE ARITHMETIC MEAN

Alameda Point
U.S. Navy Southwest Division, NAVFAC, San Diego

FIGURE G-3
DECISION TREE FOR ECOLOGICAL COPC
SELECTION FOR GROUNDWATER

Ecological Risk Assessment for
Sites 3, 4, 11, and 21



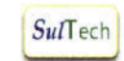
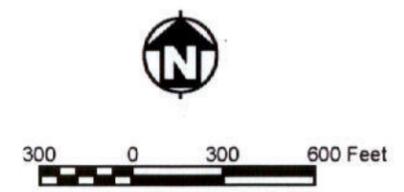
HABITAT

- Intensively Developed
- Landscaped/Developed
- Rock Breakwaters/Rip Rap
- CERCLA SITE

BUILDING

- Present
- Removed

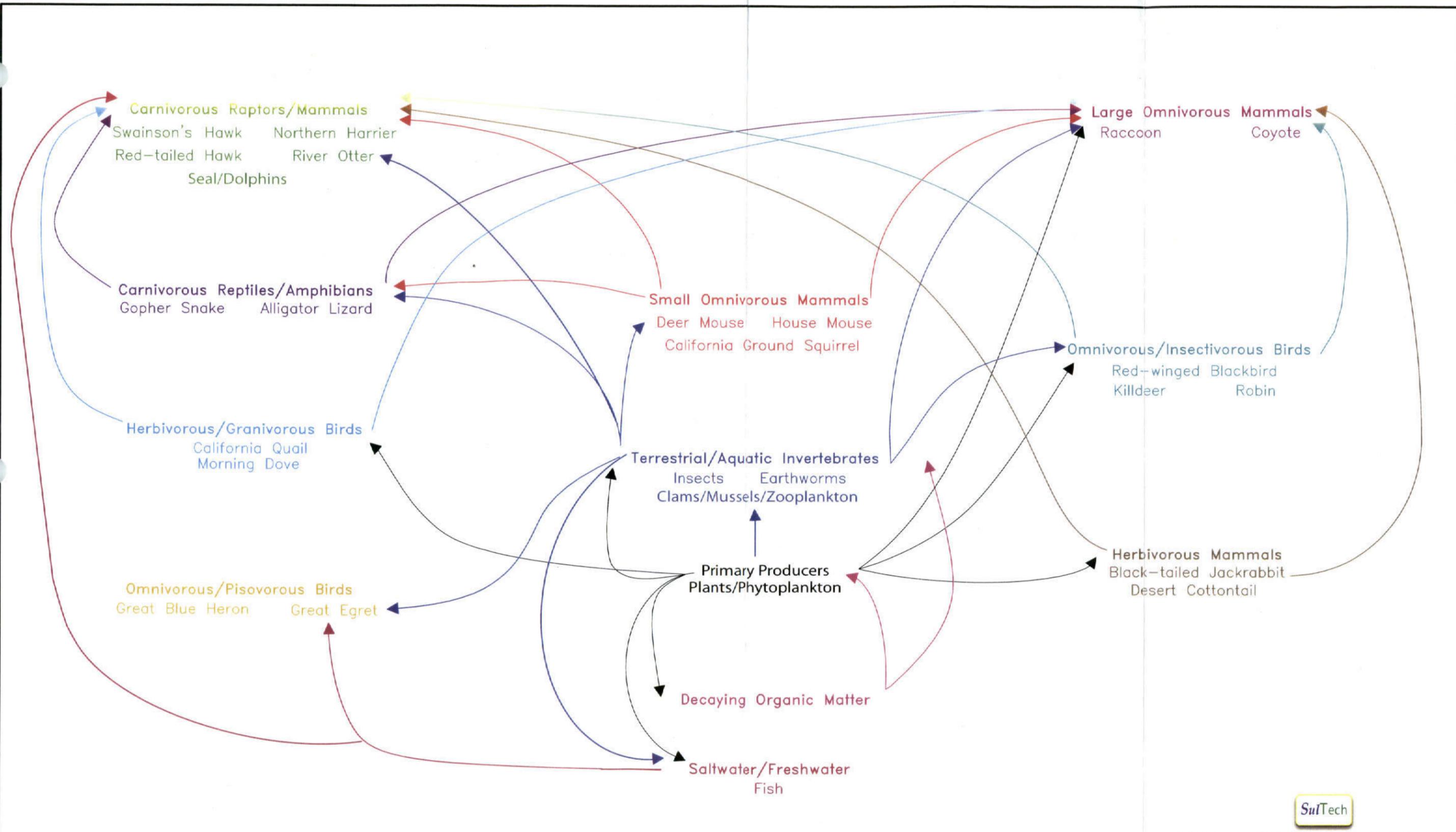
Note:
 CERCLA = Comprehensive Environmental Response, Compensation, and Liability Act of 1980



Alameda Point
 Department of the Navy, BRAC PMO West, San Diego, California

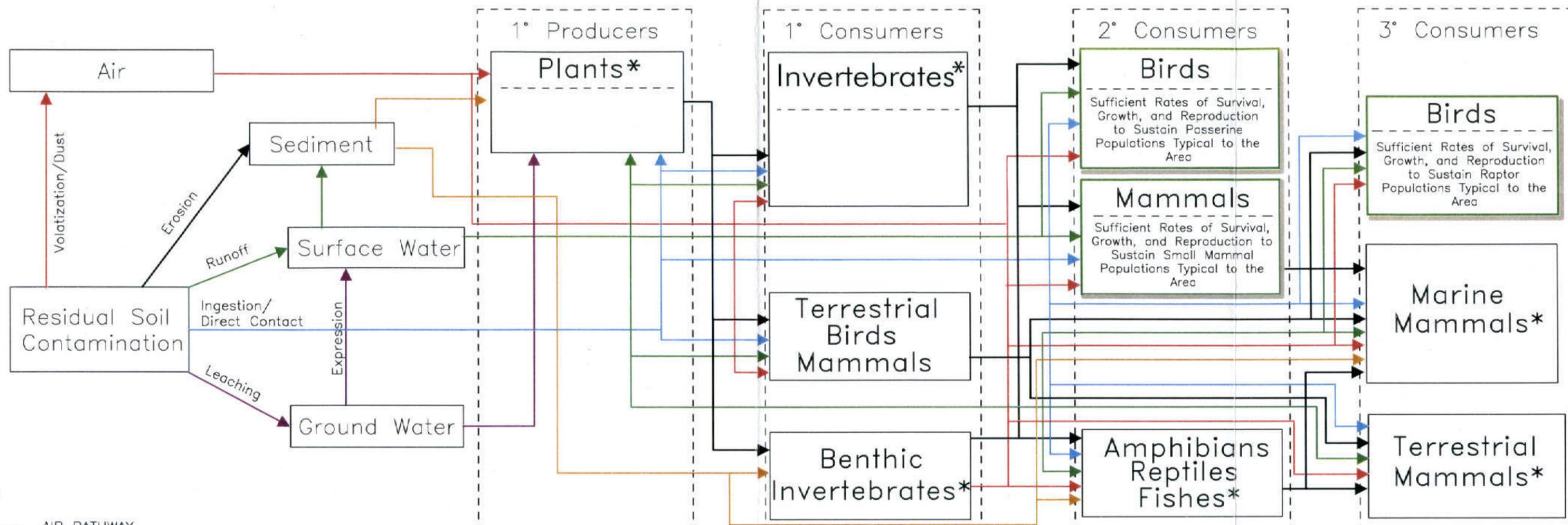
FIGURE G-4
ECOLOGICAL HABITAT MAP OF
SITES 3, 4, 11 AND 21
 Ecological Risk Assessment
 for Sites 3, 4, 11, and 21

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Alameda Point
U.S. Navy Southwest Division, NAVFAC, San Diego

FIGURE G-5
TERRESTRIAL FOOD WEB
Ecological Risk Assessment for
Sites 3, 4, 11, and 21



LEGEND

- AIR PATHWAY
- GROUNDWATER PATHWAY
- SURFACE WATER PATHWAY
- DIRECT CONTACT/SOIL INGESTION PATHWAY
- DIRECT CONTACT/SEDIMENT INGESTION PATHWAY
- FOOD INGESTION PATHWAY



ASSESSMENT ENDPOINTS

- 1° PRIMARY
- 2° SECONDARY
- 3° TERTIARY

* THESE CONSUMERS ARE PRESENT AT ALAMEDA POINT BUT WERE NOT ASSESSED AS AN ENDPOINT

Measurement Endpoint(s)

Reproductive or physiological impacts to the Alameda song sparrow (*Melospiza melodia pusillula*)

Reproductive or physiological impacts to the California ground squirrel (*Citellus beecheyi*)

Reproductive or physiological impacts to the Red-tailed hawk (*Buteo jamaicensis*)



Alameda Point
U.S. Navy Southwest Division, NAVFAC, San Diego

FIGURE G-6
GENERIC ECOLOGICAL
CONCEPTUAL SITE MODEL
Ecological Risk Assessment for
Sites 3, 4, 11, and 21

TABLES

TABLE G-1: SPECIAL STATUS SPECIES – PLANTS, FISH, REPTILES, AND MAMMALS

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

	Common Name	Scientific Name
Plants ^a	Contra Costa goldfields	<i>Lasthenia conjugens</i>
	Santa Cruz tarplant	<i>Holocarpha macradenia</i>
	Kellogg's horkelia	<i>Horkelia cuneata sericea</i>
	Point Reyes bird's beak	<i>Cordylanthus maritimus palustris</i>
	Adobe sanicle	<i>Sanicula maritima</i>
Fish ^b	Chinook salmon, winter run	<i>Oncorhynchus tshawytscha</i>
	Longfin smelt	<i>Spirinchus thaleichthys</i>
	Delta smelt	<i>Hypomesus transpacificus</i>
	Coho salmon	<i>Oncorhynchus kisutch</i>
Reptiles ^c	Alameda whipsnake	<i>Masticophis lateralis euryxanthus</i>
Mammals ^c	Saltmarsh harvest mouse ^d	<i>Reithrodonomys raviventris</i>
	San Francisco dusky-footed woodrat	<i>Neotoma fuscipes annectens</i>
	Townsend's western big-eared bat	<i>Plecotus townsendii townsendii</i>
	California mastiff bat	<i>Eumops perotis californicus</i>
	Northern (Steller) sea lion	<i>Eumetopias jubatus</i>
	Saltmarsh wandering shrew	<i>Sorex vagrens halicoetes</i>
	Alameda Island mole	<i>Scapanus latimanus parvus</i>

Notes:

- a Rare plant species listed as potentially occurring at Alameda Point. These plants were not identified during vegetation surveys performed in 1995 and 1997.
- b Rare fish species that may occur in the open water areas adjacent to Alameda Point.
- c Special status species that may occur at Alameda Point.
- d In 1995, a survey for the saltmarsh harvest mouse was conducted in the West Beach Landfill Wetland and in the Runway Area Wetland to identify potential receptors for evaluation in ecological risk assessments being conducted by the Navy for the IR program. No individuals were captured during these surveys of the West Beach Landfill Wetland and Runway Area Wetland.

Reference:

U.S. Fish and Wildlife Service (FWS). 1993. Listed and Proposed Endangered and Threatened Species and Candidate Species that May Occur in the Area of the Proposed Closure of Naval Air Station, Alameda, Alameda County, California (1-1-94-SP-192, December 31, 1993). Enclosure attached to letter from Dale A. Pierce, FWS, to John H. Kennedy, U.S. Department of Navy.

TABLE G-2: SPECIAL STATUS SPECIES – BIRDS

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

	Common Name	Scientific Name
Birds ^a	California least tern	<i>Sterna antillarum browni</i>
	American peregrine falcon	<i>Falco peregrinus anatum</i>
	Western snowy plover, coastal population	<i>Charadrius alexandrinus nivosus</i>
	Saltmarsh common yellowthroat	<i>Geothlypis trichas sinuosa</i>
	Alameda song sparrow	<i>Melospiza melodia pusillula</i>
	Double-crested cormorant, rookery sites	<i>Phalacrocorax auritus</i>
	California black rail	<i>Laterallus jamaicensis coturniculus</i>
	California clapper rail	<i>Rallus longirostris obsoletus</i>
	Caspian tern, nesting colonies	<i>Sterna caspia</i>
	Forster's tern, nesting colonies	<i>Sterna forsteri</i>
	California brown pelican, nesting colony	<i>Pelecanus occidentalis californicus</i>
	California horned lark	<i>Eremophila alpestris actia</i>
	Loggerhead shrike	<i>Lanius ludovicianus</i>
	California gull	<i>Larus californicus</i>
	Northern harrier, nesting sites	<i>Circus cyaneus</i>
	Merlin	<i>Falco columbarius</i>
	Long-billed curlew, breeding	<i>Numenius americanus</i>
	Burrowing owl, burrowing sites	<i>Athene cunicularia</i>
	Common loon, breeding	<i>Gavia imer</i>
	Fork-tailed storm petrel, rookery	<i>Ocanodroma furcata</i>
	American white pelican, nesting colony	<i>Pelicanus erythrorhynchos</i>
	Clark's grebe	<i>Aechmophorus clarkii</i>
	Western grebe	<i>Aechmophorus occidentalis</i>
	Great blue heron, rookery	<i>Ardea herodias</i>
	Great egret, rookery	<i>Casmerodius albus</i>
	Snowy egret, rookery	<i>Egreta thula</i>
	Black-crowned night heron, rookery	<i>Nycticorax nycticorax</i>
	Black-shouldered kite, nesting	<i>Elanus caeruleus</i>
	Common murre, nesting colony	<i>Uria aalge</i>

Notes:

- ^a Special status bird species and associated sensitive habitats (such as breeding, nesting, and rookery sites) that occur or may occur at Alameda Point.

Reference:

U.S. Fish and Wildlife Service (FWS). 1993. Listed and Proposed Endangered and Threatened Species and Candidate Species that may occur in the Area of the Proposed Closure of Naval Air Station, Alameda, Alameda County, California (1-1-94-SP-192, December 31, 1993). Enclosure attached to letter from Dale A. Pierce, FWS, to John H. Kennedy, U.S. Department of Navy.

TABLE G-3: TERRESTRIAL HABITAT SUMMARY FOR OU-2B SITES

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Habitat Type	Dominant Vegetation	Observed Animal Species	Relative Occurrence
Site 3			
Urban/Ornamental Landscapes	Ryegrass (<i>Lolium</i> spp.); Common plantain (<i>Plantago</i> sp.); Fennel (<i>Foeniculum vulgare</i>); Sweetclover (<i>Melilotus</i> sp.)	Canada Goose (<i>Branta canadensis</i>); American robin (<i>Turdus migratorius</i>); House sparrow (<i>Passer domesticus</i>); Mourning dove (<i>Zenaida macrowra</i>)	Common
Site 4			
Urban/Ornamental Landscapes	None (paved)	None	NA
Site 11			
Urban/Ornamental Landscapes	None (paved)	None	NA
Site 21			
Disturbed Areas	None (paved)	None	NA

Note:

NA Not applicable

TABLE G-4: HIGH TOXICITY REFERENCE VALUES FOR THE CALIFORNIA GROUND SQUIRREL (CITELLUS BEECHEYI)

Ecological Risk Assessment For Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Ecological COPC	Literature-based high TRV (mg/kg-day)	Source of Study	Study	Endpoint	Body Weight of Study Subject (g)	Allometrically converted high TRV (mg/kg-day)
Metals						
Aluminum	1.93E+01	Sample and others (1996)	Ondreicka and others (1966)	Adverse reproduction in mice	30	1.62E+01
Antimony ^a	4.70E+00	Navy (1998)	Brown and others (1976)	Decrease in water intake, kidney weight/body weight ratio, respiratory effects in Sprague-Dawley rats	110	4.26E+00
Arsenic	4.70E+00	Navy (1998)	Brown and others (1976)	Decrease in water intake, kidney weight/body weight ratio, respiratory effects in Sprague-Dawley rats	110	4.26E+00
Barium	1.98E+01	Sample and others (1996)	Borzelleca and others (1988)	Mortality in female rats	350	1.92E+01
Beryllium	6.60E+00	Sample and others (1996)	Schroeder and Mitchener (1971)	Adverse physiological effects in rats	350	6.42E+00
Cadmium	2.64E+00	Navy (1998)	Schroeder and Mitchener (1971)	Increase in young deaths and runts; failure to breed in mice	31.4	2.22E+00
Chromium	1.31E+01	Sample and others (1996)	Steven and others (1976) as cited in Eisler (1986)	Mortality in rats	350	1.28E+01
Cobalt	2.00E+01	Navy (1998)	Mollenhauer and others (1985)	Increase in testicular degeneration in rats	200	1.88E+01
Copper	6.32E+02	Navy (1998)	Hebert and others (1993)	Decreased water consumption, body weight, and increased mortality in mice	24.7	5.24E+02
Lead	2.41E+02	Navy (1998)	Wise 1981	Decrease in body weight, liver weight, and kidney weight in mice	18.7	1.96E+02
Manganese	1.59E+02	Navy (1998)	Gray and Laskey (1980)	Decrease in paired testes weight, seminal vesicle weight, and preputial gland weight in mice	29.7	1.33E+02
Mercury	4.00E+00	Navy (1998)	Wobeser and others (1976)	Adverse effects on the nervous system in rats	187.5	3.75E+00
Molybdenum	2.60E+00	Sample and others (1996)	Schroeder and Mitchener (1971)	Reduced reproductive success with a high incidence of runts in mice	30	2.18E+00
Nickel	3.16E+01	Navy (1998)	Smith and others (1993)	Increase in the number and proportion of pups born dead or dying shortly after birth during G1 in Loong-Evans rats	248.6	3.01E+01
Silver	NV	NA	NA	NA	NA	NA
Vanadium	2.10E+00	Sample and others (1996)	Domingo and others (1986)	Reproduction in rats	260	2.01E+00
Zinc	4.11E+02	Navy (1998)	Shlicker and Cox (1968)	Decreased fetus weight, fetal liver weight, and body weight in Nulliparous rats	175	3.84E+02
Pesticides						
DDT ^b	1.60E+01	Navy (1998)	EPA (1995a)	Reproductive effects in rats	320	1.55E+01

TABLE G-4: HIGH TOXICITY REFERENCE VALUES FOR THE CALIFORNIA GROUND SQUIRREL (CITELLUS BEECHEYI)

Ecological Risk Assessment For Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Ecological COPC	Literature-based high TRV (mg/kg-day)	Source of Study	Study	Endpoint	Body Weight of Study Subject (g)	Allometrically converted high TRV (mg/kg-day)
PCBs^c						
Total PCBs	1.28E+00	Navy (1998)	Linzey (1987)	Decrease in survival/litter; increase in birth interval, decrease number of young per litter in mice	22.85	1.06E+00
PAHs						
HMW PAHs ^d	3.28E+01	Navy (1998)	Rigdon and Neal (1969)	Increase in pulmonary adenoma	30.5	2.75E+01
LMW PAHs ^e	1.50E+02	Navy (1998)	Navarro and others (1991)	Decrease in weight gain during gestation period	270.2	1.44E+02
SVOCs						
Bis(2-ethylhexyl)phthalate	1.83E+02	Sample and others (1996)	Lamb and others (1987)	Adverse reproductive effects in mice	30	1.53E+02
n-Nitroso-diphenylamine	NV	NA	NA	NA	NA	NA
Pentachlorophenol	2.40E+00	Sample and others (1996)	Schwetz and others (1978)	Significant reduction in survival and growth in rats	350	2.33E+00
VOCs						
2-Butanone	NV	NA	NA	NA	NA	NA
1,1,1-Trichloroethane	1.00E+04	Sample and others (1996)	Lane and others (1982)	No observed effects to critical lifestages (reproduction) of mice	35	8.47E+03
Acetone	5.00E+02	Sample and others (1996)	EPA (1986)	Adverse physiological effects in rats	350	4.86E+02
Benzene	2.64E+02	Sample and others (1996)	Nawrot and Staples (1979)	Decrease in fetal weights, increase maternal mortality, and embryonic resorption	30	2.21E+02
Carbon Disulfide	NV	NA	NA	NA	NA	NA
Chloroform	4.10E+01	Sample and others (1996)	Palmer and others (1979)	Gonadal atrophy observed in male and female rats	350	3.99E+01
Ethylbenzene	NV	NA	NA	NA	NA	NA
Toluene	2.60E+02	Sample and others (1996)	Nawrot and Staples (1979)	Adverse effects on reproduction in mice	30	2.18E+02
Xylene	2.60E+00	Sample and others (1996)	Marks and others (1982)	Adverse effects on reproduction in mice	30	2.18E+00

Notes:

^a Individual TRV not developed for antimony. Based on arsenic TRV.

^b DDT TRV based on 4,4'-DDT; individual TRVs not developed.

^c PCB TRV based on Aroclor-1254; individual TRVs not developed.

^d HMW PAHs are defined as measured PAHs with a molecular weight greater than 200 a.u. and include: benzo(a)fluorene, fluoranthene, benzo(k)fluoranthene, benzo(b)fluoranthene, benzo(b)fluorene, benzo(g,h,i)perylene, chrysene, benzo(a)anthracene, dibenzo(a,h)anthracene, indeno(1,2,3-cd)pyrene, pyrene, and benzo(a)pyrene. TRV based on benzo(a)pyrene.

TABLE G-4: HIGH TOXICITY REFERENCE VALUES FOR THE CALIFORNIA GROUND SQUIRREL (CITELLUS BEECHEYI)
 Ecological Risk Assessment For Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Notes (Continued):

e	LMW PAHs are defined as measured PAHs with a molecular weight below 200 a.u., including naphthalene, fluorene, anthracene, phenanthrene, acenaphthene, and 2-methylnaphthalene. TRV based on naphthalene.
a.u.	Atomic unit
COPC	Chemical of potential concern
DDT	Dichlorodiphenyltrichloroethane
DDTt	Sum of concentrations of 4,4'-dichlorodiphenyldichloroethane, 4,4'-dichlorodiphenyldichloroethene, and 4,4'-dichlorodiphenyltrichloroethane
EPA	U.S. Environmental Protection Agency
g	Gram
HMW	High molecular weight
LMW	Low molecular weight
mg/kg-day	Milligram per kilogram per day
NA	Not applicable
Navy	U.S. Department of Navy
NOAEL	No observed adverse effects level
NV	No value available
PAH	Polynuclear aromatic hydrocarbon
PCB	Polychlorinated biphenyl
SVOC	Semivolatile organic chemical
TRV	Toxicity reference value
VOC	Volatile organic chemical

References:

Navy. 1998. "Interim Final Technical Memorandum, Development of Toxicity Reference Values for Conducting Ecological Risk Assessments at Naval Facilities in California." September.
 Sample, B.E., D.M. Opresko, and G.W. Suter, II. 1996. "Toxicological Benchmarks for Wildlife: 1996 Revision." ES/ER/TM-86/R3. Oak Ridge National Laboratory. Oak Ridge, Tennessee.

TABLE G-5: LOW TOXICITY REFERENCE VALUES FOR THE CALIFORNIA GROUND SQUIRREL (CITELLUS BEECHEYI)

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Ecological COPC	Literature-based low TRV (mg/kg-day)	Source of Study	Study	Endpoint	Body Weight of Study Subject (g)	Allometrically converted low TRV (mg/kg-day)
Metals						
Aluminum	1.93E+00	Sample and others (1996)	Ondreicka and others (1966)	Adverse reproduction in mice	30	1.62E+00
Antimony ^a	3.20E-01	Navy (1998)	Schroeder and others (1968)	Adverse effect on growth rates, survival, glycosuria, proteinuria, blood pressure, tumors, and heart weight and adverse effect on serum glucose in females (rats)	332	3.10E-01
Arsenic	3.20E-01	Navy (1998)	Schroeder and others (1968)	Adverse effect on growth rates, survival, glycosuria, proteinuria, blood pressure, tumors, and heart weight and adverse effect on serum glucose in females (rats)	332	3.10E-01
Barium	5.10E+00	Sample and others (1996)	Perry and others (1983)	Growth and hypertension in rats	435	5.02E+00
Beryllium	6.60E-01	Sample and others (1996)	Schroeder and Mitchener (1971)	Adverse physiological effects in rats	350	6.42E-01
Cadmium	6.00E-02	Navy (1998)	Webster (1988)	NOAEL for effects on fetal weight	32.2	5.05E-02
Chromium	3.28E+00	Sample and others (1996)	McKenzie and others (1958)	Physiological effects in rats	350	3.19E+00
Cobalt	1.20E+00	Navy (1998)	Domingo and others (1985)	Decrease in pup growth in rats	275	1.15E+00
Copper	2.67E+00	Navy (1998)	Pocino and others (1991)	Adverse effect on food ingestion rate, body weight, number of cells in the thymus, or mortality in mice	30	2.24E+00
Lead	1.00E+00	Navy (1998)	Agency for Toxic Substances and Disease Registry (1993)	Adverse effects on reproductive, hematological, and neurological systems in rats	208	9.42E-01
Manganese	1.37E+01	Navy (1998)	Gray and Laskey (1980)	Decrease in paired testes weight, seminal vesicle weight, and preputial gland weight in mice	34.6	1.16E+01
Mercury	2.50E-01	Navy (1998)	Wobeser and others (1976)	Adverse effects on the nervous system in rats	187.5	2.34E-01
Molybdenum	2.60E-01	Sample and others (1996)	Schroeder and Mitchener (1971)	Reduced reproductive success with a high incidence of runts in mice	30	2.18E-01
Nickel	1.33E-01	Navy (1998)	Smith and others (1993)	Increase in the number and proportion of G2 pups born dead or dying shortly after birth	248.6	1.27E-01
Silver	NV	NA	NA	NA	NA	NA
Vanadium	2.10E-01	Sample and others (1996)	Domingo and others (1986)	Reproduction in rats	260	2.01E-01

TABLE G-5: LOW TOXICITY REFERENCE VALUES FOR THE CALIFORNIA GROUND SQUIRREL (CITELLUS BEECHEYI)
 Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Ecological COPC	Literature-based low TRV (mg/kg-day)	Source of Study	Study	Endpoint	Body Weight of Study Subject (g)	Allometrically converted low TRV (mg/kg-day)
Metals (Continued)						
Zinc	9.61E+00	Navy (1998)	Aughey and others (1977)	Hypertrophy and vacuolation of pancreatic islets cells and fasciolata cells in the adrenal cortex	25.5	7.98E+00
Pesticides						
DDT ^b	8.00E-01	Navy (1998)	EPA (1995a)	Reproductive effects in rats	320	7.73E-01
PCBs^c						
Total PCBs	3.60E-01	Navy (1998)	Simmons and McKee (1992)	NOAEL for liver weight, drug induced sleep time, or enzyme activity in mice	20.6	2.95E-01
PAHs						
HMW PAHs ^d	1.31E+00	Navy (1998)	Neal and Rigdon (1967)	Occurrences of gastric neoplasts and change of life span in mice	30.5	1.10E+00
LMW PAHs ^e	5.00E+01	Navy (1998)	Navarro and others (1991)	Increase in maternal toxicity	276.5	4.79E+01
SVOCs						
Bis(2-ethylhexyl)phthalate	1.83E+01	Sample and others (1996)	Lamb and others (1987)	Adverse reproductive effects in mice	30	1.53E+01
n-Nitroso-diphenylamine	NV	NA	NA	NA	NA	NA
Pentachlorophenol	2.40E-01	Sample and others (1996)	Schwetz and others (1978)	Significant reduction in survival and growth in rats	350	2.33E-01
VOCs						
2-Butanone	NV	NA	NA	NA	NA	NA
1,1,1-Trichloroethane	1.00E+03	Sample and others (1996)	Lane and others (1982)	No observed effects to critical lifestages (reproduction) of mice	35	8.47E+02
Acetone	1.00E+02	Sample and others (1996)	EPA (1986)	Adverse physiological effects in rats	350	9.72E+01
Benzene	2.64E+01	Sample and others (1996)	Nawrot and Staples (1979)	Decrease in fetal weights, increase maternal mortality, and embryonic resorption	30	2.21E+01
Carbon Disulfide	NV	NA	NA	NA	NA	NA
Chloroform	1.50E+01	Sample and others (1996)	Palmer and others (1979)	Gonadal atrophy observed in male and female rats	350	1.46E+01
Ethylbenzene	NV	NA	NA	NA	NA	NA
Toluene	2.60E+01	Sample and others (1996)	Nawrot and Staples (1979)	Adverse effects on reproduction in mice	30	2.18E+01
Xylene	2.10E+00	Sample and others (1996)	Marks and others (1982)	Adverse effects on reproduction in mice	30	1.76E+00

TABLE G-5: LOW TOXICITY REFERENCE VALUES FOR THE CALIFORNIA GROUND SQUIRREL (CITELLUS BEECHEYI)
 Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Notes (Continued):

a	Individual TRV not developed for antimony. Based on arsenic TRV.
b	DDT† TRV based on 4,4'-DDT; individual TRVs not developed.
c	PCB TRV based on Aroclor-1254; individual TRVs not developed.
d	HMW PAHs are defined as measured PAHs with a molecular weight greater than 200 a.u. and include: benzo(a)fluorene, fluoranthene, benzo(k)fluoranthene, benzo(b)fluoranthene, benzo(b)fluorene, benzo(g,h,i)perylene, chrysene, benzo(a)anthracene, dibenzo(a,h)anthracene, indeno(1,2,3-cd)pyrene, pyrene, and benzo(a)pyrene. TRV based on benzo(a)pyrene.
e	LMW PAHs are defined as measured PAHs with a molecular weight below 200 a.u., including naphthalene, fluorene, anthracene, phenanthrene, acenaphthene, and 2-methylnaphthalene. TRV based on naphthalene.
a.u.	Atomic unit
COPC	Chemical of potential concern
DDT	Dichlorodiphenyltrichloroethane
DDT†	Sum of concentrations of 4,4'-dichlorodiphenyldichloroethane, 4,4'-dichlorodiphenyldichloroethene, and 4,4'-dichlorodiphenyltrichloroethane
EPA	U.S. Environmental Protection Agency
g	Gram
HMW	High molecular weight
LMW	Low molecular weight
mg/kg-day	Milligram per kilogram per day
NA	Not applicable
Navy	U.S. Department of Navy
NOAEL	No observed adverse effects level
NV	No value available
PAH	Polynuclear aromatic hydrocarbon
PCB	Polychlorinated biphenyl
SVOC	Semi-volatile organic chemical
TRV	Toxicity reference value
VOC	Volatile organic chemical

References:

Navy. 1998. "Interim Final Technical Memorandum, Development of Toxicity Reference Values for Conducting Ecological Risk Assessments at Naval Facilities in California." September.
 Sample, B.E., D.M. Opreko, and G.W. Suter, II. 1996. "Toxicological Benchmarks for Wildlife: 1996 Revision." ES/ER/TM-86/R3. Oak Ridge National Laboratory. Oak Ridge, Tennessee.

TABLE G-6: HIGH TOXICITY REFERENCE VALUES FOR THE ALAMEDA SONG SPARROW (MELOSPIZA MELODIA PUSILLULA)
 Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Ecological COPC	Literature-based high TRV (mg/kg-day)	Source of Study	Study	Endpoint	Body Weight of Study Subject (g)	Allometrically converted high TRV (mg/kg-day)
Metals						
Aluminum	1.00E+03	Sample and others (1996)	Carriere and others (1986)	Adverse reproduction in the ringed dove	155	6.63E+02
Antimony ^a	2.20E+01	Navy (1998)	Stanley, Jr., and others (1994)	Adverse effect on liver weight, glycogen depletion, number of days between pairing and first egg, whole-egg weight, duckling body and liver weights posthatching, duckling growth rate, and duckling production in mallards	1,172	9.74E+00
Arsenic	2.20E+01	Navy (1998)	Stanley, Jr., and others (1994)	Adverse effect on liver weight, glycogen depletion, number of days between pairing and first egg, whole-egg weight, duckling body and liver weights posthatching, duckling growth rate, and duckling production in mallards	1,172	9.74E+00
Barium	4.17E+01	Sample and others (1996)	Johnson and others (1960)	Adverse effects on mortality in 1-day-old chicks	121	2.91E+01
Beryllium	NV	NA	NA	NA	NA	NA
Cadmium	1.04E+01	Navy (1998)	Richardson and others (1974)	Decrease in body and testis weight, hematocrit and hemoglobin; changes in liver trace element stores; histological effects to duodenum, bone marrow, and adrenal; increase in heart weight in the Japanese quail	84	7.82E+00
Chromium	5.00E+00	Sample and others (1996)	Haaseltine and others, unpublished data	Reduction of duckling survival in black ducks	1,250	2.18E+00
Cobalt	1.59E+01 ^b	EPA (2003a)	Various	Physiological endpoints	407.5 ^b	8.69E+00
Copper	5.23E+01	Navy (1998)	Jensen and Maurice (1978)	Increase in gizzard erosion and feed to gain ratio, increase in relative gizzard and proventriculus weight in Cobb broiler chicks	409	2.86E+01
Lead	8.75E+00	Navy (1998)	Edens and Garlich (1983)	Decrease egg production in adult chickens	800	4.18E+00
Manganese	7.76E+02	Navy (1998)	Laskey and Edens (1985)	Effect on serum testosterone levels in Japanese quail	196.5	4.91E+02
Mercury	1.80E-01	Navy (1998)	Heinz and Locke (1976)	Reproductive effects in mallards	1,000	8.22E-02
Molybdenum	3.53E+01	Sample and others (1996)	Cain and Pafford (1981)	Reproductive effects in chickens	1,500	1.49E+01

TABLE G-6: HIGH TOXICITY REFERENCE VALUES FOR THE ALAMEDA SONG SPARROW (MELOSPIZA MELODIA PUSILLULA)

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Ecological COPC	Literature-based high TRV (mg/kg-day)	Source of Study	Study	Endpoint	Body Weight of Study Subject (g)	Allometrically converted high TRV (mg/kg-day)
Metals (Continued)						
Nickel	5.52E+01	Navy (1998)	Cain and Pafford (1981)	Decrease in length:weight ratio of humerus at 30 days in the mallard	580	2.81E+01
Silver	NV	NA	NA	NA	NA	NA
Vanadium	1.14E+02	Sample and others (1996)	White and Dieter (1978)	Adverse effects on mortality, body weight, and blood chemistry in mallards	1,170	5.05E+01
Zinc	1.72E+02	Navy (1998)	Gasaway and Buss (1972)	Decrease in body weight at 40 days, decrease in gonad weight, decrease in organ to body weight ratio (pancreas, adrenal, and kidney), decreases in pancreas and liver weight, leg paralysis, and diarrhea in mallard	955	7.93E+01
Pesticides						
DDT ^c	1.50E+00	Navy (1998)	Heath and others (1969), as cited in EPA 1995)	Reproductive effects in mallards	1,000	6.85E-01
PCBs^d						
Total PCBs	1.27E+00	Navy (1998)	Britton and Huston (1973)	Decrease in hatchability in chickens	1715.4	5.21E-01
PAHs						
HMW PAHs ^e	NV	NA	NA	NA	NA	NA
LMW PAHs ^f	NV	NA	NA	NA	NA	NA
SVOCs						
Bis(2-ethylhexyl)phthalate	1.10E+01	Sample and others (1996)	Peakall (1974)	Adverse reproductive effects in doves	155	7.30E+00
n-Nitroso-diphenylamine	NV	NA	NA	NA	NA	NA
Pentachlorophenol	NV	NA	NA	NA	NA	NA
VOCs						
2-Butanone	NV	NA	NA	NA	NA	NA
1,1,1-Trichloroethane	NV	NA	NA	NA	NA	NA
Acetone	NV	NA	NA	NA	NA	NA
Benzene	NV	NA	NA	NA	NA	NA
Carbon disulfide	NV	NA	NA	NA	NA	NA
Chloroform	NV	NA	NA	NA	NA	NA
Ethylbenzene	NV	NA	NA	NA	NA	NA
Toluene	NV	NA	NA	NA	NA	NA
Xylene	NV	NA	NA	NA	NA	NA

TABLE G-6: HIGH TOXICITY REFERENCE VALUES FOR THE ALAMEDA SONG SPARROW (MELOSPIZA MELODIA PUSILLULA)

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Notes:

- a Individual TRV not developed for antimony. Based on arsenic TRV.
- b High TRV and BW are the calculated geometric mean of all LOAELs and BWs presented in EPA (2003a and 2003b).
- c DDTt TRV based on 4,4'-DDT.
- d PCB TRV based on Aroclor-1254; individual TRVs not developed.
- e HMW PAHs are defined as measured PAHs with a molecular weight greater than 200 a.u. and include: benzo(a)fluorene, fluoranthene, benzo(k)fluoranthene, benzo(b)fluoranthene, benzo(b)fluorene, benzo(g,h,i)perylene, chrysene, benzo(a)anthracene, dibenzo(a,h)anthracene, indeno(1,2,3-cd)pyrene, pyrene, and benzo(a)pyrene. TRV based on benzo(a)pyrene.
- f LMW PAHs are defined as measured PAHs with a molecular weight below 200 a.u., including naphthalene, fluorene, anthracene, phenanthrene, acenaphthene, and 2-methylnaphthalene. TRV based on naphthalene.

a.u.	Atomic unit
COPC	Chemical of potential concern
DDT	Dichlorodiphenyltrichloroethane
DDTt	Sum of concentrations of 4,4'-dichlorodiphenyldichloroethane, 4,4'-dichlorodipenyldichloroethene, and 4,4'-dichlorodiphenyltrichloroethane
EPA	U.S. Environmental Protection Agency
g	Gram
HMW	High molecular weight
LMW	Low molecular weight
mg/kg-day	Milligram per kilogram per day
NA	Not applicable
Navy	U.S. Department of Navy
NOAEL	No observed adverse effects level
NV	No value available
PAH	Polynuclear aromatic hydrocarbon
PCB	Polychlorinated biphenyl
SVOC	Semi-volatile organic chemical
TRV	Toxicity reference value
VOC	Volatile organic chemical

References:

Navy. 1998. "Interim Final Technical Memorandum, Development of Toxicity Reference Values for Conducting Ecological Risk Assessments at Naval Facilities in California." September.

Sample, B.E., D.M. Opreko, and G.W. Suter, II. 1996. "Toxicological Benchmarks for Wildlife: 1996 Revision." ES/ER/TM-86/R3. Oak Ridge National Laboratory. Oak Ridge, Tennessee.

TABLE G-7: LOW TOXICITY REFERENCE VALUES FOR THE ALAMEDA SONG SPARROW (MELOSPIZA MELODIA PUSILLULA)

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Ecological COPC	Literature-based low TRV (mg/kg-day)	Source of Study	Study	Endpoint	Body Weight of Study Subject (g)	Allometrically converted low TRV (mg/kg-day)
Metals						
Aluminum	1.10E+02	Sample and others (1996)	Carriere and others (1986)	Adverse reproduction in the ringed dove	155	7.28E+01
Antimony ^a	5.50E+00	Navy (1998)	Stanley, Jr., and others (1994)	Adverse effect on liver weight, glycogen depletion, number of days between pairing and first egg, whole-egg weight, duckling body and liver weights posthatching, duckling growth rate, and duckling production in mallards	1,172	2.43E+00
Arsenic	5.50E+00	Navy (1998)	Stanley, Jr., and others (1994)	Adverse effect on liver weight, glycogen depletion, number of days between pairing and first egg, whole-egg weight, duckling body and liver weights posthatching, duckling growth rate, and duckling production in mallards	1,172	2.43E+00
Barium	2.08E+01	Sample and others (1996)	Johnson and others (1960)	Adverse effects on mortality in 1-day-old chicks	121	1.45E+01
Beryllium	NV	NA	NA	NA	NA	NA
Cadmium	8.00E-02	Navy (1998)	Cain and others (1983)	No observed adverse effect level for blood chemistry in mallards	798.5	3.82E-02
Chromium	1.00E+00	Sample and others (1996)	Haaseltine and others, unpublished data	Reduction of duckling survival in black ducks	1,250	4.37E-01
Cobalt	7.61E+00 ^b	EPA (2003a)	Various	Physiological endpoints	351 ^p	4.29E+00
Copper	2.30E+00	Navy (1998)	Norvell and others (1975)	Adverse effects on weight gain in boilers	639	1.15E+00
Lead	1.40E-02	Navy (1998)	Edens and others (1976)	Decrease in female egg production and plasma calcium; decrease in male testicular and liver weight in Japanese quail	103	1.01E-02
Lead (Alternate)	3.85E+00	Sample and others (1996)	Pattee (1984)	Adverse reproductive effects in the American kestrel	130	2.65E+00
Manganese	7.76E+01	Navy (1998)	Laskey and Edens (1985)	Effect on serum testosterone levels in Japanese quail	196.5	4.91E+01
Mercury	3.90E-02	Navy (1998)	Heinz (1974, 1975, 1976, and 1979)	Reproductive effects in mallards	1,000	1.78E-02
Molybdenum	3.50E+00	Sample and others (1996)	Cain and Pafford (1981)	Reproductive effects in chickens	1500	1.47E+00
Nickel	1.38E+00	Navy (1998)	Cain and Pafford (1981)	Adverse effects, such as tremors and edema, in toe and leg joints of mallards	613.75	6.95E-01
Silver	NV	NA	NA	NA	NA	NA

TABLE G-7: LOW TOXICITY REFERENCE VALUES FOR THE ALAMEDA SONG SPARROW (MELOSPIZA MELODIA PUSILLULA)

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Ecological COPC	Literature-based low TRV (mg/kg-day)	Source of Study	Study	Endpoint	Body Weight of Study Subject (g)	Allometrically converted low TRV (mg/kg-day)
Metals (Continued)						
Vanadium	1.14E+01	Sample and others (1996)	White and Dieter (1978)	Adverse effects on mortality, body weight, and blood chemistry in mallards	1,170	5.05E+00
Zinc	1.72E+01	Navy (1998)	Gasaway and Buss (1972)	Decrease in body weight at 40 days, decrease in gonad weight, decrease in organ to body weight ratio (pancreas, adrenal, and kidney), decreases in pancreas and liver weight, leg paralysis, and diarrhea in mallards	955	7.93E+00
Pesticides						
DDT ^c	9.00E-03	Navy (1998)	Anderson and others (1975, 1977, as cited in EPA 1995)	Reproductive effects in pelicans	3,500	3.20E-03
PCBs^d						
Total PCBs	9.00E-02	Navy (1998)	Platonow and Reinhart (1973)	Decrease in egg production in chickens	800	4.30E-02
PAHs						
HMW PAHs ^e	NV	NA	NA	NA	NA	NA
LMW PAHs ^f	NV	NA	NA	NA	NA	NA
SVOCs						
Bis(2-ethylhexyl)phthalate	1.10E+00	Sample and others (1996)	Peakall (1974)	Adverse reproductive effects in doves	155	7.30E-01
n-Nitroso-diphenylamine	NV	NA	NA	NA	NA	NA
Pentachlorophenol	NV	NA	NA	NA	NA	NA
VOCs						
2-Butanone	NV	NA	NA	NA	NA	NA
1,1,1-Trichloroethane	NV	NA	NA	NA	NA	NA
Acetone	NV	NA	NA	NA	NA	NA
Benzene	NV	NA	NA	NA	NA	NA
Carbon disulfide	NV	NA	NA	NA	NA	NA
Chloroform	NV	NA	NA	NA	NA	NA
Ethylbenzene	NV	NA	NA	NA	NA	NA
Toluene	NV	NA	NA	NA	NA	NA
Xylene	NV	NA	NA	NA	NA	NA

TABLE G-7: LOW TOXICITY REFERENCE VALUES FOR THE ALAMEDA SONG SPARROW (MELOSPIZA MELODIA PUSILLULA)

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Notes:

a	Individual TRV not developed for antimony. Based on arsenic TRV.
b	Low TRV presented in EPA (2003) and BW is the calculated geometric mean of BWs used in developing TRV as presented in EPA (2003).
c	DDTt TRV based on 4,4'-DDT; individual TRVs not developed.
d	PCB TRV based on Aroclor-1254; individual TRVs not developed.
e	HMW PAHs are defined as measured PAHs with a molecular weight greater than 200 a.u. and include: benzo(a)fluorene, fluoranthene, benzo(k)fluoranthene, benzo(b)fluoranthene, benzo(b)fluorene, benzo(g,h,i)perylene, chrysene, benzo(a)anthracene, dibenzo(a,h)anthracene, indeno(1,2,3-cd)pyrene, pyrene, and benzo(a)pyrene. TRV based on benzo(a)pyrene.
f	LMW PAHs are defined as measured PAHs with a molecular weight below 200 a.u., including naphthalene, fluorene, anthracene, phenanthrene, acenaphthene, and 2-methylnaphthalene. TRV based on naphthalene.

a.u.	Atomic unit
COPC	Chemical of potential concern
DDT	Dichlorodiphenyltrichloroethane
DDTt	Sum of concentrations of 4,4'-dichlorodiphenyldichloroethane, 4,4'-dichlorodiphenyldichloroethene, and 4,4'-dichlorodiphenyltrichloroethane
EPA	U.S. Environmental Protection Agency
g	Gram
HMW	High molecular weight
LMW	Low molecular weight
mg/kg-day	Milligram per kilogram per day
NA	Not applicable
Navy	U.S. Department of Navy
NOAEL	No observed adverse effects level
NV	No value available
PAH	Polynuclear aromatic hydrocarbon
PCB	Polychlorinated biphenyl
SVOC	Semivolatile organic chemical
TRV	Toxicity reference value
VOC	Volatile organic chemical

References:

- Navy. 1998. "Interim Final Technical Memorandum, Development of Toxicity Reference Values for Conducting Ecological Risk Assessments at Naval Facilities in California." September.
- Sample, B.E., D.M. Opresko, and G.W. Suter, II. 1996. "Toxicological Benchmarks for Wildlife: 1996 Revision." ES/ER/TM-86/R3. Oak Ridge National Laboratory. Oak Ridge, Tennessee.

