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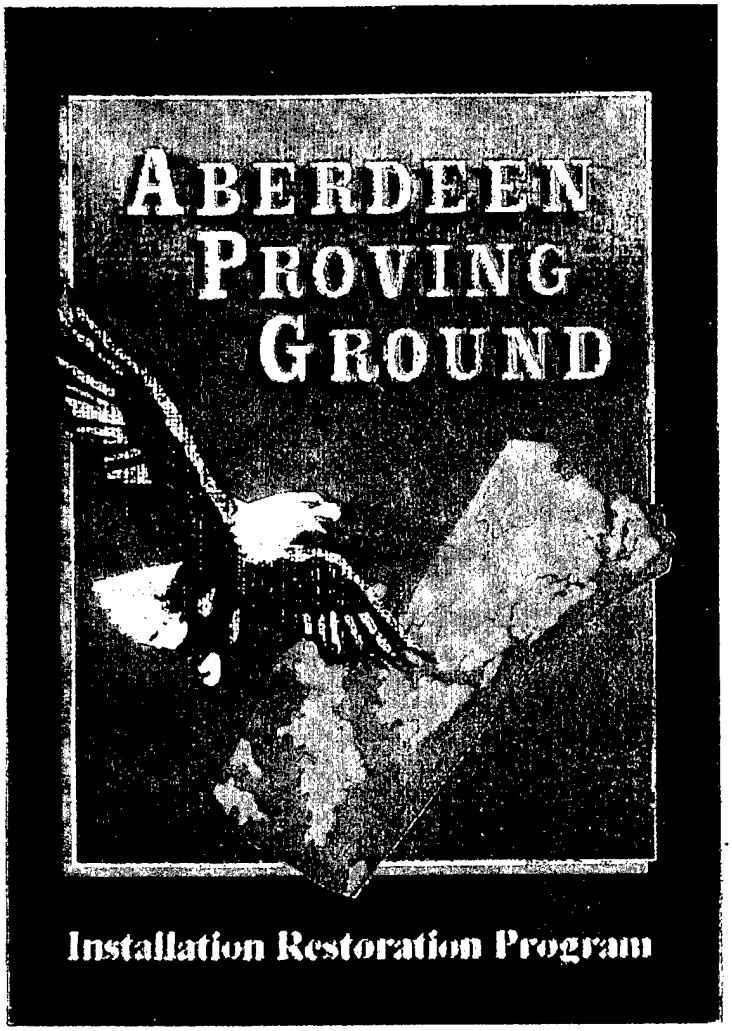

PRELIMINARY RISK ASSESSMENT FOR EIGHT SELECTED STUDY AREAS VOLUME II

FINAL DOCUMENT

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Aberdeen Proving Ground, Maryland

March 1996

10.0 NIKE SITE RISK ASSESSMENT

This chapter evaluates potential impacts on human health and the environment associated with the Nike site in the absence of remedial (corrective) actions. The RCRA facility investigation initiated by AEHA in July 1986 and summarized in AEHA (1990) is the primary source of sampling data considered in this risk assessment. This study was selected for use in this risk assessment because it was the most recent and comprehensive study conducted at the Nike site. Other sampling undertaken in this area has been discussed in Chapter 3 of this report.

These and other investigations conducted to date have not completely characterized the nature and extent of contamination at the Nike site. Therefore, this risk assessment should be considered largely preliminary and is intended as an initial step in the overall risk assessment process for the Nike site.

This assessment follows the general methodology outlined in Chapter 4 of this report, which should be consulted for the rationale and further details of the methods used in this assessment. This assessment is organized into eight primary sections:

- Section 10.1 Background Information
- Section 10.2 Selection of Chemicals of Potential Concern
- Section 10.3 Human Health Risk Assessment
- Section 10.4 Ecological Assessment
- Section 10.5 Uncertainties
- Section 10.6 Principal Data Needs
- Section 10.7 Summary and Conclusions
- Section 10.8 References

10.1 BACKGROUND INFORMATION

The Nike site is located in the northeastern portion of the Edgewood Area adjacent to the northern boundary of the installation (see Figure 2-2). The Nike site was used by the U.S. Army Chemical School for training in chemical warfare activities from 1920 to 1951. The Nike Missile Battery was subsequently constructed on some of the school fields; missiles were stored, maintained, and deployed in this area from 1954 to 1973.

Chemical warfare training activities at the school fields and the chemicals associated with these activities included (1) the use and firing of chemical ordnance; (2) identification of chemical agents and decontamination of personnel, vehicles, and equipment; (3) clothing impregnation and laundering; and (4) disposal of chemical agents, chemical ordnance, and chemical agent contaminated material. Chemical ordnance contained white phosphorus, a mixture of sulfur trioxide and chlorosulfonic acid, titanium tetrachloride, chloroacetophenone, phosgene, and mustard. Chemicals used during decontamination of personnel, vehicles, and equipment included chlorinated lime, supertropical bleach, calcium hypochlorite, 1,1,2,2-tetrachloroethane and 1,3-dichloro-5,5-dimethylhydantoin, and chlorinated solvents (e.g., chloroform). N,N'-dichloro-bis(2,4,6-trichlorophenyl)urea, 1,1,2,2-tetrachloroethane, and chlorobenzene were used for clothing impregnation and laundering. No information is available concerning disposal sites or contents resulting from disposal of chemical agents, chemical ordnance, and chemical agent contaminated material. A list of the compounds believed to have been used and/or disposed at any time at the Nike site is presented in Table 10-1.

TABLE 10-1

PRINCIPAL COMPOUNDS DISPOSED OF AND/OR USED AT THE NIKE SITE (a)

Group	Chemical Compound (Acronym) (b)
Lethal Chemical Agents	Phosgene (CG) Mustard (HD or H)
Incapacitating Agents	Chloroacetophenone (CN)
Decontaminating Agents	Decontaminating agent - noncorrosive (DANC) Chlorinated Lime Supertropical Bleach (STB) Calcium Hypochlorite (HTH)
Smoke/Incendiary Materials	White Phosphorus (WP) Sulfur Trioxide and Chlorosulfonic Acid mixture (FS) Titanium Tetrachloride (FM)
CC2 and CC3 Impregnating Materials	TCPU
Solvents	Trichloroethane Trichloroethene 1,1,2,2-Tetrachloroethane [major component of DANC] Toluene Acetone Alcohol Stoddard solvents Chlorinated solvents [e.g., chloroform] Chlorobenzene Carbon Tetrachloride Paint thinners
Oils/Fuels	Hydraulic fluid Waste oil JP-4 Inhibited red fuming nitric acid (IRFNA) Unsymmetric Dimethyl Hydrazine (UDMH) Aniline-furfuryl alcohol Ethylene Oxide Diesel fuel
Miscellaneous	Paint Battery acid/fluid Radioisotope-contaminated equipment

(a) Information obtained primarily from AEHA (1989) and AEHA (1985).
(b) See Glossary of Acronyms and Abbreviations for complete chemical name if not given in this table.

Unexploded ordnance was recovered during the construction at the launch and control areas, during range clearing surface sweeps in 1977 and 1984, and during site clearance for RFI work drilling. All areas of the Nike site may contain unexploded ordnance containing high explosive and/or military chemical agents.

Nike Ajax missiles were deployed at the site from 1954 to around 1959, and Nike Hercules missiles were deployed from around 1959 to 1973. The Nike Ajax missile was fueled by a solid propellant booster and liquid fuel and armed with high explosives. The Nike Hercules missile was solid fueled and armed with a nuclear warhead. The battery currently consists of three main source areas: the launch area, the control area, and the barracks area. These three areas can be seen on the source map in Figure 10-1 and are further discussed below.

10.1.1 LAUNCH AREA

The launch area is located at the northern end of the battery and is fenced with a road around the perimeter of the site. It contains six abandoned and water-filled missile silos, several other buildings, at least one abandoned underground fuel oil storage tank, and a septic tank and subsurface sand filter bed. Monks Creek is located east of the launch area.

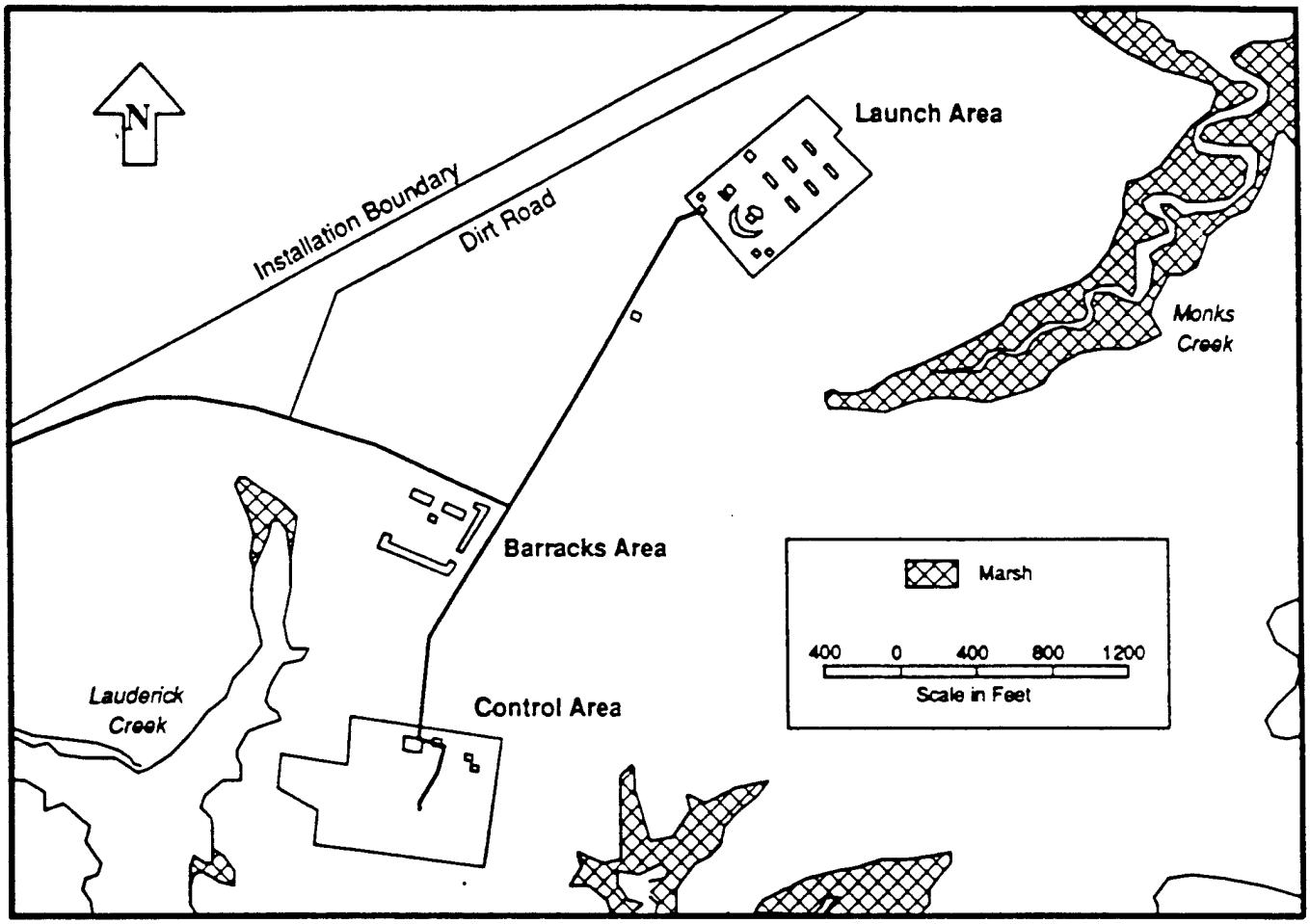
The missiles and warheads were assembled, stored, maintained, and launched at the launch area. Maintenance operations included fueling; defueling; addition/changeout of missile constituents; equipment maintenance for missile launchers, electrical generators, and vehicles; material handling; and material storage. Maintenance and fueling operations occurred at the southern end of the launch area and immediately northeast of the launch area.

Liquid fuel materials for the Nike Ajax missile included JP-4, inhibited red fuming nitric acid (IRFNA), unsymmetrical dimethyl hydrazine (UDMH), and aniline-furfuryl alcohol. Early Nike Hercules missiles used ethylene oxide. Small quantities of UDMH, aniline-furfuryl alcohol, and ethylene oxide were used and large quantities (hundreds of gallons per year) of JP-4 and IRFNA were used. Under standard practices, JP-4 should have been disposed of by burning, and IFRNA should have been neutralized in a trench with limestone or slaked lime.

An abandoned underground storage tank that was used in the past for fuel oil storage is present within the launch area. Fuel oil has been observed leaking from the above-ground filler pipe of this tank on hot days, and soil in the vicinity of the pipe is oil stained. It is reported that the stained soil is periodically removed and used to fill low areas around the property (Dames and Moore 1990). An additional underground fuel oil storage tank may also be present (Argonne National Laboratory 1990).

Wastes generated during assembly or servicing operations included hydraulic fluid, waste oil, battery acid, trichloroethane, trichloroethene, carbon tetrachloride, toluene, acetone, alcohol, and Stoddard solvents. Approximately 1,000 gallons of hydraulic fluid and 100 gallons of electric battery changeout fluid were disposed of at the launch area each year. Hundreds of gallons of trichloroethane, trichloroethene, carbon tetrachloride, and Stoddard solvents were used each year. Paints, paint thinners, and cleaning materials were used during building maintenance. Small quantities of defective radioisotope-containing electron tubes and wipes from the monitoring of nuclear warheads were also generated. The primary means of waste disposal in the launch area were probably ground dumping, discharge to ditches, and possibly dumping into the latrines to the sewer.

Figure 10-1
Nike Study Area



The disposal site southwest of the launch area was used as a demolition debris disposal site, and approximately 10 empty drums, some of which originally contained hydraulic fluid, were found to the immediate north. The disposal site south-southeast of the launch area adjacent to Monks Creek was used as a material storage area and later used as a landfill. Chipped wood and branches were placed in an area southwest of the launch area adjacent to a trail. Vegetation reportedly does not grow in the cleared area south of the launch area due to disturbance of the soil during construction.

10.1.2 CONTROL AREA

The control area is located at the southern end of the Battery. It is fenced and includes five buildings and a few other structures including a septic tank and subsurface sand filter bed. Lauderick Creek is located west, south, and east of the control area.

The control area contained the radar, electronic, and communications equipment necessary for target identification and tracking and missile launching and guidance. The four small dry wells in the control area were used for disposal of small amounts of wastes including chlorinated solvents. The fill site at the edge of the marsh east-southeast of the control area contains construction debris, but may be primarily earth and not waste.

10.1.3 BARRACKS AREA

The barracks area is located between the launch and control areas. It consists of five buildings, a septic tank, and a subsurface sand filter bed. There are also five underground fuel oil storage tanks that were installed in 1957 and are still in use (Argonne National Laboratory 1990). Lauderick Creek is located west of the barracks area. Paints, paint thinners, and cleaning materials may have discharged from the barracks area to Lauderick Creek through surface drainage.

10.2 SELECTION OF CHEMICALS OF POTENTIAL CONCERN

In this section, environmental monitoring data collected by the Army Environmental Hygiene Agency (AEHA 1990) between July 1986 and July 1989 are briefly summarized, and chemicals of potential concern selected for evaluation in this risk assessment are identified. The discussions are organized by area since different media were sampled in the launch area and the control area (no media were sampled in the barracks area).

10.2.1 LAUNCH AREA

10.2.1.1 Soil

Soil borings were made in July and August 1986 to obtain soil samples for chemical analyses, to determine soil characteristics, and to visually examine soil for hydraulic fluid or fuel oil contamination. Eight surface soil samples (0-1 foot or 0-2.5 feet) and two subsurface soil samples (2.5-3 feet and 8-9.5 feet) were collected from the launch area for chemical analyses. All soil samples were analyzed for metals: arsenic, barium, cadmium, chromium, lead, mercury, selenium, and silver. Two of the surface soil samples were also analyzed for volatile organic chemicals, semivolatile organic chemicals,

pesticides, and PCBs. Both subsurface soil samples were analyzed for volatile organic chemicals. No blank data were available for this assessment. It is not clear whether blank samples were collected.

Chemicals detected in surface and subsurface soil at the launch area are shown in Table 10-2. p-Chloro-m-cresol, 4-nitrophenol, and pentachlorophenol were detected at what were reported to be "trace" levels in the surface soil and therefore were selected as chemicals of potential concern. It was noted that during drilling at the site where these organic chemicals were detected, the soil became heated and had a strong odor characteristic of heated pentachlorophenol (AEHA 1990). AEHA hypothesized that p-chloro-m-cresol, 4-nitrophenol, and pentachlorophenol were present as a result of disposal of treated wood. Arsenic, cadmium, mercury, and selenium were present in the surface soil at concentrations above regional background levels and were therefore selected as chemicals of potential concern.

No organic chemicals were selected as chemicals of potential concern in subsurface soil. AEHA reported that the soil at the missile fueling/defueling site was visibly contaminated with organic material and had a strong organic odor (AEHA 1990). Hydrocarbons were detected at trace levels in this sample and two tentatively identified compounds, trimethylcyclohexane isomers and dimethylcyclohexane isomers, were present at a concentration of 5.0 mg/kg. A strong organic solvent odor was reported at a depth of 15-17.5 feet during drilling of the monitoring well located approximately 1,600 feet southwest of the actual launch area along the road. Selenium was present in the subsurface soil at a concentration above regional background levels and was therefore selected as a chemical of potential concern.

10.2.1.2 Groundwater

A surficial aquifer about 20 feet thick is found throughout the launch area. The bottom of the surficial aquifer is about 40-50 feet deep north and west of the launch area and 50-60 feet deep south and east of the launch area. The maximum flow velocities within the surficial aquifer are probably 40-50 feet per year. The next deeper aquifer in the launch area is approximately 30-40 feet thick with a base 120-150 feet deep. Beneath most of the launch area, there is a downward vertical gradient between the surficial and deep aquifers. The deeper aquifer becomes less defined or is not present west of the launch area. The depth to groundwater in this area is 20-25 feet, and lateral movement is expected. Groundwater flow at the launch area has not been fully defined at this time although in some areas it appears to be toward nearby surface water bodies that likely act as discharge points.

Groundwater samples were collected in July and August 1986 after the soil borings were completed. A total of five shallow boring groundwater samples were collected from the launch area and analyzed for volatile organic chemicals. No blank data are available for this sampling.

Monitoring wells were installed in 20 locations at the launch area in 1986 and 1987 and at 3 locations in 1988. At 9 of the locations, wells were installed at multiple depths. The wells installed in 1986 and 1987 were analyzed in June, October, or November 1987 for dissolved metals (arsenic, barium, cadmium, chromium, lead, mercury, selenium, and silver), inorganic and miscellaneous parameters, dissolved radionuclides, volatile organic chemicals, semivolatile organic chemicals, pesticides, and PCBs. The wells installed in 1986 and 1987 were analyzed in June 1988 only for volatile organic chemicals. Samples were collected from six wells in June and July 1989 and analyzed for volatile organic chemicals. No background groundwater samples were collected. Blank samples appear to have been collected in 1987, but these data were not available for this report. Acetone and methylene chloride were reportedly detected at low µg/L levels in the control travel blank associated with sample

TABLE 10-2

SUMMARY OF CHEMICALS DETECTED IN SURFACE AND SUBSURFACE SOIL IN THE LAUNCH AREA AT THE NIKE SITE

(Concentrations reported in mg/kg)

Chemical (a)	Frequency of Detection (b)	Range of Detected Concentrations	Background Concentration (c)
SURFACE SOIL (d)			
Organic Chemicals:			
* p-Chloro-m-cresol	1 / 1	Trace	NA
* 4-Nitrophenol	1 / 1	Trace	NA
* Pentachlorophenol	1 / 1	Trace	NA
Inorganic Chemicals:			
* Arsenic (AS)	8 / 8	7.75 - 32.3	6.0
Barium (BA)	1 / 8	67.8	500
* Cadmium (CD)	1 / 8	0.23	NA
Chromium (CR)	8 / 8	9.13 - 18.4	70
Lead (PB)	8 / 8	9.12 - 19.6	20
* Mercury (HG)	3 / 8	0.12 - 0.16	0.05
* Selenium (SE)	8 / 8	3.41 - 10.7	0.10
Sulfur (S)	1 / 1	3.0	<800
SUBSURFACE SOIL (e)			
Inorganic Chemicals:			
Arsenic (AS)	2 / 2	6.35 - 10.6	6.0
Chromium (CR)	2 / 2	9.52 - 23.2	70
Lead (PB)	2 / 2	4.90 - 5.30	20
* Selenium (SE)	2 / 2	4.28 - 8.32	0.10

(a) USATHAMA chemical codes listed in parentheses.
(b) The number of samples in which a chemical was detected divided by the total number of samples analyzed for that chemical. Detection limits were not provided for the organic chemicals and sulfur.
(c) Background concentration from Boerngen and Shacklette (1981). Location is Route 45, 2 miles north of Hereford, approximately 25 miles northwest of APG.
(d) Samples: organic chemicals -- NB13-1 and NB22-1; inorganic chemicals -- NB3-1, NB5-1, NB6-1, NB7-1, NB8-1, NB9-1, NB13-1, and NB22-1.
(e) Samples: NB2-1 and NB4-2.

* = Selected as a chemical of potential concern. See text.
NA = Not available.

20B collected in June 1987 (AEHA 1990). Blank samples were collected in 1988 and 1989, and these data are available.

Chemicals detected in groundwater at the launch area are shown in Table 10-3. The data from the shallow boring groundwater samples (only volatile organic chemicals were analyzed for) were combined with the data from the monitoring well samples. All wells were grouped together regardless of the depth of screening, since chemicals could potentially migrate between the shallow and deeper aquifers. Groundwater data for each location were averaged across time if more than one sample was collected for that location.

Gross alpha, gross beta, potassium 40, radium 226, acetone, carbon disulfide, trans-1,2-dichloroethene, 2-ethyl-1-hexanol, bis(2-ethylhexyl)phthalate, methylene chloride, and trichloroethene were detected in the groundwater and therefore were selected as chemicals of potential concern. Most of these chemicals were detected very infrequently. No chemicals were detected in the shallow boring groundwater samples for the launch area. Gross beta and bis(2-ethylhexyl)phthalate were detected relatively frequently; gross beta was detected in 25 of 30 samples at a maximum concentration of 31 pCi/L, and bis(2-ethylhexyl)phthalate was detected in 16 of 30 samples at a maximum concentration of 1,200 µg/L. Bis(2-ethylhexyl)phthalate is a common laboratory contaminant since it is a component of plastics. Both acetone (detected in 5 of 33 samples) and trichloroethene (detected in 6 of 41 samples) had maximum concentrations of 89 µg/L.

Barium was present at a concentration above the national background level and was therefore selected as a chemical of potential concern. No site-specific or regional background data were available for groundwater in this area with which to compare site levels. The use of national groundwater data, as in this case, introduces considerable uncertainty into the selection of inorganic chemicals of potential concern.

10.2.1.3 Water from Missile Silos

Water samples were collected from all six silos in July 1986, and two silos that were not prohibitively filled with water were entered and inspected. Water enters the silos through groundwater seepage and run-in of rainwater that falls on the silo doors. Silo samples were analyzed for dissolved metals (arsenic, barium, cadmium, chromium, lead, mercury, selenium, and silver), inorganic and miscellaneous parameters, dissolved radionuclides, volatile organic chemicals, semivolatile organic chemicals, pesticides, and PCBs. No blank samples were available for this sampling event.

Chemicals detected in water from the missile silos at the launch area are shown in Table 10-4. Gross alpha, gross beta, butylbenzylphthalate, and trichloroethene were detected and therefore were selected as chemicals of potential concern. Gross beta was detected in all six silos at a maximum concentration of 85 pCi/L. Gross alpha, butylbenzylphthalate, and trichloroethene were each detected in one of six silos at concentrations of 1.7 pCi/L, 130 µg/L, and 28 µg/L, respectively. Of the organic chemicals detected in groundwater, the radiologic parameters and trichloroethene were the only chemicals detected in the silo water. Lead was present at a concentration above the national groundwater levels used for comparison (no site-specific background data were available) and was therefore selected as a chemical of potential concern.