TABLE G-8: HIGH TOXICITY REFERENCE VALUES FOR THE AMERICAN ROBIN (TURDUS MIGRATORIUS)

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Ecological COPC	Literature-based high TRV (mg/kg-day)	Source of Study	Study	Endpoint	Body Weight of Study Subject (g)	Allometrically converted high TRV (mg/kg-day)
Metals						
Aluminum	1.00E+03	Sample and others (1996)	Carriere and others (1986)	Adverse reproduction in the ringed dove	155	8.78E+02
Antimony ^a	2.20E+01	Navy (1998)	Stanley, Jr., and others (1994)	Adverse effect on liver weight, glycogen depletion, number of days between pairing and first egg, whole-egg weight, duckling body and liver weights posthatching, duckling growth rate, and duckling production in mallards	1,172	1.29E+01
Arsenic	2.20E+01	Navy (1998)	Stanley, Jr., and others (1994)	Adverse effect on liver weight, glycogen depletion, number of days between pairing and first egg, whole-egg weight, duckling body and liver weights posthatching, duckling growth rate, and duckling production in mallards	1,172	1.29E+01
Barium	4.17E+01	Sample and others (1996)	Johnson and others (1960)	Adverse effects on mortality in 1-day-old chicks	121	3.85E+01
Beryllium	NV	NA	NA	NA	NA	NA
Cadmium	1.04E+01	Navy (1998)	Richardson and others (1974)	Decrease in body and testis weight, hematocrit and hemoglobin; changes in liver trace element stores; histological effects to duodenum, bone marrow, and adrenal; increase in heart weight in the Japanese quail	84	1.04E+01
Chromium	5.00E+00	Sample and others (1996)	Haaseltine and others, unpublished data	Reduction of duckling survival in black ducks	1,250	2.89E+00
Cobalt	1.59E+01 ^b	EPA (2003a)	Various	Physiological endpoints	407.5 ^b	1.15E+01
Copper	5.23E+01	Navy (1998)	Jensen and Maurice (1978)	Increase in gizzard erosion and feed to gain ratio, increase in relative gizzard and proventriculus weight in Cobb broiler chicks	409	3.78E+01
Lead	8.75E+00	Navy (1998)	Edens and Garlich (1983)	Decrease egg production in adult chickens	800	5.53E+00
Manganese	7.76E+02	Navy (1998)	Laskey and Edens (1985)	Effect on serum testosterone levels in Japanese quail	196.5	6.50E+02
Mercury	1.80E-01	Navy (1998)	Heinz and Locke (1976)	Reproductive effects in mallards	1,000	1.09E-01
Molybdenum	3.53E+01	Sample and others (1996)	Cain and Pafford (1981)	Reproductive effects in chickens	1,500	1.97E+01
Nickel	5.52E+01	Navy (1998)	Cain and Pafford (1981)	Decrease in length:weight ratio of humerus at 30 days in the mallard	580	3.72E+01
Silver	NV	NA	NA	NA	NA	NA

TABLE G-8: HIGH TOXICITY REFERENCE VALUES FOR THE AMERICAN ROBIN (TURDUS MIGRATORIUS)

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Ecological COPC	Literature-based high TRV (mg/kg-day)	Source of Study	Study	Endpoint	Body Weight of Study Subject (g)	Allometrically converted high TRV (mg/kg-day)
Metals (Continued)						
Vanadium	1.14E+02	Sample and others (1996)	White and Dieter (1978)	Adverse effects on mortality, body weight, and blood chemistry in mallards	1,170	6.68E+01
Zinc	1.72E+02	Navy (1998)	Gasaway and Buss (1972)	Decrease in body weight at 40 days, decrease in gonad weight, decrease in organ to body weight ratio (pancreas, adrenal, and kidney), decreases in pancreas and liver weight, leg paralysis, and diarrhea in mallard	955	1.05E+02
Pesticides						
DDT ^c	1.50E+00	Navy (1998)	Heath and others (1969), as cited in EPA 1995)	Reproductive effects in mallards	1,000	9.07E-01
PCBs^d						
Total PCBs	1.27E+00	Navy (1998)	Britton and Huston (1973)	Decrease in hatchability in chickens	1715.4	6.90E-01
PAHs						
HMW PAHs ^e	NV	NA	NA	NA	NA	NA
LMW PAHs ^f	NV	NA	NA	NA	NA	NA
SVOCs						
Bis(2-ethylhexyl)phthalate	1.10E+01	Sample and others (1996)	Peakall (1974)	Adverse reproductive effects in doves	155	9.66E+00
n-Nitroso-diphenylamine	NV	NA	NA	NA	NA	NA
Pentachlorophenol	NV	NA	NA	NA	NA	NA
VOCs						
2-Butanone	NV	NA	NA	NA	NA	NA
1,1,1-Trichloroethane	NV	NA	NA	NA	NA	NA
Acetone	NV	NA	NA	NA	NA	NA
Benzene	NV	NA	NA	NA	NA	NA
Carbon disulfide	NV	NA	NA	NA	NA	NA
Chloroform	NV	NA	NA	NA	NA	NA
Ethylbenzene	NV	NA	NA	NA	NA	NA
Toluene	NV	NA	NA	NA	NA	NA
Xylene	NV	NA	NA	NA	NA	NA

Notes:

- a Individual TRV not developed for antimony. Based on arsenic TRV.
- b TRV of these compounds based on 4,4'-DDT; individual TRV not developed.
- c DDT^t TRV based on 4,4'-DDT; individual TRVs not developed.
- d PCB TRV based on Aroclor-1254; individual TRVs not developed.

TABLE G-8: HIGH TOXICITY REFERENCE VALUES FOR THE AMERICAN ROBIN (TURDUS MIGRATORIUS)

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Notes (Continued):

e	HMW PAHs are defined as measured PAHs with a molecular weight greater than 200 a.u. and include: benzo(a)fluorene, fluoranthene, benzo(k)fluoranthene, benzo(b)fluoranthene, benzo(b)fluorene, benzo(g,h,i)perylene, chrysene, benzo(a)anthracene, dibenzo(a,h)anthracene, indeno(1,2,3-cd)pyrene, pyrene, and benzo(a)pyrene. TRV based on benzo(a)pyrene.
f	LMW PAHs are defined as measured PAHs with a molecular weight below 200 a.u., including naphthalene, fluorene, anthracene, phenanthrene, acenaphthene, and 2-methylnaphthalene. TRV based on naphthalene.
a.u.	Atomic unit
COPC	Chemical of potential concern
DDT	Dichlorodiphenyltrichloroethane
DDT†	Sum of concentrations of 4,4'-dichlorodiphenyldichloroethane, 4,4'-dichlorodiphenyldichloroethene, and 4,4'-dichlorodiphenyltrichloroethane
EPA	U.S. Environmental Protection Agency
g	Gram
HMW	High molecular weight
LMW	Low molecular weight
mg/kg-day	Milligram per kilogram per day
NA	Not applicable
Navy	U.S. Department of Navy
NOAEL	No observed adverse effects level
NV	No value available
PAH	Polynuclear aromatic hydrocarbon
PCB	Polychlorinated biphenyl
SVOC	Semivolatile organic chemical
TRV	Toxicity reference value
VOC	Volatile organic chemical

References:

Navy. 1998. "Interim Final Technical Memorandum, Development of Toxicity Reference Values for Conducting Ecological Risk Assessments at Naval Facilities in California." September.
 Sample, B.E., D.M. Opresko, and G.W. Suter, II. 1996. "Toxicological Benchmarks for Wildlife: 1996 Revision." ES/ER/TM-86/R3. Oak Ridge National Laboratory. Oak Ridge, Tennessee.

TABLE G-9: LOW TOXICITY REFERENCE VALUES FOR THE AMERICAN ROBIN (TURDUS MIGRATORIUS)

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Ecological COPC	Literature-based low TRV (mg/kg-day)	Source of Study	Study	Endpoint	Body Weight of Study Subject (g)	Allometrically converted low TRV (mg/kg-day)
Metals						
Aluminum	1.10E+02	Sample and others (1996)	Carriere and others (1986)	Adverse reproduction in the ringed dove	155	9.63E+01
Antimony ^a	5.50E+00	Navy (1998)	Stanley, Jr., and others (1994)	Adverse effect on liver weight, glycogen depletion, number of days between pairing and first egg, whole-egg weight, duckling body and liver weights posthatching, duckling growth rate, and duckling production in mallards	1,172	3.22E+00
Arsenic	5.50E+00	Navy (1998)	Stanley, Jr., and others (1994)	Adverse effect on liver weight, glycogen depletion, number of days between pairing and first egg, whole-egg weight, duckling body and liver weights posthatching, duckling growth rate, and duckling production in mallards	1,172	3.22E+00
Barium	2.08E+01	Sample and others (1996)	Johnson and others (1960)	Adverse effects on mortality in 1-day-old chicks	121	1.92E+01
Beryllium	NV	NA	NA	NA	NA	NA
Cadmium	8.00E-02	Navy (1998)	Cain and others (1983)	No observed adverse effect level for blood chemistry in mallards	798.5	5.06E-02
Chromium	1.00E+00	Sample and others (1996)	Haaseltine and others, unpublished data	Reduction of duckling survival in black ducks	1,250	5.79E-01
Cobalt	7.61E+00 ^b	EPA (2003a)	Various	Physiological endpoints	351 ^b	5.68E+00
Copper	2.30E+00	Navy (1998)	Norvell and others (1975)	Adverse effects on weight gain in boilers	639	1.52E+00
Lead	1.40E-02	Navy (1998)	Edens and others (1976)	Decrease in female egg production and plasma calcium; decrease in male testicular and liver weight in Japanese quail	103	1.33E-02
Lead (Alternate)	3.85E+00	Sample and others (1996)	Pattee (1984)	Adverse reproductive effects in the American kestrel	130	3.50E+00
Manganese	7.76E+01	Navy (1998)	Laskey and Edens (1985)	Effect on serum testosterone levels in Japanese quail	196.5	6.50E+01
Mercury	3.90E-02	Navy (1998)	Heinz (1974, 1975, 1976, and 1979)	Reproductive effects in mallards	1,000	2.36E-02
Molybdenum	3.50E+00	Sample and others (1996)	Cain and Pafford (1981)	Reproductive effects in chickens	1500	1.95E+00
Nickel	1.38E+00	Navy (1998)	Cain and Pafford (1981)	Adverse effects, such as tremors and edema, in toe and leg joints of mallards	613.75	9.20E-01
Silver	NV	NA	NA	NA	NA	NA

TABLE G-9: LOW TOXICITY REFERENCE VALUES FOR THE AMERICAN ROBIN (TURDUS MIGRATORIUS)

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Ecological COPC	Literature-based low TRV (mg/kg-day)	Source of Study	Study	Endpoint	Body Weight of Study Subject (g)	Allometrically converted low TRV (mg/kg-day)
Metals (Continued)						
Vanadium	1.14E+01	Sample and others (1996)	White and Dieter (1978)	Adverse effects on mortality, body weight, and blood chemistry in mallards	1,170	6.68E+00
Zinc	1.72E+01	Navy (1998)	Gasaway and Buss (1972)	Decrease in body weight at 40 days, decrease in gonad weight, decrease in organ to body weight ratio (pancreas, adrenal, and kidney), decreases in pancreas and liver weight, leg paralysis, and diarrhea in mallards	955	1.05E+01
Pesticides						
DDT ^c	9.00E-03	Navy (1998)	Anderson and others (1975, 1977, as cited in EPA 1995)	Reproductive effects in pelicans	3,500	4.24E-03
PCBs^d						
Total PCBs	9.00E-02	Navy (1998)	Platonow and Reinhart (1973)	Decrease in egg production in chickens	800	5.69E-02
PAHs						
HMW PAHs ^e	NV	NA	NA	NA	NA	NA
LMW PAHs ^f	NV	NA	NA	NA	NA	NA
SVOCs						
Bis(2-ethylhexyl)phthalate	1.10E+00	Sample and others (1996)	Peakall (1974)	Adverse reproductive effects in doves	155	9.66E-01
n-Nitroso-diphenylamine	NV	NA	NA	NA	NA	NA
Pentachlorophenol	NV	NA	NA	NA	NA	NA
VOCs						
2-Butanone	NV	NA	NA	NA	NA	NA
1,1,1-Trichloroethane	NV	NA	NA	NA	NA	NA
Acetone	NV	NA	NA	NA	NA	NA
Benzene	NV	NA	NA	NA	NA	NA
Carbon disulfide	NV	NA	NA	NA	NA	NA
Chloroform	NV	NA	NA	NA	NA	NA
Ethylbenzene	NV	NA	NA	NA	NA	NA
Toluene	NV	NA	NA	NA	NA	NA
Xylene	NV	NA	NA	NA	NA	NA

Notes:

^a Individual TRV not developed for antimony. Based on arsenic TRV.

^b TRV of these compounds based on 4,4'-DDT; individual TRV not developed.

TABLE G-9: LOW TOXICITY REFERENCE VALUES FOR THE AMERICAN ROBIN (TURDUS MIGRATORIUS)

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Notes (Continued):

- c DDTt TRV based on 4,4'-DDT; individual TRVs not developed.
- d PCB TRV based on Aroclor-1254; individual TRVs not developed.
- e HMW PAHs are defined as measured PAHs with a molecular weight greater than 200 a.u. and include: benzo(a)fluorene, fluoranthene, benzo(k)fluoranthene, benzo(b)fluoranthene, benzo(b)fluorene, benzo(g,h,i)perylene, chrysene, benzo(a)anthracene, dibenzo(a,h)anthracene, indeno(1,2,3-cd)pyrene, pyrene, and benzo(a)pyrene. TRV based on benzo(a)pyrene.
- f LMW PAHs are defined as measured PAHs with a molecular weight below 200 a.u., including naphthalene, fluorene, anthracene, phenanthrene, acenaphthene, and 2-methylnaphthalene. TRV based on naphthalene.

a.u.	Atomic unit
COPC	Chemical of potential concern
DDT	Dichlorodiphenyltrichloroethane
DDTt	Sum of concentrations of 4,4'-dichlorodiphenyldichloroethane, 4,4'-dichlorodiphenyldichloroethene, and 4,4'-dichlorodiphenyltrichloroethane
EPA	U.S. Environmental Protection Agency
g	Gram
HMW	High molecular weight
LMW	Low molecular weight
mg/kg-day	Milligram per kilogram per day
NA	Not applicable
Navy	U.S. Department of Navy
NOAEL	No observed adverse effects level
NV	No value available
PAH	Polynuclear aromatic hydrocarbon
PCB	Polychlorinated biphenyl
SVOC	Semivolatile organic chemical
TRV	Toxicity reference value
VOC	Volatile organic chemical

References: Navy. 1998. "Interim Final Technical Memorandum, Development of Toxicity Reference Values for Conducting Ecological Risk Assessments at Naval Facilities in California." September.

Sample, B.E., D.M. Opresko, and G.W. Suter, II. 1996. "Toxicological Benchmarks for Wildlife: 1996 Revision." ES/ER/TM-86/R3. Oak Ridge National Laboratory. Oak Ridge, Tennessee.

TABLE G-10: HIGH TOXICITY REFERENCE VALUES FOR THE RED-TAILED HAWK (BUTEO JAMAICENSIS)

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Ecological COPC	Literature-based high TRV (mg/kg-day)	Source of Study	Study	Endpoint	Body Weight of Study Subject (g)	Allometrically converted high TRV (mg/kg-day)
Metals						
Aluminum	1.00E+03	Sample and others (1996)	Carriere and others (1986)	Adverse reproduction in the ringed dove	155	1.49E+03
Antimony ^a	2.20E+01	Navy (1998)	Stanley, Jr., and others (1994)	Adverse effect on liver weight, glycogen depletion, number of days between pairing and first egg, whole-egg weight, duckling body and liver weights posthatching, duckling growth rate, and duckling production in mallards	1,172	2.18E+01
Arsenic	2.20E+01	Navy (1998)	Stanley, Jr., and others (1994)	Adverse effect on liver weight, glycogen depletion, number of days between pairing and first egg, whole-egg weight, duckling body and liver weights posthatching, duckling growth rate, and duckling production in mallards	1,172	2.18E+01
Barium	4.17E+01	Sample and others (1996)	Johnson and others (1960)	Adverse effects on mortality in 1-day-old chicks	121	6.52E+01
Beryllium	NV	NA	NA	NA	NA	NA
Cadmium	1.04E+01	Navy (1998)	Richardson and others (1974)	Decrease in body and testis weight, hematocrit and hemoglobin; changes in liver trace element stores; histological effects to duodenum, bone marrow, and adrenal; increase in heart weight in the Japanese quail	84	1.75E+01
Chromium	5.00E+00	Sample and others (1996)	Haaseltine and others, unpublished data	Reduction of duckling survival in black ducks	1,250	4.90E+00
Cobalt	1.59E+01 ^b	EPA (2003a)	Various	Physiological endpoints	407.5 ^b	1.95E+01
Copper	5.23E+01	Navy (1998)	Jensen and Maurice (1978)	Increase in gizzard erosion and feed to gain ratio, increase in relative gizzard and proventriculus weight in Cobb broiler chicks	409	6.41E+01
Lead	8.75E+00	Navy (1998)	Edens and Garlich (1983)	Decrease egg production in adult chickens	800	9.38E+00
Manganese	7.76E+02	Navy (1998)	Laskey and Edens (1985)	Effect on serum testosterone levels in Japanese quail	196.5	1.10E+03
Mercury	1.80E-01	Navy (1998)	Heinz and Locke (1976)	Reproductive effects in mallards	1,000	1.84E-01
Molybdenum	3.53E+01	Sample and others (1996)	Cain and Pafford (1981)	Reproductive effects in chickens	1,500	3.34E+01
Nickel	5.53E+01	Navy (1998)	Cain and Pafford (1981)	Decrease in length:weight ratio of humerus at 30 days in the mallard	580	6.31E+01
Silver	NV	NA	NA	NA	NA	NA

TABLE G-10: HIGH TOXICITY REFERENCE VALUES FOR THE RED-TAILED HAWK (BUTEO JAMAICENSIS)

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Ecological COPC	Literature-based high TRV (mg/kg-day)	Source of Study	Study	Endpoint	Body Weight of Study Subject (g)	Allometrically converted high TRV (mg/kg-day)
Metals (Continued)						
Vanadium	1.14E+02	Sample and others (1996)	White and Dieter (1978)	Adverse effects on mortality, body weight, and blood chemistry in mallards	1,170	1.13E+02
Zinc	1.72E+02	Navy (1998)	Gasaway and Buss (1972)	Decrease in body weight at 40 days, decrease in gonad weight, decrease in organ to body weight ratio (pancreas, adrenal, and kidney), decreases in pancreas and liver weight, leg paralysis, and diarrhea in mallard ducks	955	1.78E+02
Pesticides						
DDT ^c	1.50E+00	Navy (1998)	Heath and others (1969), as cited in EPA 1995)	Reproductive effects in mallards	1,000	1.54E+00
PCBs^d						
Total PCBs	1.27E+00	Navy (1998)	Britton and Huston (1973)	Decrease in hatchability in chickens	1715.4	1.17E+00
PAHs						
HMW PAHs ^e	NV	NA	NA	NA	NA	NA
LMW PAHs ^f	NV	NA	NA	NA	NA	NA
SVOCs						
Bis(2-ethylhexyl)phthalate	1.10E+01	Sample and others (1996)	Peakall (1974)	Adverse reproductive effects in doves	155	1.64E+01
n-Nitroso-diphenylamine	NV	NA	NA	NA	NA	NA
Pentachlorophenol	NV	NA	NA	NA	NA	NA
VOCs						
2-Butanone	NV	NA	NA	NA	NA	NA
1,1,1-Trichloroethane	NV	NA	NA	NA	NA	NA
Acetone	NV	NA	NA	NA	NA	NA
Benzene	NV	NA	NA	NA	NA	NA
Carbon disulfide	NV	NA	NA	NA	NA	NA
Chloroform	NV	NA	NA	NA	NA	NA
Ethylbenzene	NV	NA	NA	NA	NA	NA
Toluene	NV	NA	NA	NA	NA	NA
Xylene	NV	NA	NA	NA	NA	NA

Notes:

^a Individual TRV not developed for antimony. Based on arsenic TRV.

^b TRV of these compounds based on 4,4'-DDT; individual TRV not developed.

^c DDT TRV based on 4,4'-DDT; individual TRVs not developed.

^d PCB TRV based on Aroclor-1254; individual TRVs not developed.

TABLE G-10: HIGH TOXICITY REFERENCE VALUES FOR THE RED-TAILED HAWK (BUTEO JAMAICENSIS)

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Notes (Continued):

- e HMW PAHs are defined as measured PAHs with a molecular weight greater than 200 a.u. and include: benzo(a)fluorene, fluoranthene, benzo(k)fluoranthene, benzo(b)fluoranthene, benzo(b)fluorene, benzo(g,h,i)perylene, chrysene, benzo(a)anthracene, dibenzo(a,h)anthracene, indeno(1,2,3-cd)pyrene, pyrene, and benzo(a)pyrene. TRV based on benzo(a)pyrene.
- f LMW PAHs are defined as measured PAHs with a molecular weight below 200 a.u., including naphthalene, fluorene, anthracene, phenanthrene, acenaphthene, and 2-methylnaphthalene. TRV based on naphthalene.

a.u.	Atomic unit
COPC	Chemical of potential concern
DDT	Dichlorodiphenyltrichloroethane
DDTt	Sum of concentrations of 4,4'-dichlorodiphenyldichloroethane, 4,4'-dichlorodiphenyldichloroethene, and 4,4'-dichlorodiphenyltrichloroethane
EPA	U.S. Environmental Protection Agency
g	Gram
HMW	High molecular weight
LMW	Low molecular weight
mg/kg-day	Milligram per kilogram per day
NA	Not applicable
Navy	U.S. Department of Navy
NOAEL	No observed adverse effects level
NV	No value available
PAH	Polynuclear aromatic hydrocarbon
PCB	Polychlorinated biphenyl
SVOC	Semivolatile organic chemical
TRV	Toxicity reference value
VOC	Volatile organic chemical

References:

- Navy. 1998. "Interim Final Technical Memorandum, Development of Toxicity Reference Values for Conducting Ecological Risk Assessments at Naval Facilities in California." September.
- Sample, B.E., D.M. Opresko, and G.W. Suter, II. 1996. "Toxicological Benchmarks for Wildlife: 1996 Revision." ES/ER/TM-86/R3. Oak Ridge National Laboratory. Oak Ridge, Tennessee.

TABLE G-11: LOW TOXICITY REFERENCE VALUES FOR THE RED-TAILED HAWK (BUTEO JAMAICENSIS)

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Ecological COPC	Literature-based low TRV (mg/kg-day)	Source of Study	Study	Endpoint	Body Weight of Study Subject (g)	Allometrically converted low TRV (mg/kg-day)
Metals						
Aluminum	1.10E+02	Sample and others (1996)	Carriere and others (1986)	Adverse reproduction in the ringed dove	155	1.63E+02
Antimony ^a	5.50E+00	Navy (1998)	Stanley, Jr., and others (1994)	Adverse effect on liver weight, glycogen depletion, number of days between pairing and first egg, whole-egg weight, duckling body and liver weights posthatching, duckling growth rate, and duckling production in mallards	1,172	5.46E+00
Arsenic	5.50E+00	Navy (1998)	Stanley, Jr., and others (1994)	Adverse effect on liver weight, glycogen depletion, number of days between pairing and first egg, whole-egg weight, duckling body and liver weights posthatching, duckling growth rate, and duckling production in mallards	1,172	5.46E+00
Barium	2.08E+01	Sample and others (1996)	Johnson and others (1960)	Adverse effects on mortality in 1-day-old chicks	121	3.25E+01
Beryllium	NV	NA	NA	NA	NA	NA
Cadmium	8.00E-02	Navy (1998)	Cain and others (1983)	No observed adverse effect level for blood chemistry in mallards	798.5	8.58E-02
Chromium	1.00E+00	Sample and others (1996)	Haaseltine and others, unpublished data	Reduction of duckling survival in black ducks	1,250	9.80E-01
Cobalt	7.61E+00 ^b	EPA (2003a)	Various	Physiological endpoints	351 ^b	9.61E+00
Copper	2.30E+00	Navy (1998)	Norvell and others (1975)	Adverse effects on weight gain in boilers	639	2.58E+00
Lead	1.40E-02	Navy (1998)	Edens and others (1976)	Decrease in female egg production and plasma calcium; decrease in male testicular and liver weight in Japanese quail	103	2.26E-02
Lead (Alternate)	3.85E+00	Sample and others (1996)	Pattee (1984)	Adverse reproductive effects in the American kestrel	130	5.93E+00
Manganese	7.76E+01	Navy (1998)	Laskey and Edens (1985)	Effect on serum testosterone levels in Japanese quail	196.5	1.10E+02
Mercury	3.90E-02	Navy (1998)	Heinz (1974, 1975, 1976, and 1979)	Reproductive effects in mallards	1,000	4.00E-02
Molybdenum	3.50E+00	Sample and others (1996)	Cain and Pafford (1981)	Reproductive effects in chickens	1,500	3.31E+00
Nickel	1.38E+00	Navy (1998)	Cain and Pafford (1981)	Adverse effects, such as tremors and edema, in toe and leg joints of mallards	613.75	1.56E+00
Silver	NV	NA	NA	NA	NA	NA

TABLE G-11: LOW TOXICITY REFERENCE VALUES FOR THE RED-TAILED HAWK (BUTEO JAMAICENSIS)

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Ecological COPC	Literature-based low TRV (mg/kg-day)	Source of Study	Study	Endpoint	Body Weight of Study Subject (g)	Allometrically converted low TRV (mg/kg-day)
Metals (Continued)						
Vanadium	1.14E+01	Sample and others (1996)	White and Dieter (1978)	Adverse effects on mortality, body weight, and blood chemistry in mallards	1,170	1.13E+01
Zinc	1.72E+01	Navy (1998)	Gasaway and Buss (1972)	Decrease in body weight at 40 days, decrease in gonad weight, decrease in organ to body weight ratio (pancreas, adrenal, and kidney), decreases in pancreas and liver weight, leg paralysis, and diarrhea in mallards	955	1.78E+01
Pesticides						
DDT ^c	9.00E-03	Navy (1998)	Anderson and others (1975, 1977, as cited in EPA 1995)	Reproductive effects in pelicans	3,500	7.18E-03
PCBs^d						
Total PCBs	9.00E-02	Navy (1998)	Platonow and Reinhart (1973)	Decrease in egg production in chickens	800	9.64E-02
PAHs						
HMW PAHs ^e	NV	NA	NA	NA	NA	NA
LMW PAHs ^f	NV	NA	NA	NA	NA	NA
SVOCs						
Bis(2-ethylhexyl)phthalate	1.10E+00	Sample and others (1996)	Peakall (1974)	Adverse reproductive effects in doves	155	1.64E+00
n-Nitroso-diphenylamine	NV	NA	NA	NA	NA	NA
Pentachlorophenol	NV	NA	NA	NA	NA	NA
VOCs						
2-Butanone	NV	NA	NA	NA	NA	NA
1,1,1-Trichloroethane	NV	NA	NA	NA	NA	NA
Acetone	NV	NA	NA	NA	NA	NA
Benzene	NV	NA	NA	NA	NA	NA
Carbon disulfide	NV	NA	NA	NA	NA	NA
Chloroform	NV	NA	NA	NA	NA	NA
Ethylbenzene	NV	NA	NA	NA	NA	NA
Toluene	NV	NA	NA	NA	NA	NA
Xylene	NV	NA	NA	NA	NA	NA

Notes:

^a Individual TRV not developed for antimony. Based on arsenic TRV.

^b TRV of these compounds based on 4,4'-DDT; individual TRV not developed.

TABLE G-11: LOW TOXICITY REFERENCE VALUES FOR THE RED-TAILED HAWK (BUTEO JAMAICENSIS)

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Notes (Continued):

- c DDTt TRV based on 4,4'-DDT; individual TRVs not developed.
- d PCB TRV based on Aroclor-1254; individual TRVs not developed.
- e HMW PAHs are defined as measured PAHs with a molecular weight greater than 200 a.u. and include: benzo(a)fluorene, fluoranthene, benzo(k)fluoranthene, benzo(b)fluoranthene, benzo(b)fluorene, benzo(g,h,i)perylene, chrysene, benzo(a)anthracene, dibenzo(a,h)anthracene, indeno(1,2,3-cd)pyrene, pyrene, and benzo(a)pyrene. TRV based on benzo(a)pyrene.
- f LMW PAHs are defined as measured PAHs with a molecular weight below 200 a.u., including naphthalene, fluorene, anthracene, phenanthrene, acenaphthene, and 2-methylnaphthalene. TRV based on naphthalene.

a.u.	Atomic unit
COPC	Chemical of potential concern
DDT	Dichlorodiphenyltrichloroethane
DDTt	Sum of concentrations of 4,4'-dichlorodiphenyldichloroethane, 4,4'-dichlorodiphenyldichloroethene, and 4,4'-dichlorodiphenyltrichloroethane
EPA	U.S. Environmental Protection Agency
g	Gram
HMW	High molecular weight
LMW	Low molecular weight
mg/kg-day	Milligram per kilogram per day
NA	Not applicable
Navy	U.S. Department of Navy
NOAEL	No observed adverse effects level
NV	No value available
PAH	Polynuclear aromatic hydrocarbon
PCB	Polychlorinated biphenyl
SVOC	Semivolatile organic chemical
TRV	Toxicity reference value
VOC	Volatile organic chemical

References:

Navy. 1998. "Interim Final Technical Memorandum, Development of Toxicity Reference Values for Conducting Ecological Risk Assessments at Naval Facilities in California." September.

Sample, B.E., D.M. Opresko, and G.W. Suter, II. 1996. "Toxicological Benchmarks for Wildlife: 1996 Revision." ES/ER/TM-86/R3. Oak Ridge National Laboratory. Oak Ridge, Tennessee.

TABLE G-12: ASSESSMENT AND ASSOCIATED MEASUREMENT ENDPOINTS
 Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Assessment Endpoint	Associated Measurement Endpoint
Sufficient Rates of Survival, Growth, and Reproduction to Sustain Small Mammal Populations Typical to the Area	Reproductive or physiological impacts to the California ground squirrel (<i>Citellus beecheyi</i>), as indicated by HQs developed based on both high (LOAEL-based) and low (NOAEL-based) TRVs
Sufficient Rates of Survival, Growth, and Reproduction to Sustain Passerine Populations Typical to the Area	Reproductive or physiological impacts to the Alameda song sparrow (<i>Melospiz melodia pusillula</i>) and the American robin (<i>Turdus migratorius</i>), as indicated by HQs developed based on both high (LOAEL-based) and low (NOAEL-based) TRVs
Sufficient Rates of Survival, Growth, and Reproduction to Sustain Raptor Populations Typical to the Area	Reproductive or physiological impacts to the red-tailed hawk (<i>Buteo jamaicensis</i>), as indicated by HQs developed based on both high (LOAEL-based) and low (NOAEL-based) TRVs

Notes:

HQ	Hazard quotient
LOAEL	Lowest observed adverse effects level
NOAEL	No observed adverse effects level
TRV	Toxicity reference value

TABLE G-13: PLANT AND INVERTEBRATE BIOCONCENTRATION FACTORS FOR ECOLOGICAL CHEMICALS OF POTENTIAL CONCERN AT OU-2B SITES

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Ecological COPCs	LogK _{ow}	BCFs _{soil-to-invert} ^a	BCFs _{soil-to-plant} ^a
Metals			
Aluminum	NA	0.22	0.004
Antimony	NA	0.22	0.2
Arsenic	NA	0.11	0.036
Barium	NA	0.22	0.15
Beryllium	NA	0.22	0.01
Cadmium	NA	0.96	0.364
Chromium	NA	0.01	0.0075
Cobalt	NA	0.22	0.02
Copper	NA	0.04	0.4
Lead	NA	0.03	0.045
Manganese	NA	0.22	0.25
Mercury ^b	NA	1.1398	0.051
Molybdenum	NA	0.22	0.25
Nickel	NA	0.02	0.032
Silver	NA	0.22	0.4
Vanadium	NA	0.22	0.0055
Zinc	NA	0.56	0.0000000000012
Pesticides and PCBs			
DDT	6.51	1.26	0.00937
Total PCBs ^d	6.8	1.13 ^d	0.01 ^d
PAHs and SVOCs			
HMW and LMW PAHs ^e	3.86	0.063	0.00991
Bis(2-ethylhexyl)phthalate	7.6	1,309	0.038
n-Nitroso-diphenylamine	3.13	26.15	0.601
Pentachlorophenol	5.12	1,034	0.0449
VOCs			
2-Butanone	0.29	0.124	26.33
1,1,1-Trichloroethane	2.49	7.82	1.41
Acetone	-0.24	0.05	52
Benzene	2.13	3.97	2.27
Carbon Disulfide	1.94	2.77	2.93
Chloroform	1.97	2.93	2.81
Ethylbenzene	3.15	27.2	0.585
Toluene	2.73	12.3	1.02
Xylene	3.2	29.84	0.548

Notes:

- ^a Based on values presented in EPA 1999, or regression equations published in EPA 1999.
- ^b Based on BCF for total mercury, which assumed 87 percent consisting of divalent mercury and 13 percent consisting of methylmercury (EPA 1999).
- ^c Based on BCF for parent compound, Heptachlor (EPA 1999).
- ^d Based on BCF for Aroclor-1254 (EPA 1999).
- ^e Based on the average of the BCFs presented for PAHs presented in EPA 1999.

BCF Bioconcentration factor
 BCF_{soil-to-inverts} Bioconcentration factor for uptake of constituent from soil to invertebrate tissue
 BCF_{soil-to-plant} Bioconcentration factor for uptake of constituent from soil to plant tissue
 COPC Chemical of potential concern

TABLE G-13: BIOACCUMULATION FACTORS FOR ECOLOGICAL CHEMICALS OF POTENTIAL CONCERN AT OU-2A SITES

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Notes (Continued)

DDT	Dichlorodiphenyltrichloroethane
DDTt	Sum of the concentrations of 4,4'- Dichlorodiphenyldichloroethane, 4,4'- Dichlorodiphenyldichloroethene, and 4,4'- Dichlorodiphenyltrichloroethane
EPA	U.S. Environmental Protection Agency
HMW	High molecular weight
LMW	Low molecular weight
PAH	Polynuclear aromatic hydrocarbon
PCB	Polychlorinated biphenyl
SVOC	Semivolatile organic chemical
VOC	Volatile organic chemical

Reference:

EPA. 1999. Screening Level Ecological Risk Assessment Protocol for Hazardous Waste Combustion Facilities. Solid Waste and Emergency Response. EPA530-D-99-001A. August.

TABLE G-14: CALCULATED MAMMAL BIOCONCENTRATION FACTORS FOR ECOLOGICAL CHEMICALS OF POTENTIAL CONCERN AT OU-2B SITES

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda

Ecological COPC	Log K _{ow}	Ba _{mammal} ^a (day/kg)	BCF _{soil-to-mammal} ^b (unitless)	BCF _{plant-to-mammal} ^b (unitless)
Metals				
Aluminum ^b	NA	1.00E+00	1.99E-03	7.88E-02
Antimony ^c	NA	1.00E-03	1.99E-06	7.88E-05
Arsenic	NA	2.00E-03	3.98E-06	1.58E-04
Barium ^c	NA	1.50E-04	2.99E-07	1.18E-05
Beryllium ^c	NA	1.00E-03	1.99E-06	7.88E-05
Cadmium ^c	NA	6.50E-06	1.29E-08	5.12E-07
Chromium ^c	NA	5.50E-03	1.09E-05	4.33E-04
Cobalt ^b	NA	1.00E+00	1.99E-03	7.88E-02
Copper ^c	NA	1.00E-02	1.99E-05	7.88E-04
Lead ^c	NA	3.00E-04	5.97E-07	2.36E-05
Manganese ^b	NA	1.00E+00	1.99E-03	7.88E-02
Mercury	NA	1.40E-02 ^c	2.79E-05	1.10E-03
Molybdenum ^b	NA	1.00E+00	1.99E-03	7.88E-02
Nickel ^c	NA	6.00E-03	1.19E-05	4.73E-04
Silver ^c	NA	3.00E-03	5.97E-06	2.36E-04
Vanadium ^b	NA	1.00E+00	1.99E-03	7.88E-02
Zinc ^c	NA	9.00E-05	1.79E-07	7.09E-06
Pesticides and PCBs^d				
DDT ^e	6.51	2.04E-01	4.06E-04	1.61E-02
Total PCBs	6.8	1.59E-01	3.16E-04	1.25E-02
SVOCs^d				
HMW and LMW PAHs ^h	Varies	3.07E-02	6.11E-05	2.42E-03
Bis(2-ethylhexyl)phthalate	7.6	4.00E-03	7.96E-06	3.15E-04
n-Nitroso-diphenylamine	3.13	3.39E-05	6.75E-08	2.67E-06
Pentachlorophenol	5.12	3.31E-03	6.59E-06	2.61E-04
VOCs^d				
2-Butanone	0.29	4.90E-08	9.75E-11	3.86E-09
1,1,1-Trichloroethane	2.49	7.76E-06	1.54E-08	6.11E-07
Acetone	-0.24	1.45E-08	2.89E-11	1.14E-09
Benzene	2.13	3.39E-06	6.75E-09	2.67E-07
Carbon Disulfide	1.94	2.19E-06	4.36E-09	1.73E-07
Chloroform	1.97	2.34E-06	4.66E-09	1.84E-07
Ethylbenzene	3.15	3.55E-05	7.06E-08	2.80E-06
Toluene	2.73	1.35E-05	2.69E-08	1.06E-06
Xylene	3.2	3.98E-05	7.92E-08	3.14E-06

Notes:

^a For metals, the Ba_{mammal} value was presented in EPA 1998, unless noted otherwise. For organics, Ba_{mammal} values were calculated using the correlation equation derived by Travis and Arms (1984).

^b Calculated by multiplying the Ba_{mammal} by the soil and plant ingestion rate for the California ground squirrel of 0.00199 kg/day-DW and 0.07879 kg/day-FW, respectively (EPA 1999).

TABLE G-14: CALCULATED MAMMAL BIOCONCENTRATION FACTORS FOR ECOLOGICAL CHEMICALS OF POTENTIAL CONCERN AT OU-2B SITES

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda

Notes (Continued):

- c Default Ba_{mammal} value of 0.02 kg/day-DW for total mercury presented in EPA 1997, was used and converted to FW by assuming 70 percent moisture in mammals (EPA 1999).
- e Based on log K_{ow} for 4,4-Dichlorodiphenyltrichloroethane

Ba_{mammal}	Biotransfer factor for mammals
$BCF_{plant-to-mammal}$	Bioconcentration factor from plant food item to mammals
$BCF_{soil-to-mammal}$	Bioconcentration factor from incidental soil ingestion to mammals
COPC	Chemical of potential concern
day/kg	Day per kilogram
DDT	Dichlorodiphenyltrichloroethane
DDTt	Sum of the concentrations of 4,4'- Dichlorodiphenyldichloroethane, 4,4'- Dichlorodiphenyldichloroethene, and 4,4'- Dichlorodiphenyltrichloroethane
DW	Dry weight
EPA	U.S. Environmental Protection Agency
FW	Fresh weight
HMW	High molecular weight
kg/day	Kilogram per day
Log K_{ow}	Octanol-water partition coefficient (unitless)
LMW	Low molecular weight
NA	Not applicable
ORD	Office of Research and Development
PAH	Polynuclear aromatic hydrocarbon
PCB	Polychlorinated biphenyl
SVOC	Semi-volatile organic chemical
VOC	Volatile organic chemical

References:

EPA. 1999. Screening Level Ecological Risk Assessment Protocol for Hazardous Waste Combustion Facilities. Solid Waste and Emergency Response. EPA530-D-99-001A. August.

EPA. 1998. Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities. Solid Waste and Emergency Response. EPA530-D-98-001A. July.

EPA. 1997. Mercury Study Report to Congress, Volumes I through VIII. Office of Air Quality Planning and Standards and ORD. EPA/452/R-97-001. December.

Travis, C.C. and A.D. Arms. 1988. "Bioconcentration of Organics in Beef, Milk, and Vegetation." Environmental Science and Technology. Volume 22. Pages 271-274.

TABLE G-15: FOOD-CHAIN MULTIPLIERS BY TROPHIC LEVEL FOR ECOLOGICAL CHEMICALS OF POTENTIAL CONCERN AT OU-2B SITES

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Ecological COPC	LOG K _{ow}	Food-Chain Multiplier ^a		
		Trophic Level of Consumer		
		2	3	4
Metals^b				
Aluminum	NA	1	1	1
Antimony	NA	1	1	1
Arsenic	NA	1	1	1
Barium	NA	1	1	1
Beryllium	NA	1	1	1
Cadmium	NA	1	1	1
Chromium	NA	1	1	1
Cobalt	NA	1	1	1
Copper	NA	1	1	1
Lead	NA	1	1	1
Manganese	NA	1	1	1
Mercury	NA	1	1	1
Molybdenum	NA	1	1	1
Nickel	NA	1	1	1
Silver	NA	1	1	1
Vanadium	NA	1	1	1
Zinc	NA	1	1	1
Pesticides and PCBs				
DDTt	6.91	1	14	27
Total PCBs	6.8	1	14	27
SVOCs				
HMW and LMW PAHs ^a	5.6	1	7.1	8.6
Bis(2-ethylhexyl)phthalate	7.6	1	12	17
n-Nitroso-diphenylamine	3.13	1	1	1
Pentachlorophenol	5.12	1	3.6	3.2
VOCs				
2-Butanone	0.29	1	1	1
1,1,1-Trichloroethane	2.49	1	1	1
Acetone	-0.24	1	1	1
Benzene	2.13	1	1	1
Carbon Disulfide	1.94	1	1	1
Chloroform	1.97	1	1	1
Ethylbenzene	3.15	1	1	1
Toluene	2.73	1	1	1
Xylene	3.2	1	1	1

Notes:

- ^a Obtained from EPA 1999
- ^b FCMs were not presented for metals, assumed a ratio of 1.
- COPC Chemical of potential concern
- DDT Dichlorodiphenyltrichloroethane
- DDTt Sum of the concentrations of 4,4'- Dichlorodiphenyldichloroethane, 4,4'- Dichlorodiphenyldichloroethene, and 4,4'- Dichlorodiphenyltrichloroethane
- EPA U.S. Environmental Protection Agency
- FCM Food-chain multiplier

TABLE G-15: FOOD-CHAIN MULTIPLIERS BY TROPHIC LEVEL FOR ECOLOGICAL CHEMICALS OF POTENTIAL CONCERN AT OU-2B SITES

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Notes (Continued):

HMW	High molecular weight
K_{ow}	Octanol-water partition coefficient (unitless)
LMW	Low molecular weight
LOG	Logarithm
NA	Not applicable
PAH	Polynuclear aromatic hydrocarbon
PCB	Polychlorinated biphenyl
SVOC	Semi-volatile organic chemical
VOC	Volatile organic chemical

References:

EPA. 1999. Screening Level Ecological Risk Assessment Protocol for Hazardous Waste Combustion Facilities. Solid Waste and Emergency Response. EPA530-D-99-001A. August

TABLE G-16: VALUES FOR EXPOSURE FACTORS FOR MEASUREMENT ENDPOINT RECEPTORS

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Exposure Parameters	Abbreviation	Units	California ground squirrel	Alameda song sparrow	American Robin	Red-tailed hawk
Daily Food Ingestion Rate ^a	Total Food IR	kg/day-FW	0.0984	0.01498	0.03511	0.327
		kg/day-DW	0.03161	0.00486	0.01267	0.0898
Incidental Soil Ingestion Rate ^b	Soil IR	kg/day-DW	0.00199	0.000457	0.0012	0.00063
Plant Percentage of Diet ^c	Plant Percent	percent	80%	50%	50%	--
Plant Ingestion Rate ^c	Plant IR	kg/day-FW	0.07879	0.00749	0.0176	--
Invertebrate Percentage of Diet ^d	Invertebrate Percent	percent	20%	50%	50%	--
Invertebrate Ingestion Rate ^d	Invert IR	kg/day-FW	0.0197	0.00749	0.0176	--
Vertebrate Percentage of Diet ^e	Vertebrate Percent	percent	--	--	--	100%
Vertebrate Ingestion Rate ^e	Vert IR	kg/day-FW	--	--	--	0.327
Site Use Factor ^f	SUF	unitless	1.00	1.00	1.00	1.00
Body Weight ^g	BW	kg	0.562	0.0199	0.081	1.13

Notes:

- ^a Based on the formula presented in Nagy 2001. Dry and Fresh weight both calculated since wildlife tissues assessed on a FW basis and soil is assessed on a DW basis.
- ^b For California ground squirrel, based on estimated percent soil in diet of jack rabbits, 6.3%, multiplied by the total IR (Arthur and Gates [1988] as cited in EPA 1993); for Alameda song sparrow, based on sediment consumption by wild turkey (9.4 percent of total IR) (Beyer and others 1994); and for red-tailed hawk, based on estimated percent soil in diet of bald eagles, 0.7% (Beyer and others 1994), multiplied by the total Food IR for dry matter intake (Nagy 2001).
- ^c For California ground squirrel, plant IR based on 80% of net food IR (California EPA, 2000); for Alameda song sparrow and American robin, plant IR based on 50% of net food IR (EPA 1993).
- ^d For California ground squirrel, invertebrate IR based on 20% of net food IR (California EPA, 2000); for Alameda song sparrow and American robin, invertebrate IR based on 50% of net food IR (EPA 1993).
- ^e Vertebrate IRs for the red-tailed hawk calculated based upon 100% of the net food IR.
- ^f Site use factor based upon the conservative estimate of 100% use of all receptors at all times.
- ^g For the California ground squirrel, based on the average female body weight (Holecamp and Nunes 1989); for the Alameda song sparrow, based upon the mean value for male and female adults during nesting, post-nesting, and winter (Dunning 1993); for the American robin based on the average body weight of an adult robin; for the red-tailed hawk based on the average body weight of an adult red-tailed hawk.

TABLE G-16: VALUES FOR EXPOSURE FACTORS FOR MEASUREMENT ENDPOINT RECEPTORS

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Notes (Continued):

--	This exposure parameter is not applicable to this receptor.
BW	Body weight
FW	Fresh weight
DW	Dry weight
IR	Ingestion rate
kg	Kilogram
kg/day	Kilogram per day
SUF	Site use factor

References:

- Beyer, W.N., G.H. Heinz, and A.W. Redmon-Norwood. 1996. *Environmental Contaminants in Wildlife: Interpreting Tissue Concentrations*. CRC Press Inc. Boca Raton, Florida.
- California EPA. 2000. California Wildlife Exposure Factor and Toxicity Database. Office of Environmental Health Hazard Assessment. http://www.oehha.org/cal_ecotox/.
- Dunning, J.B. 1993. *CRC Handbook of Avian Body Masses*. CRC Press. Boca Raton, Florida.
- Holecamp, K.E., and S. Nunes. 1989. "Seasonal Variation in Body Weight, Fat, and Behaviour of California Ground Squirrels (*Spermophilus beecheyi*)."
California Journal of Zoology. Volume 67, Number 6. Pages 1425 to 1433.
- Nagy, K. A. 2001. Food Requirements of Wild Animals: Predictive Equations for Free-Living Mammals, Reptiles, and Birds. Nutrition Abstracts and Reviews, Series B71, 21R-31R
- EPA. 1993. *Wildlife Exposure Factors Handbook*. Volumes 1 and 2. EPA 600/R-93/187a. December.

TABLE G-17: OU-2B SITES 3, 11, AND 21 (THE PINK) SOIL BACKGROUND STATISTICS

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Chemical	Frequency of Detection	Minimum Concentration (mg/kg)	Maximum Concentration (mg/kg)	Median Concentration (mg/kg)	Mean Concentration (mg/kg)	UCL95 ^a (mg/kg)
Metals						
Aluminum ^b	55/55	1,760	22,600	5,230	5,799.87	6,521.2
Antimony ^c	18/55	0.7	8.6	2.6	2.77	3.82
Arsenic ^d	45/55	0.44	15.6	1.7	2.58	4.21
Barium ^b	55/55	6.91	156	32.5	41.33	47.55
Beryllium ^e	28/55	0.25	1.47	0.58	0.5	0.61
Cadmium ^c	11/55	0.1	3.19	0.33	0.36	0.71
Calcium ^d	55/55	816	66,600	2,400	3,805.34	4,704.82
Chromium ^b	55/55	15.6	66.7	29.2	30.31	32.56
Cobalt ^d	48/55	3.02	49.7	4.7	5.68	6.58
Copper ^d	52/55	3.12	49.1	6.91	8.95	10.51
Iron ^d	55/55	4,500	27,900	8,590	10,108.88	11,154.7
Lead ^d	51/55	0.47	165	3.2	7.05	9.98
Magnesium ^d	55/55	1,290	8,800	2,320	2,859.91	3,175.36
Manganese ^d	55/55	55.50	748	108	145.19	167.2
Mercury ^c	7/54	0.06	2.71	0.1	0.11	0.36
Molybdenum ^c	0/16	NA	NA	3.1	NA	NA
Nickel ^d	55/55	11.5	80.4	24.3	27.22	29.53
Potassium ^b	55/55	209	2,480	691	740.63	820.09
Selenium ^c	0/55	NA	NA	0.42	NA	NA
Silver ^c	11/55	0.32	5.64	0.54	0.53	1.12
Sodium ^b	54/55	62.6	1,580	325	411.81	495.34
Thallium ^c	0/55	NA	NA	0.3	NA	NA
Titanium ^c	1/1	518	518	518	NA	NA
Vanadium ^d	55/55	10.5	55.3	21.0	22.52	24.5
Zinc ^d	54/55	9.98	191	20.6	25.66	29.27

Notes:

^a The UCL95 may be less than the minimum detected concentration or exceed the maximum detected concentration, because one-half of the quantitation limit was used as a proxy value for non-detected results. The UCL95 was calculated using a distribution-dependent formula.

^b Distribution determined to be lognormal.

^c Distribution not tested.

^d Distribution assumed to be lognormal based on examination of probability plots and outlier box plots.

^e Distribution assumed to be normal based on examination of probability plots and outlier box plots.

mg/kg Milligram per kilogram

NC Not calculated, detection of frequency lower than 50 percent

UCL95 95th percentile upper confidence limit on the arithmetic mean

TABLE G-18: OU-2B SITE 4 (THE BLUE) SOIL BACKGROUND STATISTICS
 Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Chemical	Frequency of Detection	Minimum Concentration (mg/kg)	Maximum Concentration (mg/kg)	Median Concentration (mg/kg)	Mean Concentration (mg/kg)	UCL95 ^a (mg/kg)
Metals						
Aluminum ^b	88/88	2,880	26,800	4,965	6,417.49	7,073.7
Antimony ^c	2/88	0.89	1	2.4	2.16	2.9
Arsenic ^c	33/88	0.74	23	2.9	4.59	6.39
Barium ^b	85/88	0.3	198	38.75	53.01	63.26
Beryllium ^c	25/88	0.09	0.77	0.3	0.37	0.49
Cadmium ^c	29/88	0.1	0.82	0.3	0.4	0.49
Calcium ^b	88/88	1,360	19,200	2,600	3,683.74	4,201.93
Chromium ^b	66/88	11.4	81.7	29.5	33.5	35.74
Cobalt ^d	66/88	1.9	14	5.35	5.37	6.45
Copper ^b	83/88	4.2	89.4	9.7	13.12	15.23
Iron ^b	88/88	760	26,900	8,140	10,072.09	11,092.99
Lead ^c	27/88	1.3	41	5.9	5.31	7.54
Magnesium ^b	88/88	1,510	42,400	2,240	2,867.67	3,156.01
Manganese ^b	88/88	50	1,060	108.5	143.63	159.52
Mercury ^c	0/22	NC	NC	0.17	NC	NC
Molybdenum ^c	0/85	NC	NC	1.4	NC	NC
Nickel ^b	88/88	11.6	88.5	24	29.17	31.64
Potassium ^b	87/88	310	6,382	770	902.98	996.73
Selenium ^c	1/88	5.7	5.7	5	4.05	4.67
Silver ^c	2/88	0.44	0.61	0.7	1.07	1.88
Sodium ^b	68/88	88.1	3,510	340	422.62	718.2
Thallium ^c	1/88	5.3	5.3	3.1	3.2	4.16
Titanium ^d	66/66	223	1,020	372.5	407.1	436.76
Vanadium ^b	88/88	12.8	62.3	20	22.23	23.68
Zinc ^b	88/88	14	84	24.85	28.55	30.93

Notes:

- ^a The UCL95 may be less than the minimum detected concentration or exceed the maximum detected concentration, because one-half of the quantitation limit was used as a proxy value for non-detected results. The UCL95 was calculated using a distribution-dependent formula.
- ^b Distribution assumed to be lognormal based on examination of probability plots and outlier box plots.
- ^c Distribution not tested.
- ^d Distribution determined to be lognormal.

mg/kg Milligram per kilogram
 NC Not calculated, detection of frequency lower than 50 percent
 UCL95 95th percentile upper confidence limit on the arithmetic mean

TABLE G-19: OU-2B GROUNDWATER BACKGROUND STATISTICS

Ecological Risk Assessment for Sites 6, 7, 8, and 16, Alameda Point, Alameda, California

Chemical	SCREENING LEVELS		Frequency of Detection	Minimum Concentration	Maximum Concentration	Median Concentration	Mean Concentration	UCL95
	MARINE ^a							
	CCC	CMC ^b						
Metals (mg/L)								
Aluminum ^c	**	**	56/194	0.003	4.53	0.0408	0.1931	0.4018
Antimony ^c	0.5 ^d	NA	13/194	0.0019	0.0478	0.0065	0.0083	0.0123
Arsenic ^e	0.036	NA	107/198	0.0014	0.0407	0.0053	0.0081	0.0158
Barium ^f	**	**	161/194	0.0023	1.26	0.0425	0.1347	0.3298
Beryllium ^c	**	**	18/194	0.0009	0.003	0.001	0.0007	0.0009
Cadmium ^c	0.0093	NA	22/194	0.0002	0.0034	0.0006	0.0009	0.0013
Calcium ^f	**	**	194/198	0.62	513	21.3	57.3059	76.7281
Chromium ^c	0.05 ^g	NA	33/194	0.0006	0.0828	0.0023	0.0031	0.0056
Cobalt ^c	**	**	12/194	0.0008	0.0105	0.0061	0.0039	0.0055
Copper ^c	0.0031	NA	60/194	0.0018	0.0273	0.0059	0.006	0.0087
Iron ^f	**	**	130/198	0.0072	24.4	0.1305	2.0403	9.3908
Lead ^c	0.0081	NA	17/195	0.0012	0.0284	0.0013	0.0013	0.0024
Magnesium ^f	**	**	198/198	0.549	1,070	15.15	67.9087	98.1515
Manganese ^f	**	**	187/198	0.0011	2.48	0.1315	0.8066	1.3736
Mercury ^c	0.00094 ^{d,h}	NA	4/198	0.0002	0.0006	0.0002	0.0001	0.0001
Molybdenum ^c	**	**	12/119	0.0005	0.0194	0.0096	0.0045	0.0064
Nickel ^c	0.0082	NA	23/198	0.0007	0.151	0.0113	0.0076	0.0127
Potassium ^e	**	**	193/198	1.2	505	15	33.4114	41.749
Selenium ^c	0.071	NA	1/193	0.0025	0.0025	0.0024	0.0015	0.0029
Silver ^c	NV	0.00019	4/188	0.0002	0.0048	0.002	0.0014	0.0019
Sodium ^f	**	**	198/198	4.6	8,160	140.5	660.4794	907.9395
Thallium ^c	0.04	NA	3/193	0.0036	0.0052	0.0027	0.0021	0.0041
Vanadium ^c	**	**	72/198	0.002	0.0508	0.007	0.0073	0.0103
Zinc ^c	0.081	NA	65/198	0.0028	46.8	0.0078	0.247	1.2774

Notes:

^a Based on the California Toxics Rule Criteria (EPA) for Enclosed Bays and Estuaries, Saltwater Aquatic Life Protection, unless otherwise specified. See full reference below.

^b When the chronic criteria, the CCC, was not available, the published acute criteria, the CMC, divided by an uncertainty factor of 10 was used. The CMC was divided by 10 to estimate chronic effects.

TABLE G-19: OU-2B GROUNDWATER BACKGROUND STATISTICS

Ecological Risk Assessment for Sites 6, 7, 8, and 16, Alameda Point, Alameda, California

Notes (Continued)

c	Distribution was not tested if sample size was less than five or frequency of detection was less than 50 percent. Lognormal distribution was assumed.
d	California Toxics Rule Criteria not available; therefore, value from US EPA National AWQC, Saltwater Aquatic Life Protection as presented in the NOAA SQUIRT Tables. See full reference below.
e	Distribution determined to be lognormal, based on Shapiro-Wilk W test (alpha = 0.05).
f	Distribution unknown. Assumed to be lognormal based on examination of probability plots, box-plots, and frequency histograms.
g	Based on Chromium 6+
h	Based on inorganic Mercury

AWQC	Ambient water quality criteria
CCC	Criteria continuous concentration
CDL	Concentration not above 1/10 the diluted concentration
CMC	Criteria maximum concentration
CSB	Concentration within statistical background
CSL	Concentration within screening level concentration
EN	Essential nutrient
EPA	U.S. Environmental Protection Agency
EPC	Exposure point concentration
FOD	Frequency of detection less than 5 percent
mg/L	Microgram per Liter
NA	Not applicable, CCC value available
NB	Non-bioaccumulating
NOAA	National Oceanic and Atmospheric Administration
NV	No value available
SQUIRT	Screening Quick Reference Tables
UCL95	95th percentile upper confidence limit on the arithmetic mean

** California Toxic Rule Criteria or US EPA AWQC not available

References:

California Environmental Protection Agency, Regional Water Quality Control Board Central Valley Region. 2000. A Compilation of Water Quality Goals. August.
NOAA. 1999. NOAA SQUIRTs. Hazmat Report 99-1. Updated September.

TABLE G-20: SITE 3, 11, AND 21 - (THE PINK) BACKGROUND SURFACE SOIL HAZARD QUOTIENT BY MEASUREMENT ENDPOINT

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Ecological COPC	MEASUREMENT ENDPOINTS							
	Reproductive or physiological impacts to the California ground squirrel		Reproductive or physiological impacts to the Alameda song sparrow		Reproductive or physiological impacts to the American robin		Reproductive or physiological impacts to the Red-tailed hawk	
	HAZARD QUOTIENT							
	High TRV	Low TRV	High TRV	Low TRV	High TRV	Low TRV	High TRV	Low TRV
Aluminum	4.56E+00 ^a	4.56E+01 ^a	1.04E+00 ^a	9.49E+00 ^a	4.65E-01 ^a	4.24E+00 ^a	6.07E-02 ^a	5.55E-01 ^a
Antimony	1.31E-02 ^b	1.80E-01 ^b	4.50E-02 ^a	1.80E-01 ^a	2.01E-02 ^a	8.03E-02 ^a	2.33E-03 ^a	9.30E-03 ^a
Arsenic	7.97E-03 ^b	1.09E-01 ^b	2.85E-02 ^a	1.14E-01 ^a	1.29E-02 ^a	5.18E-02 ^a	1.34E-03 ^a	5.34E-03 ^a
Barium	3.41E-02 ^b	1.30E-01 ^b	1.84E-01 ^a	3.69E-01 ^a	8.20E-02 ^a	1.64E-01 ^a	9.69E-03 ^a	1.94E-02 ^a
Beryllium	1.09E-03 ^b	1.09E-02 ^b	NV	NV	NV	NV	NV	NV
Cadmium	1.39E-02 ^a	6.09E-01 ^a	3.64E-02 ^a	7.45E+00 ^b	1.59E-02 ^a	3.26E+00 ^b	2.28E-03 ^a	4.64E-01 ^b
Chromium	1.02E-02 ^b	4.10E-02 ^b	4.04E-01 ^b	2.02E+00 ^b	1.94E-01 ^b	9.66E-01 ^b	7.57E-03 ^b	3.79E-02 ^b
Cobalt	4.06E-03 ^a	6.63E-02 ^a	8.08E-02 ^a	1.64E-01 ^a	3.61E-02 ^a	7.30E-02 ^a	4.69E-03 ^a	9.52E-03 ^a
Copper	2.34E-04 ^b	5.48E-02 ^b	2.06E-02 ^b	5.13E-01 ^b	9.42E-03 ^b	2.34E-01 ^b	4.73E-04 ^b	1.18E-02 ^b
Lead	2.72E-04 ^b	5.67E-02 ^a	8.66E-02 ^a	3.59E+01 ^a	4.06E-02 ^a	1.69E+01 ^a	2.44E-03 ^a	1.01E+00 ^a
Lead, alternate TRV ^c	NA	NA	NA	1.37E-01 ^a	NA	6.41E-02 ^a	NA	3.86E-03 ^a
Manganese	1.94E-02 ^a	2.23E-01 ^a	3.99E-02 ^a	3.99E-01 ^a	1.78E-02 ^a	1.78E-01 ^a	2.19E-03 ^a	2.19E-02 ^a
Mercury	4.26E-03 ^b	6.82E-02 ^b	1.99E+00 ^a	9.18E+00 ^a	8.69E-01 ^a	4.01E+00 ^a	1.30E-01 ^a	5.98E-01 ^a
Molybdenum	ND	ND	ND	ND	ND	ND	ND	ND
Nickel	4.69E-03 ^a	1.11E+00 ^a	3.36E-02 ^b	1.36E+00 ^b	1.59E-02 ^b	6.41E-01 ^b	8.04E-04 ^b	3.25E-02 ^b
Silver	NV	NV	NV	NV	NV	NV	NV	NV
Vanadium	1.38E-01 ^a	1.38E+00 ^a	5.14E-02 ^b	5.14E-01 ^b	2.30E-02 ^b	2.30E-01 ^b	3.01E-03 ^b	3.01E-02 ^b
Zinc	1.77E-03 ^a	8.50E-02 ^b	8.63E-02 ^b	8.63E-01 ^b	3.80E-02 ^b	3.80E-01 ^b	5.42E-03 ^b	5.42E-02 ^b

Notes:

- ^a TRV based on an reproductive effect
- ^b TRV based on an physiological effect
- ^c The Navy established avian low TRV of 0.014 mg/kg-day is considered highly conservative. For comparison purposes an alternate, less conservative, low TRV of 3.85 mg/kg-day as referenced in Sample and Others (1996) was used.

TABLE G-20: SITE 3, 11, AND 21 - (THE PINK) BACKGROUND SURFACE SOIL HAZARD QUOTIENT BY MEASUREMENT ENDPOINT

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Notes (Continued)

mg/kg-day	Milligram per kilogram per day
NA	Not applicable
COPC	Chemical of potential concern
ND	Not detected in background samples
NV	Reference value not available, HQ could not be calculated
TRV	Toxicity reference value

Reference:

Sample, B.E., D.M. Opresko, and G.W. Suter, II. 1996. "Toxicological Benchmarks for Wildlife: 1996 Revision." ES/ER/TM-86/R3. Oak Ridge National Laboratory. Oak Ridge, Tennessee.

TABLE G-21: OU-2B SITE 4 - (THE BLUE) BACKGROUND SURFACE SOIL HAZARD QUOTIENT BY MEASUREMENT ENDPOINT
 Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Ecological COPC	MEASUREMENT ENDPOINTS							
	Reproductive or physiological impacts to the California ground squirrel		Reproductive or physiological impacts to the Alameda song sparrow		Reproductive or physiological impacts to the American robin		Reproductive or physiological impacts to the Red-tailed hawk	
	HAZARD QUOTIENT							
	High TRV	Low TRV	High TRV	Low TRV	High TRV	Low TRV	High TRV	Low TRV
Aluminum	4.94E+00 ^a	4.94E+01 ^a	1.13E+00 ^a	1.03E+01 ^a	5.04E-01 ^a	4.60E+00 ^a	6.59E-02 ^a	6.02E-01 ^a
Antimony	9.95E-03 ^b	1.37E-01 ^b	3.42E-02 ^a	1.37E-01 ^a	1.52E-02 ^a	6.10E-02 ^a	1.77E-03 ^a	7.06E-03 ^a
Arsenic	1.20E-02 ^b	1.65E-01 ^b	4.33E-02 ^a	1.74E-01 ^a	1.96E-02 ^a	7.86E-02 ^a	2.03E-03 ^a	8.10E-03 ^a
Barium	4.54E-02 ^b	1.74E-01 ^b	2.45E-01 ^a	4.91E-01 ^a	1.09E-01 ^a	2.19E-01 ^a	1.29E-02 ^a	2.59E-02 ^a
Beryllium	8.72E-04 ^b	8.72E-03 ^b	NV	NV	NV	NV	NV	NV
Cadmium	9.56E-03 ^a	4.20E-01 ^a	2.51E-02 ^a	5.14E+00 ^a	1.09E-02 ^a	2.25E+00 ^a	1.57E-03 ^a	3.20E-01 ^a
Chromium	1.12E-02 ^b	4.50E-02 ^b	4.44E-01 ^b	2.21E+00 ^b	2.12E-01 ^b	1.06E+00 ^b	8.31E-03 ^b	4.16E-02 ^b
Cobalt	3.98E-03 ^a	6.50E-02 ^a	7.92E-02 ^a	1.60E-01 ^a	3.54E-02 ^a	7.16E-02 ^a	4.60E-03 ^a	9.34E-03 ^a
Copper	3.39E-04 ^b	7.94E-02 ^b	2.99E-02 ^b	7.43E-01 ^b	1.37E-02 ^b	3.40E-01 ^b	6.86E-04 ^b	1.70E-02 ^b
Lead	2.06E-04 ^b	4.28E-02 ^a	6.55E-02 ^a	2.71E+01 ^a	3.07E-02 ^a	1.27E+01 ^a	1.84E-03 ^a	7.65E-01 ^a
Lead, alternate TRV ^c	NA	NA	NA	1.03E-01 ^a	NA	4.84E-02 ^a	NA	2.92E-03 ^a
Manganese	1.85E-02 ^a	2.13E-01 ^a	3.80E-02 ^a	3.80E-01 ^a	1.69E-02 ^a	1.69E-01 ^a	2.09E-03 ^a	2.09E-02 ^a
Mercury	ND	ND	ND	ND	ND	ND	ND	ND
Molybdenum	ND	ND	ND	ND	ND	ND	ND	ND
Nickel	5.02E-03 ^a	1.19E+00 ^a	3.60E-02 ^b	1.45E+00 ^b	1.70E-02 ^b	6.87E-01 ^b	8.62E-04 ^b	3.49E-02 ^b
Silver	NV	NV	NV	NV	NV	NV	NV	NV
Vanadium	1.34E-01 ^a	1.34E+00 ^a	4.97E-02 ^b	4.97E-01 ^b	2.22E-02 ^b	2.22E-01 ^b	2.91E-03 ^b	2.91E-02 ^b
Zinc	1.87E-03 ^a	8.98E-02 ^b	9.12E-02 ^b	9.12E-01 ^b	4.01E-02 ^b	4.01E-01 ^b	5.73E-03 ^b	5.7E-02 ^b

Notes:

- ^a TRV based on an reproductive effect
- ^b TRV based on an physiological effect
- ^c The Navy established avian low TRV of 0.014 mg/kg-day is considered highly conservative. For comparison purposes an alternate, less conservative, low TRV of 3.85 mg/kg-day as referenced in Sample and Others (1996) was used.

mg/kg-day

Milligram per kilogram per day

TABLE G-21: OU-2B SITE 4 - (THE BLUE) BACKGROUND SURFACE SOIL HAZARD QUOTIENT BY MEASUREMENT ENDPOINT
Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Notes (Continued):

COPC	Chemical of potential concern
NA	Not applicable
ND	Not detected in background samples
NV	Reference value not available, HQ could not be calculated
TRV	Toxicity reference value

Reference:

Sample, B.E., D.M. Opresko, and G.W. Suter, II. 1996. "Toxicological Benchmarks for Wildlife: 1996 Revision." ES/ER/TM-86/R3. Oak Ridge National Laboratory. Oak Ridge, Tennessee.

TABLE G-22: SITE 3 SURFACE SOIL DETECTED CONSTITUENT SCREENING-- SELECTION OF ECOLOGICAL CHEMICALS OF POTENTIAL CONCERN

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Chemical	Frequency of Detection	Sample Quantitation Limit	Minimum Concentration	Maximum Concentration	EPC ^a	Screening Evaluation	
						Rejected	Retained
Metals (mg/kg)							
Aluminum ^b	14/14	NA	3,820	22,400	11,790.93		X
Antimony ^c	2/14	0.46 - 9.9	0.92	1	1	CSB	
Arsenic ^b	8/14	0.52 - 16	1.2	21	21		X
Barium ^d	14/14	NA	17.3	1,060	220.9		X
Beryllium ^c	6/14	0.2 - 1.6	0.31	1.9	1.46	CSB	
Cadmium ^c	4/14	0.06 - 1.6	0.07	1.3	1.08	CSB	
Calcium ^d	14/14	NA	2,000	20,000	7,825.31	EN	
Chromium ^e	13/14	0.08 - 8.2	22	79	42.61	CSB	
Cobalt ^b	10/14	1.3 - 8.2	4.4	11.1	11.1		X
Copper ^b	13/14	0.4 - 8.2	4.9	119	54.64		X
Iron ^b	14/14	NA	7,050	34,300	21,613.24	EN	
Lead ^b	37/42	0.17 - 8.2	1.7	3,870	634.15		X
Magnesium ^b	14/14	NA	1,830	10,000	4,544.69	EN	
Manganese ^b	14/14	NA	76.1	887	294.47		X
Mercury ^c	1/8	0.15 - 0.24	0.82	0.82	0.6	CSB	
Nickel ^f	13/14	1.9 - 8.2	20	66	39.02	CSB	
Potassium ^b	14/14	NA	213	4,100	1,826.35	EN	
Silver ^c	1/14	0.18 - 8.2	2.4	2.4	2.4	CSB	
Sodium ^c	6/14	2.3 - 820	434	6,400	3,000.46	EN	
Titanium ^b	6/6	5.4 - 8.2	310	670	538.29	CSB	
Vanadium ^b	14/14	NA	16	69.3	40.14		X
Zinc ^d	14/14	NA	18	1,260	231.35		X
SVOCs (mg/kg)							
2-Methylnaphthalene ^d	102/156	0.00022-0.0189	0.00022	0.37	0.015		X
Acenaphthene ^c	77/156	0.00022-0.0284	0.00028	2.6	0.103		X
Acenaphthylene ^d	100/153	0.00017-0.0307	0.00023	0.048	0.016		X
Anthracene ^d	115/156	0.0002-0.026	0.00021	7.6	1.05		X
Benzo(a)anthracene ^b	137/156	0.00014-0.0426	0.00020	14	0.151		X
Benzo(a)pyrene ^b	142/156	0.00015-0.0331	0.00028	11	0.259		X
Benzo(b)fluoranthene ^b	142/156	0.00015-0.0307	0.00031	11	0.201		X
Benzo(g,h,i)perylene ^b	147/156	0.00011-0.0355	0.00030	4.1	0.231		X
Benzo(k)fluoranthene ^b	123/156	0.00016-0.0426	0.00024	10	0.337		X
Chrysene ^b	139/156	0.00016-0.0402	0.00025	14	0.211		X
Dibenz(a,h)anthracene ^d	108/156	0.00019-0.0331	0.00033	1	0.044		X
Fluoranthene ^d	134/156	0.00018-0.0449	0.00050	38	0.39		X
Fluorene ^d	93/155	0.00018-0.024	0.00019	3.9	0.025		X
Indeno(1,2,3-cd)pyrene ^b	131/156	0.00016-0.0473	0.00034	5.9	0.474		X
Naphthalene ^d	122/156	0.00022-0.024	0.00026	1.8	0.024		X
Phenanthrene ^d	130/156	0.00016-0.0402	0.00034	32	0.276		X
Pyrene ^d	142/156	0.00012-0.0378	0.00093	25	0.394		X
VOCs (mg/kg)							
2-Butanone ^c	1/13	0.01 - 13	0.24	0.24	0.24		X
Acetone ^c	2/13	0.01 - 13	0.14	0.58	0.58		X
Benzene ^c	1/13	0.005 - 13	7.5	7.5	3.19		X
Carbon disulfide ^c	1/13	0.005 - 13	0.01	0.01	0.01		X
Ethylbenzene ^c	2/13	0.005 - 13	0.94	50	20.66		X
Toluene ^c	6/13	0.005 - 13	0.002	210	86.59		X
Xylene (Total) ^c	2/13	0.005 - 13	2.3	250	103.18		X

TABLE G-22: SITE 3 SURFACE SOIL DETECTED CONSTITUENT SCREENING-- SELECTION OF ECOLOGICAL CHEMICALS OF POTENTIAL CONCERN

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Notes:

a	The EPC is the lesser of the UCL95 and the maximum detected concentration. The maximum detected concentration is used for all samples with fewer than three detected measurements.
b	Distribution determined to be lognormal.
c	Distribution not tested.
d	Distribution determined to be unknown, but assumed to be lognormal based on examination of probability plots and outlier box plots.
e	Distribution determined to be unknown, but assumed to be normal based on examination of probability plots and outlier box plots.
f	Distribution determined to be normal.
CSB	Concentrations within statistical background
EN	Essential nutrient
EPC	Exposure point concentration
mg/kg	Millogram per kilogram
NA	Not applicable, frequency of detection is 100 percent
SVOC	Semivolatile organic chemical
UCL95	95th percentile upper confidence limit on the arithmetic mean
VOC	Volatile organic chemical

TABLE G-23: SITE 4 SURFACE SOIL DETECTED CONSTITUENT SCREENING-- SELECTION OF ECOLOGICAL CHEMICALS OF POTENTIAL CONCERN

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Chemical	Frequency of Detection	Sample Quantitation Limit	Minimum Concentration	Maximum Concentration	EPC ^a	Screening Evaluation	
						Rejected	Retained
Metals (mg/kg)							
Aluminum ^b	68/68	NA	2,870	10,000	5,621.34	CSB	
Antimony ^c	12/66	0.42-9.00	0.46	134	12.27		X
Arsenic ^d	43/68	0.26-13.00	0.94	7.4	4.53		X
Barium ^d	68/68	NA	23	317	59.66		X
Beryllium ^c	29/68	0.08-1.30	0.17	1.5	0.48	CSB	
Cadmium ^c	37/79	0.02-1.30	0.1	105	9.58		X
Calcium ^d	68/68	NA	1,000	16,100	4,773.05	EN	
Chromium ^d	76/77	0.01-6.40	19.8	1,530	88.29		X
Chromium (VI) ^b	25/29	0.03-0.28	0.09	7.81	2.48		X
Cobalt ^d	56/68	0.41-6.40	1.90	26.3	9.94	CSB	
Copper ^d	70/71	0.08-6.40	4.30	326	24.26		X
Iron ^e	68/68	NA	103	14,600	9,249.67	EN	
Lead ^d	60/71	0.14-6.40	1.6	1,460	54.99		X
Magnesium ^b	68/68	NA	1,240	4,200	2,522.5	EN	
Manganese ^d	68/68	NA	72	306	141.88		X
Mercury ^c	14/50	0.05-0.18	0.05	0.24	0.12		X
Molybdenum ^c	5/48	0.42-6.40	0.72	3.1	2.24		X
Nickel ^d	71/71	NA	17	1,400	64.8		X
Potassium ^b	61/68	37.20-640.00	386	1,200	792.24	EN	
Selenium ^c	3/68	0.21-13.00	0.44	1.2	1.2	FOD	
Silver ^c	30/71	0.06-6.40	0.8	81.1	11.55		X
Sodium ^b	60/68	2.30-640.00	83.3	1,530	403.74	EN	
Titanium ^b	18/18	NA	197	729	444.32	CSB	
Vanadium ^b	68/68	NA	13	35	22.11		X
Zinc ^d	68/68	NA	13.6	283	38.32		X
SVOCs (mg/kg)							
2-Methylnaphthalene ^d	117/225	0.00022-0.0189	0.00022	0.3	0.014		X
Acenaphthene ^c	63/225	0.00022-0.0284	0.00023	0.29	0.019		X
Acenaphthylene ^d	111/222	0.00017-0.0307	0.00019	0.28	0.021		X
Anthracene ^d	129/225	0.0002-0.026	0.00021	0.74	0.025		X
Benzo(a)anthracene ^d	182/225	0.00014-0.0426	0.00015	1.6	0.122		X
Benzo(a)pyrene ^d	186/223	0.00015-0.0331	0.00023	2.1	0.141		X
Benzo(b)fluoranthene ^d	187/225	0.00015-0.0307	0.00023	1.9	0.142		X
Benzo(g,h,i)perylene ^b	201/223	0.00011-0.0355	0.00028	1.8	0.071		X
Benzo(k)fluoranthene ^d	164/225	0.00016-0.0426	0.00017	1.1	0.083		X
Bis(2-ethylhexyl)phthalate ^c	2/95	0.035-3.8	0.69	7.6	1.311		X
Chrysene ^d	190/224	0.00016-0.0402	0.00024	2.2	0.201		X
Dibenz(a,h)anthracene ^d	121/225	0.00019-0.0331	0.00021	0.18	0.027		X
Fluoranthene ^d	186/220	0.00018-0.0449	0.00022	3.9	0.189		X
Fluorene ^c	80/221	0.00018-0.024	0.00019	0.45	0.023		X
Indeno(1,2,3-cd)pyrene ^d	173/216	0.00016-0.0473	0.00029	1.7	0.108		X
n-Nitroso-di-n-propylamine ^c	2/95	0.11-3.8	0.064	0.18	0.180	FOD-NB	
n-Nitroso-diphenylamine ^c	3/95	0.086-3.8	0.073	0.32	0.320		X
Naphthalene ^d	118/225	0.00022-0.024	0.00026	0.32	0.017		X
Pentachlorophenol ^c	1/95	0.54-19	0.13	0.13	0.130		X
Phenanthrene ^d	180/225	0.00016-0.0402	0.00022	4.1	0.094		X

TABLE G-23: SITE 4 SURFACE SOIL DETECTED CONSTITUENT SCREENING-- SELECTION OF ECOLOGICAL CHEMICALS OF POTENTIAL CONCERN

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Chemical	Frequency of Detection	Sample Quantitation Limit	Minimum Concentration	Maximum Concentration	EPC ^a	Screening Evaluation	
						Rejected	Retained
SVOCs (Continued) (mg/kg)							
Phenol ^c	1/95	0.15-3.8	0.074	0.074	0.074	FOD-NB	
Pyrene ^b	194/225	0.00012-0.0378	0.0002	4.2	0.122		X
VOCs (mg/kg)							
1,1,1-Trichloroethane ^c	5/62	0.005-0.013	0.002	0.008	0.008		X
1,1-Dichloroethene ^c	2/62	0.005-0.013	0.001	0.004	0.004	FOD-NB	
1,2-Dichloroethene (Total) ^c	1/62	0.005-0.013	0.001	0.001	0.001	FOD-NB	
Acetone ^c	1/62	0.01-0.018	0.092	0.092	0.019	FOD-NB	
Carbon disulfide ^c	3/61	0.005-0.013	0.001	0.001	0.001	FOD-NB	
Ethylbenzene ^c	21/61	0.005-0.013	0.001	0.028	0.009		X
Methylene Chloride ^c	1/62	0.005-0.013	0.005	0.005	0.005	FOD-NB	
Toluene ^c	15/62	0.005-0.013	0.001	0.19	0.037		X
Trichloroethene ^c	1/62	0.005-0.013	0.059	0.059	0.011	FOD-NB	
Xylene (Total) ^c	30/61	0.005-0.013	0.001	0.17	0.036		X

Notes:

- ^a The EPC is the lesser of the UCL95 and the maximum detected concentration. The maximum detected concentration is used for all samples with fewer than three detected measurements.
- ^b Distribution determined to be lognormal.
- ^c Distribution not tested.
- ^d Distribution determined to be unknown, but assumed to be lognormal based on examination of probability plots and outlier box plots.
- ^e Distribution determined to be unknown, but assumed to be normal based on examination of probability plots and outlier box plots.

CSB	Concentrations within statistical background
EN	Essential nutrient
EPC	Exposure point concentration
FOD	Frequency of detection five percent or lower
mg/kg	Millogram per kilogram
NA	Not applicable, frequency of detection is 100 percent
NB	Non-bioaccumulating
SVOC	Semivolatile organic chemical
UCL95	95th percentile upper confidence limit on the arithmetic mean
VOC	Volatile organic chemical

TABLE G-24: SITE 11 SURFACE SOIL DETECTED CONSTITUENT SCREENING-- SELECTION OF ECOLOGICAL CHEMICALS OF POTENTIAL CONCERN

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Chemical	Frequency of Detection	Sample Quantitation Limit	Minimum Concentration	Maximum Concentration	EPC ^a	Screening Evaluation	
						Rejected	Retained
Metals (mg/kg)							
Aluminum ^b	30/30	NA	3,150	25,900	8,494.57	CSB	
Antimony ^c	3/30	0.45-2.70	0.61	2.6	1.73	CSB	
Arsenic ^b	29/30	0.24-0.53	0.53	4.2	2.36	CSB	
Barium ^b	30/30	NA	10.3	73.4	43.83	CSB	
Beryllium ^d	27/30	0.13-0.23	0.21	2	1.01		X
Cadmium ^c	11/30	0.08-0.32	0.12	4.32	1.56	CSB	
Calcium ^b	30/30	NA	1,380	28,700	6,499.11	EN	
Chromium ^e	29/30	0.08-0.61	5.3	44.7	28.98	CSB	
Cobalt ^b	30/30	NA	2.8	36.1	9.58		X
Copper ^b	28/30	0.00-0.80	4.77	83.2	28.72		X
Iron ^b	30/30	NA	6,430	32,200	14,520.95	EN	
Lead ^b	27/30	0.20-3.70	0.78	242	102.8		X
Magnesium ^b	30/30	NA	1,840	15,100	4,814.09	EN	
Manganese ^b	30/30	NA	60.6	558	254.38		X
Mercury ^c	4/30	0.08-0.25	0.12	0.63	0.23	CSB	
Nickel ^d	28/30	1.32-3.30	6.75	51.2	27.82	CSB	
Potassium ^b	28/30	57.10-140.00	176	1,220	719.92	EN	
Selenium ^c	1/30	0.19-0.60	0.28	0.28	0.28	FOD	
Silver ^c	3/30	0.18-0.53	0.65	0.82	0.63	CSB	
Sodium ^f	29/30	5.00-6.40	80.3	1,020	399.73	EN	
Vanadium ^b	30/30	NA	14.5	82.5	32		X
Zinc ^f	30/30	NA	16.1	196	47.34		X
SVOCs (mg/kg)							
2-Methylnaphthalene ^c	29/60	0.00022-0.0189	0.00028	0.061	0.019		X
Acenaphthene ^c	23/60	0.00022-0.0284	0.0003	0.42	0.05		X
Acenaphthylene ^c	20/60	0.00017-0.0307	0.00018	0.068	0.018		X
Anthracene ^f	33/60	0.0002-0.026	0.00025	1.6	0.121		X
Benzo(a)anthracene ^b	47/60	0.00014-0.0426	0.00033	11	0.601		X
Benzo(a)pyrene ^f	49/60	0.00015-0.0331	0.00035	4.7	0.414		X
Benzo(b)fluoranthene ^b	50/60	0.00015-0.0307	0.00026	9.2	0.597		X
Benzo(g,h,i)perylene ^b	50/60	0.00011-0.0355	0.00055	1.8	0.217		X
Benzo(k)fluoranthene ^b	46/60	0.00016-0.0426	0.00018	2.3	0.277		X
Chrysene ^b	50/60	0.00016-0.0402	0.00036	9.9	0.694		X
Dibenz(a,h)anthracene ^f	33/60	0.00019-0.0331	0.00022	0.7	0.056		X
Fluoranthene ^b	51/60	0.00018-0.0449	0.00026	15	0.933		X
Fluorene ^c	22/60	0.00018-0.024	0.00032	0.43	0.052		X
Indeno(1,2,3-cd)pyrene ^f	41/60	0.00016-0.0473	0.00082	1.5	0.208		X
Naphthalene ^f	36/60	0.00022-0.024	0.00022	0.05	0.024		X
Phenanthrene ^f	46/60	0.00016-0.0402	0.00028	8.2	0.625		X
Pyrene ^f	49/60	0.00012-0.0378	0.00056	12	1.23		X
VOCs (mg/kg)							
Chloroform ^c	1/15	0.0052-0.011	0.002	0.002	0.002		X

Notes:

^a The EPC is the lesser of the UCL95 and the maximum detected concentration. The maximum detected concentration is used for all samples with fewer than three detected measurements.

^b Distribution determined to be lognormal.

^c Distribution not tested.

TABLE G-24: SITE 11 SURFACE SOIL DETECTED CONSTITUENT SCREENING-- SELECTION OF ECOLOGICAL CHEMICALS OF POTENTIAL CONCERN

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Notes (Continued):

d	Distribution determined to be normal.
e	Distribution determined to be unknown, but assumed to be normal based on examination of probability plots and outlier box plots.
f	Distribution determined to be unknown, but assumed to be lognormal based on examination of probability plots and outlier box plots.
CSB	Concentrations within statistical background
EN	Essential nutrient
EPC	Exposure point concentration
FOD	Frequency of detection five percent or lower
mg/kg	Millogram per kilogram
NA	Not applicable, frequency of detection is 100 percent
SVOC	Semivolatile organic chemical
UCL95	95th percentile upper confidence limit on the arithmetic mean
VOC	Volatile organic chemical

TABLE G-25: SITE 21 SURFACE SOIL DETECTED CONSTITUENT SCREENING-- SELECTION OF ECOLOGICAL CHEMICALS OF POTENTIAL CONCERN

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Chemical	Frequency of Detection	Sample Quantitation Limit	Minimum Concentration	Maximum Concentration	EPC ^a	Screening Evaluation	
						Rejected	Retained
Metals (mg/kg)							
Aluminum ^b	12/12	NA	3,940	21,600	14,250.97		X
Antimony ^c	3/12	0.46-2.70	2.9	4.7	3.41	CSB	
Arsenic ^b	10/12	0.26-0.54	2.25	6.66	6.66		X
Barium ^b	12/12	NA	35.7	144	86.77		X
Beryllium ^d	10/12	0.13-0.23	0.87	2.5	1.61		X
Cadmium ^c	4/12	0.08-0.33	0.68	9.5	4.76	CSB	
Calcium ^b	12/12	NA	3,920	17,300	8,901.6	EN	
Chromium ^b	12/12	NA	16	67	44.98	CSB	
Cobalt ^b	12/12	NA	2.9	21.1	13.91		X
Copper ^b	12/12	NA	5.4	71.4	41.27		X
Iron ^b	12/12	NA	7,280	34,100	21,016.92	EN	
Lead ^b	10/12	0.20-3.53	2.86	416	252.16		X
Magnesium ^b	12/12	NA	1,930	11,300	7,338.56	EN	
Manganese ^d	12/12	NA	91.2	449	300.91		X
Mercury ^c	3/12	0.15-0.27	0.16	2.6	1.27	CSB	
Nickel ^b	12/12	NA	19.7	80.4	46.37	CSB	
Potassium ^b	11/12	55.70-140.00	461	2,060	1,170.82	EN	
Silver ^c	3/12	0.18-0.54	0.61	5.64	2.83	CSB	
Sodium ^b	12/12	NA	88.2	849	559.1	EN	
Vanadium ^b	12/12	NA	17.1	86.7	47.92		X
Zinc ^b	12/12	NA	16.1	267	94.47		X
Pesticides (mg/kg)							
4,4'-DDD ^c	1/10	0.0033-0.018	0.012	0.012	0.009		X
4,4'-DDT ^c	1/10	0.0033-0.018	0.058	0.058	0.033		X
Aroclor 1260 ^c	1/10	0.033-0.18	0.14	0.14	0.088		X
SVOCs (mg/kg)							
2-Methylnaphthalene ^e	39/63	0.00022-0.0189	0.00024	0.063	0.007		X
Acenaphthene ^c	7/63	0.00022-0.0284	0.00036	0.01	0.01		X
Acenaphthylene ^c	14/63	0.00017-0.0307	0.00019	0.0048	0.005		X
Anthracene ^c	26/63	0.0002-0.026	0.00022	0.019	0.011		X
Benzo(a)anthracene ^b	50/63	0.00014-0.0426	0.0002	0.084	0.02		X
Benzo(a)pyrene ^b	49/63	0.00015-0.0331	0.00041	0.087	0.028		X
Benzo(b)fluoranthene ^b	54/63	0.00015-0.0307	0.00031	0.085	0.018		X
Benzo(g,h,i)perylene ^b	53/63	0.00011-0.0355	0.00031	0.13	0.042		X
Benzo(k)fluoranthene ^e	43/63	0.00016-0.0426	0.0003	0.073	0.02		X
Chrysene ^b	52/63	0.00016-0.0402	0.00017	0.27	0.045		X
Dibenz(a,h)anthracene	27/63	0.00019-0.0331	0.00029	0.028	0.013		X
Fluoranthene ^b	60/63	0.00018-0.0449	0.00025	0.099	0.016		X
Fluorene ^c	10/63	0.00018-0.024	0.00022	0.077	0.014		X
Indeno(1,2,3-cd)pyrene ^b	45/63	0.00016-0.0473	0.00021	0.13	0.028		X
Naphthalene ^e	32/63	0.00022-0.024	0.00029	0.032	0.007		X
Phenanthrene ^e	53/63	0.00016-0.0402	0.00026	0.16	0.012		X
Pyrene ^b	56/63	0.00012-0.0378	0.00034	0.14	0.021		X

Notes:

^a The EPC is the lesser of the UCL95 and the maximum detected concentration. The maximum detected concentration is used for all samples with fewer than three detected measurements.

^b Distribution determined to be lognormal.