TABLE 10-3

SUMMARY OF CHEMICALS DETECTED IN LAUNCH AREA
GROUNDWATER AT THE NIKE SITE (a)

(Concentrations reported in ug/L)

Chemical (b)	Frequency of Detection (c)	Range of Detected Concentrations (d)	Background Concentration (e)
Radiological Parameters (pCi/L):			
* Gross Alpha (ALPHAG)	3 / 30	1.0 - 1.6	NA
* Gross Beta (BETAG)	25 / 30	1.2 - 31.0	NA
* Potassium 40 (K40)	1 / 2	45.0	NA
* Radium 226 (RA226)	1 / 1	0.6	NA
Organic Chemicals:			
* Acetone (ACET)	5 / 33	8 - 89	NA
* Carbon Disulfide (CS2)	1 / 33	17	NA
* trans-1,2-Dichloroethene (T12DCE)	1 / 22	8	NA
* 2-Ethyl-1-hexanol (2E1HXL)	1 / 1	16	NA
* bis(2-Ethylhexyl)phthalate (B2EHP)	16 / 30	10 - 1,200	NA
* Methylene Chloride (CH2CL2)	1 / 36	24	NA
* Trichloroethene (TRCLE)	6 / 41	1.8 - 89	NA
Inorganic Chemicals:			
Arsenic (AS)	3 / 30	3 - 6	100
* Barium (BA)	30 / 30	27 - 319	100
Cadmium (CD)	9 / 30	0.5 - 2	100
Chromium (CR)	9 / 30	1 - 6	100
Lead (PB)	4 / 30	1 - 7.3	100
Nitrite/Nitrate (NIT) (f)	26 / 30	50 - 980	10,000
Selenium (SE)	9 / 30	1 - 3	100

(a) Samples: 1A, 1B, 1C, 2A, 2B, 2C, 3A, 3B, 5A, 6A, 6B, 7A, 8A, 9A, 15A, 16A, 17A, 18A, 19A, 19B, 19C, 20A, 20B, 20C, 21A, 22A, 22B, 24A, 24B, 25A, 26B, 29A, 30A, 31A, 32A, NB10, NB11, NB12, NB14, and NB22.
(b) USATHAMA chemical codes listed in parentheses.
(c) The number of samples in which a chemical was detected divided by the total number of samples analyzed for that chemical.
(d) Values reported for metals are dissolved concentrations.
(e) Background concentrations from Walton (1985). Values reported are dissolved concentrations.
(f) Concentration is reported as nitrite/nitrate non-specific. The value reported is assumed to represent the total concentration of nitrite/nitrate.

* = Selected as a chemical of potential concern. See text.
NA = Not available.

TABLE 10-4

SUMMARY OF CHEMICALS DETECTED IN WATER
FROM MISSILE SILOS AT THE NIKE SITE (a)

(Concentrations reported in ug/L)

Chemical (b)	Frequency of Detection (c)	Range of Detected Concentrations (d)	Background Concentration (e)
Radiological Parameters (pCi/L):			
* Gross Alpha (ALPHAG)	1 / 6	1.7	NA
* Gross Beta (BETAG)	6 / 6	21.0 - 85.0	NA
Organic Chemicals:			
* Butylbenzylphthalate (BBZP)	1 / 6	130	NA
* Trichloroethene (TRCLE)	1 / 6	28	NA
Inorganic Chemicals:			
Cadmium (CD)	5 / 6	1 - 4	100
Chromium (CR)	2 / 6	13 - 49	100
* Lead (PB)	5 / 6	38 - 185	100
Nitrite/Nitrate (NIT) (f)	6 / 6	10 - 460	10,000

(a) Samples: 1-6.
(b) USATHAMA chemical codes listed in parentheses.
(c) The number of samples in which a chemical was detected divided by the total number of samples analyzed for that chemical.
(d) Values reported for metals are dissolved concentrations.
(e) Background concentrations from Walton (1985). Values reported are dissolved concentrations.
(f) Concentration is reported as nitrite/nitrate non-specific. The value reported is assumed to represent the total concentration of nitrite/nitrate.

* = Selected as a chemical of potential concern. See text.
NA = Not available.

10.2.1.4 Surface Water

One surface water sample was collected from the upper portion of Monks Creek and analyzed for volatile organic chemicals only. No volatile organic chemicals were detected in this surface water sample. Surface water samples were not collected from the ditches that drain the launch area since they were dry. No surface water samples were collected from Lauderick Creek.

10.2.1.5 Sediment

Three sediment samples were collected in August 1986 from the ditches that drain the launch area and analyzed for metals, volatile organic chemicals, semivolatile organic chemicals, pesticides, and PCBs. No blank samples were available for this sediment sampling. No sediment samples were collected from Monks Creek or Lauderick Creek.

Chemicals detected in sediment from the ditches that drain the launch area are shown in Table 10-5. Bis(2-ethylhexyl)phthalate, hexanedioic acid (dioctyl ester), and tetrachloroethene were the organic chemicals detected in sediment and were therefore selected as chemicals of potential concern. Bis(2-ethylhexyl)phthalate is a common laboratory contaminant, and therefore its presence in site samples may be a sampling/analytical artifact. In addition, hydrocarbon oil was detected in two of the three samples at concentrations of 0.06 mg/kg and 0.6 mg/kg, and high molecular weight hydrocarbons were detected in one sample at a concentration of 0.03 mg/kg. Barium, cadmium, chromium, lead, and selenium were present at concentrations above regional sediment background levels and were therefore selected as chemicals of potential concern. A solvent and/or hydrocarbon odor was noted at the time of sampling in the sediment sample collected from the drainage ditch northeast of the launch area, and an odor was also noted in the sample from the drainage ditch south of the launch area (AEHA 1990).

10.2.1.6 Radiation

Radiation surveys were conducted at the ground scar area southwest of the launch area and north of the barracks and the fill area southwest of the launch area. The surveys were conducted by walking 10-foot transects across the areas with portable instruments capable of detecting beta emitters in the surface soil and gamma emitters in the surface or subsurface soil. Radiation survey findings indicated no radiation levels above background for the surface and subsurface soils in either area surveyed. Nevertheless, the authors of this study pointed out that, from this limited survey, it is not possible to conclude that no radioisotope-containing electron tubes were buried on-site (AEHA 1990).

10.2.1.7 Soil Gas

Soil gas samples were collected (generally at a depth of 4-7 feet) in July and August 1986 during the soil boring sampling. A total of six soil gas samples were collected from the following locations: the launch area (three samples), south of the launch area (two samples), and southwest of the launch area (one sample). The soil gas samples were analyzed only for volatile organic chemicals. Field blanks and duplicates reportedly were collected (AEHA 1990), but this information is not available.

Chemicals detected in soil gas in the launch area are shown in Table 10-6. Chlorobenzene, chloroform, tetrachloroethene, 1,1,1-trichloroethane, and trichloroethene were detected in soil gas

TABLE 10-5

SUMMARY OF CHEMICALS DETECTED IN
SEDIMENT SAMPLES NEAR THE LAUNCH AREA AT THE NIKE SITE (a)

(Concentrations reported in mg/kg)

Chemical (b)	Frequency of Detection (c)	Range of Detected Concentrations	Range of Background Concentrations (d)
Organic Chemicals:			
* bis(2-Ethylhexyl)phthalate (B2EHP)	1 / 1	0.003	NA
* Hexanedioic Acid, Dioctyl Ester	1 / 1	0.010	NA
* Tetrachloroethene (TCLEE)	1 / 1	0.001	NA
Inorganic Chemicals:			
Arsenic (AS)	3 / 3	12.7 - 18.1	14 - 46
* Barium (BA)	2 / 3	177 - 220	NA
* Cadmium (CD)	3 / 3	0.52 - 1.61	0.005 - 1.1
* Chromium (CR)	3 / 3	57.7 - 192	50 - 69
* Lead (PB)	3 / 3	45.8 - 118	42 - 66
Mercury (HG)	2 / 3	0.12 - 0.27	0.10 - 0.30
* Selenium (SE)	3 / 3	3.69 - 5.12	0.92 - 1.3 (e)

(a) Samples: NS1, NS2, and NS3.
(b) USATHAMA chemical codes listed in parentheses.
(c) The number of samples in which a chemical was detected divided by the total number of samples analyzed for that chemical. Detection limits were not provided for the organic chemicals.
(d) Range of sediment concentrations reported for two monitoring stations in the Bush and Gunpowder Rivers, except as noted. Data derived from Maryland Chesapeake Bay Toxicant Monitoring Program.
(e) Background concentrations from 3 stations near the Chesapeake Bay Bridge and Annapolis. Data derived from NOAA (1988).

* = Selected as a chemical of potential concern. See text.
NA = Not available.

TABLE 10-6

SUMMARY OF CHEMICALS DETECTED IN SOIL GAS
IN THE LAUNCH AREA AT THE NIKE SITE (a)

(Concentrations reported in ug/m3)

Chemical (b)	Frequency of Detection (c)	Range of Detected Concentrations
Organic Chemicals:		
* Chlorobenzene (CLC6H5)	1 / 6	1.0
* Chloroform (CHCL3)	1 / 6	2.0
* Tetrachloroethene (TCLEE)	2 / 6	2.0 - 3.0
* 1,1,1-Trichloroethane (111TCE)	1 / 6	7.0
* Trichloroethene (TRCLE)	2 / 6	2.0 - 6.0

(a) Samples: NG1, NG3, NG4, NG5, NG6, and NG9.
(b) USATHAMA chemical codes listed in parentheses.
(c) The number of samples in which a chemical was detected divided by the total number of samples analyzed for that chemical.

* = Selected as a chemical of potential concern. See text.

(albeit at low concentrations) and therefore were selected as chemicals of potential concern. All of these chemicals were detected in the sample located in the ground scar area approximately 1,600 feet southwest of the actual launch area along a road. In addition, trichloroethene was detected at the launch area, and tetrachloroethene was detected approximately 800 feet south of the launch area.

10.2.2 CONTROL AREA

10.2.2.1 Soil

Soil borings were made in July and August 1986 to obtain soil samples for chemical analyses, to determine soil characteristics, and to visually examine soil for hydraulic fluid or fuel oil contamination. No surface soil samples were collected in the control area. Two subsurface soil samples (14-15 feet and 16-17 feet) were collected from the control area. One of the samples was analyzed for metals (arsenic, barium, cadmium, chromium, lead, mercury, selenium, and silver) and volatile organic chemicals, and the other sample was analyzed for volatile organic chemicals, semivolatile organic chemicals, pesticides, and PCBs. No blank samples were available from this sampling.

Chemicals detected in subsurface soil at the control area are shown in Table 10-7. Bis(2,4,6-trichlorophenyl)urea, the only organic chemical detected in this soil, was selected as a chemical of potential concern. Mercury and selenium were present at low concentrations, but slightly above regional background levels and were therefore selected as chemicals of potential concern.

10.2.2.2 Groundwater

The principal aquifer in the control area is approximately 20 feet thick starting at a depth of 35-55 feet. The depth to groundwater is about 20 feet in the center of the control area. The direction of groundwater flow is probably radially outward towards branches of Lauderick Creek.

Groundwater samples were collected in July and August 1986 after the soil borings were completed. Four shallow boring groundwater samples were collected from the control area and analyzed for volatile organic chemicals. No blank samples were available from this sampling.

Monitoring wells were installed in four locations at the launch area in 1986 and 1987. Samples collected from these wells were analyzed for dissolved metals (arsenic, barium, cadmium, chromium, lead, mercury, selenium, and silver), inorganic and miscellaneous parameters, dissolved radionuclides, volatile organic chemicals, semivolatile organic chemicals, pesticides, and PCBs in June 1987 and volatile organic chemicals in June 1988. No background groundwater samples were collected. Blank samples appear to have been collected in 1987, but these data are not available. Blank data are available for the June 1988 sampling round.

Chemicals detected in groundwater at the control area are shown in Table 10-8. The data from the shallow boring groundwater samples were combined with the data from the monitoring well samples. All wells were grouped together regardless of the depth of screening since chemicals could potentially migrate between aquifers. Groundwater data for each location were averaged across time if more than one sample was collected for that location.

TABLE 10-7

SUMMARY OF CHEMICALS DETECTED IN SUBSURFACE
SOIL IN THE CONTROL AREA AT THE NIKE SITE (a)

(Concentrations reported in mg/kg)

Chemical (b)	Frequency of Detection (c)	Detected Concentration	Background Concentration (d)
Organic Chemicals:			
* bis(2,4,6-Trichlorophenyl)urea (TCPU)	1 / 1	3.0	NA
Inorganic Chemicals:			
Arsenic (AS)	1 / 1	4.42	6.0
Chromium (CR)	1 / 1	6.73	70
Lead (PB)	1 / 1	3.61	20
* Mercury (HG)	1 / 1	0.18	0.05
* Selenium (SE)	1 / 1	3.65	0.10

(a) Samples: organic chemicals -- NB1-2 and NB20-1; inorganic chemicals -- NB1-2.
(b) USATHAMA chemical codes listed in parentheses.
(c) The number of samples in which a chemical was detected divided by the total number of samples analyzed for that chemical. Detection limits were not provided for the organic chemicals.
(d) Background concentration from Boerngen and Shacklette (1981). Location is Route 45, 2 miles north of Hereford, approximately 25 miles northwest of APG.

* = Selected as a chemical of potential concern. See text.
NA = Not available.

TABLE 10-8

SUMMARY OF CHEMICALS DETECTED IN CONTROL AREA
GROUNDWATER AT THE NIKE SITE (a)

(Concentrations reported in ug/L)

Chemical (b)	Frequency of Detection (c)	Range of Detected Concentrations (d)	Background Concentration (e)
Radiological Parameters (pCi/L):			
* Gross Beta (BETAG)	3 / 4	1.8 - 4.6	NA
Organic Chemicals:			
* trans-1,2-Dichloroethene (T12DCE)	1 / 4	18	NA
* bis(2-Ethylhexyl)phthalate (B2EHP)	4 / 4	30 - 700	NA
* 1,1,1-Trichloroethane (111TCE)	3 / 8	2.9 - 18	NA
* Trichloroethene (TRCLE)	2 / 8	7 - 25	NA
Inorganic Chemicals:			
* Barium (BA)	4 / 4	19 - 203	100
Nitrite/Nitrate (NIT) (f)	4 / 4	160 - 690	10,000
Selenium (SE)	1 / 4	2	100

(a) Samples: 10A, 11A, 12A, 13A, NB17, NB18, NB20, and NB21.
(b) USATHAMA chemical codes listed in parentheses.
(c) The number of samples in which a chemical was detected divided by the total number of samples analyzed for that chemical.
(d) Values reported for metals are dissolved concentrations.
(e) Background concentrations from Walton (1985). Values reported are dissolved concentrations.
(f) Concentration is reported as nitrite/nitrate non-specific. The value reported is assumed to represent the total concentration of nitrite/nitrate.

* = Selected as a chemical of potential concern. See text.
NA = Not available.

Gross beta, trans-1,2-dichloroethene, bis(2-ethylhexyl)phthalate, 1,1,1-trichloroethane, and trichloroethene were detected in the groundwater and therefore were selected as chemicals of potential concern. Bis(2-ethylhexyl)phthalate, a common lab contaminant, was detected in all four samples at a maximum concentration of 700 µg/L. Trans-1,2-dichloroethene was only detected in the one of the shallow boring groundwater samples. 1,1,1-trichloroethane and trichloroethene were detected in one of the shallow boring groundwater samples and in samples from the monitoring wells. Barium was present at a concentration above national background levels and was therefore selected as a chemical of potential concern. No site-specific or regional background data were available for groundwater in this area with which to compare site levels. The use of national groundwater data, as in this case, introduces considerable uncertainty into the selection of inorganic chemicals of potential concern.

10.2.2.3 Radiation

A radiation survey was conducted at the fill area east of the control area adjacent to wetlands of Lauderick Creek. The survey was conducted by walking 10-foot transects across the area with portable instruments capable of detecting beta emitters in the surface soil and gamma emitters in the surface or subsurface soil. Radiation survey findings indicated no radiation levels above background for the surface and subsurface soils in this area, but the authors of this study note that from this limited survey it is not possible to conclude that no radioisotope-containing electron tubes were buried on-site (AEHA 1990).

10.2.2.4 Soil Gas

Soil gas samples were collected (generally at a depth of 4-7 feet) in July and August 1986 during the soil boring sampling. One soil gas sample was collected from the control area, and one sample was collected from the fill area east of the control area. The soil gas samples were analyzed for volatile organic chemicals. No volatile organic chemicals were detected in either of these samples. Field blanks and duplicates reportedly were collected (AEHA 1990), but this information is not available.

10.2.3 BARRACKS AREA

No samples were collected from the barracks area, and no information is available concerning the hydrogeology beneath the area. Chemicals could potentially be present at the barracks area as a result of the five underground storage tanks or previous chemical agent testing.

10.2.4 SUMMARY OF CHEMICALS OF POTENTIAL CONCERN

Table 10-9 summarizes the chemicals of potential concern for each media sampled in the launch and control areas of the Nike site during the RCRA facility investigation. Analytes varied considerably across media and individual samples within a media. Radiological parameters were analyzed for only in groundwater and water in the missile silos. Only a few of the soil samples were analyzed for volatile organic chemicals, semivolatile organic chemicals, pesticides, and PCBs. The groundwater samples were analyzed for only volatile organic chemicals after the first sampling round. The surface water and soil gas samples were analyzed only for volatile organic chemicals.

TABLE 10-9

SUMMARY OF CHEMICALS OF POTENTIAL CONCERN FOR THE NIKE SITE

Chemical (a)	Launch Area						Control Area	
	Surface Soil	Subsurface Soil	Groundwater	Missile Silo Water	Sediment	Soil Gas	Subsurface Soil	Groundwater
Radiological Parameters:								
Gross Alpha (ALPHAG)			X	X				
Gross Beta (BETAG)			X	X				
Potassium 40 (K40)			X					X
Radium 226 (RA226)			X					
Organic Chemicals:								
Acetone (ACET)			X					
Butylbenzylphthalate (BBZP)				X				
Carbon Disulfide (CS2)			X					
p-Chloro-m-cresol	X							
Chlorobenzene (CLC6H5)						X		
Chloroform (CHCL3)						X		
trans-1,2-Dichloroethene (T12DCE)			X					
2-Ethyl-1-hexanol (2E1HXL)			X					X
bis(2-Ethylhexyl)phthalate (B2EHP)			X		X			
Hexanedioic Acid, Dioctyl Ester					X			X
Methylene Chloride (CH2CL2)			X					
4-Nitrophenol	X							
Pentachlorophenol	X							
Tetrachloroethene (TCLEE)					X	X		
1,1,1-Trichloroethane (111TCE)						X		X
Trichloroethene (TRCLE)			X	X		X		X
bis(2,4,6-Trichlorophenyl)urea (TCPU)							X	
Inorganic Chemicals:								
Arsenic (AS)	X							
Barium (BA)			X		X			X
Cadmium (CD)	X				X			
Chromium (CR)					X			
Lead (PB)				X	X			
Mercury (HG)	X						X	
Selenium (SE)	X	X			X		X	

(a) USATHAMA chemical codes listed in parentheses.

X = Selected as a chemical of potential concern.

Note: Blanks in this table indicate that a chemical was not selected as a chemical of potential concern either because (1) it was not detected in a given medium, (2) it was not included in the analyses, or (3) it was detected at background concentrations (inorganic chemicals only). See text for this information.

Of the radiological parameters, gross beta was detected the most frequently. Bis(2-ethylhexyl)phthalate, a common lab contaminant, was the most frequently detected organic chemical in both the launch and control areas. Several chlorinated chemicals were present, as might be expected given the past use of these materials at the launch and control areas. Strong solvent, hydrocarbon, and/or pentachlorophenol odors were noted in soil and sediment in several areas of the launch area. More inorganic chemicals were selected as chemicals of potential concern in surface soil and sediment at the launch area than in subsurface soil at the launch and control areas.

In addition to the chemicals of potential concern selected for each medium using the available sampling data, other chemicals are possibly present at the Nike site and may be of potential concern regarding possible exposures and risks. Table 10-10 summarizes additional chemicals potentially of concern for the Nike site that were not included in any of the chemical analyses. The chemicals listed are those that have the potential to be present based on historical information. Lethal chemical agents, incapacitating agents, smoke/incendiary materials, and explosives are of potential concern at the Nike site because of the potential for a fire or explosion and the release and dispersion of agents into the atmosphere. Agent breakdown products and fuels/oils are of concern because of their potential contribution to groundwater, soil, sediment, and/or surface water contamination.

10.3 HUMAN HEALTH RISK ASSESSMENT

This section addresses the potential human health risks associated with the Nike site in the absence of remedial actions. This human health risk assessment is divided into three sections. Section 10.3.1 evaluates and provides estimates of potential human exposures for the chemicals of potential concern at the Nike site. Section 10.3.2 summarizes relevant toxicity information for the chemicals of potential concern. Section 10.3.3 provides quantitative and qualitative estimates of human health risks.

10.3.1 EXPOSURE ASSESSMENT

This section identifies the pathways by which human populations may be exposed to chemicals of potential concern at or originating from the Nike Site, and describes the pathways selected for evaluation. Only exposure pathways considered to be complete are selected for further evaluation (see Chapter 4 for a definition of a complete pathway). Evaluations of exposure were quantitative or qualitative depending upon several factors, including the probability of exposure, the potential magnitude of exposure, and the availability of data to support quantitative evaluations. Exposure point concentrations and daily intakes were estimated for pathways selected for quantitative evaluation.

This exposure assessment is organized into three sections. Section 10.3.1.1 discusses potential exposure pathways under current land-use conditions and Section 10.3.1.2 discusses those potentially occurring under hypothetical future land-use conditions. Section 10.3.1.3 presents estimates of potential human exposures for those pathways selected for quantitative evaluation.

10.3.1.1 Potential Exposure Pathways Under Current Land-Use Conditions

The majority of the Nike Site is covered by grassy fields, and most of the perimeter is forested. The northern portion of the site borders Amtrak railroad tracks. Some sections of these railroad tracks have been fenced. Other borders of the Nike site are water: Monks Creek to the northeast; and Lauderick Creek to the west, southwest, and south. There is only one road entering the Nike Site,

TABLE 10-10

CHEMICALS OF CONCERN POTENTIALLY PRESENT AT THE NIKE SITE (a)

Group	Chemical	Comments
Lethal Chemical Agents	Mustard (HD or H) Phosgene (CG)	Mustard and phosgene were used at the Nike site in filled chemical ordnance and in training activities such as large scale decontamination. Therefore, mustard and phosgene could be present in unexploded ordnance. Mustard can persist in soil by forming an oxide coating. Phosgene is not expected to persist in the environment due to rapid hydrolysis and volatilization.
Incapacitating Agents	Chloroacetophenone (CN)	CN containing chemical ordnance were used and fired at the Nike site; therefore, CN could be present in unexploded ordnance. CN has a low solubility, a very low rate of solution, and slow hydrolysis.
Smoke/Incendiary Materials	White Phosphorus (WP) Sulfur Trioxide-Chlorosulfonic Acid (FS) Titanium Tetrachloride (FM)	WP, FS, and FM filled chemical ordnance were used and fired at the Nike site; therefore, these chemicals could still be present in unexploded ordnance.
Agent Breakdown Products	Thiodiglycol 1,4-Dithiane 1,4-Oxathiane Titanium Chloride Sulfate	Mustard will rapidly hydrolyze in water to thiodyglycol and will thermally degrade to 1,4-dithiane and 1,4-oxathiane. Titanium tetrachloride, a smoke screen, solidifies to titanium chloride when in contact with moisture. Sulfure trioxide-chlorosulfonic acid produces sulfuric acid when dispersed later breaking down to sulfate.
Explosives	1,3-Dinitrobenzene 2,4-Dinitrotoluene 2,6-Dinitrotoluene HMX Nitrobenzene RDX Tetryl 1,3,5-Trinitrobenzene 2,4,6-Trinitrotoluene	High explosive ordnance were fired at the Nike site; therefore, these chemicals could be present in unexploded ordnance, soil, groundwater, and surface water at the Nike site.
Oils/Fuels	Hydraulic fluid Waste oil JP-4 Diesel fuel Unsymmetrical Dimethyl Hydrazine (UDMH) Aniline	Large quantities of hydraulic fluid, fuel oil, and JP-4 were used at the launch area. Hydraulic fluid and fuel oil also were used at the control area. Underground fuel oil storage tanks are located at all three areas. Although aniline and UDMH were reportedly used in small quantities, both of these chemicals could potentially be present at the launch area.
UDMH Breakdown Products	Dimethylnitrosamine (DMNA)	DMNA is resistant to microbial degradation, extremely mobile in the subsurface, and could be present in groundwater. DMNA will photolyze to some extent in surface water, but the rate of photolysis will be extremely site specific.