TABLE G-25: SITE 21 SURFACE SOIL DETECTED CONSTITUENT SCREENING-- SELECTION OF ECOLOGICAL CHEMICALS OF POTENTIAL CONCERN

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Notes (Continued):

c	Distribution not tested.
d	Distribution determined to be normal.
e	Distribution determined to be unknown, but assumed to be lognormal based on examination of probability plots and outlier box plots.
CSB	Concentrations within statistical background
DDD	Dichlorodiphenyldichloroethane
DDT	Dichlorodiphenyltrichloroethane
EN	Essential nutrient
EPC	Exposure point concentration
mg/kg	Millogram per kilogram
NA	Not applicable, frequency of detection is 100 percent
SVOC	Semivolatile organic chemical
UCL95	95th percentile upper confidence limit on the arithmetic mean

TABLE G-26: OU-2B GROUNDWATER DETECTED CONSTITUENT SCREENING -- SELECTION OF ECOLOGICAL CHEMICALS OF POTENTIAL CONCERN

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Chemical	SCREENING LEVELS		Frequency of Detection	Reporting Limit	Minimum Concentration	Maximum Concentration	EPC ^c	Screening Evaluation		Hazard Quotient
	MARINE ^a							Rejected	Retained	
	CCC	CMC ^b								
Metals (mg/L)										
Aluminum	**	**	37/70	0.0046 - 1	0.0056	2.24	0.31		X	NA
Antimony	0.5 ^d	NA	42/70	0.00008 - 0.05	0.00004	0.0014	0.00072	CSL		NA
Arsenic	0.036	NA	54/70	0.0008 - 0.005	0.0012	0.083	0.015	CSL		NA
Barium	**	**	64/70	0.00005 - 0.307	0.024	0.88	0.18		X	NA
Beryllium	**	**	8/70	0.0001 - 0.002	0.00002	0.00011	0.00011	CSB		NA
Cadmium	0.0093	NA	17/78	0.00007 - 0.005	0.00005	0.0034	0.00288	CSL		NA
Calcium	**	**	70/70	0.0038 - 50	3.8	1,400	347	EN		NA
Chromium	0.05 ^e	NA	35/77	0.00013 - 0.01	0.0001	1.54	0.113		X	0.226
Chromium (VI)	0.05		1/7	0.01	0.19	0.19	0.147	CSB		NA
Cobalt	**	**	60/70	0.00021 - 0.01	0.00006	0.041	0.00553		X	NA
Copper	0.0031	NA	61/70	0.0006 - 0.01	0.00007	0.02	0.00537	CDL		NA
Iron	**	**	58/70	0.0083 - 0.5	0.0242	28	12.3	EN		NA
Lead	0.0081	NA	21/88	0.00002 - 0.003	0.00004	0.058	0.00569	CDL		NA
Magnesium	**	**	69/70	0.0038 - 50	2	2,600	682	EN		NA
Manganese	**	**	68/70	0.0002 - 1	0.00082	26	26		X	NA
Mercury	0.00094 ^{d,f}	NA	4/70	0.00004 - 0.00027	0.00004	0.00005	0.00005	CSB		NA
Molybdenum	**	**	53/70	0.00025 - 0.2	0.00074	0.39	0.0234		X	NA
Nickel	0.0082	NA	60/70	0.0003 - 0.1	0.00042	0.52	0.0266		X	0.324
Potassium	**	**	70/70	0.0037 - 5	0.91	370	56	EN		NA
Selenium	0.071	NA	47/69	0.0008 - 0.005	0.00036	0.078	0.00967	CDL		NA
Silver	NV	0.00019	2/70	0.00003 - 0.005	0.00074	0.0017	0.0017	FOD		NA
Sodium	**	**	70/70	0.0885 - 50	13	17,000	5,350	EN		NA
Thallium	0.04	NA	5/70	0.00001 - 0.0039	0.00025	0.006	0.00137	CSL		NA
Vanadium	**	**	43/70	0.00024 - 0.01	0.00072	0.046	0.0073		X	NA
Zinc	0.081	NA	36/70	0.0003 - 0.0979	0.00049	0.106	0.0138	CDL		NA
SVOCs (mg/L)										
Acenaphthylene	**	0.030 ^{d,g}	1/110	0.002 - 0.01	0.002	0.002	0.002	CSL		NA
Benzo(a)anthracene	**	0.030 ^{d,g}	3/110	0.0002 - 0.01	0.0001	0.0001	0.0001	CSL		NA
Benzo(a)pyrene	**	0.030 ^{d,g}	2/110	0.0002 - 0.0027	0.0001	0.0001	0.0001	CSL		NA
Bis(2-ethylhexyl)phthalate	**	0.030 ^{d,g}	1/76	0.004		0.003	0.00282	CSL		NA
Fluoranthene	**	0.030 ^{d,g}	5/110	0.0002 - 0.01	0.0001	0.00093	0.00093	CSL		NA
Naphthalene	**	0.030 ^{d,g}	6/219	0.0005 - 0.67	0.0002	0.0056	0.0056	CSL		NA
Pyrene	**	0.030 ^{d,g}	11/110	0.0002 - 0.01	0.0001	0.00051	0.00051	CSL		NA
VOCs (mg/L)										
1,1-Dichloroethane	**	11.3 ^{d,h}	57/256	0.0005 - 0.2	0.0001	2.5	0.0611	CSL		NA

TABLE G-26: OU-2B GROUNDWATER DETECTED CONSTITUENT SCREENING -- SELECTION OF ECOLOGICAL CHEMICALS OF POTENTIAL CONCERN

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Chemical	SCREENING LEVELS		Frequency of Detection	Reporting Limit	Minimum Concentration	Maximum Concentration	EPC ^c	Screening Evaluation		Hazard Quotient
	MARINE ^a							Rejected	Retained	
	CCC	CMC ^b								
VOCs (Continued) (mg/L)										
1,1-Dichloroethene	**	22.4 ^{d,i}	54/256	0.0005 - 0.2	0.0002	4	0.151	CSL		NA
1,1,1-Trichloroethane	**	3.12 ^d	14/256	0.0005 - 0.2	0.0004	0.048	0.00698	CSL		NA
1,1,2-Trichloroethane	**	3.12 ^{d,j}	10/256	0.0005 - 0.2	0.0004	0.065	0.00724	FOD		NA
1,2-Dichlorobenzene	0.129 ^d	NA	32/292	0.0005 - 0.17	0.0001	0.24	0.0121	CDL		NA
1,2-Dichloroethane	**	11.3 ^d	19/256	0.0005 - 0.17	0.0002	0.096	0.00603	CSL		NA
1,2-Dichloroethene (total)	**	22.4 ^d	51/122	0.002 - 0.2	0.0003	5.2	0.311	CSL		NA
1,2-Dichloropropane	**	0.079 ^d	1/256	0.0005 - 0.2	0.0001	0.0001	0.0001	FOD		NA
1,2,4-Trichloromethylbenzene	**	**	8/128	0.0005 - 0.17	0.0002	0.046	0.00841		X	NA
1,3-Dichlorobenzene	0.129 ^{d,k}	NA	6/292	0.0005 - 0.17	0.00007	0.0045	0.0045	FOD		NA
1,3,5-Trichloromethylbenzene	**	**	6/128	0.0004 - 0.17	0.0002	0.02	0.00795	FOD		NA
1,4-Dichlorobenzene	0.129 ^d	NA	22/292	0.0005 - 0.17	0.0003	0.048	0.00561	CSL		NA
2-Butanone	**	**	1/231	0.0007 - 3.3	0.0086	0.0086	0.0086	FOD		NA
2-Hexanone	**	**	1/232	0.002 - 3.3	0.002	0.002	0.002	FOD		NA
4-Methyl-2-Pentanone	**	**	5/237	0.002 - 3.3	0.0007	0.029	0.029	FOD		NA
Acetone	**	**	22/231	0.0004 - 3.3	0.0005	63	1.54		X	NA
Benzene	0.7 ^d	NA	37/256	0.0005 - 0.17	0.0001	0.55	0.0238	CSL		NA
Bromodichloromethane	6.4 ^d	**	7/256	0.0005 - 0.2	0.0009	0.01	0.00652	FOD		NA
Carbon Disulfide	**	**	42/237	0.0005 - 0.2	0.002	0.017	0.00707		X	NA
Chlorobenzene	0.129 ^d	NA	18/256	0.0005 - 0.2	0.0005	0.02	0.0067	CSL		NA
Chloroethane	**	**	9/256	0.001 - 0.33	0.0004	0.037	0.00978	FOD		NA
Chloroform	**	**	10/256	0.0005 - 0.2	0.0001	0.034	0.00664	FOD		NA
Chloromethane	**	**	4/256	0.001 - 0.33	0.0003	0.01	0.00952	FOD		NA
Ethylbenzene	**	0.043 ^d	20/256	0.0002 - 0.2	0.0003	0.15	0.0091	CSL		NA
Hexachlorobutadiene	**	**	1/185	0.0005 - 0.17	0.0004	0.0004	0.0004	FOD		NA
Isopropylbenzene	**	**	6/109	0.0005 - 0.17	0.0005	0.0055	0.0055		X	NA
Methy-T-Butyl Ether	**	**	11/229	0.0001 - 0.17	0.0001	0.0016	0.0016	FOD		NA
Methylene Chloride	6.4 ^d	**	1/256	0.0001 - 1.7	0.075	0.075	0.014	FOD		NA
Tert-Butanol	**	**	12/73	0.01 - 3.3	0.0027	0.5	0.286		X	NA
Tetrachloroethene	0.45 ^d	NA	12/256	0.0005 - 0.2	0.0002	0.003	0.003	CSL		NA
Toluene	5 ^d	NA	45/256	0.0005 - 0.2	0.0001	0.016	0.00655	CSL		NA
Trichloroethene	NV	0.2 ^d	103/256	0.0005 - 0.2	0.0002	19	0.786		X	0.393
Vinyl Chloride	**	**	62/256	0.0005 - 0.17	0.0003	1.4	0.0335		X	NA
Xylene	**	**	23/128	0.001 - 0.2	0.0003	0.32	0.0186		X	NA

TABLE G-26: OU-2B GROUNDWATER DETECTED CONSTITUENT SCREENING -- SELECTION OF ECOLOGICAL CHEMICALS OF POTENTIAL CONCERN

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Notes:

- a Based on the California Toxics Rule Criteria (EPA) for Enclosed Bays and Estuaries, Saltwater Aquatic Life Protection, unless otherwise specified. See full reference below.
- b When the chronic criteria, the CCC, was not available, the published acute criteria, the CMC, divided by an uncertainty factor of 10 was used. The CMC was divided by 10 to estimate chronic effects.
- c The EPC was the lower value of the maximum detected concentration or the UCL95.
- d California Toxics Rule Criteria not available; therefore, value from EPA National AWQC, Saltwater Aquatic Life Protection as presented in the NOAA SQUIRT Tables. See full reference below.
- e Based on chromium 6+
- f Based on inorganic mercury
- g Value is was derived for chemical class
- h Based on a similar compound, 1,2-dichloroethane
- i Based on a similar compound, 1,2-dichloroethene
- j Based on a similar compound, 1,1,1-trichloroethane
- k Based on similar compounds 1,2-dichlorobenzene and 1,4-dichlorobenzene

AWQC	Ambient water quality criteria	mg/L	Millogram per Liter
CCC	Criteria continuous concentration	NA	Not applicable
CDL	Maximum diluted concentration (1/10) within screening level concentrati	NB	Nonbioaccumulating
CMC	Criteria maximum concentration	NOAA	National Oceanic and Atmospheric Administration
CSB	Concentration within statistical background	NV	No value available
CSL	EPC within screening level concentration	SQUIRT	Screening Quick Reference Tables
EN	Essential nutrient	SVOC	Semivolatile organic chemical
EPA	U.S. Environmental Protection Agency	UCL95	95th percentile upper confidence limit on the arithmetic mean
EPC	Exposure point concentration	VOC	Volatile organic chemical
FOD	Frequency of detection less than 5 percent		

** California Toxic Rule Criteria or EPA AWQC not available

References:

California Environmental Protection Agency, Regional Water Quality Control Board Central Valley Region. 2000. A Compilation of Water Quality Goals. August.

NOAA. 1999. NOAA SQUIRTs. Hazmat Report 99-1. Updated September.

TABLE G-27: SITE 3 - SURFACE SOIL HAZARD QUOTIENT BY MEASUREMENT ENDPOINT

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Ecological COPC	MEASUREMENT ENDPOINTS							
	Reproductive or physiological impacts to the California ground squirrel		Reproductive or physiological impacts to the Alameda song sparrow		Reproductive or physiological impacts to the American robin		Reproductive or physiological impacts to the Red-tailed hawk	
	HAZARD QUOTIENT							
	High TRV	Low TRV	High TRV	Low TRV	High TRV	Low TRV	High TRV	Low TRV
Aluminum	8.24E+00 ^a	8.24E+01 ^a	1.88E+00 ^a	1.72E+01 ^a	8.42E-01 ^a	7.68E+00 ^a	1.10E-01 ^a	1.00E+00 ^a
Arsenic	3.94E-02 ^b	5.42E-01 ^b	1.42E-01 ^a	5.70E-01 ^a	6.46E-02 ^a	2.59E-01 ^a	6.67E-03 ^a	2.66E-02 ^a
Barium	1.58E-01 ^b	6.06E-01 ^b	8.54E-01 ^a	1.71E+00 ^a	3.82E-01 ^a	7.65E-01 ^a	4.50E-02 ^a	9.03E-02 ^a
Cobalt	6.84E-03 ^a	1.12E-01 ^a	1.36E-01 ^a	2.76E-01 ^a	6.09E-02 ^a	1.23E-01 ^a	7.92E-03 ^a	1.61E-02 ^a
Copper	1.22E-03 ^b	2.85E-01 ^b	1.07E-01 ^b	2.66E+00 ^b	4.91E-02 ^b	1.22E+00 ^b	2.46E-03 ^b	6.11E-02 ^b
Lead	1.73E-02 ^b	3.60E+00 ^a	5.51E+00 ^a	2.28E+03 ^a	2.58E+00 ^a	1.07E+03 ^a	1.55E-01 ^a	6.44E+01 ^a
Alternate Lead TRV ^c	NA	NA	NA	8.68E+00 ^a	NA	4.08E+00 ^a	NA	2.45E-01 ^a
Manganese	3.42E-02 ^a	3.92E-01 ^a	7.02E-02 ^a	7.02E-01 ^a	3.13E-02 ^a	3.13E-01 ^a	3.86E-03 ^a	3.86E-02 ^a
Vanadium	2.27E-01 ^a	2.27E+00 ^a	8.43E-02 ^b	8.43E-01 ^b	3.77E-02 ^b	3.77E-01 ^b	4.93E-03 ^b	4.93E-02 ^b
Zinc	1.40E-02 ^a	6.72E-01 ^b	6.82E-01 ^b	6.82E+00 ^b	3.01E-01 ^b	3.01E+00 ^b	4.28E-02 ^b	4.28E-01 ^b
HMW PAHs	5.79E-04 ^b	1.45E-02 ^b	QE	QE	QE	QE	QE	QE
LMW PAHs	6.20E-05 ^a	1.86E-04 ^a	QE	QE	QE	QE	QE	QE
2-Butanone	QE	QE	QE	QE	QE	QE	QE	QE
Acetone	1.05E-03 ^b	5.25E-03 ^b	QE	QE	QE	QE	QE	QE
Benzene	2.61E-03 ^{2a}	2.61E-02 ^a	QE	QE	QE	QE	QE	QE
Carbon disulfide	QE	QE	QE	QE	QE	QE	QE	QE
Ethylbenzene	QE	QE	QE	QE	QE	QE	QE	QE
Toluene	1.79E-01 ^a	1.79E+00 ^a	QE	QE	QE	QE	QE	QE
Xylene	5.01E+01 ^a	6.21E+01 ^a	QE	QE	QE	QE	QE	QE

Notes:

^a TRV based on a reproductive effect.

^b TRV based on a physiological effect.

^c The Navy established avian low TRV of 0.014 mg/kg-day is considered highly conservative. For comparison purposes an alternate, less conservative, low TRV of 3.85 mg/kg-day, as referenced by Sample and others (1996), was used.

TABLE G-27: SITE 3 - SURFACE SOIL HAZARD QUOTIENT BY MEASUREMENT ENDPOINT

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Notes (Continued):

COPC	Chemical of potential concern
HMW	High molecular weight
LMW	Low molecular weight
mg/kg-day	Milligram per kilogram per day
NA	Not applicable
PAH	Polynuclear aromatic hydrocarbon
QE	No TRV developed for Ecological COPC and endpoint, qualitative evaluation only
TRV	Toxicity reference value

Reference:

Sample, B.E., D.M. Opresko, and G.W. Suter, II. 1996. "Toxicological Benchmarks for Wildlife: 1996 Revision." ES/ER/TM-86/R3. Oak Ridge National Laboratory. Oak Ridge, Tennessee.

TABLE G-28: SITE 4 - SURFACE SOIL HAZARD QUOTIENT BY MEASUREMENT ENDPOINT

Ecological Risk Assessment for Sites 3, 4, 11, and, and 21, Alameda Point, Alameda, California

Ecological COPC	MEASUREMENT ENDPOINTS							
	Reproductive or physiological impacts to the California ground squirrel		Reproductive or physiological impacts to the Alameda song sparrow		Reproductive or physiological impacts to the American robin		Reproductive or physiological impacts to the Red-tailed hawk	
	HAZARD QUOTIENT							
	High TRV	Low TRV	High TRV	Low TRV	High TRV	Low TRV	High TRV	Low TRV
Antimony	4.21E-02 ^a	5.79E-01 ^a	1.45E-01 ^b	5.80E-01 ^b	6.45E-02 ^b	2.58E-01 ^b	7.48E-03 ^b	2.99E-02 ^b
Arsenic	8.51E-03 ^a	1.17E-01 ^a	3.07E-02 ^b	1.23E-01 ^b	1.39E-02 ^b	5.58E-02 ^b	1.44E-03 ^b	5.75E-03 ^b
Barium	4.28E-02 ^a	1.64E-01 ^a	2.31E-01 ^b	4.63E-01 ^b	1.03E-01 ^b	2.07E-01 ^b	1.22E-02 ^b	2.44E-02 ^b
Cadmium	1.87E-01 ^b	8.22E+00 ^b	4.91E-01 ^a	1.00E+02 ^a	2.15E-01 ^a	4.41E+01 ^a	3.07E-02 ^a	6.27E+00 ^a
Chromium	2.77E-02 ^a	1.11E-01 ^a	1.10E+00 ^b	5.47E+00 ^b	5.25E-01 ^b	2.62E+00 ^b	2.05E-02 ^b	1.03E-01 ^b
Chromium 6+	7.78E-04 ^a	3.12E-03 ^a	3.08E-02 ^b	1.54E-01 ^b	1.47E-02 ^b	7.36E-02 ^b	5.77E-04 ^b	2.88E-03 ^b
Copper	5.40E-04 ^a	1.26E-01 ^a	4.76E-02 ^a	1.18E+00 ^a	2.18E-02 ^a	5.42E-01 ^a	1.09E-03 ^a	2.71E-02 ^a
Lead	1.53E-03 ^a	3.12E-01 ^b	4.77E-01 ^b	1.98E+02 ^b	2.24E-01 ^b	9.31E+01 ^b	1.34E-02 ^b	5.58E+00 ^b
Alternate Lead TRV ^c	NA	NA	NA	7.53E-01 ^b	NA	3.54E-01 ^b	NA	2.13E-02 ^b
Manganese	1.65E-02 ^b	1.89E-01 ^b	3.88E-02 ^b	3.38E-01 ^b	1.51E-02 ^b	1.51E-01 ^b	1.86E-03 ^b	1.86E-02 ^b
Mercury	1.42E-03 ^a	2.27E-02 ^a	6.63E-01 ^b	3.06E+00 ^b	2.90E-01 ^b	1.34E+00 ^b	4.34E-02 ^b	2.00E-01 ^b
Molybdenum	1.59E-02 ^b	1.59E-01 ^b	1.76E-02 ^b	1.78E-01 ^b	7.86E-03 ^b	7.94E-02 ^b	9.67E-04 ^b	9.75E-03 ^b
Nickel	1.03E-02 ^b	2.44E+00 ^b	7.37E-02 ^a	2.98E+00 ^a	3.48E-02 ^a	1.41E+00 ^a	1.77E-03 ^a	7.14E-02 ^a
Silver	QE	QE	QE	QE	QE	QE	QE	QE
Vanadium	1.25E-01 ^b	1.25E+00 ^b	4.64E-02 ^a	4.64E-01 ^a	2.08E-02 ^a	2.08E-01 ^a	2.72E-03 ^a	2.72E-02 ^a
Zinc	2.31E-03 ^b	1.11E-01 ^a	1.13E-01 ^a	1.13E+00 ^a	4.98E-02 ^a	4.98E-01 ^a	7.10E-03 ^a	7.10E-02 ^a
HMW PAHs	2.59E-04 ^a	6.49E-03 ^a	QE	QE	QE	QE	QE	QE
LMW PAHs	8.75E-06 ^b	2.63E-05 ^b	QE	QE	QE	QE	QE	QE
Bis(2-ethylhexyl)phthalate	3.93E-01 ^b	3.93E+00 ^b	8.85E+01 ^b	8.85E+02 ^b	3.86E+01 ^b	3.86E+02 ^b	7.27E+01 ^b	7.27E+02 ^b
n-Nitroso-diphenylamine	QE	QE	QE	QE	QE	QE	QE	QE
Pentachlorophenol	2.02E+00 ^a	2.02E+01 ^a	QE	QE	QE	QE	QE	QE
1,1,1-Trichloroethane	2.85E-07 ^b	2.85E-06 ^b	QE	QE	QE	QE	QE	QE
Ethylbenzene	QE	QE	QE	QE	QE	QE	QE	QE
Toluene	7.67E-05 ^b	7.67E-04 ^b	QE	QE	QE	QE	QE	QE
Xylene	1.75E-02 ^b	2.17E-02 ^b	QE	QE	QE	QE	QE	QE

TABLE G-28: SITE 4 - SURFACE SOIL HAZARD QUOTIENT BY MEASUREMENT ENDPOINT

Ecological Risk Assessment for Sites 3, 4, 11, and, and 21, Alameda Point, Alameda, California

Notes:

- a TRV based on an physiological effect.
b TRV based on an reproductive effect.
c The Navy established avian low TRV of 0.014 mg/kg-day is considered highly conservative. For comparison purposes an alternate, less conservative, low TRV of 3.85 mg/kg-day, as referenced by Sample and others (1996), was used.

COPC	Contaminant of potential concern
HMW	High molecular weight
LMW	Low molecular weight
mg/kg-day	Milligram per kilogram per day
NA	Not applicable
PAH	Polynuclear aromatic hydrocarbon
QE	No TRV developed for Ecological COPC and endpoint, qualitative evaluation only
TRV	Toxicity reference value

Reference:

Sample, B.E., D.M. Opresko, and G.W. Suter, II. 1996. "Toxicological Benchmarks for Wildlife: 1996 Revision." ES/ER/TM-86/R3. Oak Ridge National Laboratory. Oak Ridge, Tennessee.

TABLE G-29: SITE 11 - SURFACE SOIL HAZARD QUOTIENT BY MEASUREMENT ENDPOINT

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Ecological COPC	MEASUREMENT ENDPOINTS							
	Reproductive or physiological impacts to the California ground squirrel		Reproductive or physiological impacts to the Alameda song sparrow		Reproductive or physiological impacts to the American robin		Reproductive or physiological impacts to the Red-tailed hawk	
	HAZARD QUOTIENT							
	High TRV	Low TRV	High TRV	Low TRV	High TRV	Low TRV	High TRV	Low TRV
Beryllium	1.80E-03 ^a	1.80E-02 ^a	QE	QE	QE	QE	QE	QE
Cobalt	5.91E-03 ^b	9.65E-02 ^b	1.18E-01 ^b	2.38E-01 ^b	5.33E-02 ^b	1.08E-01 ^b	6.83E-03 ^b	1.39E-02 ^b
Copper	6.40E-04 ^a	1.50E-01 ^a	5.63E-02 ^a	1.40E+00 ^a	2.64E-02 ^a	6.57E-01 ^a	1.29E-03 ^a	3.21E-02 ^a
Lead	2.81E-03 ^a	5.84-01 ^b	8.92E-01 ^b	3.69E+02 ^b	4.34E-01 ^b	1.80E+02 ^b	2.51E-02 ^b	1.04E+01 ^b
Alternate Lead TRV ^c	NA	NA	NA	1.41E+00 ^b	NA	6.85E-01 ^b	NA	3.98E-02 ^b
Manganese	2.96E-02 ^b	3.39E-01 ^b	6.06E-02 ^b	6.06E-01 ^b	2.74E-02 ^b	2.74E-01 ^b	3.33E-03 ^b	3.33E-02 ^b
Vanadium	1.81E-01 ^b	1.81E+00 ^b	6.72E-02 ^a	6.72E-01 ^a	3.05E-02 ^a	3.05E-01 ^a	3.93E-03 ^a	3.93E-02 ^a
Zinc	2.86E-03 ^b	1.37E-01 ^a	1.40E-01 ^a	1.40E+00 ^a	6.19E-02 ^a	6.19E-01 ^a	8.77E-03 ^a	8.77E-02 ^a
HMW PAHs	1.12E-03 ^a	2.81E-02 ^a	QE	QE	QE	QE	QE	QE
LMW PAHs	3.73E-05 ^b	1.12E-04 ^b	QE	QE	QE	QE	QE	QE
Chloroform	7.70E-06 ^b	2.10E-05 ^b	QE	QE	QE	QE	QE	QE

Notes:

- ^a TRV based on an physiological effect.
- ^b TRV based on an reproductive effect.
- ^c The Navy established avian low TRV of 0.014 mg/kg-day is considered highly conservative. For comparison purposes an alternate, less conservative, low TRV of 3.85 mg/kg-day, as referenced by Sample and others (1996), was used.

- COPC Chemical of potential concern
- HMW High molecular weight
- LMW Low molecular weight
- mg/kg-day Milligram per kilogram per day
- NA Not applicable
- PAH Polynuclear aromatic hydrocarbon
- QE No TRV developed for Ecological COPC and endpoint, qualitative evaluation only
- TRV Toxicity reference value

Reference:

Sample, B.E., D.M. Opresko, and G.W. Suter, II. 1996. "Toxicological Benchmarks for Wildlife: 1996 Revision." ES/ER/TM-86/R3. Oak Ridge National Laboratory.

TABLE G-29: SITE 11 - SURFACE SOIL HAZARD QUOTIENT BY MEASUREMENT ENDPOINT

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda, California

Oak Ridge, Tennessee.

TABLE G-30: SITE 21 - SURFACE SOIL HAZARD QUOTIENT BY MEASUREMENT ENDPOINT

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda

Ecological COPC	MEASUREMENT ENDPOINTS							
	Reproductive or physiological impacts to the California ground squirrel		Reproductive or physiological impacts to the Alameda song sparrow		Reproductive or physiological impacts to the American robin		Reproductive or physiological impacts to the Red-tailed hawk	
	HAZARD QUOTIENT							
	High TRV	Low TRV	High TRV	Low TRV	High TRV	Low TRV	High TRV	Low TRV
Aluminum	9.96E+00 ^a	9.96E+01 ^a	2.28E+00 ^a	2.07E+01 ^a	1.03E+00 ^a	9.40E+00 ^a	1.33E-01 ^a	1.21E+00 ^a
Arsenic	1.25E-02 ^b	1.72E-01 ^b	4.51E-02 ^a	1.81E-01 ^a	2.09E-02 ^a	8.37E-02 ^a	2.12E-03 ^a	8.45E-03 ^a
Barium	6.23E-02 ^b	2.38E-01 ^b	3.36E-01 ^a	6.73E-01 ^a	1.52-01 ^a	3.04E-01 ^a	1.77E-02 ^a	8.12E-01 ^a
Beryllium	2.86E-03 ^b	2.86E-02 ^b	QE	QE	QE	QE	QE	QE
Cobalt	8.57E-03 ^a	1.40E-01 ^a	1.71E-01 ^a	3.46E-01 ^a	7.74E-02 ^a	1.57E-01 ^a	9.92E-03 ^a	2.01E-02 ^a
Copper	9.19E-04 ^b	2.15E-01 ^b	8.09E-02 ^b	2.01E+00 ^b	3.80E-02 ^b	9.44E-01 ^b	1.86E-03 ^b	4.62E-02 ^b
Lead	6.88E-03 ^b	1.43E+00 ^a	2.19E+00 ^a	9.06E+02 ^a	1.06E+00 ^a	4.42E+02 ^a	6.17E-02 ^a	2.56E+01 ^a
Alternate Lead TRV ^c	NA	NA	NA	3.45E+00 ^a	NA	1.68E+00 ^a	NA	9.75E-02 ^a
Manganese	3.50E-02 ^a	4.01E-01 ^a	7.17E-02 ^a	7.17E-01 ^a	3.24E-02 ^a	3.24E-01 ^a	3.94E-03 ^a	3.94E-02 ^a
Vanadium	2.70E-01 ^a	2.70E+00 ^a	1.01E-01 ^b	1.01E+00 ^b	4.56E-02 ^b	4.56E-01 ^b	5.89E-03 ^b	5.89E-02 ^b
Zinc	5.70E-03 ^a	2.74E-01 ^b	2.78E-01 ^b	2.78E+00 ^b	1.24E-01 ^b	1.24E+00 ^b	1.75E-02 ^b	1.75E-01 ^b
DDTt	1.30E-04 ^a	2.60E-03 ^a	3.05E-02 ^a	6.53E+00 ^a	1.34E-02 ^a	2.87E+00 ^a	2.79E-02 ^a	5.98E+00 ^a
Total PCBs	3.60E-03 ^b	1.29E-02 ^b	7.58E-02 ^a	9.18E-01 ^a	3.33E-02 ^a	4.04E-01 ^a	6.89E-02 ^a	8.36E-01 ^a
HMW PAHs	5.40E-05 ^b	1.35E-03 ^b	QE	QE	QE	QE	QE	QE
LMW PAHs	2.71E-06 ^a	8.15E-06 ^a	QE	QE	QE	QE	QE	QE

Notes:

^a TRV based on a reproductive effect.

^b TRV based on a physiological effect.

^c The Navy established avian low TRV of 0.014 mg/kg-day is considered highly conservative. For comparison purposes an alternate, less conservative, low TRV of 3.85 mg/kg-day, as referenced by Sample and others (1996), was used.

COPC Chemical of potential concern

DDT Dichlorodiphenyltrichloroethane

DDTt Sum of 4,4-dichlorodiphenyldichloroethane, 4,4-dichlorodiphenyldichloroethene, and 4,4-dichlorodiphenyltrichloroethane

TABLE G-30: SITE 21 - SURFACE SOIL HAZARD QUOTIENT BY MEASUREMENT ENDPOINT

Ecological Risk Assessment for Sites 3, 4, 11, and 21, Alameda Point, Alameda

Notes (Continued):

HMW	High molecular weight
LMW	Low molecular weight
mg/kg-day	Milligram per kilogram per day
NA	Not applicable
PAH	Polynuclear aromatic hydrocarbon
PCB	Polychlorinated biphenyl
QE	No TRV developed for Ecological COPC and endpoint, qualitative evaluation only
TRV	Toxicity reference value

Reference:

Sample, B.E., D.M. Opresko, and G.W. Suter, II. 1996. "Toxicological Benchmarks for Wildlife: 1996 Revision." ES/ER/TM-86/R3. Oak Ridge National Laboratory. Oak Ridge, Tennessee.

**ATTACHMENT A
ECOTOXICOLOGICAL PROFILES FOR ECOLOGICAL CHEMICALS OF POTENTIAL
CONCERN AT OU-2B SITES**

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ACRONYMS AND ABBREVIATIONS

$\mu\text{g}/\text{m}^3$	Microgram per cubic meter
μg	Microgram
$\mu\text{g}/\text{L}$	Microgram per Liter
AQUIRE	Aquatic Toxicity Information Retrieval Database
ATPase	Adenosine Triphosphatase
ATSDR	Agency for Toxic Substances and Disease Registry
BCF	Bioconcentration factor
$^{\circ}\text{C}$	Degrees Celsius
COPC	Contaminants of potential concern
DCE	Dichloroethene
DDD	Dichlorodiphenyldichloroethane
DDE	Dichlorodiphenyldichloroethene
DDT	Dichlorodiphenyltrichloroethane
DNA	Deoxyribonucleic acid
EC50	Effects concentration, where 50 percent treated are effected
EPA	U.S. Environmental Protection Agency
g/day	Gram per day
g/ha	Gram per hectare
GTF	Glucose tolerance factor
HSDB	Hazardous Substances Database
IARC	International Agency Research Cancer
ICA	International Copper Association
ILL	Incipient Lethal Level
IRIS	Integrated Risk Information System
Koc	Organic carbon/water partition coefficients
Kow	Octanol-Water Coefficient
LC50	Lethal concentration where 50 percent treated were killed
LOAEL	Lowest observed adverse effect level
mg	Milligram
mg/kg	Milligram per kilogram
mg/kg/day	Milligram per kilogram per day
mg/L	Milligram per liter
mL/g	Milliliter per gram

ACRONYMS AND ABBREVIATIONS (Continued)

NAS	National Academy of Sciences
NCRR	National Center for Research Resources
NLM	National Library of Medicine
NOAEL	No observed adverse effect level
NRCC	National Research Council Canada
PAH	Polynuclear aromatic hydrocarbons
PCB	Polychlorinated biphenyl
PCE	Perchloroethylene
ppm	Parts per million
redox	Oxidation-reduction
SVOC	Semivolatile organic compound
VOC	Volatile organic compound
WHO	World Health Organization

1.0 INTRODUCTION

The following sections present identified literature data on toxicological effects of ecological contaminants of potential concern (COPC) identified at Alameda Point, Alameda, California (Alameda Point). Section 2.0 presents data on metals; Section 3.0 presents data on chlorinated pesticides; Section 4.0 presents data on polychlorinated biphenyls (PCB); Section 5.0 presents data on polynuclear aromatic hydrocarbons (PAH); Section 6.0 presents data on semivolatile organic compounds (SVOC); and Section 7.0 presents data on volatile organic compounds (VOC).

Bioconcentration in this document is defined as the net accumulation of a substance by an organism as a result of uptake from an aqueous solution. Bioaccumulation is defined as the net accumulation of a substance by an organism as a result of uptake from all environmental sources, including food and water, which are ingested. The amount of a substance that is bioaccumulated in an organism also could be defined as the body burden of the substance in an organism. Biomagnification is defined as the sequential increase in concentration of a chemical from one trophic level to the next.

2.0 METALS

The following sections present data on ecotoxicological effects of various metals identified as ecological COPCs at Alameda Point.

2.1 ALUMINUM

Aluminum is one of the most abundant metals in the earth's crust. It is used in the manufacture of many products such as aircraft, utensils, electrical conductors, and dental fillings. It also is used in combination with other chemicals to serve many purposes such as water purification, medicinals, catalysts, dyes, baking powder, fireproofing, and tanning. Because of its frequent use and common occurrence, aluminum enters the environment from point and nonpoint sources.

Acute toxic effects may include the death of animals, birds, or fish and death or low growth rate in plants. Acute effects are seen 2 to 4 days after animals or plants come in contact with a toxic chemical.

Aluminum has moderate acute toxicity to aquatic life and high acute toxicity to birds. Insufficient data are available to evaluate or predict the short-term effects of aluminum on plants or land animals.

Chronic toxic effects may include shortened lifespan, reproductive problems, lower fertility, and changes in appearance or behavior. Chronic effects can be seen long after first exposure to a toxic chemical.

Aluminum has high chronic toxicity to aquatic life. Insufficient data are available to evaluate or predict the long-term effects of aluminum to plants, birds, or land animals.

Aluminum solubility in water is dependent upon pH, as pH increases or decreases from near neutrality, solubility of aluminum will increase. Aluminum is highly persistent in water, with a half-life greater than 200 days. The half-life of a pollutant is the amount of time it takes for one-half of the chemical to degrade. Some substances increase in concentration, or bioaccumulate, in living organisms as they breathe contaminated air, drink contaminated water, or eat contaminated food. These chemicals can become concentrated in the tissues and internal organs of animals and humans.

2.1.1 Plants

Aluminum interferes with cell division in roots; decreases root respiration; fixes phosphorous in unavailable forms in roots; interferes with uptake, transport, and use of calcium, magnesium, phosphorus, potassium, and water; and interferes with enzyme activities (Foy and Others 1978). Symptoms of toxicity include stubby, brittle roots; stunting; late maturity; and collapse of growing points. Seedlings are more susceptible to damage from aluminum toxicity than older plants.

2.1.2 Invertebrates

No information was identified on the effects of aluminum on invertebrates.

2.1.3 Amphibians and Reptiles

No information was identified on the effects of aluminum on amphibians and reptiles.

2.1.4 Fish and Aquatic Organisms

Aluminum will not accumulate in fish tissues; however, not much information was identified on the effects of aluminum on toxicity to fish and aquatic organisms.

2.1.5 Birds

Chickens developed severe rickets when about 1,400 parts per million (ppm) inorganic phosphorus was ingested.

2.1.6 Mammals

The acute toxicity of aluminum and aluminum compounds is low. Absorption in the gastrointestinal tract is usually less than 1 percent and is easily excreted in the urine. Short term studies using various aluminum compounds in the diet or drinking-water, showed only minimal effects to rats, mice, and dog receptors. Aluminum is not fetotoxic, carcinogenic, or mutagenic. Inhalation studies of aluminum dust caused respiratory infections in rats and rabbits, as well as, widespread interstitial fibrosis that caused emphysema and hemorrhages. Larger species, such as pigs and dogs, exposed to aluminum can develop the bone disorder, osteomalacia, or rickets (NLM 2003).

2.2 ANTIMONY

The analysis of tissue samples from lower trophic levels (invertebrates and small mammals) in a food chain suggests that biomagnification did not occur and that mobility of antimony in food chains is low (Ainsworth 1988). This theory is further strengthened by an investigation found that antimony concentrations in small mammals resulting from dietary intake were "very low in all experiments in comparison to the antimony concentrations in the diet" (Ainsworth and Others 1991). Consequently, food chain transfer of antimony to upper trophic-level consumers appears to be negligible. Of the antimony absorbed by the body is excreted rapidly through urine and feces.

The toxic action mechanism of metals and arsenic lies in their ability to bind to thiol groups of biological molecules, destroying their function (Balazs and Others 1986). Antimony occurs in the same column of the periodic table of the elements as arsenic; antimony participates in bonding, similar to arsenic. Compared to arsenic, antimony has a lower affinity for bonding to sulfur-containing proteins and for causing protein-dioxyribnucleic acids (DNA) crosslinks (Gebel 1997). Consequently, antimony should possess a lower toxicity compared to arsenic. Therefore, the screening-level ecological risk assessment could use the arsenic toxicity reference value as a conservative approach to estimating risk.

2.2.1 Plants

Antimony is considered to be a nonessential metal and is easily taken up by plants if available in soil in soluble forms (Kabata-Pendias and Pendias 1984). A 4 ppm concentration of antimaony in culture solution produced a toxic response in cabbage plants (NLM 2003).

2.2.2 Invertebrates

No information was identified on the effects of antimony on invertebrates.

2.2.3 Amphibians and Reptiles

No information was identified on the effects of antimony on amphibians and reptiles.

2.2.4 Fish and Aquatic Organisms

No information was identified on the effects of antimony on fish and aquatic organisms.

2.2.5 Birds

No information was identified on the effects of antimony on birds.

2.2.6 Mammals

Inhalation of antimony compounds causes irritation of the respiratory tract. Teratogenic effects (abortions) were noted in female rabbits fed doses of 5 and 55 milligrams (mg) metallic antimony every other day for 30, 60, and 90 days. Animals poisoned by antimony compounds showed dyspnea, loss of weight, loss of hair, and evidence of cardiac insufficiencies. Those that survived began to regain weight after 5 to 10 days.

2.3 ARSENIC

Arsenic is a naturally occurring element that is widely distributed in the environment and is used in metallurgy to harden copper, lead, and alloys and is used in the manufacture of certain types of glass. Historically, some forms of arsenic have been used as pesticides.

Arsenic is present in the environment in both inorganic and organic forms. The bioavailability and toxicity of arsenic are dependent on the chemical and physical forms of arsenic, the exposure route, and the species of concern. Inorganic arsenic is present in the environment in two major forms, arsenate, and arsenite. In the environment, arsenate is more abundant and bioaccumulates more rapidly than arsenite; however, arsenite is the more toxic form (Sadiq 1992). Any environmental conditions that promote an increase in arsenite concentrations will increase the toxicity of arsenic in the environment. The redox environment is the most important environmental parameter that affects the bioavailability of arsenic. Reduced conditions in the environment cause the ratio of arsenite to arsenate to increase, which increases the toxicity of arsenic in the environment. In addition to redox, the pH of the aquatic environment influences arsenic toxicity. As the pH of water increases, arsenic toxicity decreases as more arsenate is produced (Sadiq 1992).

Marine organisms accumulate more arsenic than freshwater organisms. Arsenate is more readily bioaccumulated than arsenite (Sadiq 1992). Bioconcentration factors (BCF) experimentally determined for arsenic appear to be relatively low. Methylated species of arsenic are transferred efficiently in the food chain (Eisler 1988a); however, it does not appear to be biomagnified through the food chain

(Eisler 1988a; Callahan and Others 1979; U.S. Environmental Protection Agency [EPA] 1982a, 1983e, as cited in Agency for Toxic Substances and Disease Registry [ATSDR] 1993a).

2.3.1 Plants

Terrestrial plants may accumulate arsenic by root uptake from the soil or by absorption of airborne arsenic deposited on the leaves. Certain species of plants may accumulate substantial levels of arsenic (Shaw 1990). Soil concentrations of arsenic have been known to reduce crop plant yields (Eisler 1988a). If the roots of the plant absorb excess arsenic, the plant will stop growing and developing (Eisler 1988a). The chemical form of arsenic absorbed by plants will determine the type of toxic effect plants will express. Effects can range from inhibition of light activation, wilting, chlorosis, browning, dehydration, and death (Eisler 1988a).

Marine phytoplankton have the ability to bioconcentrate inorganic arsenic to high levels, then chemically transform arsenic by methylation. Methylated species are efficiently transferred in the food chain (Eisler 1988a); however, arsenic does not appear to be biomagnified through the food chain (Eisler 1988a; Callahan and Others 1979; EPA 1982a, 1983e, as cited in ATSDR 1993a).

2.3.2 Invertebrates

Arsenic has been used as a pesticide in the past. Arsenic may reduce growth and metabolism of soil microbiota, may reduce numbers of bacteria and protozoa, and may adversely affect earthworms by reducing their numbers in the soil (National Research Council Canada [NRCC] 1978, as cited in Eisler 1988a).

2.3.3 Amphibians and Reptiles

Very little information was available on the effects of arsenic on amphibians and reptiles. One report states, however, that developing toad embryos exposed to arsenic concentrations observed that 50 percent of developing embryos died or were malformed (Eisler 1988a).

2.3.4 Fish and Aquatic Organisms

Arsenic can produce both carcinogenic and mutagenic effects in aquatic organisms (Eisler 1988a). Toxic effects of arsenic in fish include impaired behavior, reduced growth, lack of appetite, and failure to metabolize food (Eisler 1988a). The chemical form of arsenic, the type of organism, and the organism's life stage all influence the organism's susceptibility to arsenic poisoning. In general, the earlier life stages and smaller organisms are more sensitive to arsenic concentrations (Sadiq 1992).

In addition to the stage of development of an organism, the toxic effect of arsenic is influenced by the type of organism. Marine organisms accumulate more arsenic than freshwater organisms. Bioaccumulation of arsenic in marine organisms is influenced by the amount of arsenic in seawater and is further influenced by the marine organism's feeding habits. Bottom feeders appear to be more sensitive to arsenic concentrations than other types of fish (Sadiq 1992). This is because bottom feeders filter the water column and ingest arsenic-containing particulate matter for food. As a result, bottom feeders, such as mollusks, accumulate more arsenic in their soft tissue than fish (Sadiq 1992). Fish, on the other hand, do not have to filter the water column for food and as a result, the main route of uptake involves diffusion of dissolved residual around the gill membrane (Sadiq 1992).

2.3.5 Birds

Some species of birds are more sensitive to arsenic exposure, while others appear to be more tolerant of arsenic exposure (Eisler 1988a). The acute oral exposure of inorganic arsenic destroys the blood vessel lining in the gut, which can result in lower blood pressure (Eisler 1988a). The acute, oral exposure of arsenite in birds was observed to cause hepatocyte damage by arsenic inhibition of the sodium pump in cells (Eisler 1988a). Acute effects of arsenite in birds include muscular incoordination, debility, slowness, spasmodic movement, falling, hyperactivity, fluffed feathers, drooped eyelids, huddled position, unkempt appearance, loss of righting reflex, immobility, and seizures (Eisler 1988a). Chronic effects include systemic, growth, behavioral, and reproductive problems (Stanley and Others 1994; Whitworth and Others 1991; Camardese and Others 1990).

2.3.6 Mammals

Arsenic is a carcinogen, teratogen, and a possible mutagen. Adverse effects produced by arsenic are highly dose-dependent. At low levels, arsenic may be an essential nutrient and substitute for phosphorous in biochemical reactions (ATSDR 1993a). At high levels, however, arsenic is recognized as an effective poison. Chronic exposure to low levels of arsenic can produce malaise and fatigue, gastrointestinal distress, anemia and basophilic stippling, neuropathy, and skin lesions, which can develop into skin cancer. Water-soluble arsenic is absorbed efficiently from the gastrointestinal tract and circulated throughout the body. Trivalent arsenic is detoxified in the liver by conversion to methylarsenic acid and dimethylarsenic acid, which are the principal forms excreted in the urine. The body burden of arsenic can reach considerable levels, because it can be sequestered in nails, hair, bones, teeth, skin, liver, kidneys, and lungs (ATSDR 1993a). In mammalian species, arsenic is a teratogen that can pass the placental barrier and produce fetal death and malformations, consisting of exencephaly, eye defects, and renal and gonadal agenesis (Eisler 1988a; ATSDR 1993a; Domingo 1994).

2.4 BARIUM

Barium is an alkaline earth metal that is widely distributed in the environment and is used to produce alloys, paints, soap, paper, rubber, ceramics, and glass. Barium is naturally present in plant and animal tissue and may be an essential element in trace amounts.

The solubility of barium compounds influences adsorption and toxicity. Soluble barium compounds are more easily adsorbed by organisms compared to their lower soluble counterparts and may be accumulated in the skeleton (Amdur and Others 1991).

Barium is found mainly in inorganic complexes and is stable in the +2 valence state. Environmental conditions in soil, such as pH, Eh, cation exchange capacity, and calcium carbonate levels, will affect the movement of barium in the environment (Agency for Toxic Substances and Disease Registry [ATSDR] 1990a). In an aquatic environment, barium will most likely precipitate out of solution as a barium sulfate or barium carbonate or the barium ion will adsorb to particulate matter (ATSDR 1990a).

Barium can bioaccumulate in terrestrial and aquatic organisms (ATSDR 1990a), and marine plants can bioconcentrate barium by a factor of 1,000 times the concentration found in water. Marine animals, plankton, and brown algae have reported bioconcentration factors (BCF) of 100, 120, and 260, respectively (ATSDR 1990a), and terrestrial plants bioconcentrate low levels of barium from the soil (ATSDR 1990a).

2.4.1 Plants

Some plants may accumulate barium from soil, for example, Brazil nuts can accumulate high levels of barium (Amdur and Others 1991; ATSDR 1990a).

Plants, probably in a manner similar to calcium and magnesium, easily take up soluble forms of barium but barium is not readily translocated from roots to shoots (Peterson and Girling 1981). Barium has been reported to inhibit seed germination, enzyme activation, and uptake of calcium and magnesium by roots. Common symptoms are brown, retarded roots and stunted foliage (Romney and Childress 1965).

2.4.2 Invertebrates

No information was identified on the effects of barium on invertebrates.

2.4.3 Amphibians and Reptiles

No information was identified on the effects of barium on amphibians and reptiles.

2.4.4 Fish and Aquatic Organisms

Little information was identified on the effects of barium on fish and aquatic organisms. In the aquatic environment, the route for the transport of metals into fish is through the gill, gut, or skin (Pulsford and Others 1992). In one study, however, barium was observed to inhibit chlorine absorption in the intestine of winter flounder (Chamey and Taglietta 1992).

2.4.5 Birds

No information was identified on the effects of barium on birds.

2.4.6 Mammals

In addition to skeletal deposition, ingestion of barium salts can result in gastrointestinal distress, muscular paralysis, lowered pulse rate, muscle stimulation, and irregular cardiac contractions (Amdur and Others 1991). Rats exposed to barium concentrations in their diet showed toxic responses. In both acute and chronic oral exposure scenarios, increased blood pressure was observed in exposed rat populations (ATSDR 1990a). In other acute exposure studies, gastrointestinal effects and respiratory weakness were observed in exposed populations (ATSDR 1990a). Ovary weight and ovary-to-brain weight ratios both decreased as a result of acute, oral exposure to barium in rat populations (Borzelleca and Other 1988, as cited in ATSDR 1990a). Published data concerning developmental, reproductive, or carcinogenic effects of barium on mammalian species are very limited (ATSDR 1990a).

2.5 BERYLLIUM

In most types of soil, beryllium will be tightly sorbed onto clay particles (ATSDR 1993a). Beryllium is expected to have limited mobility in soil, although its mobility may increase as a result of formation of soluble hydroxide complexes in soils of higher pH (Callahan and Others 1979, as cited in ATSDR 1993a).

In aquatic environments, most beryllium is usually present sorbed to suspended matter or to sediment, rather than in a dissolved form in the water column. Beryllium may precipitate into sediment as a result of a formation of insoluble complexes and usually is sorbed onto clay particles in sediment. A high percentage of beryllium is expected to be immobile in water as a result of this association with sediment particles, although at a high pH, the formation of water-soluble complexes with hydroxide ions may increase the solubility and mobility of beryllium (Callahan and Others 1979, as cited in ATSDR 1993a).

Bioconcentration of beryllium in fish is not likely because of the low absorption of beryllium from the water column by aquatic animals. Significant biomagnification of beryllium within the food chain has not been observed (ATSDR 1993a).

2.5.1 Plants

No information was identified on the effects of beryllium on plants.

2.5.2 Invertebrates

No information was identified on the effects of beryllium on invertebrates.

2.5.3 Amphibians and Reptiles

Very little information was available on the effects of beryllium on amphibians and reptiles. However, in a journal article, Jagoe and others (1993) suggest that the toxic effects of beryllium are similar to those by aluminum. In general, metals induce their toxic effects in the gill surface (skin) of amphibians. Reduced survival of adult populations of frogs and salamanders has been observed when the study organisms were chronically exposed to aluminum in the test water (Horne and Dunson 1995). In addition, aluminum exposure was shown to cause a significant increase in embryonic mortality (Horne and Dunson 1995).

2.5.4 Fish and Aquatic Organisms

Very little information was available on the effects of beryllium on fish and aquatic organisms. However, in a journal article, Jagoe and Others (1993) suggest that the toxic effects of beryllium are similar to those produced by aluminum. In general, metals induce their toxic effects in the gill surface of aquatic organisms. Like other metals, beryllium affects the physiological processes occurring at the gill surface, including ion regulation and gas exchange (Jagoe and Others 1993). At low concentrations, beryllium causes gill damage. At higher concentrations, effects include development of chloride cell apical crypts, increased mucus production, microridge loss, epithelial hyperplasia, and fusions of primary lamellae

(Jagoe and Others 1993). Beryllium water concentrations were found to cause gill abnormalities and damage to the fish species studied (Jagoe and Others 1993).

2.5.5 Birds

No information was identified on the effects of beryllium on birds.

2.5.6 Mammals

The major toxicological effects of beryllium occur when the compound is inhaled from the air and beryllium is then deposited in the lungs of mammals. The mammals that were studied include mice, rats, rabbits, and monkeys. The toxicological effects of beryllium in the lung include pneumonitis, hypersensitivity, and chronic granulomatous pulmonary disease (Amdur and Others 1991).

2.6 CADMIUM

Cadmium is a naturally occurring element. It is used in the production of nickel-cadmium batteries, metal plating, pigments, plastics, synthetics, and alloys. Cadmium in soils may leach into water, especially under acidic conditions (Callahan and Others 1979; Elinder 1985, as cited in ATSDR 1993b), and cadmium-containing soil particles may be distributed into the air or eroded into water (ATSDR 1993b). In the aquatic environment, the bioavailability of cadmium depends on such factors as pH, oxidation-reduction (redox) potential, water hardness, and the presence of other complexing agents. The most bioavailable form of cadmium is the Cd^{2+} ion. An increase in temperature or salinity will increase the bioavailable form of cadmium and as a result, increase the bioaccumulation and toxicity of cadmium to aquatic organisms. A decrease in pH will increase the amount of cadmium ions in water and increase bioaccumulation in aquatic organisms (Sadiq 1992).

Cadmium has no essential biological function and is highly toxic to plants and animals. It is a carcinogen and teratogen and a suspected mutagen. Cadmium is associated with severe sublethal effects on reproduction at relatively low environmental concentrations (Eisler 1985a). Aquatic and terrestrial organisms, at all trophic levels, will bioconcentrate and bioaccumulate cadmium (Eisler 1985a).

Bioconcentration in fish depends on the pH and the organic content of the water (John and Others 1987, as cited in ATSDR 1993b). Although some data suggest that lower trophic levels display biomagnification of cadmium, available data, particularly for animals at the top of the food chain, are inconclusive (Beyer and Others 1996; Gochfield and Burger 1982, as cited in ATSDR 1993b).

2.6.1 Plants

Cadmium is known to be toxic to plants at much lower soil concentrations than other heavy metals. Plants more readily take up cadmium than other metals (US Environmental Protection Agency [EPA] 1981) and as a result, some plants can accumulate high levels of cadmium in developing leaflets (Morishita and Boratynski 1992).

2.6.2 Invertebrates

Very little information was available concerning the effects of cadmium on invertebrates. Some insects can accumulate large quantities of cadmium without observable adverse effects (Jamil and Hussain 1992). Certain insects, such as caddis flies, can accumulate high levels of cadmium in their gill tissue (Sadiq 1992).

2.6.3 Amphibians and Reptiles

No information was identified concerning the effects of cadmium on amphibians and reptiles.

2.6.4 Fish and Aquatic Organisms

Marine organisms appear more resistant to cadmium than are freshwater organisms. Cadmium accumulates in the gill tissue of mussels, the digestive glands of scallops, and the liver and kidney of bony fish and sharks (Loring and Prosi 1986, Bryan and Gibbs 1973, and Grimanis and Others 1978, as cited in Sadiq 1992). In general, however, cadmium accumulates in the liver and kidney of fish (Sindayigaya and Others 1994; Sadiq 1992). Cadmium has been shown to be highly toxic in aquatic environments and has been implicated as the cause of deleterious effects on fish and aquatic organisms,

including increased mortality, respiratory disruptions, altered enzyme levels, abnormal muscular contractions, reduced growth, and reduced reproduction (Eisler 1985a). Cadmium concentrations in water caused damage to the reproductive organs of fish, a decrease in the survival rate of fish embryos, and a reduction of growth rates of fry (EPA 1976). Crustaceans appear to be more sensitive to cadmium concentrations, compared with fish and mollusks (Sadiq 1992), and younger stages of aquatic life appear to be more sensitive to cadmium than adults (Sadiq 1992).

2.6.5 Birds

Sublethal effects in birds include growth retardation, nephrotoxicity, anemia, damage to the testicles and absorptive epithelium of the duodenum, reduced egg production, and effects on calcium absorption (Scheuhammer 1987).

2.6.6 Mammals

In mammalian species, cadmium concentrates in the liver and kidneys and is excreted in the urine at a very slow rate. The acute toxic effects of cadmium given orally include nausea, vomiting, salivation, diarrhea, and abdominal cramps. Immediate death may be caused by shock and dehydration; renal and cardiopulmonary failure may cause death a week or so after ingestion. Chronic toxicity effects of cadmium given orally to rats are decreased motor skills, peripheral neuropathy, weakness, and muscle atrophy. When inhaled, cadmium is a carcinogen that can produce tumors in the lung, trachea, and bronchus.

Cadmium is a known developmental toxin causing teratogenic and mutagenic effects. Parental doses of cadmium have been shown to decrease testosterone and produce adverse effects on the testes and prostate of test animals. Prenatal exposure to cadmium has fetotoxic effects, such as reduced fetal weights (ATSDR 1993b), and can cause adverse effects during development of the lung, brain, testes, eye, and palate (Domingo 1994). It is believed that small amounts of cadmium could affect embryonic DNA and protein synthesis (Domingo 1994).

2.7 CHROMIUM

Chromium is a metal that is listed by EPA as one of 129 priority pollutants (Keith and Telliard 1979), and is considered to be one of the 14 most noxious heavy metals (Jenkins 1981). Chromium also is listed among the 25 hazardous substances thought to pose the most significant potential threat to human health at priority Superfund sites (Department of Health and Human Services and EPA 1987). Chromium received special attention in studies of subsurface agricultural irrigation drainage waters of the San Joaquin Valley of California, because it was determined to be a "substance of definite concern" (Moore and Others 1990).

Chromium can exist in oxidation states ranging from -2 to +6, but is most frequently found in the environment in the trivalent and hexavalent oxidation states (Eisler 1986a). The trivalent and hexavalent forms are the most important, because the +2, +4, and +5 forms are unstable and are rapidly converted to +3, which is oxidized to +6 (Eisler 1986a). Chromium compounds are stable in the trivalent state and occur in nature in this state in ores such as ferrochromite. The hexavalent state is the second most stable state. Although hexavalent chromium rarely occurs naturally, it is produced from anthropogenic sources (ATSDR 1993c). Most of the hexavalent chromium in the environment is a result of domestic and industrial emissions. Interaction of hexavalent chromic oxide, dichromate, or chromate compounds with organic compounds can result in reduction to the comparatively less toxic trivalent form (Eisler 1986a). Hexavalent chromium is often in the form of chromates, dichromates, or chromic acid; most have a yellow color, and all are toxic (Grolier Electronic Publishing 1988; Meyers 1990). Both trivalent and hexavalent chromium occur as dissolved chromium (Hem 1989).

EPA regards all chromium compounds as toxic, although the most toxic and carcinogenic chromium compounds tend to be the strong oxidizing agents, with an oxidation state of +6 (Meyers 1990).

Hexavalent chromium compounds tend to be strong oxidizers and are associated with cancer risk and kidney damage (Meyers 1990). Divalent and trivalent compounds of chromium often (not always) have a lower toxicity or biological hazard associated with them (Moore and Others 1990; Patnaik 1992). The hazards associated with chromium are highly related to chemical speciation (Long and Morgan 1990; Meyers 1990).

The toxic mechanism of action differs for hexavalent chromium as compared to trivalent chromium (Moore and Others 1990). Hexavalent chromium causes cellular damage through its role as a strong oxidizing agent, whereas trivalent chromium can inhibit various enzyme systems or react with organic molecules (Moore and Others 1990). Strong oxidizing agents can cause damage to DNA and many other tissue structures.

As in the case of other metals, the overall hazard presented by chromium may be partly related to the solubility of the specific form of chromium (Meyers 1990). Substances having a low solubility in water often are not as easily absorbed through the gastrointestinal tract, as are those substances with higher solubilities (Meyers 1990). Some hexavalent chromium compounds tend to be more toxic than the trivalent compounds, not only because the oxidizing potential is high, but because some of the hexavalent forms more easily penetrate biological membranes (Eisler 1986a). Trivalent chromium has low toxicity because of poor membrane permeability and noncorrosivity, while hexavalent chromium is highly toxic, because it possesses strong oxidizing characteristics and readily pushes through membranes (National Research Council Canada [NRCC] 1976; Hazardous Substances Database [HSDB] 1999).

Both trivalent and hexavalent chromium are significant from the standpoint of potential impacts to fish and wildlife (Eisler 1986a; Rompala and Others 1984). Most of the notoriety associated with chromium as a potentially harmful environmental contaminant, however, is caused by the toxic, carcinogenic, oxidizing agent and reproductive risk hazards of hexavalent chromium compounds (HSDB 1999; Patnaik 1992; Manahan 1992; Meyers 1990; Jones 1990).

Little is known about the relationship between concentrations of total chromium in the environment and biological effects on the organisms living there (Eisler 1986a). Depending on the physical and chemical state of the chromium, the same elemental concentration has a wide variety of mobilities and reactivates and therefore produces different effects (Eisler 1986a).

Certain hexavalent chromium compounds, when administered through inhalation at high doses, have the potential to induce lung tumors in humans and experimental animals (Jones 1990). However, at low levels of exposure, hexavalent chromium ions are reduced in epithelial lining fluid of the respiratory tract,

blood, and other fluids, before the hexavalent ions can interact with DNA, unless the dose is sufficient to overwhelm the body's reduction capacity (Jones 1990).

Small amounts of trivalent chromium are considered to be essential in animals and humans (HSDB 1999). Trivalent chromium is an essential human and animal nutrient at levels of 50 to 200 micrograms per day (Jones 1990).

Trivalent chromium is the only form of chromium known to play a beneficial, biological role. The form must be supplied as a stable complex, because trivalent chromium exists as a relatively insoluble macromolecule at normal blood pH. The known biological effect of trivalent chromium is the maintenance of normal glucose tolerance (Moore and Others 1990).

Trivalent chromium is an essential element for fungi and vertebrates in general (HSDB 1999; Manahan 1992). Trivalent chromium is considered to be essential for glucose and lipid metabolism in mammals, and a deficiency of it produces symptoms of diabetes mellitus (Manahan 1992; HSDB 1999). Trivalent chromium is essential for the maintenance of normal glucose tolerance in animals and humans, and the factor or group of factors containing trivalent chromium (called glucose tolerance factor [GTF]) has been suggested to be responsible for this favorable action of chromium (HSDB 1999).

2.7.1 Plants

The greatest chromium hazard to plants is posed in acidic, sandy soil with low organic content (HSDB 1999). In plants, chromium interferes with uptake translocation, and accumulation by plant tops of calcium, potassium, magnesium, phosphorus, boron, and copper aggravates iron deficiency chlorosis by interfering with iron metabolism (HSDB 1999).

Chromium is not an essential element in plants. The hexavalent form is more soluble and available to plants than the trivalent form and is considered to be the more toxic form. In soils within a normal Eh and pH range, hexavalent chromium, a strong oxidant, is likely to be reduced to the less-available trivalent chromium, although trivalent chromium may be oxidized to hexavalent chromium in the presence of oxidized manganese (Bartlett and James 1979). In nutrient solution, however, both trivalent and

hexavalent chromium are taken up about equally by plants and are toxic to plants (McGrath 1982).

Hexavalent chromium is more mobile in plants than trivalent chromium, but translocation varies with plant type. After plant uptake, chromium generally remains in the roots because of the many binding sites in the cell wall, particularly the trivalent chromium ions (Smith and Others 1989). Symptoms of toxicity include stunted growth, poorly developed roots, and leaf curling.

2.7.2 Invertebrates

Little information was identified on the effects of chromium on invertebrates. Polychaete worms, clams, crabs, and oysters have been shown to take up chromium; excess chromium in these species leads to decreased weight gain, increased oxygen consumption, impaired reproduction, and increased hematocrit (Moore and Others 1990).

2.7.3 Amphibians and Reptiles

No information was identified on the effects of chromium on amphibians and reptiles.

2.7.4 Fish and Aquatic Organisms

Chromium toxicity to aquatic biota is influenced significantly by abiotic variables such as water hardness, temperature, pH, and salinity. Biological factors, such as species, life stage, and potential differences in sensitivities of local populations, influence the susceptibility of the organisms to chromium toxicity.

It has been reported that freshwater fish seem to be relatively tolerant of chromium (EPA 1988; Flora and Others 1984).

Rainbow trout exposed to excessive hexavalent chromium developed severe gill damage, precipitated by hypertrophy and hyperplasia (Moore and Others 1990). Toxicity in aquatic species is primarily affected by water hardness, pH, temperature, species, and organism size (Moore and Others 1990). Hard water conditions promote the toxicity of hexavalent chromium (Moore and Others 1990).

2.7.5 Birds

No information was identified on the effects of chromium on birds.

2.7.6 Mammals

In mammals, trivalent chromium is an essential nutrient. Adequate trivalent chromium nutrition improves growth and longevity and, along with insulin, helps to maintain correct glucose, lipid, and protein metabolism (NRCC 1976; HSDB 1999). The biologically active form of chromium, called the GTF, is a complex of chromium, nicotinic acid, and possibly, amino acids (ATSDR 1993c).

Chromium is considered to be a significant potential threat to human health. However, in trace amounts, chromium is considered to be one of the least toxic elements, because normal stomach pH converts hexavalent chromium to trivalent chromium (Moore and Others 1990). One to two hundred times the normal, total body load of chromium usually can be tolerated in mammals without evidence of negative effects (Moore and Others 1990). The therapeutic dose to toxic dose ratio for trivalent chromium in rats has been calculated at about 1:10,000 (Moore and Others 1990).

Studies with mammals have suggested that trivalent chromium is not well absorbed from the intestinal tract. For example, rat studies have indicated that only a few percent of an oral chromium +3 dose crosses the intestinal wall, regardless of previous dietary history. However, in studies of small intestinal absorption in black ducks (*Anas rubripes*), Eastin and Others (Eisler 1986a), measured equal rates of absorption of trivalent hexavalent chromium. Also, it was noted that the ionic form of chromium influenced the degree of its absorption, with anionic chromium complexes being better absorbed (Moore and Others 1990).

Although trivalent chromium is an essential nutrient, exposure to high levels through inhalation, ingestion, or dermal contact may cause some health effects (ATSDR 1993c). In general, the toxicity of trivalent chromium to mammals is low, because membrane permeability is poor and it is noncorrosive (Eisler 1986a). However, chromium deficiency is unknown, and too much chromium can be harmful to humans (American Medical Association 1989).

2.8 COBALT

Sources of cobalt in the environment are both natural and anthropogenic. Cobalt is naturally present in soil, fresh water, and seawater. Anthropogenic sources of cobalt include fossil fuel burning, vehicular and aircraft exhaust, processing of cobalt-containing alloys and chemicals, sewage sludge, fertilizers derived from phosphate rocks, and copper and nickel smelting and refining (ATSDR 1992a).

Most of the cobalt emitted into the environment settles into the soil or sediment. Cobalt mobility is dependent on the amount of chelating agents in the soil, the pH, and the redox potential of the soil. Lower pH levels increase the mobility of cobalt in the soil. Metal oxides, crystalline minerals, and natural organic matter in the soil decrease the mobility of cobalt. Cobalt is slightly mobilized by normal weathering (pH 5 to 8), moderately mobile in the presence of oxidizing sulfide ore, and immobile in organic-rich environments (Perelman 1967).

In most fresh water, less than 2 percent of cobalt species are present in the dissolved form in the water column, while the rest is precipitated or adsorbed on suspended solids and sediments. Under acidic conditions and in the presence of excess chloride ions or organic and inorganic chelating agents, some mobilization of cobalt from the sediments may occur (ATSDR 1992a). In these conditions, the formation of cobalt complexes may increase the amount of cobalt in the water column.

Microorganisms, higher plants, and animals accumulate traces of cobalt. Although essential, excessive intake results in accumulation and toxicity (Considine 1976). Mollusks, crustaceans, and other bottom feeders have been reported to accumulate large quantities of cobalt (Jenkins 1980). However, a study of organisms in Ottawa River sediments showed no detectable bioaccumulation of cobalt-60 (Evans and Others 1988). Bioaccumulation factors for cobalt on a dry-weight basis were 100 to 4,000 for marine fish and 40 to 1,000 for freshwater fish (Smith and Carson 1981). The concentration of cobalt in normal rat tissues has been shown to range from 0.001 to 0.006 ppm of dry matter (Considine 1976). Cobalt appears not to bioaccumulate significantly in benthic bottom feeders in comparison to its concentration in sediment (ATSDR 1992a).

Plant uptake of cobalt from soil is not appreciable. In highly acidic soils and in some higher plants, however, significant uptake has been observed (ATSDR 1992a). The translocation of cobalt from roots to aboveground parts of plants is not significant in most soils. The soil to plant BCF for cobalt ranges from 0.01 to 0.3 (ATSDR 1992a). Plant uptake of cobalt is enhanced by low soil pH (HSDB 1999) but inhibited by complexes formed by organic complexing agents such as (EDTA) in soil (Killey and Others 1984; McLaren and Others 1986). There is little biomagnification of cobalt in animals at higher trophic levels (Jenkins 1980).

2.8.1 Plants

At elevated levels, cobalt is a phytotoxin. However, certain plants are known to develop a mechanism of cobalt tolerance and can grow on serpentinite or ore bodies (Kabata-Pendias and Pendias 1992).

2.8.2 Invertebrates

No information was identified on the effects of cobalt on invertebrates.

2.8.3 Amphibians and Reptiles

No information was identified on the effects of cobalt on invertebrates.

2.8.4 Fish and Aquatic Organisms

Very little information was available on the effects of cobalt on fish and aquatic organisms. Because of a lack of toxicological data, EPA has not determined an ambient water quality criterion for cobalt (Diamond and Others 1992).

2.8.5 Birds

No information was identified on the effects of cobalt on birds.

2.8.6 Mammals

Cardiomyopathy is the primary adverse effect of cobalt in acutely or chronically exposed animals (ATSDR 1992a). Additional studies on animals suggest that exposure to high amounts of cobalt during pregnancy affect fetal health.

Inhalation of cobalt by animals results in respiratory, cardiovascular, hematological, hepatic, renal, ocular, and body weight effects. Short-term exposure of rats to high levels of cobalt in air results in lung damage and death. Short-term exposure of rats, guinea pigs, hamsters, and pigs to lower levels of cobalt in air results in lung damage and increased red blood cells.

Testicular atrophy was reported in rats and mice exposed to cobalt concentrations in air (ATSDR 1992a). Similarly, testicular degeneration and atrophy have been reported in rats exposed to cobalt concentrations in water (ATSDR 1992a).

Stunted growth and decreased survival of offspring is noted in study rats exposed to cobalt in their diet from the third trimester of gestation through lactation (Domingo and Others 1985). Rodent fetal growth and development, however, were not adversely affected by gestation-only exposure to high cobalt levels (Paternain and Domingo 1988; Seidenberg and Others 1986), so the stunted growth and decreased survival of offspring may be attributable to cobalt transfer through milk.

The rate of absorption of ingested cobalt is variable, depending on such factors as chemical form, nutritional state of body, and preexisting foods in the stomach (ATSDR 1992a). In several studies rats absorbed 30 to 40 percent of the administered dose of cobalt (Hollins and McCulloch 1971; Taylor 1962).

2.9 COPPER

Copper is a naturally occurring element that is widely distributed in the environment. Copper is the main component of alloys, which include brass, bronze, and gunmetal. Copper is an essential trace mineral nutrient and a toxicant (ATSDR 1990b).

Copper is very mobile under oxidizing and acidic conditions and immobile in organic-rich and reducing environments. Adsorption increases with pH and higher organic matter content. In aquatic systems, copper binds primarily to organic matter and forms complexes with both organic and inorganic ligands (mainly with calcium carbonate) that settle out in sediments (Kirk-Othmer 1965). Under normal pH and redox conditions, copper tends to be present in sediments in the form of organic complexes and coprecipitates with iron and manganese oxides and cupric carbonate complexes.

Copper is an essential nutrient and is homeostatically controlled; therefore, biomagnification is not a significant fate process for copper. Bioaccumulated copper is stored in the liver, kidney, bone marrow, and hair (Talmage and Walton 1991). Fish can bioconcentrate copper, with BCFs ranging from the tens to the hundreds. Mollusks have BCFs for copper that range up to 30,000 (Perwack and Others 1980; Chapman and Others 1968; Raymont 1972).

2.9.1 Plants

Based on yield reductions of 14 to 28 percent in agronomic and grassland plants, 100 milligrams per kilogram (mg/kg) of total copper in the soil is considered to be a threshold concentration for toxicity to plants (International Copper Association [ICA] 1992).

Copper is a micronutrient essential for plant nutrition and is required as a cofactor for many enzymes. Furthermore, it is an essential part of a copper protein involved in photosynthesis. Root absorption appears to be passive, perhaps in organo-copper complexes (Jarvis and Whitehead 1983), and active through a specific carrier (Fernandes and Henriques 1991). When copper is adsorbed to cells in the root system, it may result in low-copper soils. The form in which copper is taken into the root affects its binding (Wallace and Romney 1977). Copper can be transported in the xylem and phloem of plants complexed with amino acids.

The most common toxicity symptoms include reduced growth, poorly developed root systems, and leaf chlorosis (Wong and Bradshaw 1982). The basic deleterious effect of copper is related to the root system, where it interferes with enzyme functioning (Mukherji and Das Gupta 1972), but it also strongly interferes with photosynthesis and fatty acid synthesis (Smith and Others 1985).

2.9.2 Invertebrates

Copper is used as an ingredient in many fungicides and insecticides applied to agricultural crops (Meister 1995). Based on yield reductions of 14 to 28 percent in agronomic and grassland plants, 100 mg/kg of total copper in the soil is considered to be a threshold concentration for toxicity to soil invertebrates (ICA 1992).

2.9.3 Amphibians and Reptiles

Copper is highly toxic to amphibians. Environmental conditions, including pH and water hardness, and the life stage of the amphibian exposed affect the organism's sensitivity and adverse response to exposure to metal concentrations in water. Copper increased rates of mortality in high-pH and low, water hardness aquatic environments (Horne and Dunson 1995). Although information concerning amphibians and metal toxicity is limited, it is believed that the primary mechanism of action of metal-induced toxicity and low pH environments is body loss of sodium across the gill surface (Horne and Dunson 1995). Tadpoles were affected adversely when exposed to aqueous copper concentrations (Owen 1981). Copper is toxic to certain types of frogs and salamanders during both acute and chronic exposure studies, causing embryonic curling, body loss of sodium, and mortality (Horne and Dunson 1995). Earlier life stages of amphibians appear to be more sensitive to copper toxicity than later life stages (Horne and Dunson 1995).

No information was identified on the effects of copper in reptiles.

2.9.4 Fish and Aquatic Organisms

Copper is highly toxic in aquatic environments and is a priority pollutant (EPA 1992). Copper is toxic to many fish and aquatic organisms, including mussels, striped bass, bluegill, and carp. Copper is mainly accumulated in the gill, liver, filaments, stomach, and intestine; however, the gill is the primary organ for concentrating copper in aquatic organisms (Owen 1981). Copper concentrations can significantly affect fish egg hatchability and reduce fry growth (EPA 1976). The age and species of organism influence the

toxicity characteristics of copper. In general, younger organisms are affected at lower concentration levels.

2.9.5 Birds

Copper can produce toxic effects in birds. Diets containing elevated copper levels can slow the growth rate, diminish egg production, and cause developmental abnormalities in different avian species (Owen 1981).

2.9.6 Mammals

Toxic effects of copper have been studied on many animals, including cats, dogs, cattle, sheep, rats, mice, horses, guinea pigs, pigs, and monkeys. Different species of animals display varying levels of sensitivity to copper. However, the main organ affected by exposure to copper is the liver, where copper primarily accumulates in subcellular organelles, causing liver cirrhosis. In addition to liver cirrhosis, copper exposure can cause necrotic kidney tubules and brain damage (Owen 1981). Acute, toxic effects of copper given orally include gastrointestinal irritation, vomiting (including blood), low blood pressure, jaundice caused by liver necrosis, and coma. Chronic exposure to copper can cause accumulation of copper in the body, leading to lesions in the liver, brain, and eye and hemolytic anemia.

2.10 LEAD

Lead has been characterized as a poison for centuries and environmental pollution from lead is well documented (Eisler 1988). Lead has been used in the production of solder, pipes, paint, ceramics, roofing materials, caulking, and ammunition and also was used as a gasoline additive. From a geochemical perspective, lead is ubiquitous and occurs in rocks, soils, water, plants, animals, and air. Lead is neither essential nor beneficial to living organisms, and all data show that the biological effects are adverse. Lead is a mutagen and a teratogen. When absorbed in excessive amounts, lead also is carcinogenic or cocarcinogenic, impairs reproduction, adversely impairs liver and thyroid functions, and interferes with resistance to infectious disease (Eisler 1988). In general, lead is toxic in most of its chemical forms and can be incorporated into the body through inhalation, ingestion, dermal absorption, and placental transfer.

Lead is a poison that accumulates in the body and upon reaching a certain level; it will affect behavior, as well as hematopoietic, vascular, nervous, renal, and reproductive systems (Eisler 1988).

The biological availability and fate of lead in soil is affected by such factors as soil pH, organic content, ion-exchange characteristics, and the amount of lead in the soil (National Science Foundation 1977, as cited in ATSDR 1999a). Plants and animals may bioconcentrate and bioaccumulate lead; however, biomagnification has not been well documented. Several studies have shown that invertebrates can accumulate lead in their tissues; however, the variability in the extent of lead bioaccumulation suggests that the mechanisms of lead uptake range between species. Organolead compounds, such as trialkyl and tetraalkyl lead compounds are more toxic than inorganic forms and have been shown to bioconcentrate in aquatic organisms. High accumulations of lead from ambient seawater by marine plants are well documented. Although lead is bioconcentrated from water, little evidence suggests that it is transferred through the food chain (Wong and Others 1978, Branica and Konrad 1980, and Settle and Patterson 1980, as cited in Eisler 1988). Lead concentrations tend to decrease with increasing trophic level in food chains in freshwater and marine habitats (Wong and Others 1978 and Stewart and Schulz-Baldes 1976, as cited in Eisler 1988).

2.10.1 Plants

Lead is not essential for plants, and excessive amounts can cause growth inhibition as well as a reduction in photosynthesis, mitosis, and water absorption (Demayo and Others 1982, as cited in Eisler 1988). Roots take up lead passively, and translocation to shoots is limited (Wallace and Romney 1977). It is bound to the outside of roots, in the apoplast, and in cell walls and organelles or absorbing roots (Koepe 1981). In the plant, lead may exist in a naturally chelated form or in pyro- or orthophosphate forms. The phytotoxicity of lead is relatively low compared with other trace elements. It affects mitochondrial respiration and photosynthesis by disturbing electron transfer reactions (Miles and Others 1972).

2.10.2 Invertebrates

A concentration of 12,800 mg/kg of lead in soil is associated with reductions in natural populations of decomposers such as fungi, earthworms, and arthropods (Eisler 1988).

2.10.3 Amphibians and Reptiles

Environmental conditions, including pH and water hardness, and the life stage of the amphibian exposed, affect the organism's sensitivity and adverse response to exposure to metal concentrations in water. Although information concerning amphibians and metal toxicity is limited, the primary mechanism of action of metal-induced toxicity in low pH environments is believed to be body loss of sodium across the gill surface (MacDonald and Wood 1993, as cited in Horne and Dunson 1995). Reduced rates of learning acquisition and retention were observed in tadpoles exposed to lead water concentrations. Limited evidence has been published on the effects of lead in amphibians; however, lead may be important as a toxicant to developing embryos (Horne and Dunson 1995).

2.10.4 Fish and Aquatic Organisms

Lead is toxic to most aquatic organisms; however, adverse effects are modified by environmental conditions. Fish continuously exposed to toxic concentrations of dissolved lead show various signs of poisoning, including spinal curvature, anemia, degeneration of the caudal fin, destruction of spinal neurons, reduced ability to swim against a current, destruction of the respiratory epithelium, muscular atrophy, paralysis, renal pathology, growth inhibition, retardation of sexual maturity, testicular and ovarian histopathology, decreased fry survival rate, and death (Eisler 1988; EPA 1976).

2.10.5 Birds

Absorbed lead produces a variety of effects in avian species, including damage to the nervous system, muscular paralysis, inhibition of heme synthesis, damage to kidneys, damage to the liver, and death (Mudge 1983, as cited in Eisler 1988). Sublethal lead exposure also may have adverse effects of reproduction in some avian species by decreasing plasma calcium, inhibition of growth, and reduced chick hatchability.

2.10.6 Mammals

Lead can have multiple effects in mammalian species. Lead may cause damage to the nervous system, hematological effects, kidney dysfunction, sterility, abortion, neonatal mortality, growth retardation, delay in maturation, and reduced body weight (Amdur and Others 1991; Eisler 1988). Younger mammals may have greater sensitivity to lead toxicity because of their developing blood brain barrier. Developing capillaries in the brain allow lead levels in the blood to be transported to newly formed components of the brain (Amdur and Others 1991).

2.11 MANGANESE

Manganese is a naturally occurring substance found in many types of soil. Manganese is an essential trace mineral with many oxidation states. Although its primary use is in iron and copper alloys, it is also used in dry cells, matches and pyrotechnics, fossil fuels, and a variety of chemical processes.

Manganese metal has four different oxidation states (2+, 3+, 4+, and 7+). The specific chemical form is determined by environmental factors, such as pH, Eh, microbial activity, and available anions (ATSDR 1991). The most common form of manganese is present in the divalent form (Mn^{2+}) (EPA 1984, as cited in ATSDR 1991). The ability of manganese to adsorb to soil and sediment depends on the organic content and the ion exchange capacity of the soil or sediment (Curtin and Others 1980; Hemstock and Low 1953; Kabata-Pendias 1984, McBride 1979 and Schnitzer 1969, as cited in ATSDR 1991). As a result, soil adsorption values range from 0.2 to 10,000 milliliters per gram (mL/g).

Manganese in water may be bioconcentrated by lower trophic levels (ATSDR 1991). Manganese BCFs have been reported as 2,500 to 6,300 for phytoplankton; 300 to 5,500 for marine algae; 800 to 830 for intertidal mussels; and 35 to 930 for coastal fish (Folsom and Others 1963, as cited in ATSDR 1991). In another study, BCFs similarly were reported as 10,000 to 20,000 for marine and freshwater plants, 10,000 to 40,000 for invertebrates, and 100 to 600 for fish (Thompson and Others 1972, as cited in ATSDR 1991). Although bioconcentration occurs in lower trophic levels, homeostasis is reached in higher-trophic-level receptors, and as a result, biomagnification of manganese in the food chain is believed to be insignificant (ATSDR 1991).

2.11.1 Plants

Manganese concentrations were found to be higher in the sprout stage than in the adult stage of *Spartina alterniflora* (Williams and Murdoch 1969). However, manganese concentrations increased to the highest levels after death. These concentrations and related concentration factors were similar to those found in terrestrial monocots, marine algae, and submerged grasses (Williams and Murdoch 1969).

2.11.2 Invertebrates

No information was identified on the effects of manganese on terrestrial invertebrates.

2.11.3 Amphibians and Reptiles

Very little information was identified on the effects of manganese on amphibians and reptiles. In one study, however, manganese has been observed to alter membrane sodium permeability in amphibians (Arhem 1980, as cited in Power and Others 1989). In adult frogs, the highest concentration of manganese was found in the skin (Baudo 1976, as cited in Power and Others 1989).

2.11.4 Fish and Aquatic Organisms

Reproductive effects, such as a decrease in spermatogenic activity and injury to testes, have been recorded in fish treated with manganese sulfate (Srivastava and Agrawal 1983, as cited in Joardan and Sharma 1990).

2.11.5 Birds

Manganese levels in birds were higher in bones, livers, and kidneys than in the brain, heart, or muscle (Mahoney 1978). High levels of manganese caused decreased hemoglobin and liver iron levels, mild anemia, and depressed growth in chicks (Southern and Baker 1983). Reproductive effects were minimal in birds fed 90 times the normal dietary level of manganese (Laskey and Edens 1985).

2.11.6 Mammals

Symptoms observed in rats that ingested elevated levels of manganese included a decrease in weight gain, increase in physical activity, alterations of brain chemicals, slight irritation of the stomach, and delayed testicular development (ATSDR 1991). In addition, pregnant rats that drank water containing elevated levels of manganese were observed to bear litters that weighed less than normal (ATSDR 1991).

Monkeys that drank water containing elevated levels of manganese became weak and their muscles became rigid (ATSDR 1991).

Rats and monkeys that inhaled elevated concentrations of manganese dust developed irritation and swelling in the lungs (ATSDR 1991). Monkeys also developed chemical alterations in the brain as a result of manganese inhalation (ATSDR 1991). Mice that were exposed to elevated concentrations of manganese in the air were more likely to catch pneumonia and develop behavioral changes (ATSDR 1991). The parents who inhaled air concentrations of manganese had increased behavioral malformations and smaller offspring than those of the control group (ATSDR 1991).

2.12 MERCURY

Inorganic and organic forms of mercury are relatively well characterized within an ecotoxicological context. Methyl-mercury and other organomercurial compounds (including ethyl- and phenyl-mercuricals) are among the most toxic organometals in the environment and bioaccumulate through the food chain (Fimreite 1979). The chemical form of mercury influences its distribution in the body, and comparative toxicity data suggest that the organic species (ethyl-, methyl-, and phenyl-mercury) of the metal are far more toxic than inorganic mercury (Peterle 1991). In general, methyl-mercury, as well as its metallic (Hg^0), mercurous (Hg^+), and mercuric (Hg^{++}) forms, are nonessential and exert their toxicity at the biochemical level as inhibitors of enzyme-catalyzed reactions.

Mercury is a known mutagen, teratogen, and carcinogen. Its toxicity and environmental effects vary with its form, dose, and route of ingestion and with the species, sex, age, and general condition of the organism

(Eisler 1987a; Fimreite 1979). Depending on the pH, salt content, and composition of the soil, mercury usually forms various complexes with chloride and hydroxide ions in the soil (Alloway 1990).

Biotransformation is an important fate process in the environmental partitioning of mercury. Under favorable conditions, microorganisms in soil and sediment can convert various forms of mercury to methyl-mercury, which is more available for uptake by various organisms and for transport in the food chain and more mobile than inorganic forms (Peterle 1991).

Mercury has a high potential for bioaccumulation and biomagnification (Eisler 1987a). Methylated mercury is the form most readily bioconcentrated and bioaccumulated (Kramer and Neidhart 1975). Biomagnification of methyl-mercury has been documented for both aquatic and terrestrial food chains (Eisler 1987a). Concentrations of methyl-mercury in upper trophic-level fish reportedly have been biomagnified on the order of 10,000 to 100,000 times those concentrations found in ambient waters (Callahan and Others 1979). The accumulation of mercury by aquatic organisms is enhanced at elevated water temperatures, reduced water salinity or hardness, reduced water pH, increased age of the organism, and reduced organic matter in the medium. Mercury transfers and biomagnification through mammalian food chains is well documented.

2.12.1 Plants

Plants take up mercury from the soil in relatively insignificant amounts because roots appear to act as a barrier. However, mercury compounds applied to other parts of plants appear to be readily absorbed and translocated (Adriano 1986).

Mercury and its compounds taken up by roots are translocated to only a limited extent in plants. Organic forms of mercury may be translocated to a greater degree than inorganic forms in some plants (Huckabee and Blaylock 1973). Gay (1975) reports that pea plants (*Pisum sativum*) form methyl-mercury as an intermediate product from mercury added to the soil in organic and inorganic forms.

2.12.2 Invertebrates

Some types of invertebrates can accumulate mercury through the food chain with little or no observable effects (Jamil and Hussain 1992). In certain types of flies, however, methyl-mercury can alter chromosomes, causing abnormalities in offspring (NAS 1978 and Khera 1979, as cited in Eisler 1987a). Mercury concentrations in soil have been observed to cause reduced segment regeneration in worms, and at elevated levels, mortality (Abbasi and Soni 1983, as cited in Eisler 1987a). For marine invertebrates, mercury was observed to inhibit reproduction (Eisler 1987a).

2.12.3 Amphibians and Reptiles

Little information was identified on the effects of mercury on amphibians and reptiles. In one study, frogs did not metamorphose when exposed to mercury concentrations (Eisler 1987a).

2.12.4 Fish and Aquatic Organisms

Methyl-mercury appears to be the most toxic form of mercury (MacDonald 1993). Earlier developmental stages of fish and aquatic organisms are more sensitive to mercury toxicity than more mature individuals. Effects of chronically exposed fish and aquatic organisms include loss of appetite, brain lesions, cataracts, abnormal motor coordination, and behavioral changes. In addition to chronic symptoms of mercury toxicity, mercury at comparatively low concentrations adversely affects reproduction, growth, behavior, metabolism, blood chemistry, osmoregulation, and oxygen exchange of marine and freshwater organisms (Eisler 1987a). Mercury is very toxic to aquatic organisms because of its ability to bind to sulphhydryl groups (Sindayigaya and Others 1994). Degree of toxicity is increased in elevated water temperatures and in reduced salinity environments (Eisler 1987a).

2.12.5 Birds

Sublethal effects of mercury on birds, administered by a variety of routes, include adverse effects on growth, development, reproduction, blood and tissue chemistry, metabolism, and behavior (Eisler 1987a). Reproductive effects are noted at low doses long before acute effects are noticeable in exposed adult

populations (Scheuhammer 1987). Significant reproductive effects of chronic dietary inorganic mercury exposure in birds include delayed testicular development, altered mating behavior, reduced fertility, reduced survivability and growth in young, and gonadal atresia. Mercury also is transferred to the egg in avian species, where it has adverse effects on the developing embryo (Peterle 1991).

2.12.6 Mammals

Methyl-mercury is the more acutely toxic form of mercury. Mercury can cause adverse neurological, renal, behavioral, and reproductive effects in mammals. Nephrotoxicity is the most common effect mercury has on mammals. Acute toxicity responses to the organic form of the metal include ataxia, aphagia, tremors, and diminished capacities for coordinated movements. In chronic exposures, methyl-mercury intoxication is characterized by central nervous system and peripheral nervous system neuropathies (Linstrom and Others 1991, as cited in ATSDR 1994a). Methyl-mercury also exhibits reproductive effects in both sexes as well as in the developing embryo and fetus (Cagiano and Others 1990). Methyl-mercury can cause other reproductive effects, which include diminished neurological function and behavioral deficits in newborns (Khera and Tabacova 1973).

2.13 MOLYBDENUM

Molybdenum is found in all living organisms and is considered to be an essential or beneficial nutrient. This element is used primarily in the production of steel alloys, which are used in building aircraft and weapons. Fossil fuel combustion, smelting, mining, and milling operations have all contributed to molybdenum contamination in the environment. Molybdenum chemistry is complex and not adequately understood. In water at a pH greater than 7, molybdenum exists primarily as the molybdate ion; at a pH of less than 7, various polymeric compounds are formed, including the paramolybdate ion (Busev 1969, as cited in Eisler 1989). In soils, molybdate is sorbed most readily to alkaline, high-calcium, and high-chloride soils.

Molybdenum interacts toxicologically with other trace elements, especially copper and inorganic sulfates. In mammals, molybdenum can protect against poisoning by copper, mercury, and probably other metals

and may have anticarcinogenic properties. However, ruminants are sensitive to molybdenum poisoning, particularly when accompanied by a deficiency in copper or inorganic sulfates (Eisler 1989).

Little information was found on the bioaccumulative potential of molybdenum.

2.13.1 Plants

Plants readily accumulate molybdate, except in soils with low pH, high-sulfate, low-phosphate, and high-organic-matter content (Gupta and Lipsett 1981, as cited in Eisler 1989). No toxicity of molybdenum to field-grown crops has been observed (Soon and Bates 1985, as cited in Eisler 1989).

Molybdenum is required for symbiotic nitrogen fixation by legumes and for growth of nonleguminous plants. The most important functions of molybdenum in plants are related to enzymes active in nitrogen metabolism (activation of nitrogenase and nitrate reductase). The majority of molybdenum taken up by the root system tends to remain in the roots, although significant amounts may be translocated to the shoots in some cases (Wallace and Romney 1977). Toxicity symptoms include chlorosis, apparently caused by interference with iron metabolism (Warington 1954).

2.13.2 Invertebrates

Molybdenum compounds have been used in baits to control termites. These molybdenum baits were fatal to the termite population; however, other species of insects, including fire ants, beetles, and cockroaches, were unaffected by the bait (Brill and Others 1987, as cited in Eisler 1989).

2.13.3 Amphibians and Reptiles

No information was identified on the effects of molybdenum on amphibians and reptiles.

2.13.4 Fish and Aquatic Organisms

Little information was identified on the effects of molybdenum on aquatic invertebrates and fish. Freshwater and marine fish appear to be relatively resistant to molybdenum exposure (Eisler 1989). However, rainbow trout eggs exposed to molybdenum for 28 days through day 4 posthatch caused mortality at elevated levels.

2.13.5 Birds

Data are scarce on the effects of molybdenum on avian wildlife under controlled conditions. All studies on birds have been restricted to domestic poultry, which appear to be relatively resistant to molybdenum exposure. At elevated levels, however, molybdenum has been observed to decrease the growth rate of chicks and turkey poults, reduce egg production, and decrease hatchability (Friberg and Others 1975, as cited in Eisler 1989).

2.13.6 Mammals

Molybdenum is essential in mammalian diets, can protect against poisoning by copper or mercury, and may be useful in controlling cancer. Almost all studies conducted to date on the effects of molybdenum under controlled conditions have been on livestock, particularly cattle and sheep. Molybdenum poisoning in ruminants has been observed in livestock (Eisler 1989). In certain areas of England, cattle and sheep that grazed in fields that had high levels of molybdenum and low levels of copper and inorganic sulfate developed molybdenosis (Eisler 1989). Symptoms of molybdenosis include weight loss, diarrhea, and death, in extreme cases. Data on the effects of molybdenum on mammalian wildlife are scarce. All evidence indicates that mammals other than cattle and sheep are comparatively tolerant of high dietary intakes of molybdenum (Underwood 1971, Buck 1978, Chappell and Others 1979, and Friberg and Lener 1986, as cited in Eisler 1989).

2.14 NICKEL

Nickel is a naturally occurring metal. Nickel is mined for use in electroplating, iron and steel processing, nickel-cadmium batteries, fuel combustion, and a variety of other applications.

Nickel has many oxidation states: -1, 0, +1, +2, +3, and +4. Nickel⁺², however, is the most common form present in the environment (Environment Canada 1994a). Nickel is strongly sorbed by soil, although to a lesser degree than lead, copper, and zinc (Rai and Zachara 1984; Alloway 1990). Nickel adsorption depends strongly on pH (Rai and Zachara 1984; Alloway 1990). Many forms of nickel are found in soil, and many factors affect the extent to which these different forms of nickel are adsorbed, making nickel adsorption highly site-specific. Amorphous oxides of iron, manganese, and clay minerals are the most important adsorbents of nickel in soil. In alkaline soils, adsorption may be irreversible, thereby limiting nickel's availability and mobility. Cations, such as calcium and magnesium, reportedly reduce adsorption as a result of competition for binding sites, whereas anions, like sulfate, reduce adsorption because of complexation. In an aquatic environment, environmental factors, such as pH, oxidation-reduction (redox) potential, and organic matter content, can affect the fate, transport, and biological availability of nickel (Environment Canada 1994a).

BCFs for nickel have been reported between 120 and 550 for submerged lichens; 770 and 1,500 for submerged mosses; 2,000 and 4,500 for *Daphnia*; 200 to 1,000 for clams, zooplankton, and benthos; and 230 and 330 for fish (Dietz 1973, Cowgill 1976, Mathis and Cummings 1973, Hutchison and Others 1976, as cited in Environment Canada 1994a). Recent studies of nickel levels in voles and rabbits living on sludge-amended land did not indicate any accumulation of nickel in these herbivores or in the plants on which they fed (Alberici and Others 1989, Dressler and Others 1986, as cited in ATSDR 1993d). Animals appear to be able to regulate the amount of nickel that is accumulated in the body and as a result, biomagnification in upper trophic levels is believed to be insignificant (Environment Canada 1994a).

2.14.1 Plants

Nickel tends to be less available in soils than zinc or cadmium but is generally more available to plants than copper (Alloway 1990). The soil chemistry of nickel is relatively simple and is largely based on its

occurrence as divalent nickel²⁺. Nickel becomes more soluble with decreasing pH, allowing more nickel to be bioavailable to plants. Clay content and texture of the soil also will influence bioavailability of nickel to plants. Depending on these factors and on nickel's biological role as an ultra-trace element, nickel is bioconcentrated by plants; the greatest bioconcentration occurs in soils that have naturally high levels of nickel, where some plants are classified as "hyperaccumulators" (Alloway 1990). Similar patterns of nickel bioconcentration also occur in highly contaminated soils (for example, near nickel and copper smelters).

Effects of nickel toxicity on plants include reduced growth of roots and shoots, poor branching, deformation of plant parts, decreased dry matter production, leaf spotting, abnormal flower shape, mitotic root-tip disturbance, germination inhibition, and chlorosis (Mishra and Kar 1974, Rauser 1978, McIlveen and Negusanti In Press, as cited in Environment Canada 1994a).

2.14.2 Invertebrates

Little information was identified on effects of nickel on invertebrates. However, microorganism growth and survival were reduced as a result of elevated nickel exposure (Babich and Stotzky 1982, as cited in Environment Canada 1994a).

2.14.3 Amphibians and Reptiles

Nickel exposure in amphibians has been observed to slow down the response time of the potassium system in myelinated nerve fibers (Arhem 1980, as cited in Power and Others 1989). In addition, nickel also has been observed to alter sodium permeability across cell membranes in amphibians (Arhem 1980, as cited in Power and Others 1989).

2.14.4 Fish and Aquatic Organisms

Nickel is both a carcinogen and a mutagen in the aquatic environment (EPA 1992). Observed effects of nickel exposure in an aquatic environment to fish and invertebrates include tissue damage, genotoxicity, and decreased growth (Environment Canada 1994a). Mollusks and crustaceans appear to be more sensitive to nickel exposure than other aquatic organisms (Environment Canada 1994a).

2.14.5 Birds

Elevated dietary concentrations of nickel caused growth inhibition in poultry. The expression of nickel toxicity is influenced by the age, reproductive status, nutritional content of the diet, and exposure duration in test organisms. This information should be considered in the interpretation of the risk posed by nickel to terrestrial wildlife (National Academy of Sciences [NAS] 1980).

No effects were observed on adult mallards exposed to nickel in their diet. No effects to adult mallards were observed regarding body weight, histological changes in liver and kidneys, tissue damage, blood chemistry, egg-laying ability, hatchability percentages, and hatchling survival to 14 days of age (Eastin and O'Shea 1981, as cited in Environment Canada 1994a). However, mallard ducklings fed diets containing elevated nickel concentrations up to 90 days were observed to develop tremors and ataxia (Cain and Pafford 1981, as cited in Environment Canada 1994a). Newly hatched chickens also were observed to have slower growth rates as a result of diets containing elevated nickel concentrations (Ling and Leach 1979, as cited in Environment Canada 1994a).

2.14.6 Mammals

Growth inhibition was the primary effect to domestic livestock fed elevated levels of nickel in their diet. The expression of nickel toxicity was influenced by the age, reproductive status, nutritional content of the diet, and exposure duration in test organisms. This information should be considered in the interpretation of risk posed by nickel to terrestrial wildlife (NAS 1980).

Rats, in a subchronic gavage study of nickel chloride in water, experienced lethargy, ataxia, irregular breathing, reduced body temperature, and discolored extremities (EPA 1999). Inhalation of nickel subsulfide in rats increased the incidence of lung tumors (ATSDR 1993d). The central nervous system appears to be the target organ for nickel oral toxicity, while the lung is the target organ for inhalation exposure.