(a) Based on historical information. Chemicals listed are those not analyzed for and potentially present in the greatest quantities. A large number of other chemicals could be present in smaller quantities at the Nike site.

and vehicle access to the site is controlled by a locked gate. Additionally, the launch and control areas are each surrounded by a fence. Two full-time caretakers work at the Nike Site. The caretakers occupy a trailer near the entrance gate and are responsible for general site maintenance, including cutting the grass.

The Nike Site is currently leased to the Maryland Army National Guard. The National Guard Operating Activity Center uses four large buildings in the barracks area as offices. The rest of the site, which includes the launch and control areas, is leased by the National Guard for training purposes. These training activities include village combat training, military police training and water training. Buildings in the launch area are used for equipment storage and troop embarkation and debarkation training. Occasionally designated areas of the Nike site may be used by other units of the National Guard as a bivouac stop.

The community of Edgewood is located less than 1 mile to the west of the Nike site, and the residential subdivision of Willoughby Woods is located to the north of the Nike site boundary.

There are no groundwater supply wells located in the Nike study area, and most of the residences in the off-post housing area nearest the site are on public water supply. However, a few of these residences are reported to have domestic wells; the closest well is approximately 0.6 miles from the launch area.

Hunting and trapping are allowed around the Nike site. Game species are upland game/early migratory bird, migratory game bird, wild turkey, woodchuck, and deer. Additionally, there is an approved recreational fishing area at Skipper's Point on the southern side of Lauderick Creek. Fishing is also allowed from boats in the Bush River. Fishing from the shore of the Nike site is prohibited, as is fishing in Monks Creek.

The following sections discuss the potential long-term and acute exposure pathways under current land-use conditions.

10.3.1.1.1 Potential Long-term Exposure Pathways Under Current Land-Use Conditions

Table 10-11 summarizes the pathways by which humans could be exposed to chemicals at or originating from the Nike site. Potential exposure pathways are discussed below by exposure medium.

Surface Soil. Very few monitoring data are available on the concentrations of the chemicals present in the surface soil at the Nike site. Surface soil samples have been collected only from the launch area; these samples showed trace concentrations of three organic chemicals. Four inorganic chemicals (arsenic, cadmium, mercury, and selenium) may be slightly elevated above background in some of the launch area surface soil samples (generally less than one order of magnitude). However, because the U.S. Army Chemical School used the Nike site for training in chemical warfare from 1920 to 1951, residual surface contamination may be present in many areas. While chemical contamination is likely to be present at low concentrations across the entire Nike site, the launch and control areas may have more concentrated areas of contamination since more recent activities involving handling of fuels and other chemicals have taken place here. Chemicals that may be present in surface soil, but that in general have not been analyzed for, include chemical agents, explosives and their breakdown products, and oils and fuels. A list of the specific chemicals potentially present in soil at the Nike site was presented in Table 10-10.

TABLE 10-11

POTENTIAL PATHWAYS OF HUMAN EXPOSURE UNDER CURRENT LAND-USE CONDITIONS AT THE NIKE SITE

Exposure Medium/ Source Area	Potential Exposure Pathway	Potential for Significant Exposure (a)	Adequacy of Data to Evaluate Pathway	Method of Evaluation
Surface Soil/ Nike Site	Direct contact and/or incidental ingestion by National Guard personnel, caretakers, or hunters.	Negligible, based on vegetation and probable infrequent use of outside areas.	Poor. Data not collected from barracks and control area. One sample from the launch area analyzed for organic chemicals and eight for inorganic chemicals.	None, due to low potential for exposure and lack of data.
Subsurface Soil/ Nike Site	Direct contact and/or incidental ingestion by site workers.	None. Exposures are expected to be infrequent and of short duration.	NA. Pathway not complete.	None. No complete pathway exists.
Groundwater	Ingestion, dermal contact, and/or inhalation of chemicals in groundwater.	Negligible to moderate. Although there are no on-site uses, groundwater is evaluated as a resource because it is used in off-site areas.	Moderate. Some groundwater data available for the launch and control areas.	The resource potential of groundwater in the launch and control areas is evaluated quantitatively for ingestion and qualitatively for inhalation and dermal contact.
Sediment/ Launch Area Ditches	Direct contact and/or incidental ingestion by National Guard personnel or caretakers.	Negligible, based on infrequent contact and low chemical concentrations.	Poor. Only three samples collected.	None, due to low potential for exposure and lack of data.
Surface Water/Sediment/ Lauderick Creek Monks Creek Bush River	Direct contact by National Guard personnel, fishermen, hunters, or trappers. These surface water bodies are not used for drinking water or regularly for swimming.	Negligible, based on infrequent contact and expected low chemical concentrations.	Poor. Only one surface water sample collected.	None, due to low potential for exposure and lack of data.
Fish	Ingestion by local fishermen of fish that have accumulated chemicals from Lauderick Creek, Monks Creek, or the Bush River.	Negligible. Significant accumulation is not likely given expected low concentrations and large foraging areas.	NA. No fish tissue data available.	None, due to low potential for exposure and lack of data.
Game	Ingestion of game that has accumulated chemicals from the Nike site.	Negligible. None of the chemicals detected at the Nike site bioaccumulates extensively in terrestrial wildlife.	Poor. No tissue samples available and little information on surface soil contamination.	None, due to low potential for exposure and lack of data.

See footnotes on the following page.

TABLE 10-11 (Continued)

POTENTIAL PATHWAYS OF HUMAN EXPOSURE UNDER CURRENT LAND-USE CONDITIONS AT THE NIKE SITE

Exposure Medium/ Source Area	Potential Exposure Pathway	Potential for Significant Exposure (a)	Adequacy of Data to Evaluate Pathway	Method of Evaluation
Air/ Nike Site	Inhalation of volatile organics and/or airborne soil particles by National Guard personnel, caretakers, or hunters.	Negligible. Areas are not used frequently and concentrations of volatile organics are expected to be low. Dust generation and transport is unlikely because the area is vegetated, sheltered, and moist.	NA. Only subsurface soil gas data collected. Particulate data not collected.	None, due to low potential for exposure and lack of data.
Air/ Off-post Areas	Inhalation of volatile organics or fugitive dust by off-post residents.	Negligible, based on dispersion of very low concentrations.	NA. Off-site data not collected.	None, due to low potential for exposure and lack of data.
Soil/Air/ Nike Site	Acute hazards: fire or explosion at the Nike site with subsequent dermal and inhalation exposures.	Moderate to high. Unexploded ordnance containing high explosive and/or military chemical agents are present at the Nike site.	Poor. Unexploded ordnance found during clearing of various areas.	Qualitative, with a high degree of uncertainty.
Air/ Off-post Areas	Acute hazards: fire or explosion at the Nike site with subsequent dermal and inhalation exposures.	Moderate to high. Unexploded ordnance containing high explosive and/or military chemical agents are present at the Nike site.	Poor. Unexploded ordnance found during clearing of various areas.	Qualitative, with a high degree of uncertainty.

(a) Based on considerations of the types and concentrations of chemicals present, or expected to be present, and on considerations of land use.

NA = Not applicable.

National Guard personnel and site caretakers may contact surface soils in the launch and control areas during activities such as training or cutting the grass. Although no surface soil samples have been collected from within the barracks area, this area is used for offices by National Guard personnel so contact with soil is not likely. Designated areas of the Nike site are used by National Guard personnel for training and/or bivouacs. However, the potential for exposure is low, based on the infrequency and short duration of any individual's stay at the site. Moreover, since most of the Nike site is vegetated, the potential for an individual to contact bare soil is low. Based on the above and the fact that surface soil contamination appears to be low, the potential for significant exposure of National Guard personnel and/or site caretakers from direct contact with surface soil at the Nike site is considered to be negligible, and was not evaluated.

Subsurface Soil. Four subsurface soil samples have been collected at the Nike site, and little contamination was found in those samples that were taken. See sections 10.2.1.1 and 10.2.2.1 for a discussion of the chemicals analyzed for. Concentrations of selenium were elevated above regional background levels in the launch area. Bis(2,4,6-trichlorophenyl)urea (TCPU) was detected at a concentration of 3 mg/kg, and concentrations of mercury and selenium were slightly above regional background levels. However, given the history of the land use at the Nike site, which likely resulted in release of chemicals to the subsurface from surface spills, septic systems, and underground storage tanks, the potential for greater contamination of the subsurface soil does exist. The types of chemicals that may be present in subsurface soil include chemical agents, explosives, oils, fuels, and breakdown products. The specific list of chemicals within these groups of chemicals is presented in Table 10-10.

Under current land-use conditions at the Nike site the only populations that may contact subsurface soils are workers who may need to repair any underground pipes (such as sewer or utility lines) or remove any of the underground tanks. Such activities could involve exposure to chemicals in the subsurface soil through absorption through the skin and/or incidental ingestion of soils. This type of exposure is expected to be infrequent and of short duration. Therefore, no complete pathway exists for long-term exposures, and no pathway was selected for evaluation. (Potential acute exposures to chemicals in subsurface soils are discussed separately in the following section.)

Groundwater. There are no uses of groundwater at the Nike site. As discussed in Section 10.3.1.1, the nearest groundwater well being used for domestic purposes is approximately 0.6 miles from the launch area. Because there is uncertainty as to the direction of groundwater flow beneath some areas of the Nike site, it has not been determined if off-post domestic wells could potentially be impacted by chemicals released to groundwater at the Nike site. However, this groundwater is classified as Class IIB groundwater (groundwater that is a potential source of drinking water) under the EPA Ground Water Protection Strategy (EPA 1986). [Groundwater is classified as IIB if it contains less than 10,000 mg/L total dissolved solids and will yield 150 gallons per day]. Therefore, exposure to groundwater beneath the launch and control areas was evaluated, because it is a resource that is being used off-post. Ingestion of groundwater beneath these areas was evaluated quantitatively, and exposure through dermal contact and inhalation was evaluated qualitatively.

Surface Water/Sediment. Although very few data are available for sediment in on-site drainage ditches, the chemicals that were detected in the ditches in the launch area were at relatively low concentrations. However, it should be mentioned that many chemicals that were not analyzed for may potentially be present in the ditches. These chemicals include the chemical agents, explosives, oils, fuels, and breakdown products listed in Table 10-10.

The only surface water sample from the surface water bodies that surround the Nike site was from Monks Creek. It was analyzed for volatile organic chemicals; none were detected. No sediment samples were collected from these water bodies. It is likely that, in some areas of the Nike site, chemicals present in shallow groundwater discharge to these water bodies and that chemicals may also be transported to surface water bodies through direct runoff or through outfalls.

The potential exists for National Guard personnel or site caretakers to be exposed to on-site sediment and surface water (when present) in the site drainage ditches. Additionally, National Guard personnel, hunters, fishermen, and/or trappers may potentially be exposed through direct contact to chemicals present in surface water and sediment in Monks Creek, Lauderick Creek, and the Bush River (although fishing is not allowed in Monks Creek). For example, it is reported that National Guard personnel conduct water training including assault boat launch and landing, helocasting, and heavy raft training at the Nike site. These training activities could potentially result in exposure to surface water and sediment through direct contact. None of these surface water bodies are used as a source of drinking water or for swimming on a regular basis.

The potential for significant long-term exposure to on- and off-site surface water and sediment through any of these activities is considered to be negligible based primarily on land-use considerations. Use by National Guard personnel is not expected to be frequent, and hunting, trapping, and fishing are not activities that would involve repeated long-term contact with water or sediments. This low potential for contact with surface water or sediment, combined with the expected low concentrations of chemicals, results in a negligible potential for significant exposure to humans through direct contact with surface water or sediment in on-site ditches and the water bodies surrounding the Nike site. Therefore no pathways involving human exposure to surface water or sediment were evaluated in this assessment.

Fish. It is possible that individuals fishing in Lauderick Creek or the Bush River near the Nike site could be exposed to chemicals from the Nike site through the consumption of fish in these waters. Exposure could also occur from ingestion of fish from Monks Creek, although fishing is not allowed there. As mentioned above only one surface water sample was collected from Monks Creek, and no sediment samples have been collected from the water bodies surrounding the Nike site. However, chemical concentrations are expected to be low due to low concentrations detected to date in potential source areas releasing chemicals to these water bodies. There has been no sampling of fish tissue from these waters. It is likely, however, that many of the fish species of commercial and recreational importance in these water bodies tend to forage over large areas (such as white perch and channel catfish) and are unlikely to spend all of their time in one area. These fish would therefore have limited exposure to any chemicals from the Nike site. Although chemicals may bioaccumulate in the smaller species, they would not tend to be caught by fishermen.

Therefore, although no data are available to evaluate this potential human exposure pathway, ingestion of fish that have bioaccumulated chemicals originating at the Nike site is not expected to result in significant exposures, so this pathway was not evaluated.

Game. Areas of the Nike site that are not fenced may potentially be hunted. Deer, waterfowl, and other game animals could be exposed to chemicals present at the Nike site, potentially resulting in exposure of hunters that have killed and consumed them. However, none of the metals and soluble organic chemicals, such as those detected in the groundwater and soil at the Nike site, accumulate significantly in terrestrial food chains. For this reason and because game hunted at the Nike site would spend only a small portion of its total foraging time at the Nike site, significant exposures via

the ingestion of game that may have accumulated chemicals from the Nike site is unlikely. Therefore, this potential human exposure pathway is not selected for further evaluation.

Air. Air contamination at the Nike site could result from direct volatilization of chemicals and transport by wind entrainment of chemicals present on dust particles. (Atmospheric dispersal of contaminants due to an explosion or fire is discussed in the section on acute exposures.) Migration of contaminants by wind entrainment of dust particles is unlikely to be an important transport process at the Nike site because the majority of the area is vegetated, somewhat sheltered from high winds by a perimeter forest, and likely has a high soil moisture content during most of the year.

Contamination at the Nike site may occur as a result of volatilization of chemicals to the atmosphere from groundwater where it discharges to surface water, or as a result of volatilization through soil to the atmosphere. Volatile organic chemicals have been detected in some of the groundwater samples at low concentrations. Because the shallow groundwater beneath most of the site likely discharges into the water bodies surrounding the site, this may be a source of low concentrations of volatile chemicals in the air. Additionally, trichloroethene was detected at a low concentration in a water sample from a missile silo, and this may also be a potential source of volatile chemicals in the air. Soil gas samples were collected from and within the vicinity of the launch area during soil boring sampling. Several volatile organic chemicals were detected in these samples at low concentrations (all less than 10 ug/m^3) generally at a depth of 4 to 7 feet below the ground surface, indicating that volatile chemicals in the soil may be a potential source of volatile chemicals in the air.

Given the very low concentrations of the chemicals measured in the soil gas in the launch area and that the barracks area is most frequented on a regular basis for indoor use only, this potential exposure pathway will not be evaluated in this assessment.

10.3.1.1.2 Potential Acute Exposures Under Current Land-Use Conditions

Due to past use of the Nike site by the U.S. Army Chemical School for training in chemical warfare activities, all areas of the Nike site may contain unexploded ordnance containing high explosive and/or military chemical agents. Although information concerning the quantity of unexploded ordnance at the Nike site is not available, unexploded ordnance was recovered from the Nike site during construction at the launch and control areas, during two range clearing surface sweeps, and during site clearance for RFI work drilling. Site workers who may disturb the subsurface during activities such as septic system repairs or removals, or removal of underground tanks may potentially encounter unexploded ordnance. Permits are required for excavation, however, and it is likely that a magnetometer sweep would be required before the subsurface could be disturbed at the Nike site. It is also possible that nearby individuals in off-post areas may be exposed if such a release occurs. Due to lack of data, these potential exposure pathways were addressed qualitatively.

10.3.1.2 Potential Exposure Pathways Under Future Land-Use Conditions

Future use of the Nike site for commercial or industrial uses is considered unlikely given the possibility that unexploded ordnance or other residual contamination from testing activities exists at the Nike site. Therefore, future uses are likely to continue to parallel current site uses, and no additional on-site exposure pathways are expected to occur.

10.3.1.3 Quantification of Exposure

The only human exposure pathway evaluated quantitatively in this assessment is ingestion of groundwater by off-post residents. The methodology used to quantitatively assess exposure by determining the chronic daily intake (CDI) of each chemical of concern for each complete exposure pathway being evaluated has been summarized in Chapter 4. In the following section, exposure point concentrations are first presented and then combined with the other exposure parameters to estimate intake.

Quantification of Exposure Under Current Land-Use Conditions

Exposure point concentrations of chemicals of potential concern in groundwater in the launch and control areas at the Nike site are presented in Tables 10-12 and 10-13. As discussed in Section 10.3.1.1, although groundwater is not used at the Nike site, its drinking water resource potential was evaluated, because it is used for domestic purposes off-post and any potential hydraulic connection between on-site groundwater and off-post wells has not been evaluated. Because different chemicals were detected in the launch and control areas and because these two areas are separated by at least 2,400 feet, the potential exposure to chemicals of potential concern originating in each of these areas was evaluated separately. Contaminants present in the soil or potentially being released from underground storage tanks, sewer lines, septic tanks, and sand filter beds are potential sources of groundwater contamination; therefore, concentrations of chemicals in the groundwater may change in the future as contaminants from these types of sources leach into the groundwater.

To evaluate groundwater ingestion exposures, individuals were assumed to drink 2 liters of water for 30 years. In addition, these individuals were assumed to weigh 70 kg (EPA 1989a) and live for 70 years (EPA 1989b). Drinking water exposures were calculated using these assumptions and the following equation:

$$CDI = (C_w * IR * EF * ED * Z) / (BW * DY * YL) \qquad \text{(Eq. 1)}$$

where:

CDI	=	chronic daily intake (mg/kg-day);
C_w	=	exposure point concentration in groundwater (µg/L);
IR	=	ingestion rate (2 liters/day);
EF	=	exposure frequency (365 days/year);
ED	=	exposure duration (30 years);
Z	=	conversion factor (mg/1,000 ug);
BW	=	body weight over the period of exposure (70 kg);

TABLE 10-12

EXPOSURE CONCENTRATIONS OF CHEMICALS OF POTENTIAL CONCERN
FOR THE NIKE SITE: LAUNCH AREA GROUNDWATER

(Concentrations reported in ug/L)

Chemical (a)	Arithmetic Mean	Upper 95 Percent Confidence Limit on the Arithmetic Mean (b)	Maximum Detected Concentration	RME Concentration (c)
Radiological Parameters (pCi/L):				
Gross Alpha (ALPHAG)	0.6	0.6	1.6	0.6
Gross Beta (BETAG)	5.3	8.6	31	8.6
Potassium 40 (K40)	23	NA	45	45
Radium 226 (RA226)	NA	NA	0.6	0.6
Organic Chemicals:				
Acetone (ACET)	8.9	9.1	89	9.1
Carbon Disulfide (CS2)	2.9	3.1	17	3.1
trans-1,2-Dichloroethene (T12DCE)	2.1	4.2	8.0	4.2
2-Ethyl-1-hexanol (2E1HXL)	NA	NA	16	16
bis(2-Ethylhexyl)phthalate (B2EHP)	100	310	1,200	310
Methylene Chloride (CH2CL2)	2.9	3.9	24.3	3.9
Trichloroethene (TRCLE)	4.8	8.4 (d)	89	8.4
Inorganic Chemicals:				
Barium (BA)	120	150	319	150

(a) USATHAMA chemical codes listed in parentheses.
(b) Values reflect a positively skewed distribution, except as noted.
(c) Value listed is the lower value of the upper 95 percent confidence limit on the arithmetic mean and the maximum detected value.
(d) Value reflects a normal distribution.

NA = Not applicable; single sample analyzed or less than three samples analyzed.

TABLE 10-13

EXPOSURE CONCENTRATIONS OF CHEMICALS OF POTENTIAL CONCERN
FOR THE NIKE SITE: CONTROL AREA GROUNDWATER

(Concentrations reported in ug/L)

Chemical (a)	Arithmetic Mean	Upper 95 Percent Confidence Limit on the Arithmetic Mean (b)	Maximum Detected Concentration	RME Concentration (c)
Radiological Parameters (pCi/L):				
Gross Beta (BETAG)	2.2	51	4.6	4.6
Organic Chemicals:				
trans-1,2-Dichloroethene (T12DCE)	6.4	300	18	18
bis(2-Ethylhexyl)phthalate (B2EHP)	200	950,000	700	700
1,1,1-Trichloroethane (111TCE)	4.9	9.6	17.5	9.6
Trichloroethene (TRCLE)	5.9	14	25	14
Inorganic Chemicals:				
Barium (BA)	74	7,200	203	203

(a) USATHAMA chemical codes listed in parentheses.
(b) Values reflect a positively skewed distribution.
(c) Value listed is the lower value of the upper 95 percent confidence limit on the arithmetic mean and the maximum detected value.

DY = days in a year (365 days/year); and

YL = period over which risk is being estimated (a lifetime [70 years] for potential carcinogens and the period of exposure [30 years] for noncarcinogens) (years).

CDIs calculated using these exposure assumptions are presented in Tables 10-14 and 10-15 for the launch and control areas, respectively.

10.3.2 TOXICITY ASSESSMENT

The general methodology for the classification of health effects and the development of health effects criteria has been described in Chapter 4 to provide the analytical framework for the characterization of human health impacts. The oral health effects criteria that were used to derive estimates of risk for individuals ingesting groundwater are presented in Table 10-16. No oral toxicity criteria are available for 2-ethyl-1-hexanol, gross alpha, and gross beta as discussed in Section 10.3.1.3. Therefore, potential risks associated with exposure to these chemicals were not quantitatively evaluated. However, complete toxicity summaries of these chemicals are provided in Appendix B.

No toxicity criteria are provided in this section for the chemicals of concern potentially present at the Nike site (see Table 10-10). However, complete toxicity summaries for most of these chemicals are provided in Appendix B.

10.3.3 RISK CHARACTERIZATION

In this section, potential human health risks at the Nike site are described. Risks were evaluated either quantitatively or qualitatively. To quantitatively assess risks, the CDIs calculated in Section 10.3.1 were combined with the health effects criteria presented in Section 10.3.2.

The exposure pathways quantitatively or qualitatively evaluated in this assessment under current land-use conditions are listed below.

- Dermal and/or inhalation exposure of site workers encountering unexploded ordnance during activities that disturb the subsurface was qualitatively evaluated.
- Dermal and/or inhalation exposure of nearby off-post residents resulting from a release of chemical filled unexploded ordnance at the Nike site was qualitatively evaluated.
- Ingestion of groundwater at the Nike Site was quantitatively evaluated because of its potential as a drinking water resource. Dermal contact with and inhalation of chemicals in groundwater were evaluated qualitatively.