2.15 SELENIUM

Selenium is an essential trace element but is harmful at concentrations only slightly higher than the nutritional requirement (Eisler 1985b). Results of laboratory studies and field investigations with fish, mammals, and birds have led to general agreement that elevated concentrations of selenium in diet or water are associated with reproductive abnormalities and growth retardation. Not as extensively documented, are reports of selenium-induced chromosomal aberrations, intestinal lesions, shifts in species composition of freshwater algal communities, swimming impairment of protozoa, and behavioral modifications (Eisler 1985b).

In aerobic waters, selenium is present in the quadrivalent oxidation state as selenite or in the hexavalent oxidation state as selenate. These chemical species are very soluble, and most of the selenium discharged into the aquatic environment is transported in these forms to the ocean. Selenium has a sorptive affinity for hydrous metals oxides, clays, and organic materials. Sorption by sediments or suspended solids can result in enrichment of selenium concentrations in sediment beds. Sorption or precipitation with hydrous iron oxides is probably the major control on mobility of selenium in aerobic waters. Selenium can be methylated by a variety of organisms, including benthic microflora. In a reducing environment, hydrogen selenide may be formed. Both the methylated forms and hydrogen selenide are volatile and may escape to the atmosphere. Formation of volatile selenium compounds in the sediments can remobilize sorbed selenium (Eisler 1985b).

Current understanding of selenium toxicology indicates that ecological effects are primarily caused by selenium in the food chain, rather than selenium dissolved in the water column (Philips 1988, Luoma and Others 1992, as cited in Taylor and Others 1992). Once in the water column, selenium enters the food chain through bioconcentration by phytoplankton, which are then consumed in large quantities by crustaceans and bivalves. Fish and waterfowl, in turn, eat crustaceans and bivalves. Bioconcentration, bioaccumulation, and biomagnification of selenium can increase selenium levels more than 1,000-fold from water to fish and animals (Saiki and Lowe 1987, as cited in Taylor and Others 1992). The greatest step increase occurs between water and phytoplankton and other aquatic plants; subsequent steps in the food chain typically increase selenium concentrations by a factor of 2 to 6 (Lemly and Smith 1987, as cited in Taylor and Others 1992). BCFs for various species of marine algae range from 16,000 to

337,000, depending on the species and water column levels (Zhang and Others 1990, as cited in Taylor and Others 1992).

2.15.1 Plants

Selenium has been observed to cause growth retardation in freshwater green algae (Hutchinson and Stokes 1975, Klaverkamp and Others 1983, as cited in Eisler 1985b).

Selenium is not proven to be essential for plant growth; however, plants will absorb the compound as selenite, selenate, or as an inorganic. Selenate is probably the most toxic form of selenium. It is believed that selenate is taken up actively, while selenite uptake is largely passive (Peterson and Girling 1981). Selenium is translocated to all parts of the plant, including the seed, in low-molecular-weight compounds (Broyer and Others 1972). Toxicity symptoms include chlorosis, stunting, and yellowing of the leaves. The mechanism of toxicity is thought to be indiscriminate replacement of sulphur by selenium in proteins and nucleic acids, with disruptions in metabolism (Trelease and Others 1960).

2.15.2 Invertebrates

No information was identified on the adverse effects of selenium on invertebrates.

2.15.3 Amphibians and Reptiles

Little information was identified on the effects of selenium on amphibians and reptiles. One report states that during frog development, cranial and vertebral deformities and lower survival were documented when exposed to selenium concentrations (Browne and Dumont 1979, as cited in Eisler 1985b).

2.15.4 Fish and Aquatic Organisms

In general, selenite is more toxic to earlier life stages, and the degree of the effect is increased with increasing temperatures (Klaverkamp and Others 1983, as cited in Eisler 1985b). Selenium is teratogenic, and its toxicity depends greatly on its chemical form (Eisler 1985b). It has been suggested that selenite is

more toxic than selenate and is preferentially concentrated over selenate by mussels (EPA 1990, as cited in Eisler 1985b). Signs of selenium poisoning include loss of equilibrium, lethargy, loss of coordination, muscle spasms, protruding eyes, swollen abdomen, liver degeneration and swelling, reduced blood hemoglobin levels, increased white blood cell numbers, swollen gill lamella with extensive cellular vacuolization, and necrotic and degenerating ovarian follicles (Ellis and Others 1937, Sorenson 1984, as cited in Eisler 1985b). Elevated concentrations of selenium were observed to cause reproductive failure, anemia, reduced hatch, reduced growth, reduced swimming rate, and chromosomal aberrations in aquatic organisms (Hodson and Others 1980, Adams 1976, Bovee and O'Brien 1982, and Krishnaja and Rege 1982, as cited in Eisler 1985b).

2.15.5 Birds

Selenium exposure in the diet or drinking water of avian species is associated with reproductive abnormalities, congenital malformations, selective bioaccumulation, and growth retardation (Eisler 1985b). Selenium has been observed to cause reduced hatching of eggs, decreased egg weight, decreased egg production, anemia, and embryo deformation, including deformed eyes, beaks, wings, and feet (Ort and Latshaw 1978 and Harr 1979, as cited in Eisler 1985b).

2.15.6 Mammals

Chronic effects of selenium on mammals include reproductive abnormalities such as congenital malformations; reduced numbers of young in litters; high mortality of young; infertility among surviving young in rats, mice, swine, and cattle; and intestinal lesions (Harr 1978 and National Center for Research Resources 1983, as cited in Eisler 1985b).

Chronic exposure of selenium, known as alkali disease, has been observed in cattle, hogs, and horses that graze on feed containing elevated levels of selenium. Adverse effects include deformed hooves; hair loss; lassitude; articular cartilage erosion; reduced conception; increased reabsorption of fetuses; and heart, kidney, and liver degeneration (Eisler 1985b).

2.16 SILVER

Silver occurs naturally in the environment in the form of silver nitrate, silver chloride, silver sulfide, or silver oxide (ATSDR 1990c). Some industrial discharges can introduce silver compounds into the environment, where they tend to form complexes with inorganic chemicals and humic substances in soils. Consequently, drainage and erosion potential, the presence of soil organic matter, redox potential, and pH conditions affects the mobility of silver in soils. Silver tends to be mobile in well-drained soils. Organic matter complexes with silver and causes it to become immobile. Because silver is toxic to soil microorganisms and inhibits bacterial enzymes, biotransformation is not expected to be a significant process (Domsch 1984, as cited in ATSDR 1990c).

The particular form of the compound influences the transport and partitioning of silver in surface waters and soils. The major forms of silver in water include the monovalent ion as sulfate, bicarbonate, or sulfate salts; more complex ions with chlorides and sulfates; and as an integral part of, or adsorbed onto, particulate matter and aquatic biota (Boyle 1968, as cited in ATSDR 1990c). Sorption is the dominant process leading to the partitioning of silver in sediments (Callahan and Others 1973, as cited in ATSDR 1990c), and pH and redox conditions affect sorption (Anderson and Others 1973, ATSDR 1990c). When decaying animal and plant material is abundant, silver strongly precipitates as the sulfide form or combines with humic materials (Smith and Carson 1977, as cited in ATSDR 1990c).

Silver is bioaccumulated by marine organisms (Nelson and Others 1983). No evidence exists that silver is biomagnified in terrestrial animals, although this may occur in some aquatic invertebrates (Adriano 1986).

2.16.1 Plants

Silver is not considered to be highly phytotoxic; even high concentrations in soil appear to have little or no effect on plant growth. Silver taken up by plants remains in the root system, precipitated with phosphate or chloride (Ward and Others 1979). The toxicity of silver is related to the binding potential of silver ions to enzymes and other active molecules at cell surfaces (Cooper and Jolly 1970).

2.16.2 Invertebrates

No information was identified on the effects of silver on invertebrates.

2.16.3 Amphibians and Reptiles

No information was identified on the effects of silver on amphibians and reptiles.

2.16.4 Fish and Aquatic Organisms

Silver is one of the most hazardous trace elements to aquatic species (EPA 1992). Younger life stages of development appear to be the most sensitive to silver concentrations (Klein-MacPhee and Others 1984). Silver concentrations have been observed to cause increased larvae mortality, physical abnormalities in developing larvae, reduced hatch, and reduced growth in winter flounder larvae (Klein-MacPhee and Others 1984). Juvenile mussels exposed to silver concentrations exhibited growth inhibition (Calabrese and Others 1984). Silver also has been observed to affect reproductive behavior and reduce larvae releases from gastropods (Nelson and Others 1983).

2.16.5 Birds

No information was identified on the effects of silver on birds.

2.16.6 Mammals

Inhalation studies have shown that silver can be absorbed in the lungs, while ingestion studies have shown that silver can be absorbed in the gastrointestinal tract (Amdur and Others 1991). Once in the body, silver can accumulate in the liver, while only a small amount is excreted (Amdur and Others 1991). A decrease in weight gain was observed in rats exposed to silver in drinking water (Matuk and Others 1989, as cited in ATSDR 1990c). Intravenous doses of silver to experimental animals caused pulmonary edema, congestion, and eventual death (Amdur and Others 1991).

2.17 TITANIUM

Titanium is the ninth most abundant natural element in the earth's crust. There are 5 natural isotopes of titanium, Ti-48, Ti-46, Ti-47, Ti-49, and Ti-50, with 78 percent of titanium being Ti-48. Several artificial isotopes have been manufactured, Ti-43, Ti-44, Ti-45, and Ti-51. It is a dark gray lustrous metal that can take on two forms, according to the surrounding temperature: hexagonal structure at temperatures below 882.5 C° and a cubic crystalline structure at temperatures above 882.5 C°. It is extremely resistant to corrosion and, in the form of a powder or dust, is highly flammable and explosive. The most common oxidation state of titanium is +4, but +3 and +2 states also exist. Titanium occurs in both a cationic state (e.g., titanium chlorides, phosphates, and sulfates) and an anionic state (e.g., calcium, iron, and sodium titanates) (World Health Organization [WHO] 1982).

There are many industrial and commercial uses of titanium. Titanium will form alloys with aluminum, chromium, cobalt, copper, iron, lead, nickel, and tin (Budavari 1996). Metallic titanium, titanium dioxide, and titanium tetrachloride are the compounds most widely used in industry (WHO 1982). The titanium alloys with copper and iron make up titanium bronze. Titanium is used in steel to provide greater tensile strength, in aluminum products to provide resistance to attack by salt solutions and organic acids, and to remove traces of oxygen and nitrogen from incandescent lamps (Budavari 1996). Titanium powder is used in pyrotechnics and vacuum engineering. Also, titanium metal is used as an implant material in orthopedics, oral surgery, and neurosurgery (National Library of Medicine [NLM] 2003). Titanium tetrachloride is a liquid formed from minerals that contain titanium. It is used to make titanium metal and other titanium-containing compounds, such as titanium dioxide, which is used as a white pigment in paints and other products, and as an intermediary to produce other chemicals (ATSDR 1997).

Owing to its great affinity for oxygen and other elements, Titanium does not exist in the metallic state in nature because it will easily form alloys with other elements. The average concentration in the earth's crust is 4400 mg/kg of titanium. Titanium concentrations in the urban air are mostly below 0.1 micrograms per cubic meter ($\mu\text{g}/\text{m}^3$), though levels exceeding 1.0 $\mu\text{g}/\text{m}^3$ have been reported, especially in industrialized areas. The titanium concentration in drinking-water supplies is generally low, having an approximate range of 0.5-15 microgram (μg) per liter (WHO 1982). Large variations in the concentrations of titanium in different types of foods have been reported. A typical diet may contribute

some 300-400 µg per day, but higher intakes ranging up to 2 mg per day have been reported (WHO 1982).

Titanium naturally is present in the ash of coal, in plants, and in the human body; however, there is no evidence of titanium being an essential element for man or animals (WHO 1982). Titanium compounds are poorly absorbed from the gastro-intestinal tract, which is the main route of exposure for the general population; however, for occupational exposure scenarios the main route is inhalation of dust, vapor, or fumes of titanium metal, titanium dioxide, titanium carbide, or titanium tetrachloride in the form (WHO 1982; NLM 2003). Available data on the occurrence of titanium and titanium compounds in the environment, as well as data on toxicity, indicate that the current level of exposure of the general population does not present a health risk. In the occupational environment, exposure occurs through inhalation and titanium is retained in the lungs. Dose-effect and dose-response relationships have not yet been established for any of the effects of various titanium compounds. Titanium tetrachloride is a known irritant to the eyes and respiratory tract (Budavari 1989). Titanium is known to occur in three valences: 2, 3, and 4, with the tetravalence the most common form. Generally, higher valence metals tend to be lower in toxicity because these elements will be rapidly excreted (NLM 2003).

2.17.1 Plants

Titanium, like aluminium, is found in relatively abundant quantities in the lithosphere and in soils, but is poorly absorbed and retained by plants (Underwood 1977, as cited in WHO 1982). Average titanium levels of approximately 1 mg/kg have been reported for a wide variety of plants (Bertrand and Voronca-Spirt 1929a and 1929b, as cited in WHO 1982). It has been suggested that levels in herbage samples are indicators of soil contamination (Barlow and Others 1960, as cited in WHO 1982). Mean levels of titanium of 1.8 mg/kg (dry weight) in red clover (range 0.7-3.8 mg/kg) and 2.0 mg/kg in ryegrass (range 0.9-4.6 mg/kg) grown on different soils have been reported (Mitchell 1957, as cited in WHO 1982). In Kazakhstan, grain crops absorbed titanium levels of 50-100 grams per hectare (g/ha) and legumes 123-398 g/ha from soil containing levels of titanium of 1.2-7 mg/kg (Grabarov, 1970, as cited in WHO). More titanium was found in maple and elm leaves than in the leaves of other plants while the content of titanium in brush was 50-820 mg/kg (WHO 1982).

However, no information was identified on the effects of titanium on plants.

2.17.2 Invertebrates

No information was identified on the effects of titanium on invertebrates.

2.17.3 Amphibians and Reptiles

No information was identified on the effects of titanium on amphibians and reptiles.

2.17.4 Fish and Aquatic Organisms

No information was identified on the effects of silver on fish and aquatic organisms.

2.17.5 Birds

No information was identified on the effects of silver on birds.

2.17.6 Mammals

Toxicity of titanium from acute exposures indicated that primarily only titanium tetrachloride had the potential to cause adverse effects in mammal studies. Exposure of guinea pigs to titanium dioxide aerosol did not induce any inflammatory response, while inhalation of titanium tetrachloride caused a higher death rate and more rapid development of lung oedema in mice than inhalation of an equivalent concentration of hydrogen chloride (Lehman and Herget 1927, Mel'nikova 1958, Mezenceva and others, 1963, Mogilevskaja 1973, as cited in WHO 1982). Similar effects in mice and rats were observed. Titanium tetrachloride also caused purulent conjunctivitis and corneal opacity in rabbit eyes (Sanockij 1961, as cited in WHO 1982) observed

Toxicity of titanium compounds from chronic exposures indicated very low incidence of adverse effects to mammals. Schroeder and others (1964) administered titanium potassium oxalate, at a concentration of

5 mg per liter, in the drinking water of mice, from weaning to natural death. The control group consisted of 88 female and 61 male mice compared with 53 female and 54 male mice in the treated group. The survival rate after 18 months was 75 percent females and 50 percent males for the control animals, and 70 percent females and 40 percent males for the treated group. The body weights of the animals in the titanium-fed group were higher than those in the control group (WHO 1982).

Two guinea pigs, 2 rabbits, 2 cats, and 1 dog were fed technical grade titanium dioxide for 390 days. The dog received 9 grams per day (g/day), the rabbits and cats, 3 g/day, and the guinea pigs, 0.6 g/day. Adverse effects were not seen in any of the animals and histopathological examination did not reveal any abnormalities (Lehman & Herget 1927, as cited in WHO 1982).

Christie and others (1963) did not find any evidence of pathological response in the lungs of rats that had inhaled titanium dioxide dust (air concentrations in the range of 10-328 million particles per cubic foot) 4 times daily, 5 days per week for periods up to 13 months, followed by a 7-month period of fresh air. The inhalation of titanium dioxide did not affect the weight of the rats (WHO 1982).

Mutagenic, carcinogenic, and teratogenic tendencies of titanium compounds are not well studied.

2.18 VANADIUM

Vanadium is a natural element in the earth. It is a white to gray metal that is often found as crystals. It has no particular odor. In the environment, it is usually combined with other elements such as oxygen, sodium, sulfur, or chloride.

Natural vanadium consists of two isotopes, V-50 and V-51, the former being slightly radioactive, with a half-life of 6.0 E15 years. Seven other radioisotopes of the element have been synthesized (Kerr 1988). Vanadium exhibits oxidation states of -1, 0, +1, +2, +3, +4, and +5 in a wide variety of complex ions and coordination complexes (Kerr 1988). Three oxidation states (+3 to +5) can be stable in water (Hem 1989). These three states are the only forms with any biological significance (Morrell and Others 1985). The +4 and +5 states are the most significant oxidation states in living organisms, while the +3 state occurs solely in one group of marine chordates (Morrell and Others 1985). In aqueous solution,

vanadium in the +5 state exists as various oxoions referred to as “vanadates.” The exact nature of the vanadate species in solution is pH- and concentration-dependent. Also, the exact species present cannot be determined unless the solution is allowed to stand for many days, because equilibrium among species is slowly attained (Morrell and Others 1985).

The chemistry of vanadium in the +4 state centers on the vanadium oxide ion. This species undergoes a number of hydrolysis reactions with increasing solution pH. At lower pH (<5.0), vanadium oxide and vanadium hydroxide dominate, whereas at more alkaline pH (>5.0), a variety of forms exist. In an alkaline environment, these ionic forms are also subject to air oxidation in the absence of suitable chelating agents (Morrell and Others 1985). Both the +4 and +5 oxidation states have a notable tendency to form stable chelates with a variety of organic and inorganic ligands (such as ketones, aldehydes, catechols, amino compounds, and phenols). Chelate formation can alter the oxidation state from +5 to +4 (Morrell and Others 1985).

The forms of vanadium most likely to be found at waste sites are not well known. One manmade form, vanadium oxide (vanadium bound to oxygen), is most often used by industry in making steel. Vanadium oxide can be a yellow-orange powder, dark-gray flakes, or yellow crystals. Much smaller amounts are used in making rubber, plastics, ceramics, and certain other chemicals (ATSDR 1992b).

Vanadium is a transition metal with complex aqueous geochemistry (Hem 1989). Vanadium is ubiquitous in the biosphere, resulting in detectable trace levels in most living organisms (Morrell and Others 1985). Human-induced vanadium emissions caused by the combustion of fossil fuels, have increased so that the volume of these emissions equal those caused by natural emissions, such as continental dust, marine aerosols, and volcanic activity (Morrell and Others 1985).

Vanadium is found in ore, along with uranium (Hem 1989). About 80 percent of vanadium production is used to make ferrovandium or as a steel additive (Kerr 1988).

Vanadium is found in many petroleum products, because it occurs naturally in fuel oils and coal (ATSDR 1992b). It is also a by-product of petroleum refining (Klassen and Others 1991).

Vanadium and its compounds are toxic, though this toxicity is variable (Kerr 1988; Klaasen and Others 1991; Lewis 1993). Toxicity depends on the valence; it increases with increasing valence, with pentavalent vanadium being most toxic. In addition, vanadium is toxic as a cation and as an anion (NRCC 1977; Fleishman 1988; HSDB 1999). Vanadium is considered to be 1 of the 14 most noxious heavy metals (Jenkins 1981). Vanadium toxicity is attributed to its ability to inhibit enzyme systems, such as monamine oxidase, adenosine triphosphatase (ATPase), tyrosinase, choline esterase, and cholesterol synthetase (Leland and Kuwabara 1985).

2.18.1 Plants

Small amounts of vanadium in the environment tend to stimulate plants, but large amounts are toxic (Brooks 1972). Essential for the growth of fungi and algae, it stimulates photosynthesis in higher plants (Venugopal and Luckey 1978; Schiffer 1989; HSDB 1999).

Levels of vanadium (parts per million [ppm] dry weight) considered to be phytotoxic are 150 (Warsaw, Poland), 50 (Warsaw, Poland) and 60 (Ontario, Canada) (Kabata-Pendias and Pendias 1992). Harmful concentrations of vanadium to most plants range from 10 to 20 ppm. However, higher concentrations can be tolerated by legumes, which use vanadium in the nitrogen fixation process (Brooks 1972). As little as 0.5 mg/kg of vanadium reduces the growth of flax, peas, soybeans, and cabbage affected by aqueous vanadium levels as low as 10 to 20 milligrams per liter (mg/L). Usually, vanadium induces ion deficiency chlorosis by interfering with iron absorption. Levels of other essential elements, such as manganese, copper, calcium, and phosphorus, also are reduced by vanadium. Excess vanadium can therefore affect the trace element nutritional value of plants (NRCC 1980; HSDB 1999).

The 1985 study on the potential of vanadium to inhibit sodium-potassium ATPases in plants (Morrell and Others 1985) explored the similarity between both plant uptake and transport of both the +4 and +5 ionic states of vanadium and supports the hypothesis of biotransformation of vanadium during root uptake by plants. Although the extent of reduction is unclear, this process generally seems to involve the reduction of +5 vanadium to +4. Even at a pH of 7, the presence of chelating agents (such as ketones and aldehydes) in cell walls would indicate the potential of ready reduction of vanadium by root tissues. The resulting +4 complexes would be extremely stable, rendering the bulk of any free-space vanadium

physiologically unavailable. Therefore, the interaction of vanadium with common organic constituents of living matter currently appears to control vanadate's disruptive potential.

Although this reduction of +5 vanadium to +4, leading to the immobilization of vanadium within the root tissue, is hypothesized to occur, it does not preclude the possibility that vanadium in the +5 state also is present in these tissues. Considering the ubiquitous nature of sodium-potassium ATPases in the membrane system and the ability of vanadium in the +5 state (vanadate) to inhibit sodium-potassium ATPases, the effects of vanadate on all living systems are potentially harmful. Given the marked increase in human-induced atmospheric vanadium concentrations, conditions may be created that allow free access to vanadate ions in living systems.

2.18.2 Invertebrates

Little information was available on the effects of vanadium on invertebrates. Vanadium is an oxygen-carrying metal in some invertebrates (Venugopal and Luckey 1978; Schiffer 1989; HSDB 1999).

2.18.3 Amphibians and Reptiles

No information was identified on the effects of vanadium on amphibians and reptiles.

2.18.4 Fish and Aquatic Organisms

A growth feeding trial was conducted in which juvenile rainbow trout (initial weight 2.9 grams per fish) were fed diets supplemented with 0 to 10 grams vanadium (as sodium orthovanadate) per kilogram diet for 12 weeks at 15°C. All levels of supplemented vanadium significantly reduced growth and feeding response in the trout. At high levels of dietary vanadium (> 493 mg/kg), feed avoidance and increased mortalities were apparent in the trout. The vanadium retention factor (carcass vanadium per total amount of vanadium consumed) and carcass concentration factor (carcass vanadium concentration per dietary vanadium content) increased in relation to the dietary vanadium level, indicating a bioaccumulation of vanadium in these fish. This is in direct contrast to the apparently low bioaccumulation of waterborne vanadium in trout. The minimum dietary vanadium toxicity level could not be determined. The major

biochemical and physiological effect of vanadium in the trout would appear to be increased in vivo lipid oxidation (Hilton and Bettger 1988; HSDB 1999).

2.18.5 Birds

Some vanadium compounds produce mutation effects in birds (Lewis 1993). Dietary vanadium has been shown to suppress egg production of laying hens (Rompala and Others 1984). Dietary vanadium at levels as low as 0.5 mg/kg have been shown to alter metabolism in mallards (White and Others 1980).

2.18.6 Mammals

Vanadium toxicity is attributed to its ability to inhibit enzyme systems, such as monoamine oxidase, ATPase, tyrosinase, choline esterase, and cholesterol synthetase (Luckey and Venugopal 1977; Leland and Kuwabara 1985; HSDB 1999).

Pregnant Sprague-Dawley rats given orally 20 milligrams per kilogram per day (mg/kg/day) of vanadium, as sodium metavanadate, displayed embryotoxicity but not teratogenicity (Paternain and Others 1987; HSDB 1999).

Wistar rats of both sexes received vanadium in drinking water in the amount of 23 to 29 mg/kg per body weight, in the form of ammonium metavanadate, for a period of 2, 4, and 8 weeks. Animals treated in this way ate less food and drank less ammonium metavanadate solution compared with the amount of water consumed by the controls; they suffered from diarrhea, and because of this, the increment in body weight was reduced. Vanadium decreased erythropoiesis and maturation of red blood cells, which was expressed by a reduced erythrocyte count and hemoglobin level and increased reticulocyte and polychromatophilic erythrocyte count in the peripheral blood (HSDB 1999).

2.19 ZINC

Zinc is an essential trace element for all living organisms, and zinc deficiency can be a problem for both plants and animals. Zinc is primarily used as a protective coating for metals and in the production of

alloys, such as bronze and brass. Adverse effects of zinc exposure to animals include growth retardation, testicular atrophy, skin changes, and poor appetite (Prasad 1979, as cited in Eisler 1993). Most of the zinc introduced into aquatic environments eventually is partitioned into sediment. Zinc released from sediment is enhanced under conditions of high dissolved oxygen, low salinity, and low pH. Dissolved zinc usually consists of the hydrated zinc ion and various organic and inorganic complexes. In reducing conditions, organically bound zinc typically forms insoluble sulfides (MacDonald 1993).

BCFs vary widely between and within species of aquatic organisms (Eisler 1993). In marine environments, the most effective zinc accumulators included red and brown algae, ostreid and crassostreid oysters, and scallops. Invertebrates can bioaccumulate large quantities of zinc (Jamil and Hussein 1992), which potentially could be passed on to upper trophic-level consumers. Studies show that bony structures can act as long-term repositories for zinc (Macapinlac and Others 1966). Zinc concentrations have been shown to increase with increasing trophic levels from phytoplankton to zooplankton but not to fish (Balasubramanian and Others 1995).

2.19.1 Plants

Zinc is an essential nutrient for plant growth in small amounts but is toxic to plants at elevated levels. Zinc can cause significant adverse effects on growth, survival, and reproduction in representative sensitive species of aquatic plants (Eisler 1993). Elevated levels of zinc in soil can cause mortality in some terrestrial plants and inhibit photosynthesis in others (Eisler 1993).

2.19.2 Invertebrates

Certain terrestrial and aquatic invertebrates are sensitive to zinc. Reduced growth, inhibited reproduction, and reduced survival are effects that zinc can have on both terrestrial and aquatic invertebrates (Eisler 1993).

2.19.3 Amphibians and Reptiles

Environmental conditions, including pH and water hardness, and the life stage of the amphibian exposed both affect the organism's sensitivity and adverse response to exposure to metal concentrations in water. Although information concerning amphibians and metal toxicity is limited, the primary mechanism of action of metal-induced toxicity and low pH environments is believed to be body loss of sodium across the gill surface (MacDonald and Wood 1993, as cited in Horne and Dunson 1995). In one study, no significant effects occurred of acute and chronic exposures of frogs and salamanders and their larvae to zinc (Horne and Dunson 1995). In addition, the pH of the aquatic environment had no effect on the toxicity of zinc, unlike the other metals that were studied (Horne and Dunson 1995).

However, another study observed that zinc caused significant adverse effects on growth, survival, and reproduction in representative sensitive species of amphibians (Eisler 1993). In this study, zinc was shown to cause teratogenic effects to frog embryos (Eisler 1993).

2.19.4 Fish and Aquatic Organisms

The gill epithelium is the primary route of zinc entry into the body of fish (Eisler 1993). Zinc can cause significant adverse effects on growth, survival, and reproduction in representative, sensitive species of protozoa, sponges, mollusks, crustaceans, echinoderms, and fish (Eisler 1993). Zinc has been shown to cause teratogenic effects to fish embryos (Eisler 1993).

2.19.5 Birds

Different species of birds have varying sensitivities to zinc exposure. Acute effects of zinc in ducks caused mortality and pancreatic degradation (Eisler 1993). Reduced growth and death were observed in poultry chicks fed diets containing elevated zinc levels. Younger stages of life appear to be more sensitive to zinc exposure. The pancreas and bone are primary target organs of zinc in birds (Eisler 1993). Decreased weight gain was observed in Japanese quail, chickens, and turkeys fed diets containing zinc (NAS 1980).

2.19.6 Mammals

Zinc is relatively nontoxic in mammals; however, excessive zinc intake adversely affects survival of all tested mammals and produces a wide variety of neurological, hematological, immunological, hepatic, renal, cardiovascular, developmental, and genotoxic effects (Eisler 1993). The pancreas and bone are primary target organs of zinc exposure in mammals (Eisler 1993). Toxic effects of zinc can be observed in many domestic animals, including dogs, cats, ferrets, cattle, sheep, and horses, as a result of ingesting zinc-containing objects (Eisler 1993). Zinc concentrations in the diet of pregnant rats and sheep caused increased incidence of hypocuprosis, still births, and fetal resorptions (Ketchenson and Others 1969, Campbell and Mill 1979, as cited in Domingo 1994).

3.0 PESTICIDES

The following sections present data on ecotoxicological effects of various chlorinated pesticides identified as COPECs at Alameda Point.

3.1 ALDRIN AND DIELDRIN

Aldrin was first manufactured in the United States as a pesticide (EPA 1986, as cited in ATSDR 1993e). Dieldrin was also manufactured as a pesticide; however, it is also an abiotic and biotic degradation product of aldrin. Aldrin rapidly metabolizes into dieldrin after it enters the body; therefore, effects in animals exposed to aldrin may be caused by dieldrin (Beyer and Others 1996). According to the Farm Chemicals Handbook (Meister 1995), all uses for aldrin within the United States have been cancelled. Dieldrin is registered as a general use insecticide.

Because of low water solubility and high soil organic carbon/water partition coefficients (K_{oc}), dieldrin and aldrin are extremely persistent in soils (ATSDR 1993e). Dieldrin and aldrin can reach the atmosphere through volatilization from water or soil, a process that is slower from soil than from water. Dieldrin also may be sorbed to fugitive dust. A strong tendency to sorb to soil particles generally prevents dieldrin and aldrin from leaching to groundwater; however, dieldrin will reach surface water sorbed to soil particles in runoff. Once in water, dieldrin sorbs strongly to sediments; it will not undergo

hydrolysis or biodegradation, and an aquatic half-life of greater than 4 years has been determined (HSDB 1999). Biodegradation is also insignificant in soils (HSDB 1999).

Dieldrin and aldrin have high potentials for bioconcentration and bioaccumulation, as indicated by log octanol-water coefficient (K_{ow}) ranging from 4.32 to 6.2 for dieldrin and 5.68 to 7.4 for aldrin (ATSDR 1993e). Dieldrin is extremely nonpolar and hydrophobic and consequently, partitions into organic matter, including animal fat and plant waxes (ATSDR 1993e). Dieldrin is stored in the adipose tissue, liver, brain, and muscle of mammals, fish, and birds; algae; plankton; insects; earthworms; and eggs of many bird species (International Agency Research Cancer [IARC] 1973).

3.1.1 Plants

Various crops take up dieldrin, with most of the dieldrin remaining in the roots (ATSDR 1993e). Foliar contamination of soybean plants was found to occur through root translocation and vapor sorption. Bioconcentration is receptor-specific. Soybean seeds contained dieldrin in concentrations of about 10 percent of those in soil (Stickel 1968). Peanuts concentrated aldrin and dieldrin by a factor of two to four times the concentration in soils in which they were grown (Beck and Others 1962, as cited in Stickel 1968).

3.1.2 Invertebrates

Aldrin and dieldrin are insecticides; therefore, by definition, they are toxic to insects, including corn pests and termites, by contact or ingestion (Hayes 1982, as cited in ATSDR 1993e).

3.1.3 Amphibians and Reptiles

Frog embryos treated with dieldrin resulted in accelerated growth and abnormalities in later developmental stages (Brooks 1981, as cited in Power and Others 1989). Adverse effects caused by exposure to aldrin and dieldrin, include coarse tremors, rigidity, abnormal reactions to stimulation, convulsions, and disorientation (Kaplan and Overpeck 1964, as cited in Power and Others 1989).

Inhibition of enzyme systems and greater effects from dieldrin than aldrin are reported to occur in reptiles (Phillips and Wells 1974, as cited in Hall 1980).

3.1.4 Fish and Aquatic Organisms

Although the toxicities of aldrin and dieldrin are similar in most organisms, in fish, dieldrin is an order of magnitude more toxic than aldrin (Beyer and Others 1996). In tests on guppy populations, populations treated with dieldrin showed reduced growth and reproduction. Such population effects on species that serve as prey to many ecosystem predators could reduce ecosystem productivity and cause nutrient loss (Peterle 1991).

3.1.5 Birds

Dieldrin adversely affects reproduction in birds. Eggshell thickness and egg production and fertility decreased in mallards given various doses of dieldrin (Winn 1972, Walker and Others 1969, as cited in Beyer and Others 1996). The major impact of dieldrin on raptors is reported as being direct mortality, rather than eggshell thinning (Beyer and Others 1996).

3.1.6 Mammals

The doses at which aldrin is lethal in rats are similar to lethal doses of dieldrin. Decreased survival was seen in rats, dogs, and mice consuming low doses of dieldrin or aldrin over long periods (ATSDR 1993e). Adverse effects of dieldrin and aldrin exposure include tremors, hyperexcitability, irritability, convulsions, and increases in liver weight and size. Increased postnatal mortality also was seen in mice and rats.

3.2 CHLORDANE (ALPHA AND GAMMA)

Chlordane is a broad-spectrum, organochlorine pesticide, consisting of about 45 components. Historically used to control soil invertebrate pests, chlordane also adversely affected nontarget species. Although federal law currently prohibits use of chlordane in the United States, persistent chlordane

residues and metabolites from past use continue to pose an ecological threat (Eisler 1990). Chlordane and its metabolites do not degrade to any significant extent either biotically or abiotically in either aquatic or terrestrial ecosystems. Chlordane in water does not undergo considerable hydrolysis, oxidation, or photolysis, and biodegradation is minimal (Gore and Others 1971, as cited in HSDB 1999). Because of chlordane's low water solubility, high lipid solubility, low vapor pressure, and strong tendency to sorb to soil and sediment particles, it persists in soil and sediments for extended periods (EPA 1988, as cited in Eisler 1990). Sorption of chlordane to sediment is expected to be a major aquatic fate process (Callahan and Others 1979, as cited in HSDB 1999).

Based on field tests, soil column leaching tests, and K_{oc} estimation, chlordane is expected to be immobile in soil (Cohen 1986, as cited in HSDB 1999). Surface-applied chlordane usually remains in the top 20 centimeters of most soils. Little biodegradation occurs in soil; chlordane residues have been detected in soils 14 years (EPA 1988, as cited in Eisler 1990) and 20 years (Beeman and Matsumura 1981, as cited in ATSDR 1994a) after application. Volatilization is the most important pathway for chlordane release from the surface of soil (ATSDR 1994a).

Chlordane is known to bioconcentrate, particularly in long-lived, carnivorous organisms having large amounts of body lipids (Eisler 1990). Chlordane bioconcentration has been shown to increase with trophic levels from zooplankton to marine mammals in marine ecosystems, causing biomagnification of total chlordane through the food chain, with residues peaking in marine mammals (Kawano and Others 1986). Chlordane biomagnification in carnivorous birds is also considerable.

3.2.1 Plants

Chlordane residues have been detected in vegetable crops grown in soils treated with chlordane (U.S. Department of Agriculture 1965, as cited in HSDB 1999). Chlordane is taken up by rooted, aquatic vascular plants, both from water and sediment. Chlordane bioconcentrates in roots and is translocated to shoots (ATSDR 1994a).

3.2.2 Invertebrates

Chlordane is toxic to terrestrial invertebrates. Chlordane was used extensively to control grubs, ants, and snails (Eisler 1990). Earthworms were nontarget soil species that were adversely affected by chlordane. Sediment concentrations of chlordane were fatal to sandworms (*Nereis virens*) in 12 days (McLeese and Others 1982, as cited in Eisler 1990). Chlordane effects decreased with increasing soil temperature and organic content (NRCC 1975, as cited in Eisler 1990).

3.2.3 Amphibians and Reptiles

Symptoms of chlordane poisoning in frogs include neuromuscular changes, excessive thrashing, and tremors (Kaplan and Overpeck 1964, as cited in Power and Others 1989). California newts had greatly elevated chlordane residues in the liver, as compared to the stomach and carcass.

3.2.4 Fish and Aquatic Organisms

Chlordane adversely affects survival, reproduction, and growth in aquatic invertebrates and fish. Symptoms of poisoning in fish include hyperexcitability, increased respiration rate, erratic swimming, loss of equilibrium, and convulsions (Eisler 1990). Other symptoms recorded were immobilization, impaired reproduction, and histopathology (NRCC 1975, as cited in Eisler 1990).

3.2.5 Birds

Lethal effects of chlordane in birds result primarily from chlordane metabolites, most notably oxychlordane and heptachlor epoxide; oxychlordane is the most toxic and persistent of the two (Eisler 1990). Reproductive impairment has been documented in waterfowl species from a wetland treated with chlordane. Organisms metabolize chlordane into several isomers, of which oxychlordane is the most toxic and persistent, even when compared to its parent chemicals (WHO 1984, as cited in Eisler 1990; Kawano and Others 1986). These isomers can persist in avian tissues for up to 35.4 years (Eisler 1990). Chlordane interacts with other chemicals to cause additive or synergistic effects, particularly when combined with other organochlorines, such as endrin, methoxychlor, and aldrin, in northern bobwhites

(Ludke 1976, as cited in Eisler 1990). Symptoms of poisoning include sluggishness, drooped eyelids, fluffed feathers, reduced food intake, and weight loss (Eisler 1990).

3.2.6 Mammals

Warm-blooded animals readily absorb chlordane through dermal contact, ingestion, and inhalation, and after residues are absorbed, they concentrate in the fat, liver, kidney, brain, and muscle (WHO 1984, as cited in Eisler 1990). Chlordane is a nerve stimulant whose physiological target sites are nerve and muscle membranes (Greenhalgh 1986, as cited in Eisler 1990). Chronic, dietary chlordane adversely affects physiology, growth, and fertility of laboratory mammals, including enlargement and increased pathology of the liver in rats and mice. It also elevates residues in cow's milk, reduces sexual activity and litter viability in rats, depresses growth, delays development, decreases immune competence, and decreases viability of offspring in mice (Talamantes and Jang 1977, WHO 1984, as cited in Eisler 1990). Chlordane interacts with other chemicals to cause additive or synergistic effects, particularly when combined with other organochlorines such as endrin, methoxychlor, and aldrin in mice (Klaassen and Others 1986, as cited in Eisler 1990).

3.3 DICHLORODIPHENYLTRICHLOROETHANE

Historically, dichlorodiphenyltrichloroethane (DDT) and related organochlorine insecticides were used worldwide in pest control. In 1972, DDT was banned in the United States, because DDT was highly toxic to nontarget species. DDT generally occurs as a constituent of a mixture of chlorinated, organic compounds, two of the most important being dichlorodiphenyldichloroethene (DDE) and dichlorodiphenyldichloroethane (DDD). DDE and DDD are also metabolites of DDT. All three have similar fate and transport properties. DDT, like many organochlorine contaminants, is characterized by its persistence in the environment, potential for volatilization and transport to unaffected areas, lipophilic nature, toxicity of metabolites, and variability of species responses to chronic exposure to DDT and its metabolites (Peterle 1991). DDT, DDE, and DDD are only slightly soluble in water and adhere to soil and sediment particles (Thibodeaux 1979). DDT and related organochlorine chemicals are slowly biodegraded in soil and in aquatic ecosystems (Alexander 1965, as cited in Forsyth and Others 1983).

These chlorinated hydrocarbons tend to be highly persistent in soil and often have half-life estimates of greater than 15 years (Lichtenstein and Others 1959, Stewart and Others 1971, as cited in ATSDR 1992c).

In addition to their relatively long half-life, organochlorine chemicals like DDT are highly lipophilic and as a result, DDT and similar chemicals have very high BCFs. Biomagnification also has been observed for DDT and its metabolites (Bevenue 1976, as cited in Forsyth and Others 1983). The biomagnification of DDT (and related organochlorine pesticides) has been well documented (Hoffman and Others 1995), and the deleterious effects associated with trophic-level transfer in both aquatic and terrestrial food chains remains a concern (EPA 1995a). DDT is most widely known for its reproductive effects on birds, primarily piscivorous species, and for its toxicity to aquatic fish and invertebrates.

3.3.1 Plants

Terrestrial plants can accumulate elevated levels of DDT (Forsyth and Others 1983). DDT can reduce photosynthesis in plants and reduce the ability of the plant to withstand environmental changes. In addition, DDT can decrease the amount of oxygen produced by aquatic plants (EPA 1975).

3.3.2 Invertebrates

DDT was once used as an insecticide. Once invertebrates have adapted to DDT, they can accumulate it at high levels (Forsyth and Others 1983). DDT can cause immobilization, reproductive impairment, embryo deformation, reduced growth, and mortality in both terrestrial and aquatic invertebrates (EPA 1975).

3.3.3 Amphibians and Reptiles

DDT has been observed to cause developmental deformities and hyperactivity in tadpoles (Osborn and Others 1981). DDT causes developmental deformities by disrupting the organization of epithelial cells into glands. This can cause blunt snouts and deformed brains in exposed tadpoles (Osborn and Others 1981).

3.3.4 Fish and Aquatic Organisms

DDT is very toxic to fish and aquatic organisms. Even at very low concentrations, DDT can cause mortality. In fish, DDT inhibits ATPase activity in cellular synthesis and osmoregulation (EPA 1975). DDT affects thyroid activity, alters behavior and activity, affects learning processes, causes developmental defects, and disrupts cellular energy use in fish (EPA 1975). DDT also adversely affects the reproductive success of fish because of its chemical characteristics that allow DDT to become concentrated in the egg yolk. When this happens, developing fry feed on the DDT-containing yolk, eventually causing increased fry mortality after birth (EPA 1975).

3.3.5 Birds

Reproductive effects of DDT and its metabolites on birds have been well characterized. Organochlorines, including DDT, have been implicated in the thinning of eggshells of at least 54 species of 10 orders of birds (Stickel 1975, as cited in Peterle 1991). DDT is believed to affect the calcium transport and deposition from the female to the egg, resulting in eggshell thinning. In addition to eggshell thinning other birds treated with DDE showed delayed egg laying, reduced hatchability, and alteration of egg size, weight, and content (Vangilder and Peterle 1980, 1981, and 1983, as cited in Peterle 1991).

3.3.6 Mammals

Low levels of exposure to DDT and organochlorines have caused reproductive effects in mink, bats, shrews, and sea lions (Peterle 1991). In several animal species, acute, oral exposure to high doses of DDT has been associated with DDT-induced tremors or myoclonus (abrupt, involuntary contractions of skeletal muscles), hyperexcitability, convulsions, and reduced fertility (EPA 1975). Chronic exposure of experimental animals to DDT is associated with tremors and hyperirritability. DDT-induced developmental effects include decreased fetal body weight, decreased fetal brain and kidney weight, embryotoxicity, fetotoxicity, and neonatal mortality. Reproductive effects of chronic exposure produced abortions, stillbirths, increased maternal and fetal mortality, delayed estrus, a reduction in male libido, and a lack of mammary gland development.

3.4 ENDRIN, ENDRIN ALDEHYDE, AND ENDRIN KETONE

Endrin is a neurotoxin that has been used as an insecticide, an avicide, and a rodenticide. Endrin aldehyde, an impurity of technical-grade endrin, has fate and transport properties similar to endrin. Like most organochlorines, endrin is very persistent in soil and may remain relatively unchanged for 14 years or longer. Endrin's soil persistence results from its high K_{oc} values (8,500 to 45,000), its low water solubility, its resistance to biodegradation, and its slow volatilization rate. Calculated half-lives in soil range from 4 to 14 years or more (Menzie 1972, Nash and Others 1967, Alexander 1973, and Kenaga 1980, as cited in HSDB 1999).

Endrin reaches the atmosphere by slow evaporation and sorbed to fugitive dust. Because of its strong tendency to sorb to soil particles and its low solubility in water, endrin generally does not leach to groundwater. Like dieldrin, however, endrin can reach surface water sorbed to soil particles in runoff (Southeastern Research Center 1980, as cited in HSDB 1999). In water, endrin sorbs to sediment and does not evaporate, hydrolyze, or biodegrade to any great extent. A half-life of greater than 4 years has been calculated for endrin in water (Eichelberger 1971, EPA 1979, as cited in HSDB 1999). Photodegradation in water of endrin to endrin ketone will yield low concentrations of this product (HSDB 1999).

Like other organochlorine pesticides, endrin, owing to its high lipophilicity and low water solubility, has a great potential for bioconcentration and bioaccumulation. For example, in microcosm and mosquito fish studies, endrin has been found to bioconcentrate to a great extent in aquatic organisms, and its high partitioning into lipids enhances its transfer through the food chain by the diet. As with other organochlorines, endrin is prohibited from routine use, but its relatively long half-life in the environment has extended its potential to exert its adverse chronic effects in aquatic and terrestrial food chains (Hoffman and Others 1995).

3.4.1 Plants

No information was identified on the effects of endrin on plants.

3.4.2 Invertebrates

Endrin was one of the most highly toxic organochlorine pesticides when routinely applied to agricultural crops.

3.4.3 Amphibians and Reptiles

No information was identified on the effects of endrin on amphibians and reptiles.

3.4.4 Fish and Aquatic Organisms

Until its restriction, endrin was one of the most highly toxic organochlorine pesticides in agricultural use. Its neurotoxic effects, however, were relatively nonspecific with respect to target and nontarget impacts, and nontarget aquatic organisms were exposed through surface water runoff, as well as accidental releases. A 28-day lethal concentration 50 (LC50) of 0.42 nanograms per liter has been observed for isopods (Anderson and Others 1980, as cited in HSDB 1999).

3.4.5 Birds

Data from studies focused on the toxicity of endrin to avian species are limited, in part reflecting the relatively underdeveloped discipline of ecotoxicology in the late 1960s and early 1970s. In wildlife, however, observation studies in the early 1970s clearly indicated that reproductive effects similar to those for other organochlorines were expressed in birds exposed in the field (Hoffman and Others 1995). For example, lethal diagnostic residues in fish-eating birds (for example, pelicans) were considered to be greater than or equal to 0.8 grams per gram (Stickel 1968).

3.4.6 Mammals

Limited information is available on the effects of endrin to mammals. However, wildlife observation studies in the early 1970s clearly indicated that reproductive effects similar to those for other organochlorines were expressed in mammals exposed in the field (Hoffman and Others 1995).

3.5 METHOXYCHLOR

Methoxychlor is an organochlorine insecticide related to DDT, and the two have similar transport properties in the environment. Unlike DDT, methoxychlor is not persistent in the environment and can be broken down metabolically by receptors. Metabolic breakdown results in a compound that is not as readily bioaccumulated in the environment (Beyer and Others 1996). In soil, methoxychlor is expected to remain immobilized in the upper layers, although a small percentage may migrate to lower depths. Residues have been observed to remain in soil for up to 14 months (NRCC 1975, as cited in HSDB 1999). Depending on the type of soil, K_{oc} values for methoxychlor can range from 73,000 to 100,000 (Karickhoff and Others 1979, as cited in HSDB 1999). This range of K_{oc} values suggests that methoxychlor would be moderately mobile to immobile in soil and will adsorb significantly to suspended solids and sediments in water (Swann and Others 1983, as cited in HSDB 1999).

Under anaerobic conditions, biodegradation appears to be the dominant removal mechanism; however, under aerobic conditions, biodegradation is negligible (Castro and Others 1971, as cited in HSDB 1999). The half-life for methoxychlor in anaerobic sediments has been reported as less than 28 days; in aerobic sediments, it was reported at greater than 100 days (Muir and Others 1984, as cited in HSDB 1999). In water, methoxychlor adsorbs to suspended solids and sediments and bioaccumulates in certain aquatic organisms. The K_{oc} in a water-sediment system has been estimated at 620 (Wolfef and Others 1977, as cited in HSDB 1999). Oxidation and hydrolysis are not expected to be significant fate processes, although some photolysis may occur in water, soil, and sediment.

BCFs have been estimated for methoxychlor in many different ecological receptors, including fungi, algae, stoneflies, snails, mussels, and soft shell clams (HSDB 1999). Fish, birds, and mammals are known, in general, to metabolize this compound fairly rapidly and, as a result, do not readily accumulate this compound (Paris and Others 1973, NRCC 1975, as cited in HSDB 1999). Methoxychlor has been estimated to be stored in fat at only 0.01 to 0.1 times its chronic intake, and the half-life of stored methoxychlor in rats is 1 to 2 weeks, as compared to an estimated value of 6 months to 1 year for DDT (Casarett and Doull's Toxicology 1986, as cited in HSDB 1999). The bioaccumulation ability of methoxychlor is very low (Beyer and Others 1996).

3.5.1 Plants

No information was identified on the effects of methoxychlor on plants.

3.5.2 Invertebrates

Methoxychlor is an insecticide that has been used to control many insects (Gardner and Bailey 1975, as cited in Beyer and Others 1996).

3.5.3 Amphibians and Reptiles

Methoxychlor was observed to be moderately toxic to tadpoles (Sanders 1970, as cited in Power and Others 1989). Changes in organ weights, feeding, behavior, and survival were not observed in amphibians exposed to methoxychlor (Hall and Swineford 1979, as cited in Power and Others 1989). In amphibians, methoxychlor is more readily accumulated through dermal exposure than through ingestion and as a result, is readily bioconcentrated and not readily biomagnified. Residue levels in amphibians were not related to the exposure duration, which could indicate that methoxychlor is rapidly equilibrated in the body (Power and Others 1989).

3.5.4 Fish and Aquatic Organisms

Methoxychlor has been observed to cause a variety of responses in exposed fish populations. Behavioral changes, including slow movement and slow response to stimuli, have been observed in exposed salmon fry (Dean and Others 1977). In addition to behavioral alterations, methoxychlor has caused reproductive impairment in exposed fish. Reproductive effects caused by methoxychlor exposure include decreased egg production, decreased hatchability, and increased abnormalities in offspring (Holdway and Dixon 1986).

3.5.5 Birds

Birds metabolize methoxychlor relatively easily and as a result, do not accumulate methoxychlor to elevated levels (Gardner and Bailey 1975, as cited in Beyer and Others 1996). No evidence has been found regarding methoxychlor residues in relation to increased mortality rates (Beyer and Others 1996). Methoxychlor has caused feminization in male gull chick embryos. Methoxychlor was injected into developing gull eggs, which resulted in abnormal development of ovarian tissue in male hatchlings (Fry and Toone 1981, as cited in Peterle 1991).

3.5.6 Mammals

Methoxychlor is an organochlorine, so its toxic effects are similar to other organochlorines such as DDT. The primary sites of toxic action are the sensory and motor nerve fibers and the motor cortex in the brain. Symptoms of poisoning include dizziness, disturbed equilibrium, tremors, and convulsions. Liver damage may also occur in mammals exposed to high levels of methoxychlor (Amdur and others 1991).

4.0 DIOXINS AND FURANS

Polychlorodibenzodioxins (PCDD) and polychlorodibenzofurans (PCDF) are chemically classified as halogenated aromatic hydrocarbons. Chlorinated and brominated dibenzodioxins and dibenzofurans are structurally similar tricyclic aromatic compounds, with similar physical and chemical properties. The terms "dioxins" and "furans" refer to the 210 possible congeners of chlorine substitution in PCDDs and PCDFs. These compounds are grouped together because of their chemical, physical, and toxicological similarities. There are 75 possible PCDD congeners and 135 possible PCDF congeners. Only 7 of the 75 possible PCDD congeners and 10 of the 135 possible PCDF congeners contain four chlorines, and substitutions in the 2,3,7,8 positions are the most toxic. Furthermore, 2,3,7,8-tetrachlorodibenzodioxin (TCDD) is considered to be the most potent and also is the most widely studied of all these compounds.

In general, these compounds have very low water solubility, high octanol-water partition coefficients, and low vapor pressure and tend to bioaccumulate. Despite a growing body of literature from laboratory, field, and monitoring studies examining the environmental fate and environmental distribution of PCDDs

and PCDFs, the fate of these environmentally ubiquitous compounds is not well understood. In soil, sediment, and the water column, PCDDs and PCDFs primarily are associated with particulate and organic matter because of their high hydrophobicity or low water solubility. Higher chlorinated congeners, specifically the hexa- through hepta-congeners, principally are sorbed to airborne particulates, whereas the tetra- and penta-congeners significantly, if not predominantly, partition to the vapor phase. Dioxin-like compounds exhibit little potential for significant leaching or volatilization once sorbed to particulate matter. The available evidence indicates that PCDDs and PCDFs, particularly the tetra- and higher-chlorinated congeners, are extremely stable compounds under most environmental conditions. The only environmentally significant transformation process for these congeners is probably photodegradation of nonsorbed species in the gaseous phase, at the soil-air interface, or in association with organic cosolvents. PCDDs and PCDFs entering the atmosphere are removed either by photodegradation or by deposition. Burial in place, resuspension back into the air, or transportation into water bodies through erosion of soil are the predominant fates of PCDDs and PCDFs sorbed to soil (MacCrady and Maggard 1993).

Bioavailability of PCDDs and PCDFs is quantified as an estimate relative to that of TCDDs. For example, the bioavailability of PCDDs and PCDFs in water is evaluated using bioaccumulation equivalency factors. The following sections present additional data on ecotoxicological effects of PCDDs and PCDFs.

4.1 PLANTS

The primary mechanism by which dioxin-like compounds enter the terrestrial environment is through atmospheric deposition, after which they become bioavailable to terrestrial organisms. Deposition can occur directly onto plant surfaces or onto soil. PCDDs and PCDFs in soil can become available to plants by volatilization and vapor absorption or through particle resuspension and adherence to plant surfaces. In addition, PCDDs and PCDFs in soil can adsorb directly to underground portions of plants. Uptake from soil through the roots into aboveground portions of plants, however, is thought to be insignificant (MacCrady and Maggard 1993).

4.2 INVERTEBRATES

No information was identified on the effects of PCDDs and PCDFs on invertebrates.

4.3 AMPHIBIANS AND REPTILES

No information was identified on the effects of PCDDs and PCDFs on amphibians and reptiles.

4.4 FISH AND AQUATIC ORGANISMS

No information was identified on the effects of PCDDs and PCDFs on fish and aquatic organisms.

4.5 BIRDS

Dioxins can elicit a broad range of toxic effects; however, their mode of action is structure-specific and receptor-mediated (ATSDR 1987). Exposure to dioxins (and other sterically similar halogenated aromatics) results in their interaction with the Ah receptor and leads to altered enzymatic activity, for example, induced cytochrome P₄₅₀ activity (Hansen 1994). Furthermore, dioxins have been found to be antiestrogenic and antiandrogenic (Hansen 1994). In birds, dioxin exposure can lead to weight loss and death (wasting syndrome); embryotoxicity is one of the more sensitive endpoints (EPA 1995b). Reproductive failure in wild populations of herring gulls, terns, and other birds has been linked to exposure to dioxins and other dioxin-like chemicals that can interact with the receptor such as PCBs (Hoffman and Others 1995).

4.6 MAMMALS

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toxicity, and effects to bone marrow and epithelial tissue (EPA 1995b). Subchronic and chronic effects on mammals include those described for acute exposure, as well as impaired reproduction and modified social behavior.

5.0 POLYCHLORINATED BIPHENYLS

PCBs, or Aroclors®, are among the most persistent anthropogenic compounds in the environment, resisting degradation for years. This family of compounds contains 209 individual congeners. PCBs have been widely used as coolants and lubricants because of their insulating and nonflammable properties. PCB manufacturing was halted in 1977 because of the ability of these compounds to bioaccumulate in the environment and adversely affect wildlife.

PCBs remain in the environment for long periods of time, because physicochemical properties of PCBs allow them to resist environmental degradation processes. The degree of chlorination and the position of chlorine substituents of PCBs influence their susceptibility to environmental degradation processes. Because of their physicochemical characteristics, PCBs partition into soil and sediment particles and organic matter. Depending on the particular PCB congener, K_{oc} range from 510 to 13,300,000. As a general rule, most K_{oc} for PCB mixtures are greater than 5,000 (Sklarew and Others 1987, as cited in HSDB 1999).

The environmental persistence of PCBs, combined with their hydrophobic nature, allows them to bioconcentrate, bioaccumulate, and biomagnify in the environment. Physicochemical properties of PCBs influence the extent of uptake and metabolic fate of PCBs in an organism. The ability of animals to metabolize PCBs tends to increase in the following order: fish < birds < mammals (Matthews and Others 1984, as cited in HSDB 1999). PCBs are absorbed from the stomach, skin, and lungs. PCBs initially concentrate in the liver, blood, and muscle, and if they are resistant to metabolism, they are sequestered into fat tissue, where they persist.

In general, bioaccumulation of PCBs is rapid, depuration is slow, and diet appears to be an important route of PCB accumulation. PCB body burdens in marine organisms, particularly benthic organisms, appear to be directly related to log PCB concentrations in sediments (Shaw and Connell 1982, as cited in

Eisler 1986b). In terrestrial or semi-aquatic vertebrates, PCBs tend to bioaccumulate to their highest concentrations in the livers of fish-eating birds, followed by species that feed on small birds and mammals, worms, and insects (NAS 1979, as cited in Eisler 1986b).

5.1 PLANTS

Soil algae exhibit depressed photosynthesis and growth inhibition when exposed to concentrations of PCBs (Eisler 1986b). In other studies, algae exposed to PCB concentrations had an increased mortality rate, decreased reproduction, and impaired growth (Beyer and Others 1996).

5.2 INVERTEBRATES

No information was identified on the effects of PCBs on terrestrial invertebrates.

5.3 AMPHIBIANS AND REPTILES

No information was identified on the effects of PCBs on amphibians and reptiles.

5.4 FISH AND AQUATIC ORGANISMS

Adverse effects associated with exposure to PCBs in aquatic organisms include decreased growth, reproductive toxicity, mutagenicity, histopathology, and a variety of biochemical perturbations (Eisler 1986b; Beyer and Others 1996). Reproductive toxicity has been reported for several aquatic species, and effects include reduced survival of developing eggs, increased mortality in eggs and fry, and reduced fertilization success (Eisler 1986b).

5.5 BIRDS

Birds exposed to PCB concentrations have shown the following effects: disruptions in normal patterns of growth, reproduction, metabolism, and behavior (Peakall and Others 1972, as cited in Eisler 1986b). PCBs can damage the liver, kidney, spleen, and thyroid in birds (Beyer and Others 1996). However,

reproductive impairment is the most sensitive endpoint for birds exposed to PCBs. Reproductive effects caused by PCB exposure include embryo mortality, teratogenic effects, decreased hatching success, and reduced eggshell thickness (Beyer and Others 1996).

5.6 MAMMALS

PCB exposures in mammals are associated with adverse effects, including reproductive impairment and failure, physiological, altered behavior, and mutagenic, carcinogenic, and teratogenic (Eisler 1986b; Beyer and Others 1996). In mammals, the most consistent, pathological changes occur in the liver. In females, however, PCBs are transferred through lactation and placenta from adult to offspring; therefore, in utero effects and fetotoxicity may be expressed.

6.0 POLYNUCLEAR AROMATIC HYDROCARBONS

PAH range from low-molecular-weight PAHs composed of two aromatic rings, including naphthalene, to high-molecular-weight PAHs, bearing three or more aromatic rings, including phenanthrene and benzo(a)pyrene. There are thousands of PAH compounds, each differing in the number and position of the aromatic rings and the amount of substitutions in their basic structure. These substitutions may include methyl, oxygen, or other elemental groups. PAH solubility decreases with increasing molecular weight and increasing log K_{ow} values (Eisler 1987).

In aquatic environments, PAHs may evaporate, disperse in the water column, become incorporated into bottom sediments, concentrate in aquatic biota, or experience photodegradation, chemical oxidation, or biodegradation (Suess 1976, as cited in Eisler 1987). Depending on the physicochemical characteristics of the individual compound, the compound will sorb primarily to suspended sediment or colloids, while only about 30 to 35 percent will dissolve into the water column (Lee and Grant 1981, as cited in Eisler 1987). PAHs in aquatic sediments degrade very slowly in the absence of penetrating radiation (Suess 1976, as cited in Eisler 1987) and may persist for long time periods in oxygen-poor basins or in anoxic sediments (Neff 1979, as cited in Eisler 1987).

PAHs cause a wide variety of adverse biological effects in numerous organisms under laboratory conditions, including effects on survival, growth, metabolism, and tumor formation. Inter- and intraspecies responses to PAHs are quite variable and are modified by interaction with other inorganic and organic compounds, including other PAHs (Eisler 1987). PAHs also have been observed to enhance cytochrome P₄₅₀ levels in the liver. Cytochrome P450 and mixed function oxidases primarily are responsible for detoxifying chemicals in the liver, and when these chemicals are altered, the toxicity of other compounds that the organisms encounter may be greater than they would be otherwise. In this way, PAHs lower the organism's resistance to other toxins (Amdur and Others 1991). Higher-molecular-weight PAHs are known to be carcinogenic, mutagenic, and teratogenic to a wide variety of organisms, including aquatic life, amphibians, birds, and mammals. Lower-molecular-weight PAHs generally are not carcinogenic but are more acutely toxic than their higher-molecular-weight relatives (Eisler 1987). Lower-molecular-weight PAHs show little tendency to biomagnify in food chains and are relatively rapidly metabolized in vertebrates, provided that toxicity is not expressed.

Low concentrations of PAHs are readily and rapidly bioconcentrated and bioaccumulated by many species of aquatic organisms in freshwater or marine settings. The ability of PAHs to bioconcentrate and bioaccumulate is chemical-specific. Molecular weight, the position of the aromatic rings, and the type of substitutions all influence the ability of a compound to bioconcentrate and bioaccumulate in ecological receptors. Species-specific characteristics also are critical in evaluating the bioconcentration potential of PAHs; organisms with relatively high lipid contents tend to bioconcentrate PAHs to greater amounts than others (Eisler 1987).

6.1 PLANTS

The biological fate of PAHs in soils is poorly understood. Much of the available information suggests that plants, fungi, and soil bacteria absorb lower-molecular-weight PAHs from soils. These materials may be available for uptake from the rhizosphere through the root (ATSDR 1995a). In general, phytotoxic effects caused by PAH exposure have not been observed; however, very little data have been published on this subject (Eisler 1987). One study observed that PAH concentrations inhibited algae growth (Bastian and Toetz 1982, as cited in Environment Canada 1994b).

6.2 INVERTEBRATES

No information was identified on the effects of PAHs on invertebrates.

6.3 AMPHIBIANS AND REPTILES

No information was identified on the effects of PAHs on amphibians and reptiles.

6.4 FISH AND AQUATIC ORGANISMS

PAHs vary substantially in their toxicity to aquatic organisms. Toxicity of PAHs tends to increase with increasing molecular weight, particularly increasing alkyl substitution. PAHs have been linked to various liver tumors in fish (Environment Canada 1994b), and increased mortality has been observed in water fleas, nymphs, and mayflies exposed to PAHs (Krantzberg and Boyd 1992, Murphy and Others 1993, as cited in Environment Canada 1994b). Sediment PAH concentrations have been observed to cause liver lesions in flatfish and brown bullheads (Eisler 1987). PAHs and their metabolites also suppress the immune system in aquatic organisms.

6.5 BIRDS

For birds, several investigations have suggested that the presence of PAHs in petroleum causes embryo toxicity in avian species (Hoffman and Gay 1981, Albers 1983, as cited in Eisler 1987). Embryo effects include reduced embryo growth, incomplete skeletal ossification, and defects in the eye, brain, liver, and bill.

6.6 MAMMALS

PAH compounds are distinct in their ability to produce tumors in skin and in most epithelial tissues of mammals. Some PAHs have been linked to increased occurrence of leukemia, lung adenoma, and stomach tumors in mice. PAHs have resulted in altered hematopoietic function, as well as development of pathological conditions in lymphoid tissues. Impaired reproduction is frequently observed in chronic

exposures where ovotoxicity and antispermatogenic effects have been noted. Other systemic effects include adrenal necrosis, as well as changes in the intestinal and respiratory epithelia. Many PAHs induce metabolic enzymes, including the mixed-function oxidases present in the liver. Some PAHs and their metabolites also suppress the immune system, which has resulted in decreased responsiveness to mitogens and an increased susceptibility to disease. The majority of available information on toxicological properties of PAHs is focused on their carcinogenicity to mammals (EPA 1980, Lee and Grant 1981, as cited in Eisler 1987).

7.0 SEMIVOLATILE ORGANIC COMPOUNDS

The following sections present data on ecotoxicological effects of various SVOCs identified as ecological COPCs at Alameda Point.

7.1 4-CHLORO-3-METHYLPHENOL

Chlorophenols are a group of chemicals that are produced by adding chlorines to phenol. Some chlorophenols are used as pesticides, while others are used in antiseptics (ATSDR 1999b). 4-chloro-3-methylphenol can be formed inadvertently in waters that have undergone chlorination treatment and by evaporation or waste releases from product formulation or end products containing the compound (NLM 2003).

Chlorophenols will stick to soil and sediments at the bottom of lakes, streams, and rivers. Low levels of chlorophenols in water, soil, or sediment are broken down and removed from aerobic environments in a few days to weeks by microorganisms. However, chlorophenols are not readily degradable in anaerobic environments and are relatively persistent in groundwater aquifers. Aquatic hydrolysis, bioconcentration, and volatilization are not important processes of the environmental fate of 4-chloro-3-methylphenol (NLM 2003; ATSDR 1999b).

7.1.1 Plants

A study using lettuce seeds were exposed for duration of 21 days to a direct application of 4-chloro-3-methylphenol, which determined an effects concentration 50 (EC50) of 2.3 mg/L (EPA 2003).

7.1.2 Invertebrates

No information was identified on the effects of 4-chloro-3-methylphenol on invertebrates.

7.1.3 Amphibians and Reptiles

No information was identified on the effects of 4-chloro-3-methylphenol on amphibians and reptiles.

7.1.4 Fish and Aquatic Organisms

Various toxicity tests for 4-chloro-3-methylphenol have been conducted using fish and other aquatic organisms. These studies have determined EC50 and LC50 concentrations. LC50 concentrations for such species as the water flea and the great pond snail after a 96-hour exposure duration range from 3.1 mg/L to 17.2 mg/L (EPA 2003).

7.1.5 Birds

A study using 16-week old northern bobwhite quail were exposed for duration of 14 days to 4-chloro-3-methylphenol through the oral exposure route. This study determined a LC50 of 1,540 mg/kg (EPA 2003).

7.1.6 Mammals

In laboratory studies, animals that received high levels of chlorophenols in food or water developed liver and immune system effects. They did not gain as much weight as animals not fed the compounds (ATSDR 1999b).

High levels of chlorophenols given to pregnant female rats in their drinking water reduced the number of babies they had, and caused low birth weights. Chlorophenols have not been shown to cause birth defects in animals (ATSDR 1999b).

7.2 BIS(2-ETHYLHEXYL)PHTHALATE

Bis(2-ethylhexyl)phthalate is used widely to make plastics. Bis(2-ethylhexyl)phthalate is a component of many products found in homes and automobiles, as well as in the medical and packaging industries. Its wide use and distribution, as well as its high volatility and persistence, lead to its common occurrence in fish, water, and sediments.

Acute toxic effects may include the death of animals, birds, or fish and death of, or low growth rate in, plants. Acute effects are seen 2 to 4 days after animals or plants come in contact with a toxic chemical substance. Bis(2-ethylhexyl)phthalate has low acute toxicity to aquatic life. Insufficient data are available to evaluate or predict the short-term effects of bis(2-ethylhexyl)phthalate to plants, birds, or land animals.

Chronic toxic effects may include shortened lifespan, reproductive problems, lower fertility, and changes in appearance or behavior. Chronic effects can be seen long after first exposure to a toxic chemical. Bis(2-ethylhexyl)phthalate has low chronic toxicity to aquatic life. Insufficient data are available to evaluate or predict the long-term effects of bis(2-ethylhexyl)phthalate to plants, birds, or land animals.

Bis(2-ethylhexyl)phthalate is slightly soluble in water. Concentrations of less than 1 milligram will mix with a liter of water. Bis(2-ethylhexyl)phthalate is slightly persistent in water, with a half-life of between 2 to 20 days. About 42.8 percent of bis(2-ethylhexyl)phthalate eventually will end up in terrestrial soil; about 40 percent will end up in aquatic sediments; and about 17 percent will end up in air. Some substances increase in concentration, or bioaccumulate, in living organisms as they breathe contaminated air, drink contaminated water, or eat contaminated food. These chemicals can become concentrated in the tissues and internal organs of animals and humans.

7.2.1 Plants

No information was identified on the effects of bis(2-ethylhexyl)phthalate on plants.

7.2.2 Invertebrates

No information was identified on the effects of bis(2-ethylhexyl)phthalate on invertebrates.

7.2.3 Amphibians and Reptiles

No information was identified on the effects of bis(2-ethylhexyl)phthalate on amphibians and reptiles.

7.2.4 Fish and Aquatic Organisms

The concentration of bis(2-ethylhexyl)phthalate found in fish tissues is expected to be much higher than the average concentration of in the water from which the fish was taken; however, little information was identified on the effects of bis(2-ethylhexyl)phthalate on the toxicity of fish and aquatic organisms.

7.2.5 Birds

No information was identified on the effects of bis(2-ethylhexyl)phthalate on birds.

7.2.6 Mammals

While significant reproductive effects were observed among mice on diets containing 0.1 percent and 0.3 percent bis(2-ethylhexyl)phthalate, no adverse effects were observed among the 0.01 percent dose group. Because the study considered exposure during critical lifestage, the 0.01 percent dose was considered to be a chronic no observed adverse effect level (NOAEL). The 0.3 percent dose was considered to be a chronic lowest observed adverse effect level (LOAEL).

7.3 BUTYLBENZYLPHthalate

Butylbenzylphthalate is used as a plasticizer for polyvinyl chloride, nitrocellulose resin, and polyurethane; as an organic intermediate; and as a coating for electric wire. Butylbenzylphthalate is expected to have low mobility in soil and will readily volatilize from wet soils, but not from dry. Biodegradation is expected to occur rapidly with estimated half-lives in the range of 4 to 13 days. In aquatic environments, butylbenzylphthalate will adsorb to sediment or particulate matter. Volatilization from water surfaces will occur, as well. The potential for bioconcentration in aquatic organisms is considered high based on experimental BCF values of 772 measured for bluegill sunfish (NLM 2003).

7.3.1 Plants

No information was identified on the effects of butylbenzylphthalate on plants.

7.3.2 Invertebrates

Invertebrates readily metabolize butylbenzylphthalate; however, no information was identified on the effects of this compound on soil invertebrates.

7.3.3 Amphibians and Reptiles

No information was identified on the effects of butylbenzylphthalate on amphibians and reptiles.

7.3.4 Fish and Aquatic Organisms

Butylbenzylphthalate was observed to be acutely toxic to a variety of algae, invertebrates, and fish in the 0.5 to 5 mg/L range and can be chronically toxic to daphnia and fathead minnows in the 0.1 to 0.8 mg/L range. A 48-hour EC50 concentration was observed at 1 mg/L. The 96-hour median lethal concentration values of butylbenzylphthalate for English sole were 0.66 mg/L and 0.55 mg/L with lethal threshold concentrations of 0.45 mg/L and 0.3 mg/L, respectively (NLM 2003).

7.3.5 Birds

No information was identified on the effects of butylbenzylphthalate on birds.

7.3.6 Mammals

Recent extensive dose toxicity studies, primarily in rats, have been conducted for butylbenzylphthalate. Effects observed have been 1) decreases in body weight gain; 2) increases in organ to body weight ratios, particularly for the kidney and liver; 3) degenerative effects on the testes, at higher doses. Chronic toxicity and carcinogenicity in rats and mice have indicated that butylbenzylphthalate is carcinogenic in female rats, indicated by pancreatic and bladder tumors. Only minor evidence of carcinogenicity in male rats was indicated. However, no indication of carcinogenicity in mice was observed. Teratogenic studies in male rats have indicated that only at doses higher than those that induce effects on other organs, adversely affect the testes and consequently fertility (NLM 2003)

7.4 DI-N-BUTYLPHthalate

Di-n-butylphthalate is used as a plasticizer, solvent for resins, fuel propellant, and insect propellant (NLM 2003). It is only slightly soluble in water and does not easily evaporate (or volatilize) (ATSDR 2001). Di-n-butylphthalate is expected to have low mobility in soil. Biodegradation is expected to occur under both aerobic and anaerobic conditions. In aquatic environments, di-n-butylphthalate will adsorb to sediment or particulate matter. Volatilization from water surfaces will be slow. The compound is expected to biodegrade in aquatic environments with estimated half-lives of about 3 and 28 days for aerobic and anaerobic conditions, respectively (NLM 2003, ATSDR 2001). Teratogenic effects from exposure to di-n-butylphthalate have been observed in several studies, as noted in the following sections.

7.4.1 Plants

Cabbage seedlings were killed when the volume of di-n-butylphthalate reached toxic levels (concentrations of up to 2,010 picograms per liter) in the air (NLM 2003).

7.4.2 Invertebrates

Di-n-butylphthalate was not toxic to female houseflies when applied at high doses, either topically or by injection (NLM 2003).

7.4.3 Amphibians and Reptiles

No information was identified on the effects of diethylphthalate on amphibians and reptiles.

7.4.4 Fish and Aquatic Organisms

Based on experimental BCF values ranging from 12 to 117 measured in oysters, fish, and shrimp, bioconcentration in aquatic organisms is low to moderate (NLM 2003).

Toxicities to fecundity in *Daphnia magna* and the fathead minnow were assessed and it was determined that 1.8 milligrams per liter of di-n-butylphthalate showed significant reduction in reproduction for *Daphnia magna* and decrease in the survival of fathead minnow embryos (NLM 2003).

7.4.5 Birds

Mallard ducks fed a continuous diet of duck mash containing 10 mg/kg of di-n-butylphthalate for 5 months, showed no significant accumulation of the chemical.

7.4.6 Mammals

Di-n-butylphthalate is not classified as a human or animal carcinogen. Teratogenic effects have been seen in rat studies during gestation. Effects such as skeletal abnormalities, such as, absence of tail, twisted legs, and abnormal skull bones, as well as, reduced weight of fetuses were observed compared to controls (NLM 2003).

7.5 PENTACHLOROPHENOL

Pentachlorophenol (PCP) is a semivolatile, chlorinated phenolic compound and is a priority pollutant. PCP is an anthropogenic substance, made from other chemicals, and does not occur naturally in the environment. At one time, it was one of the most widely used biocides in the United States. PCP is a restricted-use insecticide, fungicide, molluscicide, defoliant, herbicide, and wood preservative, and it is no longer available to the general public. PCP has two forms: pentachlorophenol and the sodium salt of pentachlorophenol. The sodium salt dissolves easily in water, but PCP does not. This compound is among 31 substances classified by the Worker Health and Safety Unit of the California Department of Food and Agriculture as having high carcinogenic or oncogenic potential and is listed by EPA as a carcinogen. PCP is a substance whose widespread use, mainly in wood protection and pulp and paper mills, has led to extensive environmental contamination.

7.5.1 Plants

Hulzebos and Others (1993) evaluated the effects of PCP on lettuce growth in two soils and in solution. Soils with high clay content produced higher EC₅₀ values for PCP. Toxic levels of PCP caused reduced fresh weight of shoots of oats (*Avenas sativa*) after 14 days of exposure and turnips (*Brassica rapa*) after 10 days of exposure in a sandy loam soil. The EC₅₀s for oats and turnips were 20 and 10 ppm, respectively.

7.5.2 Invertebrates

Fitzgerald and Others (1996) investigated the role of species, temperature, and soil type on toxicity of PCP to earthworms. Researchers estimated incipient lethal levels (ILL), which they define as a time-dependent LC₅₀. ILLs for *Eisenia fetida* grown for 14 days in an artificial soil at 24 or 15 °C were 37 and 27 ppm, respectively. The ILL for *Eisenia fetida* grown for 14 days in a clay soil at 24°C was 72 ppm. The ILL for *Eisenia fetida* at 24 °C was 168 ppm. *Lumbricus terrestris* grown at 15 °C had an ILL of 191 ppm.

7.5.3 Amphibians and Reptiles

PCP may affect growth and survival in sensitive aquatic species.

7.5.4 Fish and Aquatic Organisms

Little information was identified on the effects of PCP on fish and aquatic organisms.

7.5.5 Birds

Avian fatalities have been reported for low oral doses of PCPs. Chlorophenols appear to be mildly hepatotoxic, and studies in animals indicate that PCP may reduce humoral and cell-mediated immunity, as well as act as a cocarcinogen (HSDB 1999). Acute, dietary studies (using oral doses high enough to cause relatively quick toxicity), feeding this compound to mammal and bird species, place it in the "moderately toxic" category for mallards and pheasants and the "relatively nontoxic" category for Japanese quail.

7.5.6 Mammals

Animal studies indicate that rat embryos are most susceptible to the toxic effect of PCP during the early phases of organogenesis (HSDB 1999). Teratogenic activities of highly purified PCP and pentachloroanisole, administered in the diet of Sprague-Dawley rats of both sexes, revealed embryonic deaths following treatment with PCP at the rate of 43 mg/kg/day, while lower doses of the compound induced dose-related reductions in body weight. At the rate of 13 mg/kg/day, PCP reduced the crown to rump length and increased the skeletal alterations of the fetus. Decreased numbers of corpora lutea and embryonic death were recorded following the administration of PCP at the rate of 4 and 41 mg/kg/day. At the same dose, PCP reduced the body weight and crown to rump length of male fetuses, while their female counterparts were not affected.

8.0 VOLATILE ORGANIC COMPOUNDS

The following sections present data on ecotoxicological effects of the VOCs identified as ecological COPCs at Alameda Point.

8.1 1,1-DICHLOROETHENE AND 1,2-DICHLOROETHENE

1,1-dichloroethene (DCE) and 1,2-DCE are clear, colorless, flammable liquids, consisting of a mixture of the cis- and trans-isomers. It is used as an intermediate to make other chemicals and as a cleaning solvent. It may enter the environment from industrial or municipal discharges or spills.

Acute effects may include shortened lifespan, reproductive problems, lower fertility, and changes in appearance or behavior. Chronic toxicity of DCE is moderate in aquatic life. Insufficient data are available to evaluate or predict the long-term effects of DCE to plants, invertebrates, birds, or terrestrial animals.

8.1.1 Plants

No information was identified on the effects of DCE on plants.

8.1.2 Invertebrates

No information was identified on the effects of DCE on invertebrates.

8.1.3 Amphibians and Reptiles

No information was identified on the effects of DCE on amphibians and reptiles.

8.1.4 Fish and Aquatic Organisms

No information was identified on the effects of DCE on fish and aquatic organisms.

8.1.5 Birds

No information was identified on the effects of DCE on birds.

8.1.6 Mammals

No information was identified on the effects of DCE on mammals.

8.2 ACETONE

Acetone is a colorless, liquid VOC that has a pungent, sweet odor and taste (Budavari 1996). Acetone is used as a solvent for fats, oils, waxes, resins, rubber, plastics, lacquers, varnishes, and rubber cement; in the manufacture of acetyl acid, chloroform, and other chemicals; for extraction of plant and animal substances; in purifying paraffin; and a main ingredient in nail polish remover (Budavari 1996).

Acetone is considered to be the least toxic solvent used in industry, but it is highly volatile, making inhalation the primary route of exposure. Dermal contact is a secondary route of exposure. The mechanism by which acetone enters the environment is through wastewater from industries who use and produce the compound. Acetone is not persistent in the environment and once released, it will volatilize quickly into the air from both soils and water (Benkelbberg and Others 1995, Alarie and Others 1995, as cited in HSDB 1999). It is not expected to adsorb to sediments or soils and because of its hydrophilic characteristic, it is not expected to bioconcentrate in wildlife receptors (HSDB 1999). As a vapor, acetone will undergo photodegrade (HSDB 1999)

8.2.1 Plants

No information was identified on the effects of acetone on plants.

8.2.2 Invertebrates

No information was identified on the effects of acetone on invertebrates.

8.2.3 Amphibians and Reptiles

No information was identified on the effects of acetone on amphibians and reptiles.

8.2.4 Fish and Aquatic Organisms

Results of various ecotoxicity tests of aquatic organisms produced LC₅₀ and LD₅₀ values from acetone exposure. The LC₅₀ for brine shrimp and *Daphnia magna* is 2,100 mg/L 24 to 48 hr and 10 mg/L 24 to 48 hr, respectively. In fish, LC₅₀ values for the bluegill sunfish, mosquito fish, and the guppy were 8,300 mg/L 96 hr; 13,000 mg/L 24 to 96 hr; and 7,032 mg/L 14 days, respectively. The LD₅₀ for the goldfish was 5,000 mg/L 24 hr (Verschuere 1996). From this data, the lower trophic levels of aquatic organisms seem to exhibit greater sensitivity for adverse effects from acetone exposure than the higher trophic-level organisms.

8.2.5 Birds

Very few studies were identified on the effects of acetone on birds. Two ecotoxicity studies, on the ring-necked pheasant and the Japanese quail, established the LC₅₀ for these organisms at 40,000 mg/L of acetone in the diet at an age of 10 to 14 days. No mortality of these birds was seen at concentrations below 40,000 mg/L (Hill and Camardese 1986, [USFWS] 1975, as cited in HSDB 1999). Ameenuddin and Sunde (1984) investigated the sensitivity of developing chicken embryos to various solvents and found that acetone, at a concentration of 0.10 mL per egg injected, significantly reduced the percentage hatchability and caused high embryonic mortality during the first week of incubation.

8.2.6 Mammals

Studies of various mammal receptors exposed to acetone vapors have shown very few (if any) ill effects, other than slight irritation of the eyes and nose (in cats), loss of reflexes (in guinea pigs), moderate corneal injury (in rabbits and rats), and cataracts (in guinea pigs) (Clayton and Clayton 1982). Very little information was found on the effects of ingestion of acetone-saturated water; however, this is the least likely route of exposure for wildlife.

8.3 BENZENE

Benzene is present in gasoline as an additive, and is manufactured to produce a variety of chemicals such as polymers, detergents, pesticides pharmaceuticals, dyes, plastics, and resins. Also, benzene can be used as a solvent for waxes, resins, oils, and natural rubbers. Benzene is naturally present in volcanos, as a constituent of crude oil, in forest fires, and as a plant volatile. Benzene is highly water-soluble.

Generally, benzene will be removed from the atmosphere by rain. Volatility from moist soils is greater than from dry soils, and volatility from water surfaces is an important fate process. Soil mobility of benzene is relatively high. Abiotic degradation of benzene in the atmosphere will occur, with the half-life of the reaction estimated at 13 days. Biodegradation of benzene in water is expected. Benzene is not expected to bioconcentrate in aquatic organisms; however, bioconcentration in mammals (humans) has been observed. Benzene has been classified as a known human carcinogen for all routes of exposure (NLM 2003).

8.3.1 Plants

No information was identified on the effects of benzene on plants.

8.3.2 Invertebrates

No information was identified on the effects of benzene on invertebrates.

8.3.3 Amphibians and Reptiles

No information was identified on the effects of benzene on amphibians and reptiles.

8.3.4 Fish and Aquatic Organisms

Adverse growth effects in blue crab juveniles were observed when exposed to sublethal concentrations (0.1 or 5 ppm) of benzene. The 96-hour LC 50 concentration for grass shrimp was measured at 27 ppm; 1,108 ppm for crab larvae; and 5.8 to 11 mg/l for bass (NLM 2002).

8.3.5 Birds

No information was identified on the effects of benzene on birds.

8.3.6 Mammals

Many experimental animal studies have indicated that benzene is a carcinogen. Benzene increases the risk of cancer in multiple organ systems, including the hematopoietic system, oral and nasal cavities, liver, stomach, lung, ovaries, and mammary gland.

Respiratory effects have been noted in cats, primates, rabbits, and rats when exposed to benzene in acute inhalation studies.

Teratogenic effects have been noted in rats and mice studies. Such effects include decrease in pup survival and decrease in mean pup weights. No effects were noted in these studies between treated and control animals for maternal mortality, pregnancy rates, mean number of dead pups, and maternal body weight.

8.4 CARBON DISULFIDE

Carbon disulfide is used as a process solvent for phosphorus, sulfur, selenium, bromine, iodine, fats, resins, and rubbers, and as a chemical intermediate in the manufacture of rayon, carbon tetrachloride, xanthogenates, soil disinfectants, and electronic vacuum tubes. The ocean, primarily the coastal area or other areas of high biological productivity, is a major global source of carbon disulfide. This "natural" source of carbon disulfide is generated by the reduction of sulfates in soil. Carbon disulfide is only slightly mobile in soils. Volatilization from moist soils occurs more rapidly than from dry soils, and volatilization from water surfaces is an important process in the fate and transport of carbon disulfide. Carbon disulfide does not significantly bioaccumulate in aquatic organisms (NLM 2002).

8.4.1 Plants

No information was identified on the effects of carbon disulfide on plants.

8.4.2 Invertebrates

No information was identified on the effects of carbon disulfide on invertebrates.

8.4.3 Amphibians and Reptiles

No information was identified on the effects of carbon disulfide on amphibians and reptiles.

8.4.4 Fish and Aquatic Organisms

No information was identified on the effects of carbon disulfide on fish and aquatic organisms.

8.4.5 Birds

No information was identified on the effects of carbon disulfide on birds.

8.4.6 Mammals

Teratogenic effects were studied in rats. Rats were exposed to 642 ppm carbon disulfide for 2 hours per day for the entire pregnancy. The results indicated that the reproductive success of treated groups was less than that of the control groups (NLM 2002).

8.5 ETHYLBENZENE

Ethylbenzene is a colorless liquid that occurs together with other aromatics. A typical, complex mixture of aromatics may be more toxic, hazardous, or carcinogenic in general than this compound would be alone.

Except for short-term hazards from concentrated spills, this compound has been associated more frequently with risk to humans than with risk to nonhuman species, such as fish and wildlife. This difference occurs partly because plants, fish, and birds take up only very small amounts and because this volatile compound tends to evaporate into the atmosphere, rather than persisting in surface waters or soils (ATSDR 1990d). However, VOCs, such as ethylbenzene, can pose a drinking water hazard when they accumulate in groundwater.

Ethylbenzene releases to water occur as a result of industrial discharges, use of gasoline fuel for boating, fuel spillage, leaking underground storage tanks, landfill leachate, and the inappropriate disposal of waste (ASTDR 1990d). Ocean releases occur as a result of offshore oil production, hydrocarbon venting, oil field brines, and tanker oil spills (ASTDR 1990d).

Effects of this volatile solvent to nonhuman biota often result from high concentrations immediately after a spill (before the compound has volatilized into the atmosphere) or as the indirect result of contamination of groundwater. For example, if highly polluted groundwater enters surface waters from spring or seeps, local effects may occur in the mixing zone.

In comparison to chemicals such as PCBs, DDT, and other chlorinated pesticides, which are of great concern with respect to bioaccumulation, ethylbenzene does not bioaccumulate significantly in aquatic food species (ATSDR 1990d).

8.5.1 Plants

Little information was identified on the effects of ethylbenzene on plants.

8.5.2 Invertebrates

Little information was identified on the effects of ethylbenzene on invertebrates.

8.5.3 Amphibians and Reptiles

Little information was identified on the effects of ethylbenzene on amphibians and reptiles.

8.5.4 Fish and Aquatic Organisms

Little information was identified on the effects of ethylbenzene on fish and aquatic organisms.

8.5.5 Birds

Little information was identified on the effects of ethylbenzene on birds.

8.5.6 Mammals

Little information was identified on the effects of ethylbenzene on mammals.

8.6 TETRACHLOROETHENE

Tetrachloroethene, also known as perchloroethylene (PCE), is a carcinogenic priority pollutant that is used in dry cleaning and as an industrial solvent (EPA 1986, 1991).

This synthetic compound is widely used for dry cleaning fabrics and for metal degreasing operations. It also is used as a starting material for making other chemicals and is used in some consumer products. It evaporates easily into the air and has a sharp, sweet odor. Most people can smell PCE when it is present in the air at a level of 1 ppm or more.

PCE enters the environment mostly by evaporating into the air during use. It also can enter water supplies and soil during disposal of sewage sludge and factory waste. PCE also may enter the air, soil, or water by leaking or evaporating from storage and waste sites (ATSDR 1995b).

PCE has received more publicity and attention as a potential hazard to humans than to fish and wildlife; therefore, more literature related to humans is available, and information on other species is comparatively sparse.

Effects of this volatile solvent to nonhuman biota often result from high concentrations immediately after a spill (before the compound has volatilized into the atmosphere) or as the indirect result of groundwater contamination. For example, if highly polluted groundwater comes into surface waters from springs or seeps, effects may occur in the mixing zone.

PCE has been shown to release lysosomal enzymes from granular fractions prepared from nematodes. Because the nematode gut seems to be specialized for lysosomal, intracellular digestion of nutrients, interference with this process may explain PCE action. It has been assumed that affected worms are paralyzed sufficiently to release their attachment to the intestinal wall (Goodman and Gilman 1975).

One potentially important aspect of PCE is its ability to break down into other hazardous compounds. PCE can be transformed by reductive dehalogenation to trichloroethylene, DCE, and vinyl chloride under anaerobic conditions. Therefore, when PCE levels have been reduced to acceptable levels, it is still

necessary to verify that concentrations of suspected hazardous breakdown products also are acceptably low.

EPA classified PCE as a probable human carcinogen (EPA 1986). It has been treated as a carcinogen for EPA preliminary remediation goals and risk-based concentration modeling (EPA 1995b, 1996).

PCE is likely to enter the environment through fugitive air emissions from dry cleaning and metal degreasing industries and by spills or accidental releases to air, soil, or water. If PCE is released to soil, it will evaporate into the atmosphere and leach to the groundwater. Biodegradation may be an important process in anaerobic soils based on laboratory tests with methanogenic columns. Slow biodegradation may occur in groundwater, where acclimated populations of microorganisms exist. If PCE is released to water, it will be subject to rapid volatilization, with estimated half-lives ranging from less than 1 day to several weeks. It will not be expected to significantly biodegrade, bioconcentrate in aquatic organisms, or significantly adsorb to sediment. PCE will not be expected to significantly hydrolyze in soil or water under normal environmental conditions. If PCE is released to the atmosphere, it will exist mainly in the gas phase and will be subject to photo-oxidation, with estimates of degradation time ranging from an approximate half-life of 2 months to complete degradation in an hour. Some PCE in the atmosphere may be subject to washout in rain, based on the solubility of PCE in water; PCE has been detected in rain.

Low to moderate potential exists for bioconcentration of PCE (Environment Canada 1993a). Using a reported log K_{ow} of 3.40, a BCF of 226 was estimated. Based on the reported and estimated BCFs, PCE will not be expected to significantly bioconcentrate in aquatic organisms (Barrows and Others 1978).

8.6.1 Plants

A study was designed to assess the effects of PCE on the phytoplankton community at initial concentrations of 1.2 and 0.44 mg/L in separated compartments of an experimental pond. Measurements in surrounding water were made simultaneously to detect possible effects of compartmentalization. Residues as low as 0.1 mg/L could be analyzed in 5-day (low-dose) and 38-day (high-dose) postapplication tests. The phytoplankton community showed an increase in relative abundance and a decrease in species diversity. Studies of the frequency distribution of six selected phytoplankton species (*Spirogyra species*, *Microcystis flosaquae*, *Stichococcus bacillaris*, *Nitzschia acicularis*, *Chilomonas*

paramecium, and *Actinophrys species*) demonstrated the total elimination of at least four species from the treated compartments. Despite different dosing, only weak differences were found in toxic effects between the low- and high-dosed compartments. No significant, chemically induced effect was observed on the physicochemical properties of the treated water (Lay and Others 1984).

8.6.2 Invertebrates

Adverse effects to *Daphnia magna* occurred at concentrations as low as 0.44 mg/L (Environment Canada 1993a). A study was designed to evaluate the effects of PCE on the zooplankton community at initial concentrations of 1.2 and 0.44 mg/L in separated compartments of an experimental pond. Measurements in surrounding water were made simultaneously to detect possible effects of compartmentalization. Residues as low as 0.1 mg/L could be analyzed in 5-day (low-dose) and 38-day (high-dose) postapplication tests. In all applied biotopes, a lethal effect on the *Daphnia magna* population was detected. No significant, chemically induced effect was observed on the physicochemical properties of the treated water (Lay and Others 1984).

8.6.3 Amphibians and Reptiles

Little information was identified on the effects of PCE on amphibians and reptiles.

8.6.4 Fish and Aquatic Organisms

Long-term adverse effects to brook trout growth occurred at concentrations as low as 1.52 mg/L (Environment Canada 1993a).

LC50 values for *Cyprinodon variegatus* (sheepshead minnow) were between 29 and 52 mg/L for 4-day exposures. Death was not observed at concentrations below 29 mg for a 96-hour exposure (EPA 1997).

LC50 values for *Jordanella floridae* (flagfish) were 11.5 mg/L for a 24-hour exposure, 10.9 mg/L for a 48-hour exposure, 8.9 mg/L for a 72-hour exposure, and 4.0 and 8.4 mg/L for two 96-hour exposures.

The LOAEL for death were 3.1 and 3.7 mg/L for a 10-day and a 28-day exposure, respectively (EPA 1997).

8.6.5 Birds

Little information was identified on the effects of PCE on birds.

8.6.6 Mammals

Results from inhalation studies in animals suggest that PCE is fetotoxic, but not teratogenic, at concentrations that are also maternally toxic (ATSDR 1995b).

Pregnant mice and rats were exposed to a concentration of 300 ppm. Both species were exposed for periods of 7 hours daily on days 6 through 15 of gestation. No fetal toxicity or teratogenicity was detected (Shepard 1986; Kafafi and Others 1993).

Only one reproductive study in animals was identified, and it had serious limitations in design and conduct. Therefore, it provides no conclusive evidence for reproductive effects (ATSDR 1995b).