Potential Risks Under Current Land-Use Conditions

Table 10-17 presents estimated CDIs and risks for individuals ingesting groundwater containing chemicals from the launch area. For ingestion of groundwater containing contaminants originating at the launch area, the upper-bound excess lifetime cancer risk is estimated to be 7×10^{-5}. This risk is

TABLE 10-14

EXPOSURE POINT CONCENTRATIONS AND CHRONIC DAILY INTAKES FOR INGESTION OF GROUNDWATER: NIKE SITE LAUNCH AREA (a)

Chemical (b)	RME Concentration (ug/L)	Estimated Chronic Daily Intake (CDI) (mg/kg-day) (c)
Chemicals Exhibiting Carcinogenic Effects		
bis(2-Ethylhexyl)phthlate (B2EHP)	310	3.8E-03
Methylene Chloride (CH2CL2)	3.9	4.8E-05
Trichloroethene (TRCLE)	8.4	1.0E-04
Potassium 40 (K40)	45 (pCi/L)	9.9E+05 (pCi)
Radium 226 (RA226)	0.6 (pCi/L)	1.3E+04 (pCi)
Chemicals Exhibiting Noncarcinogenic Effects		
Acetone (ACET)	9.1	2.6E-04
Carbon Disulfide (CS2)	3.1	8.9E-05
trans-1,2-Dichloroethene (T12DCE)	4.2	1.2E-04
bis(2-Ethylhexyl)phthlate (B2EHP)	310	8.9E-03
Methylene Chloride (CH2CL2)	3.9	1.1E-04
Trichloroethene (TRCLE)	8.4	2.4E-04
Barium (BA)	150	4.3E-03

(a) CDIs have been calculated only for those chemicals of potential concern with toxicity criteria. The following chemicals of potential concern are not presented due to lack of toxicity criteria: 2-ethyl-1-hexanol, gross alpha, gross beta.
(b) USATHAMA chemical codes listed in parentheses.
(c) See text for exposure assumptions.

TABLE 10-15

EXPOSURE POINT CONCENTRATIONS AND CHRONIC DAILY INTAKES FOR INGESTION OF GROUNDWATER: NIKE SITE CONTROL AREA (a)

Chemical (b)	RME Concentration (ug/L)	Estimated Chronic Daily Intake (CDI) (mg/kg-day) (c)
Chemicals Exhibiting Carcinogenic Effects		
bis(2-Ethylhexyl)phthlate (B2EHP)	700	8.6E-03
Trichloroethene (TRCLE)	14	1.7E-04
Chemicals Exhibiting Noncarcinogenic Effects		
trans-1,2-Dichloroethene (T12DCE)	18	5.1E-04
bis(2-Ethylhexyl)phthlate (B2EHP)	700	2.0E-02
1,1,1-Trichloroethane (TRCLE)	9.6	2.7E-04
Trichloroethene (TRCLE)	14	4.0E-04
Barium (BA)	203	5.8E-03

(a) CDIs have been calculated only for those chemicals of potential concern with toxicity criteria. The following chemical of potential concern is not presented due to lack of toxicity criteria: gross beta.
(b) USATHAMA chemical codes listed in parentheses.
(c) See text for exposure assumptions.

TABLE 10-16

ORAL CRITICAL TOXICITY VALUES FOR CHEMICALS OF POTENTIAL CONCERN
AT THE NIKE SITE

Chemical	Chronic Reference Dose (mg/kg/day)	Uncertainty Factor (a)	Target Organ (b)	Reference Dose Source	Cancer Slope Factor (mg/kg/day)-1	EPA Weight of Evidence Classification (c)	Slope Factor Source
Organic Chemicals:							
Acetone	1.00E-01	1,000	Kidney/Liver	IRIS	--	D	IRIS
Carbon Disulfide	1.00E-01	100	Fetus	IRIS	--	--	IRIS
trans-1,2-Dichloroethene	2.00E-02	1,000	Blood	IRIS	--	--	--
2-Ethyl-1-hexanol	--	--	--	--	--	--	--
bis(2-Ethylhexyl)phthalate	2.00E-02	1,000	Liver	IRIS	1.40E-02	B2	IRIS
Methylene Chloride	6.00E-02	100	Liver	IRIS	7.50E-03	B2	IRIS
1,1,1-Trichloroethane	9.00E-02	1,000	Liver	IRIS	--	D	IRIS
Trichloroethene	7.35E-03	1,000	Liver	HA	1.10E-02	B2	HEAST
Inorganic Chemicals:							
Barium	7.00E-02	3	Cardiovascular System	IRIS	--	--	IRIS
Radiological Parameters:							
Gross Alpha	--	--	--	--	--	--	--
Gross Beta	--	--	--	--	--	--	--
Potassium 40	--	--	--	--	1.10E-11 (pCi/L)-1	A	HEAST
Radium 226	--	--	--	--	1.20E-10 (pCi/L)-1	A	HEAST

(a) Safety factors are the products of uncertainty factors and modifying factors. Uncertainty factors used to develop reference doses generally consist of multiples of 10, with each factor representing a specific area of uncertainty in the data available. The standard uncertainty factors include the following:
- a 10-fold factor to account for the variation in sensitivity among the members of the human population;
- a 10-fold factor to account for the uncertainty in extrapolating animal data to the case of humans;
- a 10-fold factor to account for the uncertainty in extrapolating from less than chronic NOAELs to chronic NOAELs; and
- a 10-fold factor to account for the uncertainty in extrapolating from LOAELs to NOAELs.

Modifying factors are applied at the discretion of the reviewer to cover other uncertainties in the data.

(b) A target organ is the organ most sensitive to a chemical's toxic effect. RfDs are based on toxic effects in the target organ. If an RfD was based on a study in which a target organ was not identified, an organ or system known to be affected by the chemica is listed.

(c) EPA Weight of Evidence for Carcinogenic Effects:
[A] = Human carcinogen based on adequate evidence from human studies;
[B2] = Probable human carcinogen based on inadequate evidence from human studies and adequate evidence from animal studies; and
[D] = Not classified as to human carcinogenicity.

NOTE: IRIS = Integrated Risk Information System - December 1, 1990.
HA = Health Advisory.
HEAST = Health Effects Assessment Summary Tables - July 1, 1990.
-- = No information available.

TABLE 10-17

POTENTIAL RISKS ASSOCIATED WITH INGESTION OF GROUNDWATER:
NIKE SITE LAUNCH AREA (a)

Chemicals Exhibiting Carcinogenic Effects (b)	Estimated Chronic Daily Intake (CDI) (mg/kg-day)	Slope Factor (mg/kg-day)-1	Weight of Evidence Class (c)	Upper Bound Excess Lifetime Cancer Risk
bis(2-Ethylhexyl)phthlate (B2EHP)	3.8E-03	1.4E-02	B2	5E-05
Methylene Chloride (CH2CL2)	4.8E-05	7.5E-03	B2	4E-07
Trichloroethene (TRCLE)	1.0E-04	1.1E-02	B2	1E-06
Potassium 40 (K40)	9.9E+05 (pCi)	1.1E-11 (pCi)-1	A	1E-05
Radium 226 (RA226)	1.3E+04 (pCi)	1.2E-10 (pCi)-1	A	2E-06
TOTAL	--	--	--	7E-05

Chemicals Exhibiting Noncarcinogenic Effects (b)	Estimated Chronic Daily Intake (CDI) (mg/kg-day)	Reference Dose (RfD) (mg/kg-day)	Uncertainty Factor (d)	Target Organ (e)	CDI:RfD Ratio
Acetone (ACET)	2.6E-04	1.0E-01	1,000	Kidney/Liver	3E-03
Carbon Disulfide (CS2)	8.9E-05	1.0E-01	100	Fetus	9E-04
trans-1,2-Dichloroethene (T12DCE)	1.2E-04	2.0E-02	1,000	Blood	6E-03
bis(2-Ethylhexyl)phthlate (B2EHP)	8.9E-03	2.0E-02	1,000	Liver	4E-01
Methylene Chloride (CH2CL2)	1.1E-04	6.0E-02	100	Liver	2E-03
Trichloroethene (TRCLE)	2.4E-04	7.35E-03	1,000	Liver	3E-02
Barium (BA)	4.3E-03	7.0E-02	3	Cardiovasc. Sys.	6E-02
HAZARD INDEX	--	--	--	--	< 1 (5E-01)

(a) Risks are calculated only for chemicals with toxicity criteria. The following chemicals of potential concern are not presented due to lack of toxicity criteria: 2-ethyl-1-hexanol, gross alpha, gross beta.
(b) USATHAMA chemical codes listed in parentheses.
(c) EPA Weight of Evidence for Carcinogenic Effects:
[A] = Human carcinogen based on adequate evidence from human studies; and
[B2] = Probable human carcinogen based on inadequate evidence from human studies and adequate evidence from animal studies.
(d) Factor which reflects the uncertainty in the estimate of the RfD. Larger factors are associated with greater uncertainty.
(e) A target organ is the organ most sensitive to a chemical's toxic effect. RfDs are based on toxic effects in the target organ. If an RfD was based on a study in which a target organ was not identified, an organ or organ system known to be affected by the chemical is listed.

primarily due to bis(2-ethylhexyl)phthalate and potassium 40, although trichloroethene and radium 226 also contribute to the risk. Potassium 40 and radium 226 are known human carcinogens (Class A) and bis(2-ethylhexyl)phthalate and trichloroethene are probable human carcinogens (Class B2). The Hazard Index associated with ingestion of groundwater containing chemicals from the launch area is less than 1 indicating that noncarcinogenic effects are not likely.

Table 10-18 presents estimated CDIs and risks for individuals ingesting groundwater containing chemicals from the control area. For ingestion of groundwater containing contaminants originating at the control area, the upper-bound excess lifetime cancer risk is estimated to be $1x10^{-4}$. This risk is primarily due to bis(2-ethylhexyl)phthalate, although trichloroethene also contributes to the risk. Both of these chemicals are classified as probable human carcinogens (Class B2). The Hazard Index for evaluation of potential noncarcinogenic effects is equal to one, indicating noncarcinogenic effects are possible. The Hazard Index is equal to 1 based on the presence of bis(2-ethylhexyl)phthalate.

In addition to those chemicals detected in the groundwater to date, other chemicals may be present. These chemicals are listed in Table 10-10 and are components of chemical agents, explosives, oils, fuels, and breakdown products.

In addition to exposure through ingestion of groundwater, individuals may potentially be exposed to the organic chemicals in groundwater through direct contact during activities such as bathing. Additional exposure to volatile organic chemicals may result through inhalation during activities such as showering. Given the chemicals that are present in the groundwater beneath the launch and control areas, these exposures and risks are likely to be of the same order of magnitude as those estimated for groundwater ingestion.

Under current land-use conditions site workers disturbing the subsurface may potentially encounter explosive or chemical-filled unexploded ordnance. Chemicals that could be present in chemical ordnance as a result of chemical school training activities at the Nike site include white phosphorus, a mixture of sulfur trioxide and chlorosulfonic acid, titanium tetrachloride, chloroacetophenone, phosgene, and mustard. The potential human risks from fire and explosion include burns, injury, dismemberment, and death. Exposure to chemical-filled ordnance may result in acute injuries to the eyes, skin, nose, throat, and/or lungs. For example, dermal contact with mustard may result in malaise, vomiting, fever, inflammation, and/or blistering of the eyes, skin, nose, throat, trachea, bronchi, and lung tissue. Higher doses of mustard may result in death or injury to bone marrow, lymph nodes, and spleen. Additionally, exposure to phosgene, a choking agent, may result in damage to the capillaries and scarring of the lungs. Exposure to phosgene may also result in death from oxygen deficiency.

While the potential for off-post residents to be exposed as a result of a chemical release is much less likely than for individuals on the Nike site, the close proximity of some off-post areas to the Nike site warrants mentioning. If a release resulting in acute hazards were to occur on the Nike site near the installation boundary, under some conditions, off-post individuals may potentially be affected. The conditions under which off-post individuals are most likely to be affected by a chemical release would be calm light winds from the direction of the release and stable atmospheric conditions. Under such conditions, exposure through dermal contact with airborne chemicals and/or inhalation may occur. However, the effects are likely to be much less extreme than encountered closer to the release.

TABLE 10-18

POTENTIAL RISKS ASSOCIATED WITH INGESTION OF GROUNDWATER:
NIKE SITE CONTROL AREA (a)

Chemicals Exhibiting Carcinogenic Effects (b)	Estimated Chronic Daily Intake (CDI) (mg/kg-day)	Slope Factor (mg/kg-day)-1	Weight of Evidence Class (c)	Upper Bound Excess Lifetime Cancer Risk
bis(2-Ethylhexyl)phthlate (B2EHP)	8.6E-03	1.4E-02	B2	1E-04
Trichloroethene (TRCLE)	1.7E-04	1.1E-02	B2	2E-06
TOTAL	--	--	--	1E-04

Chemicals Exhibiting Noncarcinogenic Effects (b)	Estimated Chronic Daily Intake (CDI) (mg/kg-day)	Reference Dose (RfD) (mg/kg-day)	Uncertainty Factor (d)	Target Organ (e)	CDI:RfD Ratio
trans-1,2-Dichloroethene (T12DCE)	5.1E-04	2.0E-02	1,000	Blood	3E-02
bis(2-Ethylhexyl)phthlate (B2EHP)	2.0E-02	2.0E-02	1,000	Liver	1E+00
1,1,1-Trichloroethane (TRCLE)	2.7E-04	9.0E-02	1,000	Liver	3E-03
Trichloroethene (TRCLE)	4.0E-04	7.35E-03	1,000	Liver	5E-02
Barium (BA)	5.8E-03	7.0E-02	3	Cardiovasc. Sys.	8E-02
HAZARD INDEX	--	--	--	--	- 1 (1E+00)

(a) Risks are calculated only for chemicals with toxicity criteria. The following chemical of potential concern is not presented due to lack of toxicity criteria: gross beta.
(b) USATHAMA chemical codes listed in parentheses.
(c) EPA Weight of Evidence for Carcinogenic Effects: [B2] = Probable human carcinogen based on inadequate evidence from human studies and adequate evidence from animal studies.
(d) Factor which reflects the uncertainty in the estimate of the RfD. Larger factors are associated with greater uncertainty.
(e) A target organ is the organ most sensitive to a chemical's toxic effect. RfDs are based on toxic effects in the target organ. If an RfD was based on a study in which a target organ was not identified, an organ or organ system known to be affected by the chemical is listed.

10.4 ECOLOGICAL ASSESSMENT

This section assesses potential ecological impacts associated with the chemicals of potential concern at the Nike site in the absence of remediation. The methods used to assess ecological impacts follow those outlined in Chapter 4 and roughly parallel those used in the human health risk assessment. Below, potentially exposed populations (receptors) are identified. Then information on exposure and toxicity is combined to derive estimates of potential impact in these populations. It is emphasized that this ecological assessment is a predictive assessment. Comprehensive field studies of ecological impacts have not yet been conducted at the Nike site.

This ecological assessment is divided into four principal sections. Section 10.4.1 describes the habitat of the area and identifies the potential receptor species or species groups selected for evaluation. Section 10.4.2 evaluates and provides estimates of potential exposures for the chemicals and receptors of potential concern. Section 10.4.3 summarizes relevant toxicity information for the chemicals of potential concern and Section 10.4.4 provides quantitative and qualitative estimates of ecological impact.

10.4.1 RECEPTOR CHARACTERIZATION

The Nike site is composed mostly of forested land (including maple, sweetgum, white oak, and pine). Habitat characteristics of the Nike site are shown in Figure 10-2. Grassy fields and some limited shrub growth are found around the launch, control, and barracks areas. There are wetlands along Monks Creek, Lauderick Creek, and parts of the Bush River. Based on the varied habitat, the Nike site is expected to support a variety of wildlife.

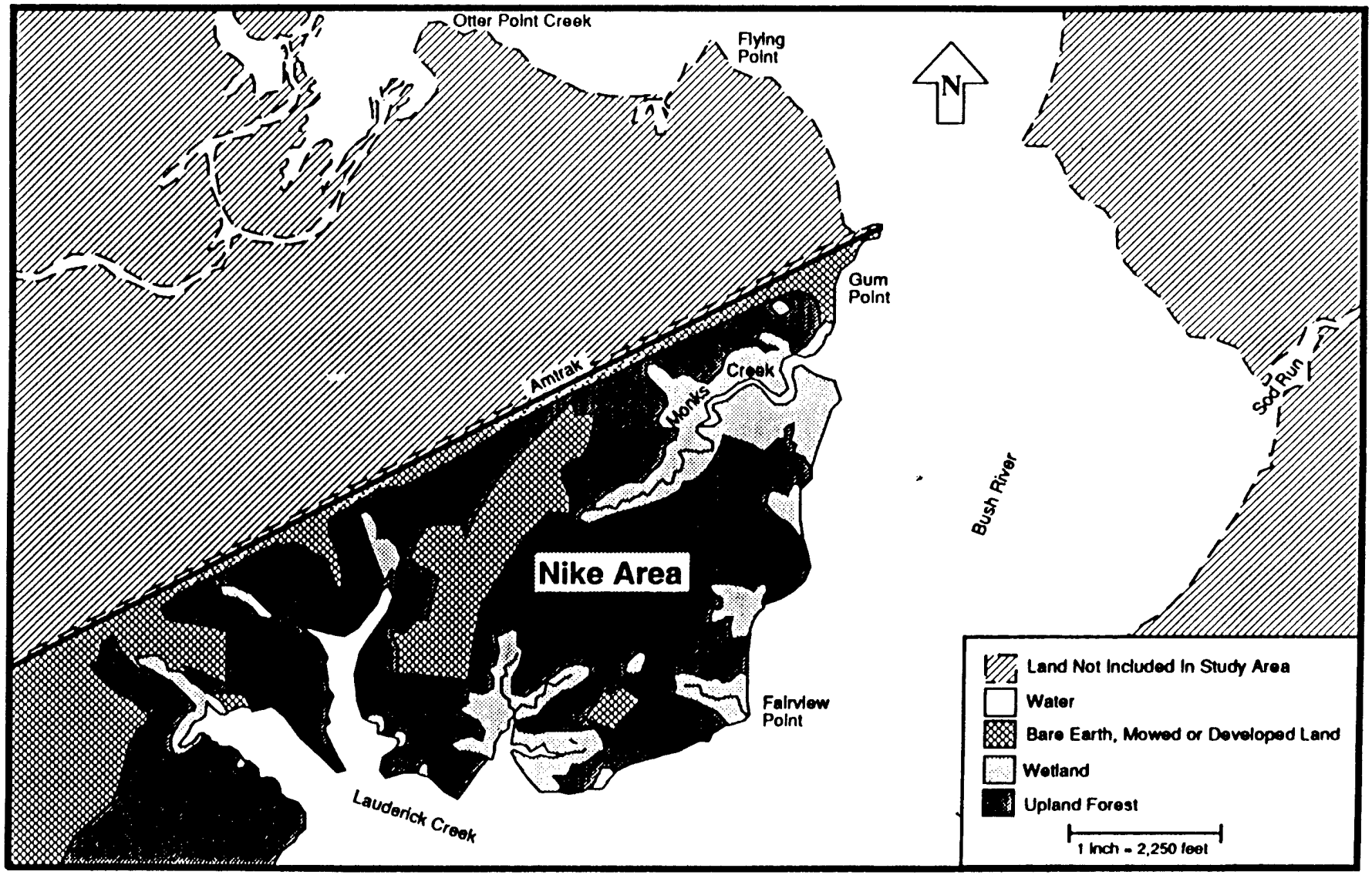
Mammalian species expected to occur in forested areas of the site include squirrel, fox, and deer. Woodpeckers and owls also are expected to occur in the forested areas. Field mice, rabbits, doves, hawks, and a variety of song birds are expected to occur in the grassy fields. In the wetland areas muskrats (*Ondatra zibethicus*), raccoons (*Procyon lotor*), voles, herons, shorebirds, and wood ducks (*Aix sponsa*) are expected to occur. During a site visit by Clement in May 1990, turkey vultures (*Cathartes aura*), starlings (*Sturnus vulgaris*), eastern bluebirds (*Sialia sialis*), killdeers (*Charadrius vociferus*), barn swallows (*Hirundo rustica*), great blue herons (*Ardea herodias*), mockingbirds (*Mimus polyglottos*), robins (*Turdus migratorius*), and crows (*Corvus* spp.) were observed.

A variety of freshwater and estuarine aquatic life are likely to be found in Monks Creek and Lauderick Creek. These creeks are tidal tributaries to Bush River. Fish species expected in these areas include catfish, suckers, sunfish (*Lepomis cyanellus*), and killifish. Aquatic life in the ditches that drain the launch area is expected to be limited in abundance and variety, because the ditches are dry at least part of the year and the bottom substrate may be of limited quality.

As discussed in Chapter 4, it is not feasible to assess the potential impacts on each of the species potentially present at the Nike site. For this reason indicator species or species groups were selected for further evaluation. The selection of indicator species for the Nike site was driven by several factors including the potential for exposure, the sensitivity or susceptibility to chemical exposures, the availability of toxicity data, the availability of chemical data for potential exposure media, ecological significance, and societal value. The indicator species selected for evaluation at the Nike site, benthic invertebrates, is among those identified as potential indicators in Chapter 4. Benthic invertebrates were selected since they may occur in the ditches on site and may be exposed to chemicals in sediments.

Figure 10-2

Habitat Characteristics of the Nike Study Area At Aberdeen Proving Ground



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10.4.2 POTENTIAL EXPOSURE PATHWAYS AND QUANTIFICATION OF EXPOSURE

In this section, the potential pathways by which the selected indicator species could be exposed to the chemicals of potential concern at the Nike site are discussed and exposure is quantified for selected exposure pathways. This exposure assessment focuses on potential exposures to chemicals in sediments. One surface water sample was collected from the upper portion of Monks Creek and was analyzed for volatile organic chemicals, but no volatiles were detected. No other surface water sampling data are available. No pathways exist by which wildlife could be exposed to chemicals of potential concern in groundwater or subsurface soils. Environmental receptors may be exposed to chemicals in surface soils. However, exposures to surface soil were not evaluated in this assessment, because of the limitations of the sampling data and limitations of exposure and toxicity information.

Aquatic Life Exposures. As discussed in Chapter 4, aquatic life could be exposed to chemicals in sediment by several pathways. In this assessment potential risks from sediments are based on RME chemical concentrations in sediments given in Table 10-19. Sediment samples were collected from ditches that drain the launch area. These earthen or concrete ditches drain into Monks Creek. The wetlands of Monks Creek are about 800 feet southeast of the launch area. The ditches were dry at the time of sampling, so no surface water samples were taken. As previously mentioned, aquatic life in the ditches is expected to be limited in abundance and variety because the ditches are dry at least part of the year. Aquatic organisms from Monks Creek are not expected to use these ditches to a significant extent, because the habitat is probably poor compared to Monks Creek. Aquatic life in the ditches could be exposed to chemicals in the sediments through several pathways. In addition, aquatic forms that are capable of surviving dry conditions in the ditches could be exposed to contaminants in sediments by direct contact. However, under these conditions, the potential absorption of chemicals is quite low.

10.4.3 TOXICITY ASSESSMENT

Limited information is available on the toxicity of the chemicals of potential concern in sediment. Selected sediment toxicity values are presented in Table 10-20 for cadmium, chromium, and lead. Sediment toxicity information is not sufficient to assess potential risks from the organic chemicals of potential concern or from selenium.

10.4.4 ESTIMATES OF IMPACTS

Impacts to aquatic invertebrates exposed to chemicals of potential concern in sediments from the launch area ditches were evaluated by comparing sediment toxicity values with the RME concentrations in the sediments reported in Table 10-21. The comparisons show that the RME concentrations for cadmium, chromium, and lead are all less than the selected sediment toxicity values. Thus, most types of aquatic organisms that may inhabit the launch area ditches are not expected to be adversely affected by the chemicals in the sediments.