Male Swiss-Cox mice were administered PCE by gavage at doses of 0; 20; 100; 200; 500; 1,000; 1,500; and 2,000 mg/kg, 5 days per week for 6 weeks. Liver toxicity was evaluated by several parameters, including liver weight-to-body weight ration, hepatic triglyceride concentrations, DNA content, histopathological evaluation, and serum enzyme levels. Increased liver triglycerides were first observed in mice treated with 100 mg/kg. Liver weight-to-body weight ratios were significantly higher than controls for the 100-mg/kg-dose level and slightly higher than controls in the 20-mg/kg-dose level. A NOAEL of 20 mg/kg/day was identified based on the absence of hepatotoxic effects. After 5 days of exposure, a NOAEL of 20 mg/kg/day was identified (EPA 1996).

8.7 TOLUENE

Toluene is an organic solvent that is widely used, particularly in the paint, printing, and adhesive industries; toluene also is abused by "recreational" inhalation. Additionally, toluene is used as a gasoline additive (HSDB 1999). Toluene often occurs together with other aromatics (often including PAHs, alkyl PAHs, and benzene), and a typical complex mixture of such aromatics may be more toxic or hazardous, in general, than this compound would be alone.

Toluene can be released to soil through petroleum spills and from leaking underground storage tanks (Environment Canada 1993b). Toluene can be released into water through chemical and petroleum product spills and from discharges of industrial and municipal effluents (Environment Canada 1993b).

Toluene is dangerous to aquatic life in high concentrations. It may be dangerous if it enters water intakes (HSDB 1999). Water uses threatened are recreational, potable supply, fisheries, and industrial (EPA 1994).

Except for short-term hazards from concentrated spills, toluene frequently has been more associated with risk to humans than with risk to other species such as fish and wildlife. This is partly because plants, fish, and birds take up only very small amounts and because this VOC tends to evaporate into the atmosphere rather than persist in surface waters or soils (ATSDR 1994b). However, volatiles, such as toluene, can pose a drinking water hazard as a contaminant in groundwater.

Toluene has been studied for reproductive effects in laboratory animals and generally has not been teratogenic but has been fetotoxic at high doses that also were toxic to the mothers (Dabney 1994).

Toluene does not persist in water or soil, because it biodegrades and volatilizes rapidly to the atmosphere (Environment Canada 1993b). Accumulation of toluene is not expected to be important in any terrestrial or aquatic organism, and no reports exist indicating any significant organism bioconcentration or food chain biomagnification (Environment Canada 1993b).

BCFs for toluene in biota have been predicted to be between 15 and 70 (values less than 100 generally indicate that a compound is unlikely to undergo significant bioconcentration in organisms or biomagnification along food chains) (Environment Canada 1993b).

8.7.1 Plants

No information was identified on the effects of toluene on amphibians and reptiles.

8.7.2 Invertebrates

No information was identified on the effects of toluene on invertebrates.

8.7.3 Amphibians and Reptiles

No information was identified on the effects of toluene on amphibians and reptiles.

8.7.4 Fish and Aquatic Organisms

For aquatic biota, the most sensitive organism identified in long-term tests was the early life stages of rainbow trout. The reported LC50 was 0.02 mg/L for continuous, 27-day exposure of the embryo-larval stages. Coho salmon fry were the most sensitive aquatic organisms in acute tests, with a 96-hour LC50 of 5.5 mg/L. The 40-day, no observed effect concentration for growth of coho salmon fry was 1.4 mg/L, and the LOAEL was 2.8 mg/L (Environment Canada 1993b).

Based on its lipophilic properties, toluene is expected to have a moderate tendency to bioconcentrate in fatty tissues of aquatic organisms. The BCF was estimated to be about 10.7 in fish and about 4.2 in mussels. Levels that accumulate in the flesh of aquatic species also depend on the degree to which the species metabolize toluene. The highest tissue levels of toluene tend to occur in species such as eels, crabs, and herring that have a low rate of toluene metabolism (ATSDR 1994b). Metabolism of toluene limits its tendency to biomagnify in the food chain (ATSDR 1994b).

8.7.5 Birds

No information was identified on the effects of toluene on birds.

8.7.6 Mammals

The weight of evidence indicates that toluene is not mutagenic in mammalian or microbial systems, and results concerning its potential to act as a promoter are inconclusive (Environment Canada 1993b). Results of chromosomal assays have been mixed (Dabney 1994).

8.8 TRICHLOROETHENE

TCE is a clear, colorless, nonflammable liquid. It is heavier than water. It is widely used as a solvent for fats, waxes, resins, ores, rubber, paints, and varnishes. It also is used in dry cleaning, in degreasing, and as an intermediate to make other organic chemicals. TCE may enter the environment from industrial discharges, municipal waste treatment plant discharges, or spills.

Acute, toxic effects may include the death of animals, birds, or fish, and death or low growth rate in plants. Acute effects are seen 2 to 4 days after animals or plants come in contact with TCE. TCE has moderate, acute toxicity to aquatic life. Insufficient data are available to evaluate or predict the short-term effects of TCE to plants, birds, or land animals.

Chronic, toxic effects may include shortened lifespan, reproductive problems, lower fertility, and changes in appearance or behavior. Chronic effects can be seen long after first exposures to a toxic chemical. TCE has moderate chronic toxicity to aquatic life. Insufficient data are available to evaluate or predict the long-term effects of TCE to plants, birds, or land animals.

8.8.1 Plants

No information was identified on the effects of TCE on plants.

8.8.2 Invertebrates

No information was identified on the effects of TCE on invertebrates.

8.8.3 Amphibians and Reptiles

No information was identified on the effects of TCE on amphibians and reptiles.

8.8.4 Fish and Aquatic Organisms

No information was identified on the effects of TCE on fish and aquatic organisms.

8.8.5 Birds

No information was identified on the effects of TCE on birds.

8.8.6 Mammals

No information was identified on the effects of TCE on mammals.

8.9 XYLENE

Xylene, a widely used industrial solvent, is a mixture of *ortho*-, *meta*-, and *para*-isomers (HSDB 1999). While *o*-xylene is recognized as a distinct product in chemical analyses, the *m*- and *p*- isomers are generally not separated during most routine analyses. Therefore, results of xylene analyses in environmental samples usually are presented as the concentration of the *o*-isomer and the total concentration of the combine *m*- and *p*- isomers (Environment Canada 1993c).

Although most xylenes are released into the air, concentrations exposed to wildlife are at least 1,000 times less than the effects threshold estimated for inhalation of xylenes by mammals. Concentrations in ambient air are at least 1 million times less than the effects threshold recorded for plants. Except for

short-term hazards from concentrated spills, this compound frequently has been more associated with risk to humans than with risk to other species such as fish and wildlife. This is partly because plants, fish, and birds take up only very small amounts, and because this VOC tends to evaporate into the atmosphere, rather than persisting in surface waters or soils (ATSDR 1993f); however, VOCs, like xylenes, can pose a drinking water hazard when they occur in groundwater.

Like benzene and toluene, xylenes are fairly volatile, and significant xylenes tend to quickly evaporate if exposed to the atmosphere (ATSDR 1993f). However, xylenes can be more persistent when in groundwater, sediment, or soil media not directly exposed to the atmosphere. Xylenes tend to migrate to groundwater, and persistence is an issue in groundwater, where in some cases, they may persist for months or years (ATSDR 1993f).

Most xylene in surface water evaporates into the air in less than a day. The rest of it biodegrades slowly into other chemicals. Plants, fish, and birds take up only very small amounts. Research has shown that it persists longer in groundwater than in lakes and rivers, probably because it can evaporate from surface water (ATSDR 1993f).

Xylene evaporates from soil surfaces. Xylene below the soil surface persists for several days and may travel down through the soil and enter groundwater. In soil and groundwater, it may be slowly biodegraded into less harmful compounds. It is not clearly known how long xylene trapped deep underground in soil or groundwater persists, but it may be months or years. Xylene persists longer in wet soil than in dry soil (ATSDR 1993f).

8.9.1 Plants

Field concentrations in shallow groundwater or pore water below 100 mg/L for any aqueous solution in contact with terrestrial plants are not considered to present an ecological risk. Toxicity of groundwater to plants may be affected by many variables (pH, Eh, cation exchange capacity, moisture content, organic content of soil, clay content of soil, differing sensitivities of various plants, and various other factors). Therefore, the solution benchmark should be used as a screening benchmark only, and site-specific tests

would be necessary to develop a more rigorous benchmark for various combinations of specific soils and plant species (Will and Suter 1994).

Growth of the alga *Selenastrum capricornutum* was reduced by 50 percent after 72 hours of exposure to 3.2 to 4.9 mg/L of each of the xylene isomers. Exposure for 30 minutes to 300 mg/L resulted in a 65 to 100 percent kill of the freshwater macrophytes *Elodea spp.* and *Potamogeton* (Environment Canada 1993c).

8.9.2 Invertebrates

No information was identified on the effects of xylene on invertebrates.

8.9.3 Amphibians and Reptiles

No information was identified on the effects of xylene on amphibians and reptiles.

8.9.4 Fish and Aquatic Organisms

Xylenes are bioconcentrated in aquatic organisms to a limited extent. Although more information on bioconcentration would be helpful, the phenomenon of biomagnification is not expected to be important for xylene (ATSDR 1993f).

LC₅₀ for *Carassius auratus* (goldfish) were 75.0, 30.55, and 36.81 mg/L for 24-hour exposures; 25.1 and 36.81 mg/L for 48-hour exposure; 20.72 mg/L for a 72-hour exposure; and 36.81 mg/L for a 96-hour exposure (EPA 1997).

8.9.5 Birds

No information was identified on the effects of xylene on birds.

8.9.6 Mammals

Large amounts of xylene can cause changes in the liver and harmful effects in the kidneys, heart, lungs, and nervous system (ATSDR 1993f).

Long-term exposures of animals to low doses of xylene have not been well studied (ATSDR 1993f). Available animal data on the carcinogenicity of xylenes are inadequate to permit an evaluation (HSDB 1999).

Xylene does not appear to be particularly genotoxic (ATSDR 1993f). In limited studies to date, individual isomers were not found to be genotoxic when tested in a number of short-term tests (Fishbein 1985; HSDB 1999).

In rats, exposure to xylene (50 or 500 milligrams per cubic meter [mg/m^3]) resulted in embryotoxic and teratogenic effects. The brain, liver, lung, and heart were affected. The number of postimplantation losses increased by 9.7 and 168 percent in the 50- and 500- mg/m^3 xylene groups, respectively. The incidence of fetal skeletal abnormalities was increased by 62 and 177 percent (Mirkova and Others 1983; HSDB 1999).

The placental crossing of benzene and its alkyl derivatives, their embryotoxic effects, and incidence of fetal anomalies were investigated in rats, mice, and rabbits. In rats, all of the components crossed the placenta and also appeared in the fetal blood and amniotic fluid. Concentrations were higher in the fetal blood than in the amniotic fluid, but both were lower than in the maternal blood. Xylenes and ethylbenzene increased the postimplantation loss. All of the organic solvents caused skeletal retardations of mouse fetuses and increased the incidence of retarded fetuses, at least at higher concentrations. The exposure of rabbits to 1,000 mg/m^3 of solvent caused a mild, toxic effect on mothers; fetal loss by abortion, and often a decrease in the weight of female fetus (Ungvary and Tatrai 1985; HSDB 1999).

REFERENCES

- Adriano, D.C. 1986. *Trace Elements in the Terrestrial Environment*. Springer Verlag. New York.
- Agency for Toxic Substances and Disease Registry (ATSDR). 1990a. "Toxicological Profile for Barium." U.S. Public Health Service (USPHS). Atlanta, Georgia.
- Ainsworth, N. 1988. Distribution and Biological Effects of Antimony in Contaminated Grasslands. Ph.D. Dissertation, Council for National Academic Awards, United Kingdom.
- Ainsworth, N., Cooke, J.A. Johnson, M.S. 1991. "Behavior and Toxicity of Antimony in the Short-tailed Field Vole (*Microtus agrestis*)." *Ecotoxicology and Environmental Safety*, Volume 21, Number 2, Pages 165-170.
- ATSDR. 1990b. "Toxicological Profile for Copper." USPHS. Atlanta, Georgia.
- ATSDR. 1990c. "Toxicological Profile for Silver." USPHS. Atlanta, Georgia.
- ATSDR. 1990d. "Toxicological Profile for Ethylbenzene." USPHS. Atlanta, Georgia.
- ATSDR. 1991. "Toxicological Profile for Manganese." USPHS. Atlanta, Georgia
- ATSDR. 1992a. "Toxicological Profile for Cobalt." USPHS. Atlanta, Georgia.
- ATSDR. 1992b. "Toxicological Profile for Vanadium." USPHS. Atlanta, Georgia.
- ATSDR. 1992c. "Toxicological Profile for p,p'-DDT, DDE, DDD." USPHS. Atlanta, Georgia.
- ATSDR. 1993a. "Toxicological Profile for Beryllium." USPHS. Atlanta, Georgia
- ATSDR. 1993b. "Toxicological Profile for Cadmium." USPHS. Atlanta, Georgia.
- ATSDR. 1993c. "Toxicological Profile for Chromium." USPHS. Atlanta, Georgia.
- ATSDR. 1993d. "Toxicological Profile for Nickel." USPHS. Atlanta, Georgia.
- ATSDR. 1993e. "Toxicological Profile for Aldrin/Dieldrin." USPHS. Atlanta, Georgia.
- ATSDR. 1993f. Toxicological Profile for Xylenes. 209 Pages Plus Appendices. Atlanta, Georgia.
- ATSDR. 1994a. "Toxicological Profile for Chlordane." USPHS. Atlanta, Georgia.
- ATSDR. 1994b. Toxicological profile for toluene. USPHS. 221 Pages. TP-93/14. Atlanta, Georgia.
- ATSDR. 1995a. "Toxicological Profile for Polycyclic Aromatic Hydrocarbons." USPHS. Atlanta, Georgia.

REFERENCES (Continued)

- ATSDR. 1995b. "Toxicological Profile for Tetrachloroethylene." USPHS. Atlanta, Georgia.
- ATSDR. 1997. "Toxicological profile for titanium tetrachloride." USPHS. Atlanta, Georgia.
- ATSDR. 1999a. "Toxicological Profile for Lead." USPHS. Atlanta, Georgia.
- ATSDR. 1999b. "Toxicological Profile for Chlorophenols." USPHS. Atlanta, Georgia.
- ATSDR. 2001. "Toxicological Profile for Di-n-butylphthalate." USPHS. Atlanta, Georgia.
- Alloway, B.J. (ed.). 1990. *Heavy Metals in Soils*. John Wiley & Sons. New York.
- Amdur, M. O., J. Doull, and C.D. Klaassen. 1991. *Casarett and Doull's Toxicology: The Basic Science of Poisons, Fourth Edition*. McGraw-Hill Inc. New York.
- American Medical Association. 1989. *Home Medical Encyclopedia*. Two Volumes. Random House Publishers. New York, New York. 1,184 Pages.
- Balasubramanian, S., R. Pappathis, and S. P. Raj. 1995. "Bioconcentration of Zinc, Lead, and Chromium in Serially-Connected Sewage-Fed Fish Ponds." *Bioresource Technology*. Volume 51, No. 2-3. Pages 193 through 197.
- Balazs, T., Hanig, J.P., Herman, E.H. 1986. Toxic Responses of the Cardiovascular System. In Casarett and Doull's Toxicology, the Basic Science of Poisons, Third Edition. C.D. Klaassen, M.O. Amdur, and J. Doull, Editors. Macmillan Publishing Company, New York.
- Barrows, M.E., S.R. Petrocelli, K.J. Macek, and Others. 1978. "Bioconcentration and Elimination of Selected Water Pollutants by Bluegill Sunfish." Haque, R., Ed. Dynamics, Exposure and Hazard Assessment of Toxic Chemicals. Ann Arbor Science Publishers, Inc. Pages 379 through 392. Ann Arbor, Michigan.
- Bartlett, R.J., and B. James. 1979. "Behavior of Chromium in Soils: III. Oxidation." *Journal of Environmental Quality*. 8:31-35.
- Beyer, W. N., G. H. Heinz, and A. W. Redmon-Norwood. 1996. *Environmental Contaminants in Wildlife: Interpreting Tissue Concentrations*. CRC Press Inc. Boca Raton, Florida.
- Brooks, R.R. 1972. *Geobotany and Biogeochemistry in Mineral Exploration*. Harper and Row Publishers Inc. New York, New York. 290 Pages.
- Broyer, T.C., C.M. Johnson, and R.P. Huston. 1972. "Selenium and Nutrition of *Astragalus*. I. Effects of Selenite or Selenate Supply on Growth and Selenium Content." *Plant Soil* 36:635-649.
- Budavari. 1996. *The Merck Index - An Encyclopedia of Chemicals, Drugs, and Biologicals*. Whitehouse Station, NJ: Merck and Co., Inc.

REFERENCES (Continued)

- Callahan, M.A., and Others. 1979. "Water-Related Fate of 129 Priority Pollutants, Volumes I and II." U.S. Environmental Protection Agency (EPA), Office of Water Planning and Standard, Washington, DC by Versar, Inc. EPA-440/4-79-029a and 029b.
- Calardese, M.B., D.J. Hoffman, L.J. LeCaptain, and G.W. Pendleton. 1990. "Effects of Arsenate on Growth and Physiology in Mallard Ducks." *Environmental Toxicology and Chemistry*. Volume 9. Pages 785 through 795.
- Chamey, A. N., and A. Taglietta. 1992. "Effects of pH, Barium and Copper on Intestinal Chloride Transport in the Winter Flounder (*Pseudopleuronectes americanus*)." *Bulletin Mountain Desert Island Biological Laboratory*. Volume 31. Pages 60 - 61.
- Chapman, W.H. and Others. 1968. "Concentration Factors of Chemical Elements in Edible Aquatic Organisms." Lawrence Livermore Radiation Laboratory. OCRL-50564.
- Considine, D. M. 1976. *Van Nostrand's Scientific Encyclopedia, 5th Edition*. Van Nostrand/Reinhold Company, New York.
- Cooper, C.F., and W.C. Jolly. 1970. Ecological Effects of Silver Iodide and Other Weather Modification Agents: A Review. *Water Resources*. Res. 6:88-98.
- Curtin, D., Ryan, J., and Chaudhary, R.A. 1980. "Manganese Adsorption and Desorption in Calcareous Lebanese Soils." *Soil Sci. Soc. Am. J.* Volume 44. Pages 947-950.
- Dabney, B. 1994. Reprotext Data Base. In: TOMES™ REPROTEXT Electronic Database, Tomes Plus™ CD-ROM, Volume. 22, Micromedex Inc. Denver, Colorado.
- Department of Health and Human Services and US Environmental Protection Agency (EPA). 1987. Notice of the First Priority List of Hazardous Substances that will be the Subject of Toxicological Profiles. In Federal Register 52:12866-12874.
- Diamond, J. M.; E. L. Winchester; D. G. Mackler; W. J. Rasnake; J. K. Fanelli; and D. Gruber. 1992. "Toxicity of Cobalt to Freshwater Indicator Species as a Function of Water Hardness." *Aquatic Toxicology* (Amsterdam). Volume 22, Number 3. Pages 163-180.
- Domingo, J. L. 1994. "Metal-induced Developmental Toxicity in Mammals: A Review." *Journal of Toxicology and Environmental Health*. Volume 42. Pages 123 through 141.
- Domingo, J.L., J.L. Paternain, J.M. Llobet, and J. Corbella. 1985. "Effects of Cobalt on Postnatal Development and Late Gestation in Rats Upon Oral Administration." *Rev. Esp. Physiol.* Volume 41. Pages 293-298.
- Eisler, R. 1985a. "Cadmium Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review." U.S. Fish and Wildlife Service (USFWS). Biological Report 85(1.2).

REFERENCES (Continued)

- Eisler, R. 1985b. "Selenium Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review." U.S. Fish and Wildlife Service. (USFWS). Biological Report 85 (1.5) Report No. 5.
- Eisler, R. 1986a. "Chromium Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review." USFWS. Biol. Rep. 85(1.6). 60 Pages.
- Eisler, R. 1986b. "Polychlorinated Biphenyl Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review." USFWS. Biological Report 85(1.7).
- Eisler, R. 1987. "Polycyclic Aromatic Hydrocarbon Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review." USFWS. Biological Report 85(1.11).
- Eisler, R. 1988. "Lead Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review." USFWS. Biological Report 85(1.14).
- Eisler, R. 1989. "Molybdenum Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review." USFWS. Biological Report 85. 1.19.
- Eisler, R. 1990. "Chlordane Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review." USFWS. Biological Report 85(1.21).
-
- Eisler, R. 1993. "Zinc Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review." USFWS. Biological Report 10.
- Environment Canada. 1993a. "Priority Substances List Assessment Report: Tetrachloroethylene." 55 Pages. Available from Commercial Chemicals Branch, Environment Canada, 14th Floor, Place Vincent Massey, 351 St. Joseph Boulevard, Hull, Quebec, Canada KIA OH3.
- Environment Canada. 1993b. "Priority Substances List Assessment Report on Toluene." 26 Pages. Available from Chemicals Evaluation Division, Environment Canada, Quebec, Canada.
- Environment Canada. 1993c. "Priority Substances List Assessment Report, Xylenes." 32 Pages. Available from Commercial Chemicals Division, Environment Canada, 351 St. Joseph Boulevard Hull, Quebec, Canada KIA OH3.
- Environment Canada. 1994a. "Priority Substances List Assessment Report: Nickel and Its Compounds." Canadian Environmental Protection Act. National Printers (Ottawa) Inc.
- Environment Canada. 1994b. "Priority Substances List Assessment Report, Polycyclic Aromatic Hydrocarbons." Minister of Supply and Services. Canada.
- Evans, R. D., D. Andrews, and R. J. Cornett. 1988. "Chemical Fractionation and Bioavailability of Cobalt-60 to Benthic Deposit Feeders." *Canadian Journal of Fish and Aquatic Science*. Volume 45. Pages 228-236.

REFERENCES (Continued)

- Fernandes, J.C., and F.S. Henriques. 1991. "Biochemical, Physiological, and Structural Effects of Excess Copper in Plants." *Botany Reviews* 57(3):246-272.
- Fishbein, L. 1985. *Science Total Environment*; 43, 1-2: Pages 165 through 83
- Fitzgerald, D.G., K.A. Warner, R.P. Lanno, and D.G. Dixon. 1996. "Assessing the Effects of Modifying Factors on Pentachlorophenol Toxicity to Earthworms: Applications of Body Residues." *Environmental Toxicology Chemistry*. 15:2299-2304.
- Fleishman, S. 1988. Chapter on Isomers. In the *Electronic Encyclopedia*™. The 21 Volume Academic American Publishing, Inc., on CD-ROM, © 1988 Grolier Electronic Publishing, Inc. Danbury, Connecticut. Written Permission to Excerpt Copyright Material Granted to Roy Irwin, Conditioned on Proper Documentation of the Source as the Electronic Encyclopedia of Grolier Electronic Publishing, Inc.
- Flora, M.D., T.E. Ricketts, J. Wilson and S. Kunkle. 1984. "Water Quality Criteria: An Overview for Park Natural Resources Specialists." WRFSL Report No. 84-4. Water Resources Field Support Laboratory. National Park Service. Colorado State University. Fort Collins, Colorado.
-
- Forsyth, D.J., T.J. Peterle, and W. Bandy. 1983. "Persistence and Transfer of CI-DDT in the Soil and Biota of an Old Field Ecosystem: A Six Year Balance Study." *Ecology*. 64: 1620-1636.
- Foy, C. D., R. L. Chaney, and M. C. White. 1978. "The Physiology of Metal Toxicity in Plants." *Ann. Review Plant Physiol*. 29:511-566.
- Gebel, T. 1997. Arsenic and Antimony: Comparative approach on Mechanistic Toxicology. *Chemico-Biological Interactions*. Volume 107, Number 3, pages 131-144.
- Goodman, L.S., and A. Gilman., Eds. 1975. *The Pharmacological Basis of Therapeutics*, 5th Ed. Macmillan Publishing Co., Inc. New York, New York.
- Grolier Electronic Publishing. 1988. Chapter on chromium. In The Electronic Encyclopedia™. The 21-Volume Academic American Encyclopedia on CD-ROM, © 1988 Grolier Electronic Publishing, Inc. Danbury, Connecticut. Written Permission to Excerpt Copyright Material Granted to Roy Irwin, Conditioned on Proper Documentation of the Source as the Electronic Encyclopedia of Grolier Electronic Publishing, Inc.
- Hall, R. J. 1980. "Effects of Environmental Contaminants on Reptiles: A Review." U.S. Department of the Interior. US Fish and Wildlife Service. Special Scientific Report. Wildlife No. 228. Washington, DC.
- Hazardous Substance Databank. 1999. "Hazardous Substance Databank." National Library of Medicine. National Toxicology Information Program. Bethesda, MD.
- Hem, J.D. 1989. "Study and Interpretation of the Chemical Characteristics of Natural Water, 3rd Edition." U.S. Geological Survey (USGS) Water-Supply Paper 2253. Government Printing

REFERENCES (Continued)

- Office. Available from the Distribution Branch, Text Products Section, USGS. 263 Pages. Alexandria, Virginia.
- Hemstock, G.A., and G.F. Low. 1953. "Mechanisms Responsible for Retention of Manganese in the Colloidal Fraction of Soil." *Soil Science*. Volume 76. Pages 331-343.
- Hilton, J.W., W.J. Bettger. 1988. *Aquatic Toxicology* 12 (1): 63-72.
- Hoffman, D.J., B.A. Rattner, G.A. Burton, Jr., and J.C. Cairns, Jr. (Eds). 1995. *Handbook of Ecotoxicology*. Lewis Publishers, Inc. Boca Raton, Florida.
- Hollins, J.G., and R.S. McCullough. 1971. "Radiation Dosimetry of Internal Contamination by Inorganic Compounds of Cobalt: An Analysis of Cobalt Metabolism in Rats." *Health Physics*. Volume 21. Pages 233-246.
- Horne, M. T., and W. A. Dunson. 1995. "Effects of Low pH, Metals, and Water Hardness on Larval Amphibians." *Archives of Environmental Contamination and Toxicology*. Volume 29. Pages 500 through 505.
- Hulzebos, E. M., D. M. M. Adema, E. M. Dirven-van Breemen, L. Henzen, W. A. van Dis, H.A. Herbold, J. A. Hoekstra, R. Baerselman, and C. A. M. van Gestel. 1993. "Phytotoxicity Studies with *Lactuca Sativa* in Soil and Nutrient Solution." *Environmental Toxicology Chemistry* 12:1079-1094.
- International Agency Research Cancer (IARC). 1973. "Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man." International Agency Research Cancer. Volume 3. Pages 46 through 68.
- IARC and World Health Organization (WHO). 1983. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man. International Agency for Research on Cancer. Geneva. Vol. 32, pp. 239-245; Supplement 7, p. 59.
- International Copper Association. 1992. "The Biological Importance of Copper - A Literature Review." International Copper Association, Limited.
- Jagoe, C. H.; V. E. Matey, T. A. Haines, and V. T. Komov. 1993. "Effect of Beryllium on Fish in Acid Water is Analogous to Aluminum Toxicity." *Aquatic Toxicology* (Amsterdam). Volume 24, Number 3-4. Pages 241 - 256.
- Jamil, K., and S. Hussain. 1992. "Biotransfer of Metals to the Insect *Neochetina eichhornae* via Aquatic Plants." *Archives of Environmental Contamination and Toxicology*. Volume 22. Pages 459-463.
- Jarvis, S.C., and D.C. Whitehead. 1983. The Absorption, Distribution and Concentration of Copper in White Clover Grown on a Range of Soils. *Plant Soil* 75:427-434.

REFERENCES (Continued)

- Jenkins, D. W. 1980. "Biological Monitoring of Toxic Trace Metals. Volume 2: Toxic Trace Metals in Plants and Animals of the World." U. S. EPA. EPA 600/3-80-090.
- Jenkins, D. W. 1981. Ediological Monitoring of Toxic Trace Elements. EPA Report 600/S3-80-090. Pages 1 through 9.
- Joardan, M., and A. Sharma. 1990. "Comparison of Clastogenicity of Inorganic Manganese Administered in Cationic and Anionic Forms in Vivo." *Mutation Research*. Volume 240. Pages 159-63.
- Jones, R. E. 1990. "Hexavalent Chrome: Threshold Concept for Carcinogenicity." *Biomedical-Environmental-Science*. 3:20-34.
- Kabata-Pendias, A., and H. Pendias. 1984. *Trace Elements in Soils and Plants*. CRC Press, Inc. Boca Raton, Florida.
- Kabata-Pendias, A., and H. Pendias. 1992. *Trace Elements in Soils and Plants*. CRC Press, Inc. Boca Raton, FL.
- Kafafi, S.A., H.Y. Afeefy, A.H. Ali, H.K. Said, and G. Kafafi. 1993. "Binding of Polychlorinated Biphenyls to the Aryl hydrocarbon Receptor." *Environmental Health Perspectives* 101:422-425.
- Kawano, M., S. Matsushita, T. Inoue, H. Tanaka, and R. Tatsukawa. 1986. "Biological Accumulation of Chlordane Compounds in Marine Organisms from Northern North Pacific and Bering Sea." *Marine Pollution Bulletin*. Volume 17, No. 11. Pages 512 through 516.
- Keith, L.H., and W.A. Telliard. 1979. "Priority Pollutants: I- A Perspective View." *Environmental Science and Toxicology*. 13:416-423.
- Kerr, J.A. 1988. Chapter on Vanadium. In *The Electronic Encyclopedia*™. The 21-Volume Academic American Encyclopedia on CD-ROM, © 1988 Grolier Electronic Publishing, Inc. Danbury, Connecticut. Written Permission to Excerpt Copyright Material Granted to Roy Irwin, Conditioned on Proper Documentation of the Source as *The Electronic Encyclopedia* of Grolier Electronic Publishing, Inc.
- Killey, R. W. D., J. O. McHugh, and D. R. Champ. 1984. "Subsurface Cobalt-60 Migration from a Low-level Waste Disposal Site." *Environmental Science and Technology*. Volume 6. Pages 28-41.
- Kirk-Othmer. 1965. "Copper." *Encyclopedia of Chemical Technology*. John Wiley and Sons. 2nd Edition. Volume 6. New York, New York.
- Klassen, C.D., M.O. Amdur, and J. Doull (Eds). 1991. *Casarett and Doull's Toxicology*, 4th Edition, Perganom Press of the Maxwell Macmillan Perganom Publishing Company. New York, New York. 1033 Pages.

REFERENCES (Continued)

- Klein-MacPhee, G., J. A. Cardin, and W. J. Berry. 1984. "Effects of Silver on Eggs and Larvae of the Winter Flounder." *Transactions of the American Fisheries Society*. Volume 113, No. 2. Pages 247 through 251. March.
- Koeppel, D.E. 1981. *Lead: Understanding the Minimal Toxicity of Lead in Plants*. In *Effects of Heavy Metal Pollution on Plants, Vol. 1. Effects of Trace Metals on Plant Function*. N. W. Lepp (ed) Applied Science Publishers. New Jersey. Pages 55 through 76.
- Laskey, J.W., and F.W. Edens. 1985. "Effects of Chronic High-level Manganese Exposure on Male Behavior in the Japanese Quail." (*Coturnix coturnix japonica*). *Poultry Science*. Volume 64. Pages 579-84.
- Lay, J.P., and Others. 1984. *Archives Environmental Contamination and Toxicology*. 13 (2): 135-42.
- Leland, H.V., and J.S. Kuwabara. 1985. "Trace metals." G.M. Rand and S.R. Petrocelli, Eds., *Fundamentals of Aquatic Toxicology*. Hemisphere Publishing Company. New York, New York. 666 Pages.
- Lewis, R.J., Sr. 1993. *Hazardous Chemicals Desk Reference. 3rd Edition*. Van Nostrand Reinhold Publishers. New York, New York. 1,742 Pages.
- Long, E., and L.G. Morgan. 1990. "The Potential for Biological Effects of Sediment-Sorbed Contaminants Tested in the National Status and Trends Program." *NOAA Technical Memorandum NOS OMA 52*, National Ocean Service. Seattle, Washington. 175 Pages.
- Luckey, T.D., and B. Venugopal. 1977. *Metal Toxicology in Mammals, 1*. Plenum Press. New York.
- Macapinlac, M.P., W.N. Pearson, and W.J. Derby. 1966. "Some Characteristics of Zinc Deficiency in the Albino Rat." *Zinc Metabolism*. A.S. Prasad, Editor Charles Thomas. Springfield, Illinois.
- MacDonald, A. 1993. "Development of an Approach to the Assessment of Sediment Quality in Florida Coastal Waters." Florida Department of Environmental Regulation, Tallahassee, Florida. MacDonald Environmental Sciences Ltd. Ladysmith, British Columbia, Canada.
- Mahoney, J.S. 1978. "Environmentally Acquired Lead, Cadmium, and Manganese in the Cattle Egret, *Bubulcus ibis*." Thesis, Master of Science. The University of Texas Health Science Center at Houston. June. 57 pages.
- Manahan, S.E. 1992. *Toxicological Chemistry*. Lewis Publishers, an Imprint of CRC Press. Boca Raton, Florida, 449 Pages.
- McGrath, S.P. 1982. "The Uptake and Translocation of Tri- and Hexa-Valent Chromium and Effects on the Growth of Oat in Flowing Nutrient Solution." *New Phytology*. 92:381-390.
- McLaren, J. W., D. M. Lawson, and R. S. Swift. 1986. "Sorption and Desorption of Cobalt by Soils and Soil Components." *Journal of Soil Science*. Volume 37. Pages 413-426.

REFERENCES (Continued)

- Meister, R.T. 1995. *Farm Chemicals Handbook. Volume 81*. Meister Publishing Company. Ohio. Pages 97 through 100.
- Meyers, E. 1990. *Chemistry of Hazardous Materials*. Prentice Hall Career and Technology. Prentice-Hall Inc. Englewood Cliffs, New Jersey. 509 Pages.
- Miles, C.D., J.R. Brandle, D.J. Daniel, O. Chu-Der, P.D. Schnore, and D.J. Uhlik. 1972. "Inhibition of Photosystem II in Isolated Chloroplasts by Lead." *Plant Physiology* 49:820-825.
- Mirkova E., and Others. 1983. *Journal of Hygiene, Epidemiology, Microbiology, and Immunology*. 27, 3: 337-43.
- Moore, S.B., J. Winckell, S.J. Detwiler, S.A. Klasing, P.A. Gaul, N.R. Kanim, B. E. Kesser, A.B. Debeveck, K. Beardsley, and L.K. Puckett. 1990. "Fish and Wildlife Resources and Agricultural Drainage in the San Joaquin Valley, California. San Joaquin Drainage Program, Sacramento, California." Six Sections and Two Appendixes. Portions Reproduced with the permission of Senior Author Stephen Moore, USFWS, Regional Office. Portland, Oregon.
- Morishita, T. and J. K. Boratynski. 1992. "Accumulation of Cadmium and Other Metals in Organs of Plants Growing Around Metal Smelters in Japan." *Soil Science and Plant Nutrition*. Volume 38, Number 4. Pages 781-785.
- Morrell, B.G., N.W. Lepp, and D.A. Phipps. 1985. "Vanadium Uptake by Higher Plants: Some Recent Developments." *Environmental Geochemistry and Health*. 8:14-18.
- Mukherji, S., and B. Das Gupta. 1972. "Characterization of Copper Toxicity in Lettuce Seedlings." *Physiology of Plants*. 27:126-129.
- National Academy of Sciences (NAS). 1980. "Mineral Tolerances of Domestic Animals." NAS. National Research Council. Washington, DC.
- National Library of Medicine. 2003. TOXNET Database. Website: <http://toxnet.nlm.nih.gov/cgi-bin/sis/search/f?/>
- National Research Council Canada (NRCC). 1976. "Effects of Chromium in the Canadian Environment. p. 15. NRCC No. 15017.
- Nelson, D.A., A. Calabrese, R. A. Greig, P. P. Yevich, and S. Chang. 1983. "Long-term Silver Effects on the Marine Gastropod (*Crepidula fornicata*)." *Marine Ecology - Progress Series*. Volume 12, Issue 2. Pages 155 through 165.
- NRCC. 1977. *Drinking Water & Health Volume 1*. National Academy Press. Washington, DC.
- NRCC. 1980. "Effects of Vanadium in the Canadian Environment" Page 38. NRCC No. 18132.

REFERENCES (Continued)

- Osborn, D.; A. S. Cooke, and S. Freestone. 1981. "Histology of a Teratogenic Effect of DDT on *Rana Temporaria* Tadpoles." *Environmental Pollution* (Series A). Volume 25, No. 4. Pages 305 through 319.
- Owen, C.A. 1981. *Copper Deficiency and Toxicity: Acquired and Inherited, in Plants, Animals, and Man*. Noyes Publications. New Jersey. Pages 84 through 102.
- Paternain, J.L., and Others. 1987. *Rev Esp Fisiol* 43 (2): 223-8.
- Paternain, J.L., and J.L. Domingo. 1988. "Developmental Toxicity of Cobalt in the Rat." *Journal of Toxicology and Environmental Health*. Volume 24. Pages 193-200.
- Patnaik, P. 1992. *A Comprehensive Guide to the Hazardous Properties of Chemical Substances*. Van Nostrand Reinhold Publishers. New York, New York. 763 Pages.
- Perelman, A. I. 1967. *Geochemistry of Epigenesis*. Plenum Press, New York.
- Perwack, J. and Others. 1980. "Exposure and Risk Assessment for Copper." EPA. EPA-400/4-81-015. NTIS PB85-211985.
- Peterle, T. J. 1991. *Wildlife Toxicology*. Van Nostrand Reinhold. New York.
- Peterson, P.J., and C.A. Girling. 1981. *Other Trace Metals. In Effects of Heavy Metal Pollution on Plants, Volume 1. Effects of Trace Metals on Plant Function*. N.W. Lepp (Editor). Applied Science Publishers. New Jersey. Pages 279-342.
- Power, T., K.L. Clark, A. Harfenist, and D.B. Peakall. 1989. "A Review and Evaluation of the Amphibian Toxicological Literature Technical Report No. 61." Canadian Wildlife Service, Headquarters.
- Pulsford, A. L., P. Ryan, and J. A. Nott. 1992. "Metals and Melanomacrophages in Flounder, *Platichthys Flesus*, Spleen and Kidney." *Journal of the Marine Biology Association*. United Kingdom. Volume 72, No. 2. Pages 483 through 498.
- Rai, D., and J. M. Zachara. 1984. "Chemical Attenuation Rates, Coefficients, and Constants in Leachate Migration. Volume I: A Critical Review." Electric Power Research Institute, Palo Alto, CA.
- Raymont, J.E.G. 1972. "Pollution in Southampton Water." *Proceedings of the Royal Society Series*. Series B. Volume 180, No. 106. Pages 451-468.
- Romney, E.M., and J.D. Childress. 1965. "Effects of Beryllium in Plants and Soil." *Soil Science* 100(2):210-17.
- Rompala, J.M., F.W. Rutosky, and D.J. Putnam. 1984. "Concentrations of Environmental Contaminants from Selected Waters in Pennsylvania." USFWS Report. State College, Pennsylvania.

REFERENCES (Continued)

- Sadiq, M. 1992. *Toxic Metal Chemistry in Marine Environments*. Marcel Dekker. New York.
- Seidenberg, J.M, and Others. 1986. "Validation of an in vivo Developmental Toxicity Screen in the Mouse." *Teratogen Carcinogen Mutagen*. Volume 6. Pages 361-374.
- Scheuhammer, A.M. 1987. "The Chronic Toxicity of Aluminum, Cadmium, Mercury, and Lead in Birds: A Review." *Environmental Pollution*. Volume 46. Pages 263 through 295.
- Schiffer, D.M. 1989. "Effects of Highway Runoff on the Quality of Water and Bed Sediments of Two Wetlands in Central Florida." USGS Water-Resources Investigations Report 88-4200. Department of the Interior. USGS. Tallahassee, Florida. 63 Pages.
- Shepard, T.H. 1986. *Catalog of Teratogenic Agents* 5th Edition. The Johns Hopkins University Press. Baltimore, Maryland.
- Sindayigaya, E.; R. V. Cauwenbergh; H. Robberecht; and H. Deelstra. 1994. "Copper, Zinc, Manganese, Iron, Lead, Cadmium, Mercury and Arsenic in Fish from Lake Tanganyika, Burundi." *The Science of the Total Environment*. Volume 144. Pages 103 through 115.
- Smith, I. C., and B. L. Carson. 1981. Trace Metals in the Environment, Volume 6: Cobalt, An Appraisal of *Environmental Exposure*. Ann Arbor Science Publications, MI.
- Smith, K.L., G.W. Bryan, and J.L. Harwood. 1985. "Changes in Endogenous Fatty Acids and Lipid Synthesis Associated with Copper Pollution in *Fucus*." *Journal of Experimental Botany*. 36:663-669.
- Smith, S.P.J. Peterson, and K.H.M. Kwan. 1989. "Chromium Accumulation, Transport and Toxicity in Plants." *Toxicology and Environmental Chemistry*. 24:241-251.
- Southern, L.L., and D.H. Baker. 1983. "Excess Manganese Ingestion in the Chick." *Poultry Science*. Volume 62. Pages 642-646.
- Stickel, L.F. 1968. "Organochlorine Pesticides in the Environment." USFWS. Special Scientific Report No. 119. Washington, D.C.
- Talmage, S.S., and B.T. Walton. 1991. "Small Mammals as Monitors of Environmental Contaminants." *Review of Environmental Contamination and Toxicology*. Volume 119. Pages 47-145.
- Taylor, D.M. 1962. "The Absorption of Cobalt from the Gastrointestinal Tract of the Rat." *Physico. Med. Biol.* Volume 6. Pages 445-451.
- Taylor, K., and Others. 1992. "Mass Emissions Reduction Strategy for Selenium, Staff Report." Basin Planning and Protection Unit. San Francisco Regional Water Quality Control Board. Oakland, California. October 12.
- Thibodeaux, L.J. 1979. *Chemodynamics*. Wiley & Sons. New York.

REFERENCES (Continued)

- Trelease, S.F., A. A. Di Somma, and A.L. Jacobs. 1960. Seleno-Amino Acid Found in *Astragalus bisulcatus*. *Science* 132:618.
- U.S. Environmental Protection Agency (EPA). 1975. "DDT: A Review of Scientific and Economic Aspects of the Decision to Ban Its Use as a Pesticide." EPA. Washington, D.C. EPA-540/1-75-022. July.
- EPA. 1976. "Effects of Exposure to Heavy Metals on Selected Fresh Water Fish: Toxicity of Copper, Cadmium, Chromium and Lead to Eggs and Fry of Seven Fish Species." Environmental Research Laboratory. Office of Research and Development. Duluth, Minnesota. 600/3-76-105.
- EPA. 1981. Health Assessment Document for Cadmium. EPA 60/8-81023. EPA. Washington, D.C.
- EPA. 1986. "Quality Criteria for Water." EPA Report 440/5-86-001. Office of Water Regulations and Standards. Washington, DC.
- EPA. 1988. State Water Quality Standards Summaries. EPA Publication Number 440/5-88-031. EPA Office of Water, Washington, D.C. Available through NTIS, Springfield, VA - order number PB89-141634.
- EPA. 1991. Water Quality Criteria Summary List (Poster Format). Based on Quality Criteria for Water and Updates 1 (1986), 2 (1987), and 3 (1990). Distributed by the EPA, Office of Science and Technology, Health and Ecological Criteria Division, Ecological Risk Assessment Branch (WH-585), Human risk Assessment Branch (WH-550D), 401 M Streets S.W., Washington, DC. 20460. Published May 1.
- EPA. 1992. "Quality Criteria for Water." EPA. Office of Water. Washington, DC.
- EPA. 1994. Oil and Hazardous Material/Technical Assistance Data System (OHM/TADS). Public Domain Information Available from the Government; Additional Commercially Available Copies of this Public Domain Information Have Included (But May Not Be Limited To, No Government Dedorsment Implied): The Tomes Plus™ CD-ROM Database Volume 22, Micromedex Inc. Denver, Colorado.
- EPA. 1995a. Great Lakes Water Quality Initiative Criteria Documents for the Protection of Wildlife. DDT, Mercury, 2,3,7,8-TCDD, PCBs. EPA 820-B-95-008.
- EPA. 1995b. "Region IX Preliminary Remediation Goals - Second Half 1995." EPA. San Francisco, California.
- EPA. 1996. EPA Integrated Risk Information System (IRIS) Electronic Database. Public Domain Information Available from the Government. EPA Criteria and Assessment Office. Cincinnati, Ohio.

REFERENCES (Continued)

- EPA. 1997. Aquatic Toxicity Information Retrieval Database (AQUIRE). EPA, ERL-Duluth, Duluth, Minnesota. Information Obtained from EPA Internet Source. Republished AQUIRE Information is also Available from Private Vendors.
- EPA. 1999. IRIS On-line Database. Washington, DC.
- EPA. 2003. EcoTox Database. Website Address: http://www.epa.gov/cgi-bin/ecotox_quick_search.
- Ungvary G., E. Tatrai. 1985. *Archives of Toxicology*. 8:425-30.
- Venugopal, B., and T.D. Luckey. 1978. *Metal Toxicity in Mammals, 2*. Plenum Press. New York.
- Wallace, A. and E.M. Romney. 1977. "Roots of Higher Plants as a Barrier to Translocation of Some Metals to Shoots of Plants." In *Biological Implications of Metals in the Environment. Proceeding of the Fifteenth Annual Hanford Life Sciences Symposium, Richland, WA*. Technical Information Center, ERDA, Washington, D.C. Pages 370-379.
- Ward, N.I., E. Roberts, and R.R. Brooks. 1979. "Silver Uptake by Seedlings of *Lolium perene* L. and *Trifolium Repens*. *Library of New Zealand Journal of Science*. 22:129-132.
- Warington, K. 1954. "The influence of iron supply on toxic effects of manganese, molybdenum, and vanadium on soybeans, peas, and flax. *Ann. Appl. Biol.* 41(1):1-22. 912.
- Will, M.E., and G.W. Suter II. 1994. "Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Terrestrial Plants: 1994 Revision." Publication No. ES/ER/TM-85/R1. Oak Ridge National Laboratory. Oak Ridge, Tennessee.
- Williams, R.B., and M.B. Murdoch. 1969. "The Potential Importance of *Spartina alterniflora* in Conveying Zinc, Manganese, and Iron into Estuarine Food Chains." In: *Proceeding of the Second National Symposium on Radioecolog.* D.J. Nelson and F.C. Evans (eds.). Pages 431-439.
- White, D.H., K.A. King, and R.M. Prouty. 1980. "Significance of Organochlorine and Heavy Metal Residues in Wintering Shorebirds at Corpus Christi, Texas, 1976-1977." *Pesticide Monitoring Journal*. 14:58-63.
- WHO. 1982. Environmental Health Criteria 24: Titanium. Internation Programme on Chemical Safety.
- Wong, M.H., and A.D. Bradshaw. 1982. "A Comparison of the Toxicity of Heavy Metals, Using Root Elongation of Rye Grass, *Lolium Perenne*. *New Phytology*. 92: 255-61.