10.5 UNCERTAINTIES

As in any risk assessment, there is a large degree of uncertainty associated with the estimates of human health and ecological risks for the Nike site. Consequently, these estimates should not be regarded as absolute estimates of risk but rather as conditional estimates based on a number of

TABLE 10-19

EXPOSURE CONCENTRATIONS OF CHEMICALS OF POTENTIAL CONCERN IN SEDIMENT
AT THE NIKE SITE LAUNCH AREA

(Concentrations reported in mg/kg)

Chemical (a)	Arithmetic Mean	Upper 95 Percent Confidence Limit on the Arithmetic Mean (b)	Maximum Detected Concentration	RME Concentration (c)
Organic Chemicals:				
bis(2-Ethylhexyl)phthalate (B2EHP)	NA	NA	0.003	0.003
Hexanedioic Acid, Dioctyl Ester	NA	NA	0.01	0.01
Tetrachloroethene (TCLEE)	NA	NA	0.001	0.001
Inorganic Chemicals:				
Barium (BA)	160	3,900	220	220
Cadmium (CD)	0.9	25	1.61	1.61
Chromium (CR)	110	3,400	192	192
Lead (PB)	85	840	118	118
Selenium (SE)	4.3	6.4	5.12	5.12

(a) USATHAMA chemical codes listed in parentheses.
(b) Values reflect a positively skewed distribution.
(c) Value listed is the lower value of the upper 95 percent confidence limit on the arithmetic mean and the maximum detected value.

NA = Not applicable; single sample.

TABLE 10-20

TOXICITY VALUES FOR ASSESSMENT OF AQUATIC LIFE IMPACTS
FROM EXPOSURE TO CHEMICALS IN SEDIMENTS AT THE NIKE SITE LAUNCH AREA

Chemical (a)	Sediment Toxicity Value (mg/kg)	Basis for Value (b)	Reference
Organic Chemicals:			
bis(2-Ethylhexyl)phthalate (B2EHP)	--	--	--
Hexandioic Acid, Dioctyl Ester	--	--	--
Tetrachloroethene (TCLEE)	--	--	--
Inorganic Chemicals:			
Barium (BA)	--	--	--
Cadmium (CD)	5.1	Concentration at or above level at which significant biological effects will occur; based on studies with benthic invertebrates	Barrick and Beller (1989)
Chromium (CR)	260	Concentration at or above level at which significant biological effects will occur; based on studies with oysters	Barrick and Beller (1989)
Lead (PB)	450	Concentration at or above level at which significant biological effects will occur; based on studies with benthic invertebrates	Barrick and Beller (1989)
Selenium (SE)	--	--	

(a) USATHAMA chemical codes listed in parentheses.
(b) See Appendix C for more study details.

-- = No information available.

TABLE 10-21

COMPARISON OF SEDIMENT TOXICITY VALUES WITH EXPOSURE CONCENTRATIONS FOR CHEMICALS IN SURFACE WATER AT THE NIKE SITE LAUNCH AREA

(Concentrations reported in mg/kg)

Chemical (a)	Toxicity Value (b)	Exposure Concentration (c)	Toxicity Value Exceeded?
Inorganic Chemicals:			
Cadmium (CD)	5.1	1.61	No
Chromium (CR)	260	192	No
Lead (PB)	450	118	No

(a) USATHAMA chemical codes listed in parentheses. Only chemicals with toxicity values are listed. The following chemicals are not presented due to lack of toxicity criteria: bis(2-ethylhexyl)phthalate; hexanedioic acid, dioctyl ester; tetrachloroethene; barium; and selenium.
(b) Reported previously in Table 10-20.
(c) Reported previously in Table 10-19.

-- No toxicity value exceeded.

assumptions regarding exposure and toxicity. A complete understanding of the uncertainties associated with the risk estimates is critical to understanding the true nature of the predicted risks and to placing the predicted risks in proper perspective. The principal sources of uncertainty associated with the APG risk assessments were discussed in general in Chapter 4. Some of the key sources of uncertainty associated with the estimates of risk for the Nike site are summarized below.

10.5.1 UNCERTAINTIES RELATED TO SELECTION OF CHEMICALS FOR EVALUATION

Site-specific background data were not collected from any of the media sampled. Therefore, the site-relatedness of inorganic chemicals in these media was determined by comparing site concentrations with background data collected from various sources. Concentrations in groundwater and water from the missile silos were compared to national background groundwater levels. Concentrations in soil and sediment were compared to regional soil and sediment background levels. The degree to which these background data are representative is limited. As a result, chemicals that may not be site-related were selected for evaluation, even though historical information provides no indication that they were associated with past activities at the Nike site. Including chemicals in the risk assessment that are present at natural levels could result in overestimates of impact associated with the Nike site.

Information regarding laboratory, trip, and/or field blanks is available in very few instances. This is a particular concern because bis(2-ethylhexyl)phthalate, a common laboratory contaminant, contributed to risks from ingestion of groundwater at the launch area and was primarily responsible for risks at the control area. If bis(2-ethylhexyl)phthalate was present in some of the groundwater samples as a result of field or laboratory contamination, the risks associated with the ingestion of groundwater may have been greatly overestimated at the Nike site.

Sampling limitations associated with all media at the Nike site result in uncertainty regarding the true nature of contamination. They stem primarily from a general lack of data due to the lack of samples collected and/or the lack of analyses performed on samples that were collected. For example, only eight surface soil samples were collected from the launch area, and only two of these were analyzed for volatile organic chemicals, semivolatile organic chemicals, pesticides, and PCBs. Although groundwater is potentially discharging into surface water bodies near the site, only one surface water sample was collected and analyzed for volatile organic chemicals, and no sediment samples were collected from those surface water bodies. No samples were analyzed for agent and explosive-related compounds. Due to this lack of data, potential risks associated with these media and air were not evaluated in this assessment.

No tentatively identified compounds were included in the risk assessment because of the large degree of uncertainty regarding the identity and concentrations of these compounds. In addition, classes of chemicals such as hydrocarbons were not evaluated. Exclusion of these chemicals from the risk assessment probably has resulted in underestimates of risk. The magnitude to which risks are underestimated depends on the concentrations of the chemicals as well as their toxicity.

10.5.2 UNCERTAINTIES ASSOCIATED WITH THE ASSUMPTIONS USED TO ESTIMATE EXPOSURES

A large degree of uncertainty in this risk assessment is associated with the estimates of exposure point concentrations. As discussed previously, there are potential sources of groundwater contamination in the subsurface at the Nike site. Releases from septic systems or underground tanks could potentially result in increases in concentrations of chemicals of concern in the groundwater.

Assuming that the concentrations of chemicals detected to date will persist throughout the period of exposure may over- or underestimate the potential risks.

10.5.3 UNCERTAINTIES IN THE TOXICITY ASSESSMENT

Uncertainties in the toxicity assessment are primarily discussed in Chapter 4. For this assessment, of the chemicals of potential concern that were selected for quantitative evaluation to assess potential risks from ingestion of groundwater, several could not be evaluated due to lack of toxicity criteria. These were 2-ethyl-1-hexanol, gross alpha, and gross beta in groundwater beneath the launch area, and gross beta in groundwater beneath the control area. The overall effect of not evaluating these chemicals results in an underestimate of risk. However, given the conservative nature of the exposure parameters, the estimated risk from ingestion of groundwater is likely to be an overestimation.

There is uncertainty associated with the sediment toxicity values used in this assessment. The toxicity of chemicals in sediment may be affected by a variety of factors including pH, organic carbon content, and particle size of sediments. In general, factors that may affect a chemical's form or biological availability can influence toxicity. Thus, the potential toxicity of the chemicals in the sediments at the launch area may differ from the selected toxicity values depending upon differences in sediment and water quality. It is also important to note that the ditches are dry part of the year, so exposure durations would be limited for most aquatic organisms. The toxicity values used in this assessment are based on studies with wet sediments that have an overlying water column. These values are not appropriate for estimating sediment toxicity under dry conditions. In addition, sediment toxicity values are not available for all of the chemicals of potential concern. Thus, potential risks may be somewhat underestimated.

10.6 PRINCIPAL DATA NEEDS

Investigations to date have not provided a complete and exhaustive characterization of the type and degree of contamination at the Nike site. As a result, additional investigation is needed to assess more definitively existing or potential impacts associated with the Nike site. The additional data needed to better evaluate impacts at the Nike site are summarized below.

- For all of the environmental sampling recommended below, analyses of samples should be for inorganic chemicals, volatile organic chemicals, semivolatile organic chemicals, PCBs, pesticides, radionuclides, and for the range of military unique compounds potentially present at the Nike site as well as their degradation products.

- For each media sampled, background samples should also be collected for analysis. Because of the potential for widespread contamination at APG, it may not be possible to collect representative background samples from, or close to, the Nike site. However, an attempt should be made to characterize background concentrations as well as possible. For example, soil background samples should be of the same soil type and surface water and sediment background samples should be collected from similar tidal creek systems as those surrounding the Nike site. A sufficient number of samples should be collected to permit statistical evaluation.

- Additional surface and subsurface soil samples should be collected from the launch, control, and barracks areas as well as from the Nike site in general to fully characterize the extent of contamination. Subsurface soil samples should be collected from the vicinity of the sumps, drains, septic sewer lines, septic tanks, and sand filter beds associated with the septic systems, as well as in the vicinity of the underground storage tanks, and dump and landfill areas near the launch area.

- Sediment samples (and surface water where present) should be collected from the drainage ditch network on the Nike site. Additionally, sediment and surface water samples should be collected from Monks Creek, Lauderick Creek, and the Bush River in order to provide a more detailed evaluation of potential risks to environmental receptors. Samples should include locations where discharge is known to have occurred. These areas include the effluent outfall pipeline from the barracks area sand filter bed to Lauderick Creek, and the outfall from the launch area drainage ditches to Monks Creek.

- Additional groundwater wells should be installed on the Nike site to better define the hydrogeology beneath the site with regard to defining groundwater flow, contaminant migration, and groundwater surface water interaction.

10.7 SUMMARY AND CONCLUSIONS

This baseline risk assessment addressed potential impacts on human health and the environment associated with the Nike site in the absence of remedial actions. Environmental monitoring data collected by Army Environmental Hygiene Agency (AEHA 1990) between July 1986 and July 1989 was used in this assessment. With the exception of groundwater, very few samples were collected from different media, and different media were sampled at the launch and control areas of the Nike site. No samples were collected from the barracks area. Of the samples that were collected, chemical analyses were limited primarily to inorganic, volatile, and semivolatile organic chemicals. A few samples were analyzed for dissolved radionuclides, but no samples were analyzed for agent and explosive-related compounds.

10.7.1 HUMAN HEALTH RISK ASSESSMENT SUMMARY

Many of the potential exposure pathways by which human populations could be exposed to chemicals of potential concern under current land-use conditions at the Nike site were not evaluated due to their negligible potential for significant exposure. This was based primarily on the infrequent use of specific areas and the low concentrations of chemicals. Although they were not likely to be significant, several exposure pathways could not be evaluated due to lack of data. It was concluded that the primary pathways by which individuals could be exposed under current land-use conditions was through (1) ingestion, dermal contact, or inhalation of chemicals of potential concern in groundwater, and (2) acute dermal and/or inhalation exposure by site workers resulting from the disturbance of explosive or chemical-filled ordnance. Although groundwater is not currently used at the Nike site, its potential as a resource was evaluated, because it is used for domestic purposes off-post and potential hydraulic connection between on-site groundwater and off-post wells has not been evaluated. It was determined that there would be no new exposure pathways under future land-use conditions.

The estimated human health risks associated with the exposure pathways under current land-use conditions are as follows:

- The upper-bound excess lifetime cancer risks to individuals through ingestion of chemicals of potential concern in groundwater originating at the launch and control areas are $7x10^{-5}$ and $1x10^{-4}$, respectively. However, bis(2-ethylhexyl)phthalate, a common laboratory contaminant, contributes substantially to these risks. (Blank sample data were not available for much of the groundwater data). The Hazard Index is less than 1 for ingestion of contaminants in groundwater from the launch area, and equal to 1 for ingestion of contaminants in groundwater from the control area, primarily due to the presence of bis(2-ethylhexyl)phthalate. Additional exposures and risks from dermal contact and inhalation of chemicals of concern in groundwater are likely to be of the same order of magnitude as those estimated for groundwater ingestion.

- Site workers excavating the subsurface may potentially suffer acute dermal and/or inhalation exposures as a result of disturbing explosive or chemical-filled ordnance. Potential human risks include burns, injury, dismemberment, lungs, and death. If a release were to occur near the installation boundary, under some conditions, off-post individuals may also potentially be affected. However, off-post effects are likely to be much less extreme than on the Nike site.

10.7.2 ECOLOGICAL ASSESSMENT SUMMARY

Impacts to aquatic life from exposure to sediments in the ditches associated with the launch area were evaluated by comparing sediment concentrations with available toxicity values. These values were not exceeded, and therefore no risks to aquatic organisms exposed to sediments in these ditches are expected. However, there are uncertainties associated with the selected sediment toxicity values, and toxicity values are not available for all of the chemicals of potential concern.

The site and its associated water bodies may support a variety of terrestrial and aquatic receptors. However, the sampling data for this site are very limited, so potential risks to environmental receptors could not be adequately characterized in this assessment.

10.7.3 CONCLUSIONS OF THE RISK ASSESSMENT

Past activities at the Nike site have potentially resulted in significant contamination of some media at the site. However, of the limited chemical data that have been collected to characterize the site, many of the chemicals expected to be present based on past activities have not been analyzed for. Few human health exposure pathways are considered to be complete due primarily to the infrequent use of many areas of the site. The resource potential of groundwater beneath the launch and control areas of the Nike site was evaluated because it is currently being used for domestic purposes in a nearby residential area. The potential interconnection between groundwater beneath the Nike site and off-post domestic wells is unknown at this time. It was determined that ingestion of groundwater from the Nike site would result in potential carcinogenic risks in the launch and control areas in excess of 10^{-6}. Additionally, noncarcinogenic risks from ingestion of groundwater in the launch area are likely. An additional potential human health risk posed by the Nike site is acute hazards to site workers resulting from contact with explosive and chemical-filled ordnance during soil disturbing activities.

Based on the limited data available, no risks are expected to aquatic organisms from exposure to sediments in the launch area drainage ditches. Potential risks to terrestrial wildlife receptors could not be adequately evaluated in this assessment due to the very limited data available.

10.8 REFERENCES

ARMY ENVIRONMENTAL HYGIENE AGENCY (AEHA). 1985. Remedial Investigation Sampling Plan, Nike Missile Site, Aberdeen Proving Ground, Maryland. Waste Disposal Engineering Division. Aberdeen Proving Ground, Maryland.

ARMY ENVIRONMENTAL HYGIENE AGENCY (AEHA). 1989. RCRA Facility Assessment Report, Edgewood Area, Aberdeen Proving Ground, Maryland. Waste Disposal Division. Aberdeen Proving Ground, Maryland. Report No. 39-26-0490-90.

ARMY ENVIRONMENTAL HYGIENE AGENCY (AEHA). 1990. RCRA Facility Investigation, Nike Missile Battery Site. Aberdeen Proving Ground, Maryland. Geohydrologic Study No. 38-26-1353-90.

ARGONNE NATIONAL LABORATORY. 1990. Enhanced Preliminary Report: Former Nike Site, Aberdeen Proving Ground, Maryland. Environmental Assessment and Information Science Division. Argonne, Illinois.

BARRICK, R.C. and BELLER, H.R. 1989. Reliability of sediment quality assessment in Puget Sound. In: Oceans '89 Proceedings. Institute of Electrical and Electronic Engineers. Piscataway, New Jersey. Publication No. 89CH2780-5.

BOERNGEN, J.G. and SHACKLETTE, H.T. 1981. Chemical Analysis of Soils and Other Surficial Materials of the Conterminous United States. U.S. Department of the Interior, U.S. Geological Survey. Open-File Report 81-197.

DAMES and MOORE. 1990. Task Order No. 21: Sampling Design Plan for Remedial Investigation/Feasibility Study at Former Nike Site, Aberdeen Proving Ground, Maryland (Draft Report). Bethesda, Maryland.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Guidelines for Ground Water Classification Under the EPA Ground Water Protection Strategy. Office of Ground Water Protection. Washington. D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989a. Exposure Factors Handbook. Office of Health and Environmental Assessment. Washington, D.C. EPA/600/8-89/043.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989b. Risk Assessment Guidance for Superfund. Volume I: Human Health Evaluation Manual. Interim Final. Office of Emergency and Remedial Response. Washington, D.C. OSWER Directive 9285.7-01a.

NATIONAL OCEANIC AND ATMOSPHERIC ADMINISTRATION (NOAA). 1988. A Summary of Selected Data on Chemical Contaminants in Sediments Collected During 1984, 1985, 1986, and 1987. Rockville, Maryland. NOAA Technical Memorandum NOX OMA 44.

WALTON, W.C. 1985. Practical Aspects of Groundwater Modeling. National Water Well Association. Worthington, Ohio.

11.0 MICHAELSVILLE LANDFILL RISK ASSESSMENT

This chapter evaluates potential impacts on human health and the environment associated with the Michaelsville Landfill in the absence of remedial (corrective) actions. The Michaelsville Landfill Hydrogeologic Assessment, conducted from May 1987 to April 1990 by the U.S. Army Engineer Waterways Experiment Station (USAEWES 1990), is the primary source of sampling data considered in this risk assessment. This study was selected for use in this risk assessment because it was the most recent and comprehensive study conducted at the Michaelsville Landfill.

These and other investigations conducted to date have not completely characterized the nature and extent of contamination at the Michaelsville Landfill. Therefore, this risk assessment should be considered largely preliminary and is intended as an initial step in the overall risk assessment process for the Michaelsville Landfill.

This assessment follows the general methodology outlined in Chapter 4 of this report, which should be consulted for the rationale and further details of the methods used in this assessment. This assessment is organized into eight primary sections:

- Section 11.1 Background Information
- Section 11.2 Selection of Chemicals of Potential Concern
- Section 11.3 Human Health Risk Assessment
- Section 11.4 Ecological Assessment
- Section 11.5 Uncertainties
- Section 11.6 Principal Data Needs
- Section 11.7 Summary and Conclusions
- Section 11.8 References

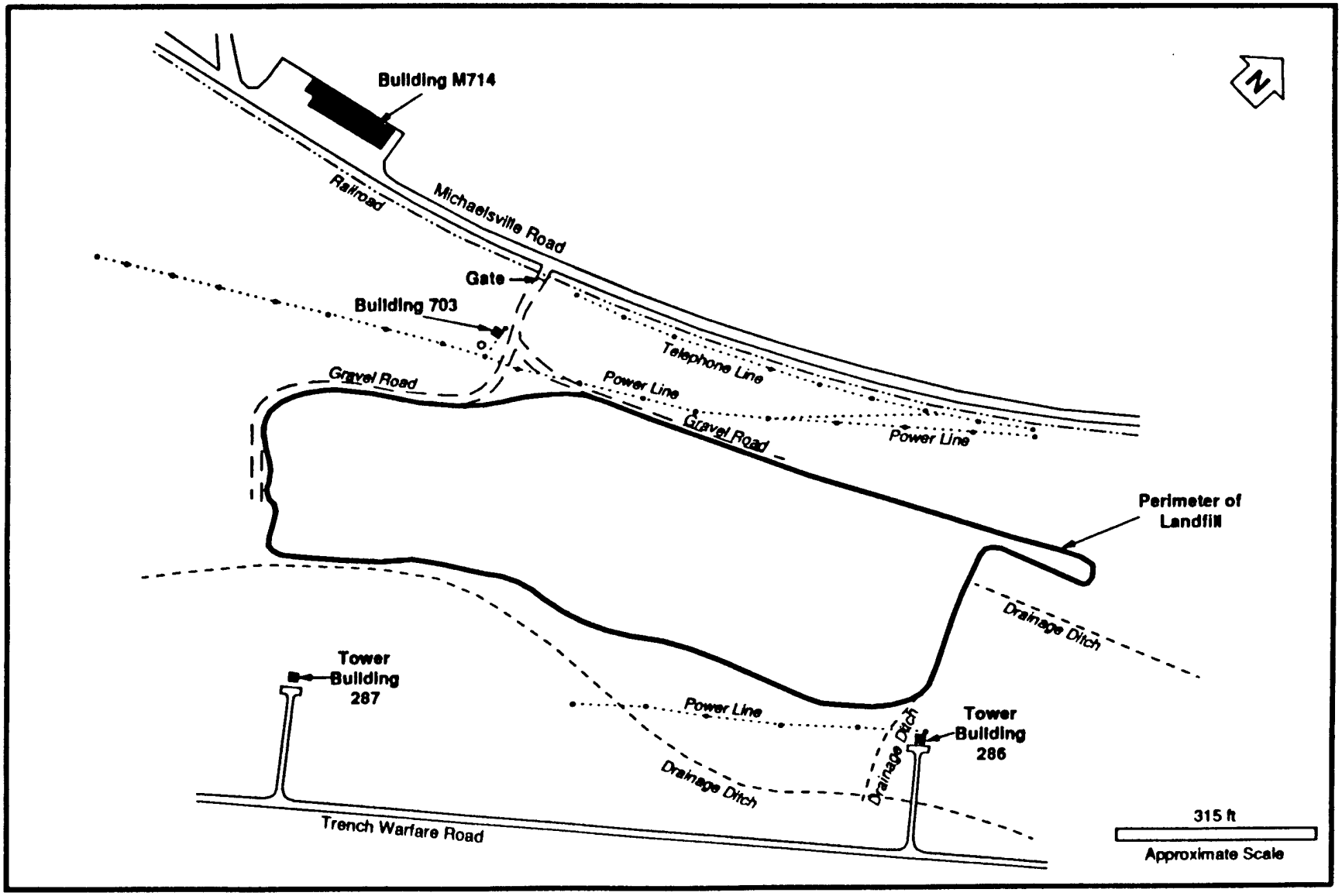
11.1 BACKGROUND INFORMATION

The Michaelsville Landfill, a closed landfill mainly used for domestic and nonindustrial wastes from the Aberdeen Area, is located in the north central portion of the Aberdeen Area of APG (see Figure 11-1). The landfill is situated between Michaelsville Road and Trench Warfare Road in a fenced, controlled area. It is approximately 20 acres in size. The northern portion is covered with grass, and the rest with grass, trees, and shrubs.[1] Many erosional rills and gullies cut the southern edge of the landfill, and several seeps are located around the perimeter. Two low-lying areas and a pond are located adjacent to the southwestern portion of the landfill. Isolated depressions occur in the areas covered with scrub brush and grass. A drainage ditch flows into the northeastern edge of the property and south along the landfill until it flows into the drainage ditch intercepting the seeps from the southern edge of the landfill. The landfill is located in the Romney Creek watershed.

Active landfilling operations were conducted from around 1969 through 1980 using the trench and fill method. The filled area is 7-16 feet above the original ground surface, and trenches were 10-16 feet deep and 40 feet wide (ESE 1981). Some of the trenches at the Michaelsville Landfill have been dug

[1]It should be noted that the Michaelsville Landfill Hydrogeologic Assessment (USAEWES 1990) appears to assume that north is site north as opposed to true north. Locations noted in this report are based on true north.

Figure 11-1
Michaelsville Landfill



into the uppermost aquifer, since this aquifer is found at a depth of 5-16 feet below the natural ground surface. The cover and cap of the landfill consists of 1.5-6 feet of compacted clay.

The majority of the waste materials disposed of in the Michaelsville Landfill are domestic and nonindustrial wastes from the Aberdeen Area. The following items have been reported as being disposed of in the landfill: waste water treatment plant sludge, tires, batteries, pesticide containers, rabbit droppings (1/2 dump truck two or three times a week during the months before closure), swimming pool paint (4-5 metric tons in 5-gallon cans), asbestos shingles, solvents, waste motor oils, grease, PCB transformer oils, and small amounts of excess chemical reagents. A list of the wastes believed to have been disposed of in the Michaelsville Landfill is presented in Table 11-1.

11.2 SELECTION OF CHEMICALS OF POTENTIAL CONCERN

In this section, environmental monitoring data collected by U.S. Army Engineer Waterways Experiment Station (USAEWES 1990) are briefly summarized, and chemicals of potential concern selected for further evaluation are described. Sampling data are available for soil, groundwater, seeps, and surface water. The discussions are organized below by environmental medium.

11.2.1 SOIL

In October 1989, two surface soil samples were taken from the top of the landfill. Since the landfill is covered (presumably with clean fill), these samples are unlikely to be representative of landfill contamination. Two other soil samples were taken approximately 700 feet east of the landfill to serve as "background" samples. Although the two background samples help to characterize levels of chemicals in nearby areas (out of the fill area), they are probably not representative of "natural" background since they were collected from sites located between the DRMO scrap metal yard and the landfill, an area that would not be expected to be unaffected by human activities. Soil samples were analyzed for volatile organic chemicals, semivolatile organic chemicals, pesticides, PCBs, and inorganic chemicals.

In this soil study, one method blank sample was collected as a QA/QC measure, but, as is typical with soil blanks, concentrations are in volume units (µg/L) making a direct comparison to soil concentrations in (mg/kg) inappropriate.[2] Therefore, these blank data were used only qualitatively. The following chemicals were detected in the soil method blank: diethylphthalate, methylene chloride, aldrin, heptachlor, cadmium, lead, and thallium. Phthalates and methylene chloride are common laboratory contaminants, but the pesticides aldrin and heptachlor are not normally detected in blanks. This raises some question as to the extent or actual presence of these chemicals in site samples.

Chemicals detected in Michaelsville Landfill surface soil are shown in Table 11-2. Organic chemicals detected were acetone, methylene chloride, and several pesticides. With the exception of acetone, all organic chemicals detected in these samples were detected in the background samples at similar levels, which may mean that their presence in cover soils is indicative of general area contamination, which may or may not be related to landfill operations. Methylene chloride, which was also present in the soil blank, is a common laboratory contaminant, and therefore may not actually be present in the landfill cover soils. Acetone, also a common laboratory contaminant, was not present in the soil

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[2] This comparison could be made if details of the sampling and analytical protocols were known.

TABLE 11-1

PRINCIPAL WASTES DISPOSED OF AT
MICHAELSVILLE LANDFILL (a)

Domestic and non-industrial waste
Waste water treatment plant sludge
Tires
Batteries
Pesticide containers
Rabbit droppings
Paint
Asbestos shingles
Solvents
Waste motor oil
Grease
PCB transformer oils
Excess chemical reagents

(a) Information obtained primarily from USAEWES (1990).

Table 11-2

SUMMARY OF CHEMICALS DETECTED IN
SURFACE SOIL AT MICHAELSVILLE LANDFILL (a)

(Concentrations reported in ug/kg for organics, and in mg/kg for inorganics)

Chemical (b)	Frequency of Detection (c)	Range of Detected Concentrations	Range of Background Concentrations (d)
Organic Chemicals:			
* Acetone (ACET)	1 / 2	41,000	ND
* DDT (total)	1 / 1	14	6.00 - 117
* 4,4'-DDD (PPDDD)	1 / 1	7.00	1.00 - 10.0
* 4,4'-DDE (PPDDE)	1 / 1	3.00	5.00 - 44.0
* 4,4'-DDT (PPDDT)	1 / 1	4.00	63.0
* Endosulfan Sulfate (ESFSO4)	1 / 1	6.00	1.00 - 2.00
* Endrin Aldehyde (ENDRNA)	1 / 1	13.0	1.00
* Heptachlor (HPCL)	2 / 2	1.00 - 11.0	1.00 - 2.00
* Heptachlor Epoxide (HPCLE)	1 / 1	2.00	1.00
* Methylene Chloride (CH2CL2)	2 / 2	170 - 810	1,000 - 1,400
Inorganic Chemicals:			
Cadmium (CD)	2 / 2	0.200 - 0.500	0.300 - 0.900
* Chromium (CR)	2 / 2	18.0 - 19.7	8.40 - 16.0
* Copper (CU)	2 / 2	8.40 - 12.5	6.40 - 8.50
Lead (PB)	2 / 2	15.0 - 25.2	25.9 - 31.1
Nickel (NI)	2 / 2	10.3 - 11.6	5.00 - 11.7
Thallium (TL)	2 / 2	0.100 - 0.300	0.200 - 0.300
* Zinc (ZN)	2 / 2	41.8 - 44.4	28.8 - 41.1

(a) Samples: MVSOIL-1 and MVSOIL-2.
(b) USATHAMA chemical codes listed in parentheses.
(c) The number of samples in which a chemical was detected divided by the total number of samples analyzed for that chemical.
(d) Samples: MVBG-1 and MVBG-2.

* = Selected as a chemical of potential concern. See text.
ND = Not detected.

blanks. Based on the difficulties associated with determining the site-relatedness of these organic chemicals, all of the organic chemicals detected in soils, acetone, DDT (total), endosulfan sulfate, endrin aldehyde, heptachlor, heptachlor epoxide, and methylene chloride, were selected as chemicals of potential concern for surface soil at the Michaelsville Landfill. Of the inorganic chemicals detected in landfill soils, the maximum concentrations of chromium, copper, and zinc were present at levels above, although only slightly above, the maximum concentration detected in background samples. Therefore these chemicals were selected as chemicals of potential concern.

11.2.2 GROUNDWATER

The base of the aquifers beneath the Michaelsville Landfill is defined by a consistent, hard, waxy clay aquiclude layer that was found at elevations of 130 feet in the northern part of the site and 100 feet in the southern part of the site. Overlying the clay layer is an approximately 10-foot-thick layer of interlaminated brown, organic clays, silts, and fine-grained sands. Overlying the interlaminated layer is the lower sand aquifer, an approximately 30-foot-thick fine-grained carbonaceous sand layer. Overlying the sand layer are 50-65 feet of interbedded clays, silts, and sands that act as an aquitard. In the eastern portion of the site, there are two sand layers within the interbedded clays, silts, and sands, which are possible minor aquifers. Overlying the interbedded clays, silts, and sands are 20-30 feet of depositional layers of gravel and sand with clay lenses in some areas. This gravel and sand layer is considered the uppermost aquifer and varies from a water table aquifer to a confined aquifer. A silty clay layer ranging in thickness from 5 to 16 feet is consistently found over the surface of the Michaelsville Landfill site.

The flow of groundwater in the uppermost aquifer is generally to the south and east, although in the April 1989 and October 1989 sampling rounds, flows were northeast and north-northeast, respectively. Groundwater flow in the lower sand aquifer, based on the five deep USAEWES wells, is south and west. Regional groundwater flow is reportedly southeast towards the Chesapeake Bay.

Thirty-three groundwater wells have been installed around the Michaelsville Landfill. In 1980, eight monitoring wells were installed in the uppermost (shallow) aquifer by the Baltimore District (NAB) of the North Atlantic Division of the Corps of Engineers. In 1988, 25 monitoring wells were installed at the landfill by USAEWES; 20 of these wells are screened in the upper gravel and sand aquifer, the uppermost aquifer, 3 are screened in the underlying laminated zone, 1 is screened between the laminated zone and the lower sand aquifer, and 1 is screened in the lower sand aquifer. These latter five wells are considered to be deep wells. The eight NAB wells were sampled in January 1988 and September 1988, 24 of 25 USAEWES wells were sampled in September 1988, 22 of 25 USAEWES wells were sampled in December 1989, and all 25 USAEWES wells were sampled in April 1989. Intermediate well WES-M-15 was sampled on June 2, 1988 and analyzed for explosives.

Groundwater samples were analyzed for volatile organic chemicals, semivolatile organic chemicals, pesticides, PCBs, and dissolved inorganic chemicals. As noted above, one groundwater sample from an intermediate well was also analyzed for explosives. Blank data were available for all sampling periods except January 1988. Three equipment blanks were analyzed for the September 1988 data, three equipment blanks and two method blanks were analyzed for the December 1988 data, and two equipment blanks and three method blanks were analyzed for the April 1989 data. A large number of chemicals were detected in groundwater sampling blanks including several phthalates, many pesticides, PCBs (Aroclor-1254), ammonia nitrogen, as well as several inorganics. Phthalates and methylene chloride are common laboratory contaminants, but pesticides and PCBs are not. This raises a question as to the validity of the Michaelsville Landfill groundwater data.

The chemicals detected in shallow and deep groundwater wells at the Michaelsville Landfill are shown in Tables 11-3 and 11-4, respectively. Thirty organic chemicals were detected in shallow groundwater and were therefore selected as chemicals of potential concern. About half of these chemicals were, however, detected in fewer than 10% of the samples and at low concentrations. The predominant organic groups present in this groundwater were pesticides, phthalate esters, and chlorinated aliphatics. Methylene chloride was the most frequently detected chemical. PCBs were also detected relatively frequently but at very low levels (less than 1 µg/L). Deep groundwater showed fewer organic chemicals, but a similar array at generally lower concentrations. Acetone was an exception since it was present at much higher concentrations in deep groundwater; it was detected in 1 of 28 samples in shallow groundwater at a concentration of 70 µg/L and in 2 of 5 samples at a maximum concentration of 2,310 µg/L in deep groundwater.

Several inorganic chemicals were identified as being potentially elevated above background levels in both shallow and deep groundwater and therefore were selected as chemicals of potential concern as shown in Tables 11-3 and 11-4. However, no site-specific or regional background data were available for groundwater in this area with which to compare site levels. The use of national data, as was done in this case, introduces considerable uncertainty into this determination. Beryllium, iron, manganese, thallium, and zinc in shallow groundwater and beryllium and iron in deep groundwater were selected as chemicals of potential concern based on this comparison. Ammonia nitrogen was selected as a chemical of potential concern in shallow and deep groundwater by default in the absence of background data for this chemical.

With respect to spatial distribution of groundwater contamination, in general, the highest groundwater concentrations are south and east of Michaelsville Landfill (i.e., generally downgradient of the landfill).

11.2.3 SEEPS

Multiple erosional rills and gullies cut the southern edge of the landfill and several seeps are located around the perimeter of the landfill. Flow from the seeps is intermittent depending on rainfall. Seeps in the southern portion of the landfill drain into a nearby drainage ditch (discussed below in Section 11.2.4). Ten samples were collected from seeps: May 1988 (1 sample), September 1988 (1 sample), April 1989 (4 samples), and October 1989 (4 samples). Data from seep samples were grouped together for this risk assessment.

Seep samples were analyzed for volatile organic chemicals, semivolatile organic chemicals, pesticides, PCBs, and inorganic chemicals. Blank data were only available for the October 1989 sampling round. The following chemicals were detected in the October 1989 method blank: butylbenzylphthalate, di-n-octylphthalate, endrin aldehyde, bis(2-ethylhexyl)phthalate, heptachlor, methylene chloride, and PCBs (Aroclor-1254). Again, as was the case with groundwater, the question of validity of this seep data is raised due to the contamination of blank samples with chemicals (e.g., pesticides and PCBs) that are not common blank contaminants.

The chemicals detected in seeps from the Michaelsville Landfill are shown in Table 11-5. A relatively large number of organic chemicals were detected in seep water, although generally infrequently. These chemicals include volatiles such as acetone, methylene chloride, and vinyl chloride, as well as phthalates, pesticides, and PCBs. Although several inorganic chemicals were identified as being potentially elevated above background levels and were therefore selected as chemicals of potential concern, no appropriate background data were available with which to compare site seep levels. In lieu of more appropriate data to characterize levels of inorganics seeping out of natural soils in the

TABLE 11-3

SUMMARY OF CHEMICALS DETECTED IN SHALLOW GROUNDWATER AT MICHAELSVILLE LANDFILL (a)

(Concentrations reported in ug/L)

Chemical (b)	Frequency of Detection (c)	Range of Detected Concentrations (d)	Background Concentration (e)
Organic Chemicals			
• Acetone (ACET)	1 / 28	70 0	NA
• Aldrin (ALDRN)	2 / 28	0 0100	NA
• Benzene (C6H6)	1 / 28	3 40	NA
• alpha-BHC (ABHC)	2 / 28	0 0100	NA
• beta-BHC (BBHC)	1 / 28	0 0100	NA
• delta-BHC (DBHC)	1 / 28	0 0200	NA
• Butylbenzylphthalate (BBZP)	20 / 28	4 00 - 17 9	NA
• Chloroethane (C2H5CL)	3 / 28	11 0 - 13 0	NA
• Chloroform (CHCL3)	1 / 28	4 4	NA
• DDT [Total]	12 / 28	0 0100 - 0 0700	NA
* 4,4'-DDD (PPDDD)	1 / 28	0 0300	NA
* 4,4'-DDT (PPDDT)	12 / 28	0 0100 - 0 0600	NA
• Dibutylphthalate (DNPH)	20 / 28	3 10 - 28 2	NA
• 1,1-Dichloroethane (11DCL)	2 / 28	3 10 - 24 0	NA
• 1,2-Dichloroethane (12DCLE)	2 / 28	3 40 - 4 30	NA
• cis-1,2-Dichloroethene (C12DCE)	6 / 28	2 50 - 22	NA
• trans-1,2-Dichloroethene (T12DCE)	1 / 28	5 10	NA
• Dieldrin (DLDRN)	6 / 28	0 0100 - 0 0500	NA
• Diethylphthalate (DEP)	12 / 28	2 90 - 18 7	NA
• 2,4-Dimethylphenol (24DMPN)	1 / 27	8 00	NA
• 1,3-Dinitrobenzene (13DNB) (f)	1 / 1	4 0	NA
• Di-n-octylphthalate (DNOP)	20 / 28	4 10 - 12 4	NA
• 1,2-Diphenylhydrazine (12DPH)	1 / 28	1 30	NA
• Endosulfan I (AENSLF)	8 / 28	0 0100 - 0 0700	NA
• Endosulfan II (BENSLF)	2 / 28	0 0100	NA
• Endosulfan Sulfate (ESFSO4)	2 / 28	0 0100	NA
• bis(2-Ethylhexyl)phthalate (B2EHP)	28 / 28	5 60 - 255	NA
• Heptachlor (HPCL)	19 / 28	0 0100 - 0 0500	NA
• Heptachlor Epoxide (HPCLE)	7 / 28	0 0100 - 0 0600	NA
• Methylene Chloride (CH2CL2)	21 / 28	2 50 - 503	NA
• PCBs (g)	13 / 28	0 170 - 0 700	NA
Inorganic Chemicals			
• Ammonia Nitrogen (NH3N2)	28 / 28	179 - 66,700	NA
Antimony (SB)	24 / 28	3 80 - 33 0	100
Arsenic (AS)	7 / 28	4 00 - 31 5	100
• Beryllium (BE)	3 / 28	3 70 - 4 30	1 0
Cadmium (CD)	27 / 28	0 0800 - 1 75	100
Calcium (CA)	8 / 8	2 160 - 17 800	1 000 000
Chloride (CL)	28 / 28	4 910 - 439,000	1,000 000
Chromium (CR)	21 / 28	0 700 - 20	100
Copper (CU)	26 / 28	0 800 - 67 7	100
• Iron (FE)	27 / 27	34 0 - 39 600	10 000
Lead (PB)	24 / 28	0 800 - 9 0	100
Magnesium (MG)	8 / 8	862 - 60 000	1 000 000
• Manganese (MN)	8 / 8	18 0 - 2 470	100
Nickel (NI)	28 / 28	4 00 - 62 7	100
Nitrate (NO3)	28 / 28	36 5 - 447	10,000
Orthophosphate (PO4ORT)	28 / 28	37 7 - 2,220	NA
Potassium (K)	8 / 8	759 - 25,900	10 000
Selenium (SE)	16 / 28	15 3 - 22 0	100
Sodium (NA)	8 / 8	2 250 - 132,000	1,000 000
Sulfate (SO4)	28 / 28	5 950 - 44,000	1,000 000
• Thallium (TL)	13 / 28	1 30 - 11 7	1 0
• Zinc (ZN)	28 / 28	10 7 - 176	100

(a) Samples MW01-MW07, MW16 WES-M-01 WES-M-02 WES-M-03, WES-M-05, WES-M-06, WES-M-08 WES-M-09 WES-M-10 WES-M-11, WES-M-13, WES-M-14 WES-M-15 WES-M-17, WES-M-18, WES-M-19, WES-M-21, WES-M-22, WES-M-23 WES-M-24 and WES-M-25

(b) USATHAMA chemical codes listed in parentheses

(c) The number of samples in which a chemical was detected divided by the total number of samples analyzed for that chemical

(d) Values reported are total concentrations, except for metals for which dissolved concentrations are given

(e) Background concentrations from Walton (1985) Values reported are dissolved concentrations

(f) Well WES-M-15 was analyzed for explosives on June 2, 1988

(g) Aroclor-1254 (PCB254)

* = Selected as a chemical of potential concern See text

NA = Not available

TABLE 11-4

SUMMARY OF CHEMICALS DETECTED IN DEEP
GROUNDWATER AT MICHAELSVILLE LANDFILL (a)

(Concentrations reported in ug/L)

Chemical (b)	Frequency of Detection (c)	Range of Detected Concentrations (d)	Background Concentrations (e)
Organic Chemicals:			
* Acetone (ACET)	2 / 5	463 - 2,310	NA
* delta-BHC (DBHC)	1 / 5	0.0100	NA
* Butylbenzylphthalate (BBZP)	5 / 5	4.70 - 8.50	NA
* DDT [Total]	3 / 5	0.0500 - 0.0700	NA
* 4,4'-DDD (PPDDD)	1 / 5	0.0200	NA
* 4,4'-DDT (PPDDT)	3 / 5	0.0500 - 0.0700	NA
* Dibutylphthalate (DNPH)	5 / 5	5.80 - 19.4	NA
* Dieldrin (DLDRN)	2 / 5	0.0100	NA
* Diethylphthalate (DEP)	3 / 5	3.80 - 10.7	NA
* Di-n-octylphthalate (DNOP)	5 / 5	4.50 - 8.20	NA
* Endosulfan I (AENSLF)	2 / 5	0.0100	NA
* bis(2-Ethylhexyl)phthalate (B2EHP)	5 / 5	18.7 - 70.8	NA
* Heptachlor (HPCL)	4 / 5	0.0100 - 0.0400	NA
* Heptachlor Epoxide (HPCLE)	2 / 5	0.0200	NA
* Methylene Chloride (CH2CL2)	4 / 5	3.50 - 28.3	NA
* 4-Methylphenol (4MP)	2 / 5	1.60 - 4.90	NA
* PCBs (f)	2 / 5	0.170 - 0.270	NA
* Phenol (PHENOL)	1 / 5	12.7	NA
Inorganic Chemicals			
* Ammonia Nitrogen (NH3N2)	5 / 5	2,040 - 3,830	NA
Antimony (SB)	5 / 5	14.5 - 20.5	100
Arsenic (AS)	3 / 5	5.30 - 11.3	100
* Beryllium (BE)	1 / 5	3.30	1.0
Cadmium (CD)	5 / 5	0.200 - 0.600	100
Chloride (CL)	5 / 5	2,570 - 4,400	1,000,000
Chromium (CR)	3 / 5	0.670 - 10.0	100
Copper (CU)	5 / 5	25.3 - 52.3	100
* Iron (FE)	5 / 5	162 - 11,700	10,000
Lead (PB)	5 / 5	1.00 - 9.20	100
Nickel (NI)	5 / 5	5.70 - 18.7	100
Nitrate (NO3)	5 / 5	115 - 399	10,000
Orthophosphate (PO4ORT)	5 / 5	1,740 - 2,730	NA
Selenium (SE)	5 / 5	17.3 - 20.0	100
Sulfate (SO4)	5 / 5	7,670 - 11,200	1,000,000
Zinc (ZN)	4 / 5	11.0 - 40.0	100

(a) Samples WES-M-04, WES-M-07, WES-M-12, WES-M-16, and WES-M-20.

(b) USATHAMA chemical codes listed in parentheses

(c) The number of samples in which the a chemical was detected divided by the total number of samples analyzed for that chemical.

(d) Values reported are total concentrations, except for metals, for which dissolved concentrations are given.

(e) Background concentrations from Walton (1985). Values reported are dissolved concentrations

(f) Arocior-1254 (PCB254)

* = Selected as a chemical of potential concern See text

NA = Not available

TABLE 11-5

SUMMARY OF CHEMICALS DETECTED IN
SEEPS AT MICHAELSVILLE LANDFILL (a)

(Concentrations reported in ug/L)

Chemical (b)	Frequency of Detection (c)	Range of Detected Concentrations (d)			Background Concentration (e)
Organic Chemicals:					
* Acetone (ACET)	1 / 9		13		NA
* Aldrin (ALDRN)	1 / 10		0.03		NA
* Butylbenzylphthalate (BBZP)	2 / 7	8.50	-	10.0	NA
* 4-Chloroaniline (4CANIL)	1 / 8		1.5		NA
* DDT [Total]	2 / 10	0.0500	-	0.0900	NA
* 4,4'-DDD (PPDDD)	2 / 10		0.05		NA
* 4,4'-DDE (PPDDE)	1 / 10		0.04		NA
* Dibutylphthalate (DNPH)	4 / 10	29.2	-	56.8	NA
* cis-1,2-Dichloroethene (C12DCE)	1 / 9		620		NA
* Dieldrin (DLDRN)	2 / 10	0.0200	-	0.0400	NA
* Diethylphthalate (DEP)	2 / 10	1.80	-	4.50	NA
* Di-n-octylphthalate (DNOP)	3 / 7	4.40	-	9.80	NA
* 1,2-Diphenylhydrazine (12DPH)	1 / 10		7.4		NA
* Ethyl Benzene (ETC6H5)	2 / 10	9.50	-	21.0	NA
* bis(2-Ethylhexyl)phthalate (B2EHP)	5 / 6	2.20	-	28.9	NA
* 3-Methyl-4-chlorophenol (4CL3C)	1 / 10		6.00		NA
* Methylene Chloride (CH2CL2)	4 / 6	7.70	-	9.60	NA
* 4-Methylphenol (4MP)	3 / 8	44.8	-	147	NA
* Methoxychlor (MEXCLR)	1 / 8		0.04		NA
* PAHs [noncarcinogenic] [Total]	1 / 10		2.6		NA
Naphthalene (NAP)	1 / 10		2.6		NA
* PCBs (f)	3 / 10	0.200	-	0.500	NA
* Phenol (PHENOL)	1 / 10		11.1		NA
* Tetrachloroethene (TCLEE)	1 / 10		5.8		NA
* Toluene (MEC6H5)	1 / 10		108		NA
* Vinyl Chloride (C2H3CL)	1 / 10		53		NA
* Xylenes [Total] (TXYLEN)	2 / 9	48.0	-	72.0	NA
Inorganic Chemicals:					
* Ammonia Nitrogen (NH3N2)	9 / 10	150	-	14,900	NA
* Antimony (SB)	3 / 10	130	-	210	100
Arsenic (AS)	1 / 10	2			100
Cadmium (CD)	7 / 10	0.300	-	5.20	100
Calcium (CA)	1 / 1		67,300		1,000,000
Chloride (CL)	9 / 9	10,600	-	50,600	1,000,000
Chromium (CR)	7 / 10	3.00	-	57.0	100
Copper (CU)	9 / 10	4.00	-	64.0	100
* Iron (FE)	2 / 2	130,000	-	198,000	10,000
* Lead (PB)	7 / 10	1.00	-	164	100
Magnesium (MG)	1 / 1		19,700		1,000,000
* Manganese (MN)	1 / 1		1,900		100
* Nickel (NI)	7 / 10	22.0	-	195	100
Nitrate (NO3)	10 / 10	14.0	-	389	10,000
Orthophosphate (PO4ORT)	10 / 10	33.0	-	1,300	NA
Potassium (K)	1 / 1		10,200		10,000
Sodium (NA)	1 / 1		12,200		1,000,000
Sulfate (SO4)	8 / 9	6,100	-	63,100	1,000,000
* Thallium (TL)	4 / 10	4.00	-	27.0	1.0
* Zinc (ZN)	8 / 10	45.0	-	1,180	100

(a) Samples: APG-S1, SS8-9, SEEP-1 - SEEP-4, and SEEP-A - SEEP-D.
(b) USATHAMA chemicals codes listed in parentheses.
(c) The number of samples in which a chemical was detected divided by the total number of samples analyzed for that chemical.
(d) Total concentrations reported.
(e) Background concentrations from Walton (1985). Values reported are dissolved concentrations. concentrations.
(f) Aroclor-1221 (PCB221).

* = Selected as a chemical of potential concern. See text.
NA = Not available.

area, national groundwater data were used. This however introduces considerable uncertainty into this determination. In addition, background groundwater concentrations are dissolved concentrations, whereas seep concentrations are total concentrations. Seven inorganic chemicals were selected as chemicals of potential concern based on this comparison, including antimony, iron, lead, manganese, nickel, thallium, and zinc. Ammonia nitrogen was selected as a chemical of potential concern by default in the absence of background data.

11.2.4 SURFACE WATER

A drainage ditch, which receives runoff from the DRMO scrap metal yard area, flows into the northeastern edge of the landfill property and then south adjacent to the landfill. Two low-lying areas and a pond are located adjacent to the southwestern portion of the landfill. One surface water sample was collected from each of the following locations: upgradient approximately 500 feet east of the site in the drainage ditch that flows south of the landfill, downgradient near the southwestern corner of the landfill in the same drainage ditch, and the small pond near the southwestern corner of the landfill. With the exception of the upgradient sample, data from this surface water sampling were grouped together for the purposes of this assessment.

Surface water samples were analyzed for volatile organic chemicals, semivolatile organic chemicals, pesticides, PCBs, and inorganic chemicals. No associated blank samples were collected.

The chemicals detected in surface water are shown in Table 11-6. Low levels of pesticides (all BHC isomers) as well as bis(2-ethylhexyl)phthalate (a common laboratory contaminant) were detected in site samples. None of these chemicals were detected in the upgradient sample (although the detection limits were probably very close to the detected values on-site) except beta-BHC, which was detected at a higher, but still low, concentration in the upgradient sample than in the site sample. All organic chemicals detected in surface water were selected as chemicals of potential concern, although, based on the above discussion, there is some question as to their association with landfill activities. A comparison of downstream surface water concentrations of inorganics with those detected in the upstream sample showed that iron, lead, and nitrate exceeded upstream concentrations by a factor of two. These chemicals were therefore selected as chemicals of potential concern. In addition, antimony, selenium, and silver were selected as chemicals of potential concern by default since they were not detected in the background sample.

11.2.5 AIR

Ambient air monitoring surveys were conducted in the area of the landfill in April 1989 using an OVA and in March 1990 using an HNU photoionization detector. USAEWES reported that "no gases" were detected in either survey. It should be noted that this kind of air data is only useful for a qualitative assessment. Therefore, no chemicals of potential concern were selected for air.

During the hydrogeologic assessment, the headspace of each monitoring well was monitored for methane and volatile organic gases prior to sampling. The highest volatile organic headspace reading was 2 ppm, and the highest methane headspace reading was 45%. Methane levels of 90-5,971 ppm were found in the headspace of the five deep wells sampled in July 1988. Methane also has been detected at a maximum concentration of 51.8% in the 12 methane gas monitoring wells installed in 1989. The highest methane concentrations have been found north and northwest of the landfill. Methane is a nontoxic gas generated by the decay of organic matter. At waste sites methane is of

TABLE 11-6

SUMMARY OF CHEMICALS DETECTED IN
SURFACE WATER AT MICHAELSVILLE LANDFILL

(Concentrations reported in ug/L)

Chemical (a)	Frequency of Detection (b)	Range of Detected Concentrations (c)	
		On-Site (d)	Background (e)
Organic Chemicals:			
* alpha-BHC (ABHC)	1 / 2	0.0100	ND
* beta-BHC (BBHC)	1 / 2	0.0100	0.410
* delta-BHC (DBHC)	1 / 2	0.0300	ND
* gamma-BHC (LIN)	1 / 2	0.0100	ND
* bis(2-Ethylhexyl)phthalate (B2EHP)	1 / 2	33.0	ND
Inorganic Chemicals:			
Ammonia Nitrogen (NH3N2)	2 / 2	284 - 391	296
* Antimony (SB)	1 / 2	1.00	ND
Arsenic (AS)	2 / 2	2.00 - 3.00	2.00
Calcium (CA)	2 / 2	8,920 - 8,960	9,050
Chromium (CR)	2 / 2	5.00	3.00
* Iron (FE)	2 / 2	1,330 - 3,220	735
* Lead (PB)	2 / 2	2.00 - 3.00	1.00
Magnesium (MG)	2 / 2	3,550 - 3,630	3,310
Manganese (MN)	2 / 2	259 - 389	723
Nickel (NI)	2 / 2	6.00 - 9.00	7.00
* Nitrate (NO3)	2 / 2	76.0 - 103	34.0
Orthophosphate (PO4ORT)	2 / 2	22.0 - 292	508
Potassium (K)	2 / 2	1,910 - 8,000	2,070
* Selenium (SE)	2 / 2	19.0 - 28.0	ND
* Silver (AG)	1 / 2	21.0	ND
Sodium (NA)	2 / 2	7,420 - 7,470	7,480

(a) USATHAMA chemicals codes listed in parentheses.
(b) The number of samples in which a chemical was detected divided by the total number of samples analyzed for that chemical.
(c) Total concentrations reported.
(d) Samples: APG-DS and APG-POND.
(e) Sample: APG-UP.

* = Selected as a chemical of potential concern. See text.
ND = Not detected.

concern if there are nearby buildings to which it can migrate in the subsurface (due to its explosion potential) and for its propensity to facilitate the transport of more toxic volatile chemicals through soils to the ambient air.

11.2.6 SUMMARY OF CHEMICALS OF POTENTIAL CONCERN

Table 11-7 summarizes the chemicals of potential concern selected for each media sampled at the Michaelsville Landfill. These chemicals include an array of volatile and semivolatile organic chemicals, many pesticides, PCBs, and one explosive (1,3-dinitrobenzene) as well as inorganic chemicals. As noted above, the quality of the hydrogeologic assessment data is somewhat questionable since blank samples contained many chemicals that are not common sampling or analytical artifacts (e.g., pesticides and PCBs).

Surface soil and surface water contained the smallest number of chemicals of potential concern. However, the two surface soil samples collected at the Michaelsville Landfill would not be expected to be highly contaminated since they were taken from the soils used to cover the landfill when it was closed. In general, the highest groundwater concentrations were south and east of the Michaelsville Landfill. Concentrations of organic chemicals in the shallow wells were generally higher with the exception of acetone, which was present at 2,310 µg/L in a deep well (compared to 70 µg/L in the shallow wells). Concentrations of ammonia nitrogen and chloride were significantly higher in the shallow wells than in the deep wells. Seeps show similar contaminants as groundwater. Although seeps on the southern portion of the landfill drain into the drainage ditch, the available data suggest that there is little impact on ditch waters; virtually no organic chemicals were detected in surface water samples. However, the ditch sediments, which were not sampled, may be impacted by this discharge.

In addition to the chemicals of potential concern selected for each medium using the available sampling data, other chemicals are possibly present in environmental media at the Michaelsville Landfill and may be of concern regarding potential exposures and risks. Detailed records were not kept of the materials disposed of in the Michaelsville Landfill; therefore, other chemicals that could also contribute to exposures and risks could be present. In addition, explosives were analyzed for only in one shallow groundwater sample in June 1988. 1,3-Dinitrobenzene was detected in this sample.

11.3 HUMAN HEALTH RISK ASSESSMENT

This section addresses the potential human health risks associated with the Michaelsville Landfill in the absence of remedial actions. This human health risk assessment is divided into three principal sections. Section 11.3.1 evaluates and provides estimates of potential human exposures for the chemicals of potential concern at the Michaelsville Landfill. Section 11.3.2 summarizes relevant toxicity information for the chemicals of potential concern. Section 11.3.3 provides quantitative and qualitative estimates of human health risks.

11.3.1 EXPOSURE ASSESSMENT

This section identifies the pathways by which human populations may be exposed to chemicals of potential concern at or originating from the Michaelsville Landfill and selects pathways for further evaluation. Only complete pathways are selected for further evaluation (see Chapter 4 for the definition of a complete exposure pathway). Evaluations of exposures may be quantitative or

TABLE 11-7

SUMMARY OF CHEMICALS OF POTENTIAL CONCERN FOR MICHAELSVILLE LANDFILL

Chemical (a)	Surface Soil	Shallow Groundwater	Deep Groundwater	Seeps	Surface Water
Organic Chemicals:					
Acetone (ACET)	X	X	X	X	
Aldrin (ALDRN)		X		X	
Benzene (C6H6)		X			
alpha-BHC (ABHC)		X			X
beta-BHC (BBHC)		X			X
delta-BHC (DBHC)		X	X		X
gamma-BHC (LIN)					X
Butylbenzylphthalate (BBZP)		X	X	X	
4-Chloroaniline (4CANIL)				X	
Chloroethane (C2H5CL)		X			
Chloroform (CHCL3)		X			
DDT (total)	X	X	X	X	
4,4'-DDD (PPDDD)	X	X	X	X	
4,4'-DDE (PPDDE)	X			X	
4,4'-DDT (PPDDT)	X	X	X		
Dibutylphthalate (DNPH)		X	X	X	
1,1-Dichloroethane (11DCL)		X			
1,2-Dichloroethane (12DCLE)		X			
cis-1,2-Dichloroethene (C12DCE)		X		X	
trans-1,2-Dichloroethene (T12DCE)		X			
Dieldrin (DLDRN)		X	X	X	
Diethylphthalate (DEP)		X	X	X	
2,4-Dimethylphenol (24DMPN)		X			
1,3-Dinitrobenzene (13DNB)		X			
Di-n-octylphthalate (DNOP)		X	X	X	
1,2-Diphenylhydrazine (12DPH)		X		X	
Endosulfan I (AENSLF)		X	X		
Endosulfan II (BENSLF)		X			
Endosulfan Sulfate (ESFSO4)	X	X			
Endrin Aldehyde (ENDRNA)	X				
Ethyl Benzene (ETC6H5)				X	
bis(2-Ethylhexyl)phthalate (B2EHP)		X	X	X	X
Heptachlor (HPCL)	X	X	X		
Heptachlor Epoxide (HPCLE)	X	X	X		
3-Methyl-4-chlorophenol (4CL3C)				X	
Methylene Chloride (CH2CL2)	X	X	X	X	
4-Methylphenol			X	X	
Methoxychlor (MEXCLR)				X	
PAHs [noncarcinogenic] [Total]				X	
Naphthalene (NAP)				X	
PCBs		X	X	X	
Aroclor-1221 (PCB221)				X	
Aroclor-1254 (PCB254)		X	X		
Phenol (PHENOL)			X	X	
Tetrachloroethene (TCLEE)				X	
Toluene (MEC6H5)				X	
Vinyl Chloride (C2H3CL)				X	
Xylenes [Total] (TXYLEN)				X	

See footnotes on the following page.

TABLE 11-7 (Continued)

SUMMARY OF CHEMICALS OF POTENTIAL CONCERN FOR MICHAELSVILLE LANDFILL

Chemical (a)	Surface Soil	Shallow Groundwater	Deep Groundwater	Seeps	Surface Water
Inorganic Chemicals:					
Ammonia Nitrogen (NH3N2)		X	X	X	
Antimony (SB)				X	X
Beryllium (BE)		X	X		
Chromium (CR)	X				
Copper (CU)	X				
Iron (FE)		X	X	X	X
Lead (PB)				X	X
Manganese (MN)		X		X	
Nickel (NI)				X	
Nitrate (NO3)					X
Selenium (SE)					X
Silver (AG)					X
Thallium (TL)		X		X	
Zinc (ZN)	X	X		X	

(a) USATHAMA chemical codes listed in parentheses.

X = Selected as a chemical of potential concern.

Note: Blanks in this table indicate that a chemical was not selected as a chemical of potential concern either because (1) it was not detected in a given medium, (2) it was not included in the analyses, or (3) it was detected at background concentrations (inorganic chemicals only). See text for this information.

qualitative depending upon several factors, including the probability of exposure, the potential magnitude of exposure, and the availability of data to support quantitative evaluations. Exposure point concentrations and daily intakes are estimated for all pathways selected for quantitative evaluation.

This exposure assessment is organized into three principal sections. Section 11.3.1.1 discusses potential exposure pathways under current land-use conditions, and Section 11.3.1.2 discusses those potentially occurring under hypothetical future land-use conditions. Section 11.3.1.3 presents estimates of potential human exposures for those pathways selected for quantitative evaluation.

11.3.1.1 Potential Exposure Pathways Under Current Land-Use Conditions

The Michaelsville Landfill was closed in December 1980. Access is restricted as it is within the fenced, controlled area of the Aberdeen Area in which badges must be worn. The landfill itself is not fenced, and there are no control measures to prevent access once personnel are within the controlled area of the Aberdeen Area. The main industrial sector of the Aberdeen Area is approximately 3,300 feet north of the Michaelsville Landfill. Several operations are situated around the landfill. A large firing range is located immediately south and east of the landfill. Firing is parallel to the landfill, and observation towers are located on Trench Warfare Road near each end of the landfill. An ammunition receiving and shipping building is located approximately 500 feet west of the landfill; most of the landfill is located within the 1,800-foot safety clearance range of the ammunition receiving and shipping building. An unused concrete observation tower is located approximately 150 feet northwest of the landfill. The DRMO scrap metal yard is located approximately 1,300 feet northeast of the landfill and a pistol range is located approximately 1,500 feet north of the landfill.

Upland game/early migratory bird hunting, wild turkey hunting, and bow and arrow woodchuck and deer hunting are allowed north and west of Michaelsville Landfill. Deer hunting with firearms is allowed all around the landfill. Trapping is allowed around a tributary of Romney Creek approximately 3,000 feet southwest of the landfill.

APG barracks are located approximately 1 mile north of the landfill, and on-post family housing is located about 2 miles north of the landfill. The City of Aberdeen is approximately 4 miles north of the landfill, and the City of Perryman is approximately 1.75 miles west of the landfill. All of these residential areas are outside of the fenced, controlled area of the Aberdeen Area.

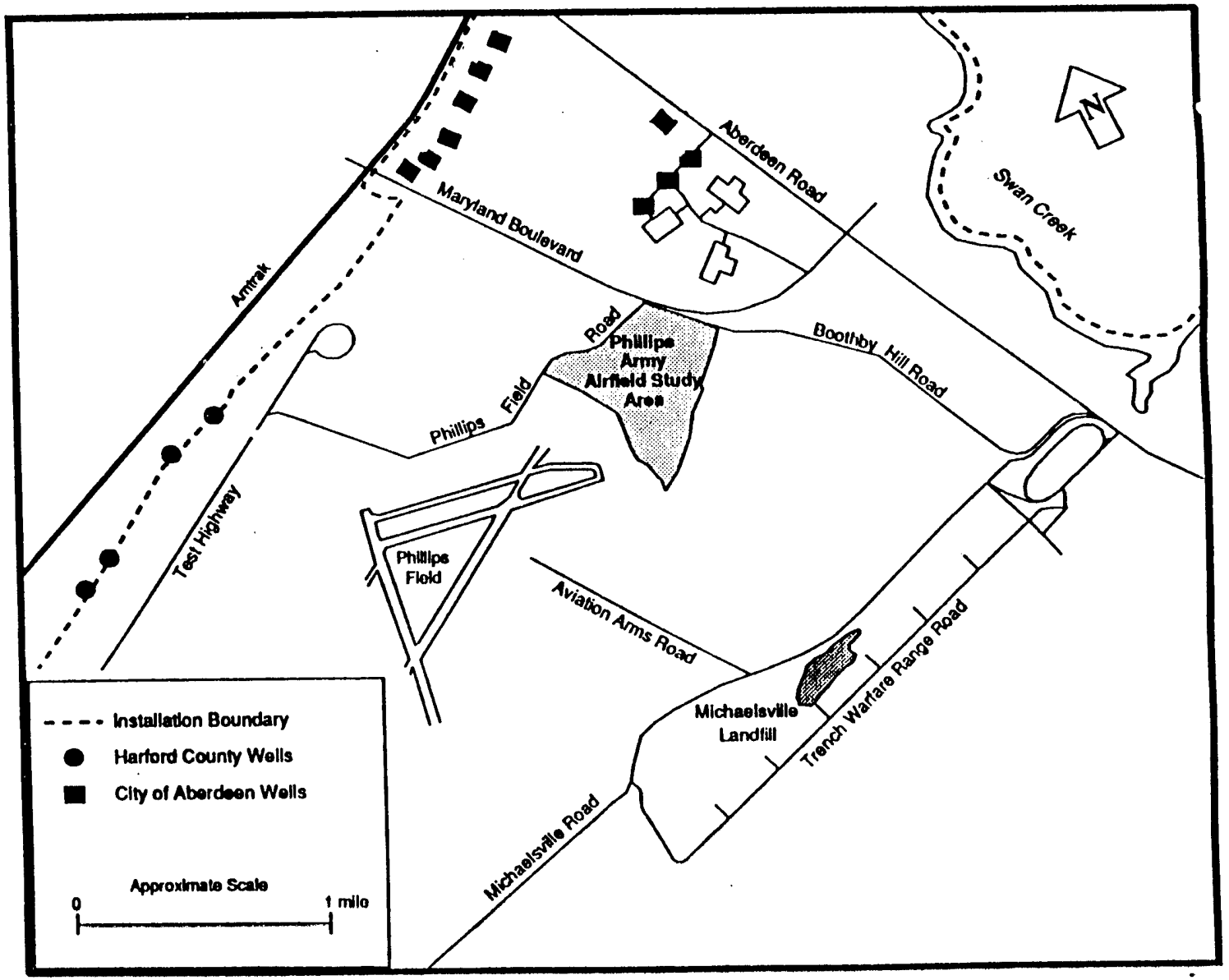
The Aberdeen Area drinking water supply is from a surface water source, Deer Creek at Churchville. However, there are ten City of Aberdeen wells approximately 2 miles northwest of the Michaelsville Landfill within the Aberdeen Area, and four Harford County wells are located along the APG boundary approximately 2 miles west-northwest of the Michaelsville Landfill. The locations of these wells are shown in Figure 11-2.

11.3.1.1.1 Potential Long-Term Exposure Pathways Under Current Land-Use Conditions

Table 11-8 summarizes the pathways by which humans could be exposed to chemicals at or originating from the Michaelsville Landfill under current land-use conditions. Potential exposure pathways are discussed below by exposure medium.

Surface Soil. There are no known activities that occur on the landfill itself. Contact with surface soil at the landfill by nearby workers or hunters who might stray onto the landfill is possible although probably unlikely. The limited surface soil sampling at the landfill (two samples) indicated

Figure 11-2
Wells Located in the Aberdeen Area of APG



only low-level contamination in the cover soils. However, regardless of whether these data are representative of the surface soil contamination at the landfill, this pathway is unlikely to result in significant exposures because of the negligible potential for any populations to contact landfill soils under current land-use conditions. This pathway was therefore not evaluated.

Subsurface Soil. Although subsurface soils are likely to be contaminated as a result of past waste disposal, no activities that involve contact with subsurface soil (e.g., excavation) take place at the Michaelsville Landfill under current land-use conditions. Therefore, dermal contact and incidental ingestion of subsurface soil was not evaluated.

Groundwater. There are no production wells in the Michaelsville Landfill study area itself, so there is no potential for exposure to groundwater in this area under current land-use conditions. A review of potentiometric surface maps in the Michaelsville Landfill study area (USAEWES 1990) indicated that there is no apparent gradient reversal caused by pumping the City of Aberdeen wells and the Harford County wells in the vicinity of the landfill under current pumping conditions; the shallow groundwater generally flows to the south and east and the deep groundwater to the south and west. Therefore, no groundwater-related pathways were evaluated under current land-use conditions.

Seeps/Surface Water/Sediment. Virtually no organic chemicals were detected in the ditch that runs south of the landfill or in the pond at the southwestern corner of the landfill. Organic chemica's were detected infrequently in the seep samples. Sediment samples have not been collected in the study area. Therefore, it is not possible to determine the impact of the landfill on soil or sediment in seep areas or in surface water bodies. However, the limited activities around the landfill are not expected to involve contact with seeps, surface water, or sediment and nearby surface waters are not used as a source of drinking water nor are they used for swimming, fishing, or other recreational activities. Dermal contact and incidental ingestion of seep water, surface water, or sediment were therefore not evaluated.

Air. APG personnel using the observation towers near each end of the landfill, as well as hunters in the area, could be exposed via inhalation to chemicals transported to ambient air from groundwater and subsurface soil. However, "no gases" were detected in the two ambient air monitoring surveys conducted using an OVA or an HNU photoionization detector (USAEWES 1990). Methane, a nontoxic gas generated from organic materials and commonly found in marshy areas (hence its common name, marsh gas), was the only gas detected in the subsurface environment at the Michaelsville Landfill. Dust generation and transport from the study area are unlikely because the Michaelsville Landfill is vegetated. Therefore, no significant air exposures to landfill contaminants are expected under current land-use conditions, and this pathway was not evaluated.

Game. Since hunting occurs in the area of the landfill, there may potentially be exposure to chemicals originating from the Michaelsville Landfill through ingestion of game in which chemicals have accumulated. Of the chemicals detected in relevant environmental media (surface water, soil, and seeps) at and near the landfill, chlorinated pesticides and PCBs are the chemical groups with the greatest tendency to bioaccumulate in organisms. Based on the above, ingestion of game that has accumulated chemicals at or originating from Michaelsville Landfill was evaluated qualitatively in this assessment.

TABLE 11-8

POTENTIAL PATHWAYS OF HUMAN EXPOSURE UNDER CURRENT LAND-USE CONDITIONS AT MICHAELSVILLE LANDFILL

Exposure Medium/ Source Area	Potential Exposure Pathway	Potential for Significant Exposure (a)	Adequacy of Data to Evaluate Pathway	Method of Evaluation
Surface Soil	Dermal contact and/or incidental ingestion of soil by hunters or nearby workers.	Negligible. Michaelsville Landfill is not frequented by hunters or nearby workers, and infrequent exposure to low-level contamination is likely to result in insignificant exposures.	Poor. Only two soil samples collected from the cover.	None, due to low potential for exposure.
Subsurface Soil	None. Although subsurface soils are likely to be contaminated as a result of past waste disposal, no activities that involve contact with subsurface soils (e.g., excavation) take place at Michaelsville Landfill.	No potential for exposure. Pathway not complete.	NA. Pathway not complete.	None. No complete pathway exists.
Groundwater/ - Shallow - Deep	None. Although chemical transport to groundwater from Michaelsville Landfill has occurred, there are no human uses of groundwater at the the site or in downgradient areas. No onsite gradient reversal is occuring due to current upgradient use of groundwater.	No potential for exposure. Pathway not complete.	NA. Pathway not complete.	None. No complete pathway exists.
Seeps/Surface Water/ Sediment	None. Although landfill chemicals are discharging to the surface, the limited activities around the landfill do not involve contact with seeps, surface water, or sediment. Nearby surface waters are not used as a source of drinking water or for swimming or fishing.	No potential for exposure. Pathway not complete.	NA. Pathway not complete.	None. No complete pathway exists.
Air	None. Volatile organic chemicals were not detected in the two air monitoring surveys conducted at the landfill and dust generation and transport is unlikely because Michaelsville Landfill is vegetated.	No potential for exposure. Pathway not complete.	NA. Pathway not complete.	None. No complete pathway exists.
Game	Ingestion of game, that has accumulated chemicals from Michaelsville Landfill, by hunters.	Low to moderate. Chemicals such as pesticides detected in surface waters and seeps near the landfill bioaccumulate in wildlife. However, seeps are intermittent and ditches are unlikely to be significant sources of water for large game animals.	Poor. No tissue or sediment samples available and surface soil data inadequate.	Qualitative, due to low potential for exposure.

See footnotes on the following page.

TABLE 11-8 (Continued)

POTENTIAL PATHWAYS OF HUMAN EXPOSURE UNDER CURRENT LAND-USE CONDITIONS AT MICHAELSVILLE LANDFILL

Exposure Medium/ Source Area	Potential Exposure Pathway	Potential for Significant Exposure (a)	Adequacy of Data to Evaluate Pathway	Method of Evaluation
Soil/Air	Acute Hazards: Hunters or nearby workers encountering unexploded ordnance.	Moderate. Explosives may be present on-site as a result of testing at the Trench Warfare Range located adjacent to Michaelsville Landfill.	Poor. Information on the amount of unexploded ordnance possibly present at Michaelsville Landfill not available.	Qualitative, with a high degree of uncertainty.

(a) Based on considerations of the types and concentrations of chemicals present, or expected to be present, and on considerations of land use.
NA = Not applicable.

11.3.1.1.2 Potential Acute Hazards Under Current Land-Use Conditions

No acute hazards are expected to occur under current land-use conditions.

11.3.1.2 Potential Exposure Pathways Under Future Land-Use Conditions

Future land-use in the immediate area of the Michaelsville Landfill is not expected to differ from current land-use, because the area around the Michaelsville Landfill is already actively used for military operations. It is considered highly unlikely that the landfill itself would be used in the future, even if military operations were to increase, since there is no scarcity of land nearby. With respect to off-post land use, it is possible that the population density might increase bringing with it the need for increased water supply.

Table 11-9 summarizes the additional exposure pathways by which humans could be exposed to chemicals at or originating from the Michaelsville Landfill under future land-use conditions. The only potential exposure pathway under future land-use conditions involves the future off-site use of groundwater at the study area.

Exposure to chemicals in groundwater originating from the Michaelsville Landfill by off-post residents might occur if a sufficient volume of groundwater was pumped by either the Harford County well field (approximately 2.5 miles west-northwest), the City of Aberdeen well field (approximately 2 miles northwest), or both, resulting in reversal of the groundwater gradient in the area of the Michaelsville Landfill. No information is available concerning the possibility of a groundwater flow reversal and off-site groundwater concentrations resulting from this reversal. In the absence of this type of information, this pathway was evaluated using data from groundwater in the study area itself. This provided a highly conservative estimate since concentrations that might reach off-site wells are much lower than those detected on-site, because of the dilution that would be caused by dispersion during transport to these wells and the fact that supply wells would draw groundwater from all directions. For this pathway, ingestion of groundwater was quantitatively evaluated, and dermal contact with and inhalation of chemicals in groundwater was evaluated qualitatively evaluated.

11.3.1.3 Quantification of Exposure

The exposure pathways that were quantitatively or qualitatively evaluated in this assessment are listed below.

Current Land-Use:

- Ingestion of game that has accumulated chemicals from the study area was evaluated qualitatively.

TABLE 11-9

POTENTIAL PATHWAYS OF HUMAN EXPOSURE UNDER FUTURE LAND-USE CONDITIONS AT MICHAELSVILLE LANDFILL

Exposure Medium/ Source Area	Potential Exposure Pathway	Potential for Significant Exposure (a)	Adequacy of Data to Evaluate Pathway	Method of Evaluation
Groundwater/ - Shallow - Deep	Ingestion, dermal contact, and inhalation by off-post residents under conditions of increased pumping.	Low to moderate, due to the chemicals present in shallow groundwater and the potential for the reversal of groundwater flow through Michaelsville Landfill towards the Harford County wells and the City of Aberdeen wells under increased pumping from these well fields.	Poor. Information concerning (1) the possibility of a groundwater flow reversal and for site groundwater to reach these wells, or (2) the concentrations of chemicals in well water if this were to occur, not available.	On-site groundwater data will be used as a conservative estimate of potential impact. The ingestion route will be quantitatively evaluated, and the dermal contact and inhalation routes will be qualitatively evaluated.

(a) Based on considerations of the type and concentration of chemicals present, or expected to be present, and on consideration of land use.

- Hunters or nearby workers around Michaelsville Landfill encountering unexploded ordnance will be evaluated qualitatively.

Future Land-Use:

- Ingestion of shallow and deep groundwater at the site by off-post residents will be quantitatively evaluated and dermal contact with and inhalation of chemicals in shallow and deep groundwater will be evaluated qualitatively.

The methodology used to quantitatively assess exposure by determining the chronic daily intake (CDI) of each chemical of potential concern for each complete exposure pathway being evaluated has been summarized in Chapter 4. No human exposure pathways are being evaluated quantitatively under current land-use conditions. Exposure point concentrations for groundwater are first presented and then are combined with the other exposure parameters to estimate intake. Arithmetic mean concentrations and the 95% upper confidence limits on the arithmetic means were calculated, and exposure point concentrations of chemicals of potential concern in shallow and deep groundwater at the Michaelsville Landfill are presented in Table 11-10 and Table 11-11, respectively.

To evaluate groundwater ingestion exposures, residents were assumed to drink 2 liters of water per day for 30 years. In addition, residents were assumed to weigh 70 kg (EPA 1989a) and live for 70 years (EPA 1989b). Drinking water exposures were calculated using these assumptions and the following equation:

$$CDI = (C_w * IR * EF * ED * Z) / (BW * DY * YL) \qquad \text{(Eq. 1)}$$

where:

CDI	=	chronic daily intake (mg/kg-day);
C_w	=	exposure point concentration in groundwater (µg/L);
IR	=	ingestion rate (2 liters/day);
EF	=	exposure frequency (365 days/year);
ED	=	exposure duration (30 years);
Z	=	conversion factor (mg/1,000 ug);
BW	=	body weight over the period of exposure (70 kg);
DY	=	days in a year (365 days/year); and
YL	=	period over which risk is being estimated (a lifetime [70 years] for potential carcinogens and the period of exposure [30 years] for noncarcinogens) (years).

CDIs calculated using these exposure assumptions are presented in Tables 11-12 and 11-13.

TABLE 11-10

EXPOSURE CONCENTRATIONS OF CHEMICALS OF POTENTIAL CONCERN FOR MICHAELSVILLE LANDFILL: SHALLOW GROUNDWATER

(Concentrations reported in ug/L)

Chemical (a)	Arithmetic Mean	Upper 95 Percent Confidence Limit on the Arithmetic Mean (b)	Maximum Detected Concentration	RME Concentration (c)
Organic Chemicals:				
Acetone (ACET)	51	52	70	52
Aldrin (ALDRN)	0.01	0.01	0.01	0.01
Benzene (C6H6)	2.5	2.6	3.4	2.6
alpha-BHC (ABHC)	0.01	0.01	0.01	0.01
beta-BHC (BBHC)	0.01	0.01	0.01	0.01
delta-BHC (DBHC)	0.01	0.01	0.02	0.01
Butylbenzylphthalate (BBZP)	6.2	6.8	17.9	6.8
Chloroethane (C2H5CL)	5.8	6.3	13	6.3
Chloroform (CHCL3)	2.6	2.7	4.4	2.7
DDT [Total]	0.02	0.03	0.07	0.03
4,4'-DDD (PPDDD)	0.01	0.01	0.03	0.01
4,4'-DDT (PPDDT)	0.02	0.03	0.06	0.03
Dibutylphthalate (DNPH)	10	13	28.2	13
1,1-Dichloroethane (11DCL)	3.3	3.5	24	3.5
1,2-Dichloroethane (12DCLE)	2.6	2.7	4.3	2.7
cis-1,2-Dichloroethene (C12DCE)	3.6	4.1	22	4.1
trans-1,2-Dichloroethene (T12DCE)	2.6	2.7	5.1	2.7
Dieldrin (DLDRN)	0.01	0.01	0.05	0.01
Diethylphthalate (DEP)	6.4	7.3	18.7	7.3
2,4-Dimethylphenol (24DMPN)	5.1	5.3	8.0	5.3
1,3-Dinitrobenzene (13DNB)	NA	NA	4.0	4.0
Di-n-octylphthalate (DNOP)	5.5	5.9	12.4	5.9
1,2-Diphenylhydrazine (12DPH)	NA	NA	1.3	1.3
Endosulfan I (AENSLF)	0.01	0.01	0.07	0.01
Endosulfan II (BENSLF)	0.01	0.01	0.01	0.01
Endosulfan Sulfate (ESFSO4)	0.01	0.01	0.01	0.01
bis(2-Ethylhexyl)phthalate (B2EHP)	65	130	255	130
Heptachlor (HPCL)	0.02	0.02	0.05	0.02
Heptachlor Epoxide (HPCLE)	0.01	0.01	0.06	0.01
Methylene Chloride (CH2CL2)	27	27	502.9	27
PCBs	0.18	0.22	0.7	0.22
Inorganic Chemicals:				
Ammonia Nitrogen (NH3N2)	3,000	7,000 (d)	66,700	7,000
Beryllium (BE)	2.7	2.8	4.3	2.8
Iron (FE)	7,300	86,000	39,600	39,600
Manganese (MN)	720	38,000	2,470	2,470
Thallium (TL)	2.0	2.9	11.7	2.9
Zinc (ZN)	46	58	176	58

(a) USATHAMA chemical codes listed in parentheses.
(b) Values reflect a positively skewed distribution, except as noted.
(c) Value listed is the lower value of the upper 95 percent confidence limit on the arithmetic mean and the maximum detected value.
(d) Value reflects a normal distribution.

NA = Not applicable; single sample.

TABLE 11-11

EXPOSURE CONCENTRATIONS OF CHEMICALS OF POTENTIAL CONCERN FOR MICHAELSVILLE LANDFILL: DEEP GROUNDWATER

(Concentrations reported in ug/L)

Chemical (a)	Arithmetic Mean	Upper 95 Percent Confidence Limit on the Arithmetic Mean (b)	Maximum Detected Concentration	RME Concentration (c)
Organic Chemicals:				
Acetone (ACET)	580	1,300,000	2,310	2,310
delta-BHC (DBHC)	0.01	0.01	0.01	0.01
Butylbenzylphthalate (BBZP)	7.0	9.1	8.5	8.5
DDT [Total]	0.04	0.66	0.07	0.07
4,4'-DDD (PPDDD)	0.01	0.03	0.02	0.02
4,4'-DDT (PPDDT)	0.03	2.9	0.07	0.07
Dibutylphthalate (DNPH)	12	24	19.4	19.4
Dieldrin (DLDRN)	0.01	0.01	0.01	0.01
Diethylphthalate (DEP)	5.8	9.9	10.7	9.9
Di-n-octylphthalate (DNOP)	6.2	8.0	8.2	8.0
Endosulfan I (AENSLF)	0.01	0.01	0.01	0.01
bis(2-Ethylhexyl)phthalate (B2EHP)	34	77	70.8	70.8
Heptachlor (HPCL)	0.02	0.08	0.04	0.04
Heptachlor Epoxide (HPCLE)	0.01	0.04	0.02	0.02
Methylene Chloride (CH2CL2)	8.7	72	28.3	28.3
4-Methylphenol (4MP)	3.2	NC	4.9	4.9
PCBs	0.15	0.27	0.27	0.27
Phenol (PHENOL)	6.5	11	12.7	11
Inorganic Chemicals:				
Ammonia Nitrogen (NH3N2)	2,800	3,700	3,830	3,700
Beryllium (BE)	2.7	3.0	3.3	3.0
Iron (FE)	6,900	35,000,000	11,700	11,700

(a) USATHAMA chemical codes listed in parentheses.
(b) Values reflect a positively skewed distribution.
(c) Value listed is the lower value of the upper 95 percent confidence limit on the arithmetic mean and the maximum detected value.

TABLE 11-12

EXPOSURE POINT CONCENTRATIONS AND CHRONIC DAILY INTAKES FOR HYPOTHETICAL FUTURE INGESTION OF SHALLOW GROUNDWATER AT MICHAELSVILLE LANDFILL (a)

Chemical (b)	RME Concentration (ug/L)	Estimated Chronic Daily Intake (CDI) (mg/kg-day) (c)
Chemicals Exhibiting Carcinogenic Effects		
Aldrin (ALDRN)	0.01	1.2E-07
Benzene (C6H6)	2.6	3.2E-05
alpha-BHC (ABHC)	0.01	1.2E-07
beta-BHC (BBHC)	0.01	1.2E-07
Chloroform (CHCL3)	2.7	3.3E-05
4,4'-DDD (PPDDD)	0.01	1.2E-07
4,4'-DDT (PPDDT)	0.03	3.7E-07
1,2-Dichloroethane (12DCLE)	2.7	3.3E-05
Dieldrin (DLDRN)	0.01	1.2E-07
1,2-Diphenylhydrazine (12DPH)	1.3	1.6E-05
bis(2-Ethylhexyl)phthlate (B2EHP)	130	1.6E-03
Heptachlor (HPCL)	0.02	2.4E-07
Heptachlor Epoxide (HPCLE)	0.01	1.2E-07
Methylene Chloride (CH2CL2)	27	3.3E-04
PCBs	0.22	2.7E-06
Beryllium (BE)	2.8	3.4E-05
Chemicals Exhibiting Noncarcinogenic Effects		
Acetone (ACET)	52	1.5E-03
Aldrin (ALDRN)	0.01	2.9E-07
Butylbenzylphthalate (BBZP)	6.8	1.9E-04
Chloroform (CHCL3)	2.7	7.7E-05
4,4'-DDT (PPDDT)	0.03	8.6E-07
Dibutylphthlate (DNPH)	13	3.7E-04
1,1-Dichloroethane (11DCLE)	3.5	1.0E-04
cis-1,2-Dichloroethene (C12DCE)	4.1	1.2E-04
trans-1,2-Dichloroethene (T12DCE)	2.7	7.7E-05
Dieldrin (DLDRN)	0.01	2.9E-07
Diethylphthlate (DEP)	7.3	2.1E-04
2,4-Dimethylphenol (24DMPN)	5.3	1.5E-04
1,3-Dinitrobenzene (13DNB)	4.0	1.1E-04
Di-n-octylphthalate (DNOP)	5.9	1.7E-04
Endosulfans (AENSLF and BENSLF)	0.02 (d)	5.7E-07
bis(2-Ethylhexyl)phthlate (B2EHP)	130	3.7E-03
Heptachlor (HPCL)	0.02	5.7E-07
Heptachlor Epoxide (HPCLE)	0.01	2.9E-07
Methylene Chloride (CH2CL2)	27	7.7E-04
PCBs	0.22	6.3E-06
Ammonia (NH3)	7,000 (e)	2.0E-01
Beryllium (BE)	2.8	8.0E-05
Manganese (MN)	2,470	7.1E-02
Thallium (TL)	2.9	8.3E-05
Zinc (ZN)	58	1.7E-03

(a) CDIs have been calculated only for those chemicals of potential concern with toxicity criteria. The following chemicals of potential concern are not presented due to lack of toxicity criteria: delta-BHC, chloroethane, endosulfan sulfate, and iron.
(b) USATHAMA chemical codes listed in parentheses.
(c) See text for exposure assumptions.
(d) Value reported is the sum of the RME concentrations for endosulfans I and II.
(e) Value reported is the RME concentration for ammonia nitrogen.

TABLE 11-13

EXPOSURE POINT CONCENTRATIONS AND CHRONIC DAILY INTAKES FOR HYPOTHETICAL FUTURE INGESTION OF DEEP GROUNDWATER AT MICHAELSVILLE LANDFILL (a)

Chemical (b)	RME Concentration (ug/L)	Estimated Chronic Daily Intake (CDI) (mg/kg-day) (c)
Chemicals Exhibiting Carcinogenic Effects		
4,4'-DDD (PPDDD)	0.02	2.4E-07
4,4'-DDT (PPDDT)	0.07	8.6E-07
Dieldrin (DLDRN)	0.01	1.2E-07
bis(2-Ethylhexyl)phthlate (B2EHP)	70.8	8.7E-04
Heptachlor (HPCL)	0.04	4.9E-07
Heptachlor Epoxide (HPCLE)	0.02	2.4E-07
Methylene Chloride (CH2CL2)	28.3	3.5E-04
PCBs	0.27	3.3E-06
Beryllium (BE)	3.0	3.7E-05
Chemicals Exhibiting Noncarcinogenic Effects		
Acetone (ACET)	2,310	6.6E-02
Butylbenzylphthalate (BBZP)	8.5	2.4E-04
4,4'-DDT (PPDDT)	0.07	2.0E-06
Dibutylphthlate (DNPH)	19.4	5.5E-04
Dieldrin (DLDRN)	0.01	2.9E-07
Diethylphthlate (DEP)	9.9	2.8E-04
Di-n-octylphthalate (DNOP)	8.0	2.3E-04
Endosulfan I (AENSLF)	0.01	2.9E-07
bis(2-Ethylhexyl)phthlate (B2EHP)	70.8	2.0E-03
Heptachlor (HPCL)	0.04	1.1E-06
Heptachlor Epoxide (HPCLE)	0.02	5.7E-07
Methylene Chloride (CH2CL2)	28.3	8.1E-04
4-Methylphenol (4MP)	4.9	1.4E-04
PCBs	0.27	7.7E-06
Phenol (PHENOL)	11	3.1E-04
Ammonia (NH3)	3,700 (d)	1.1E-01
Beryllium (BE)	3.0	8.6E-05

(a) CDIs have been calculated only for those chemicals of potential concern with toxicity criteria. The following chemicals of potential concern are not presented due to lack of toxicity criteria: delta-BHC and iron.
(b) USATHAMA chemical codes listed in parentheses.
(c) See text for exposure assumptions.
(d) Value reported is the RME concentration for ammonia nitrogen.

11.3.2 TOXICITY ASSESSMENT

The general methodology for the classification of health effects and the development of health effects criteria has been described in Chapter 4 to provide the analytical framework for the characterization of human health impacts. The oral toxicity criteria that were used to derive estimates of risk for future off-post residents ingesting groundwater are presented in Table 11-14. No oral toxicity criteria are available for delta-BHC, chloroethane, endosulfan sulfate, ammonia nitrogen, and iron as discussed in Section 11.3.1. The toxicity criteria for ammonia was used in the absence of criteria for ammonia nitrogen. In the absence of toxicity criteria, potential risks associated with exposure to delta-BHC, chloroethane, endosulfan sulfate, and iron were not quantitatively evaluated. However, complete toxicity summaries of these chemicals are provided in Appendix B.

11.3.3 RISK CHARACTERIZATION

In this section, the human health risks potentially associated with the Michaelsville Landfill are presented. Risks were evaluated either quantitatively or qualitatively. To quantitatively assess risks, the CDIs calculated in Section 11.3.1 were combined with the health effects criteria presented in Section 11.3.2. Potential risks under current land-use conditions are presented in Section 11.3.3.1, and potential risks under future land-use conditions are presented in Section 11.3.3.2.

11.3.3.1 Potential Risks Under Current Land-Use Conditions

The only potential exposure pathway evaluated in this risk assessment under current land-use conditions is ingestion of game that has accumulated chemicals from the study area by hunters. Since hunting occurs in the area of the Michaelsville Landfill, people may potentially be exposed by ingestion of game in which chemicals originating from the landfill have accumulated. Chlorinated pesticides were detected at low levels in surface soil and surface water, and chlorinated pesticides and PCBs were detected at low levels in seep samples; these chemical groups tend to bioaccumulate in organisms. It is difficult to evaluate the relative magnitude and extent of these chemicals and their potential impact on biota at the Michaelsville Landfill, because limited sampling data are available for surface soil and surface water, and because biota at the Michaelsville Landfill are expected to be exposed to chemicals in seeps only intermittently. Significant exposure and therefore risks from ingestion of game is not expected, since game would probably only spend a small portion of its total foraging time at the Michaelsville Landfill.

11.3.3.2 Potential Risks Under Future Land-Use Conditions

The only potential exposure pathway evaluated under future land-use conditions is exposure to shallow and deep groundwater at the study area. As noted previously, this evaluation of study area groundwater was performed not because it is likely that groundwater in this area will be used in the future but as a means of evaluating the potential impacts of on-site groundwater on off-site well fields if pumping conditions were to cause a gradient reversal at the site. The nearest well field is approximately 2 miles northwest of the Michaelsville Landfill.

Table 11-15 presents the estimated carcinogenic and noncarcinogenic risks associated with ingestion of chemicals in shallow groundwater by future off-post residents using on-site concentrations. The upper-bound excess lifetime cancer risk is 2×10^{-4} under the RME exposure case due primarily to

TABLE 11-14

ORAL CRITICAL TOXICITY VALUES FOR CHEMICALS OF POTENTIAL CONCERN
AT MICHAELSVILLE LANDFILL

Chemical	Chronic Reference Dose (mg/kg/day)	Uncertainty Factor (a)	Target Organ (b)	Reference Dose Source	Cancer Slope Factor (mg/kg/day)-1	EPA Weight of Evidence Classification (c)	Slope Factor Source
)rganic Chemicals:							
Acetone	1.00E-01	1,000	Kidney/Liver	IRIS	--	D	IRIS
ldrin	3.00E-05	1,000	Liver	IRIS	1.70E+01	B2	IRIS
enzene	--	--	--	IRIS	2.90E-02	A	IRIS
alpha-BHC (alpha-HCCH)	--	--	--	IRIS	6.30E+00	B2	IRIS
beta-BHC (beta-HCCH)	--	--	--	IRIS	1.80E+00	C	IRIS
delta-BHC (delta-HCCH)	--	--	--	IRIS	--	D	IRIS
utylbenzylphthalate	2.00E-01	1,000	Testes/Liver/ Kidney	IRIS	--	C	IRIS
hloroethane	--	--	--	--	--	--	--
Chloroform	1.00E-02	1,000	Liver	IRIS	6.10E-03	B2	IRIS
4,4'-DDD	--	--	--	IRIS	2.40E-01	B2	IRIS
4'-DDT	5.00E-04	100	Liver	IRIS	3.40E-01	B2	IRIS
n-butylphthalate	1.00E-01	1,000	Mortality	IRIS	--	--	IRIS
,1-Dichloroethane	1.00E-01	1,000	Kidney	HEAST	--	--	--
1,2-Dichloroethane	--	--	--	IRIS	9.10E-02	B2	IRIS
cis-1,2-Dichloroethene	1.00E-02	3,000	Blood	HEAST	--	--	--
ans-1,2-Dichloroethene	2.00E-02	1,000	Blood	IRIS	--	--	--
eldrin	5.00E-05	100	Liver	IRIS	1.60E+01	B2	IRIS
ethylphthalate	8.00E-01	1,000	Body weight	IRIS	--	--	IRIS
2,4-Dimethylphenol	2.00E-02	3,000	Blood	IRIS	--	--	--
1,3-Dinitrobenzene	1.00E-04	3,000	Spleen	IRIS	--	--	IRIS
n-octylphthalate	2.00E-02	1,000	Liver/Kidney	HEAST	--	--	IRIS
2-Diphenylhydrazine	--	--	--	IRIS	8.00E-01	B2	HEAST
dosulfans	5.00E-05	3,000	Kidney	IRIS	--	--	IRIS
Endosulfan Sulfate	--	--	--	IRIS	--	--	IRIS
bis(2-Ethylhexyl)phthalate	2.00E-02	1,000	Liver	IRIS	1.40E-02	B2	IRIS
Heptachlor	5.00E-04	300	Liver	IRIS	4.50E+00	B2	IRIS
hlor Epoxide	1.30E-05	1,000	Liver	IRIS	9.10E+00	B2	IRIS
ne Chloride	6.00E-02	100	Liver	IRIS	7.50E-03	B2	IRIS
otal)	1.00E-04	100	Fetus	CLEMENT	7.70E+00	B2	IRIS
Phenol	6.00E-01	100	Fetus	IRIS	--	D	IRIS
rganic Chemicals:							
mmonia	3.40E+01 mg/l	--	(d)	HEAST	--	--	--
mmonia Nitrogen	--	--	--	--	--	--	--
imony	4.00E-04	1,000	Blood	IRIS	--	--	IRIS
llium	5.00E-03	100	Various Organs (Tumors)	IRIS	4.3E+00	B2	--
ron	--	--	--	HEAST	--	--	--
anganese	1.00E-01	1	CNS	IRIS	--	--	--
nious Acid/Selenium	3.00E-03	15	Skin	IRIS	--	--	IRIS
lium	7.00E-05	3,000	Blood/Hair	HEAST	--	--	HEAST
	2.00E-01	10	Blood (Anemia)	HEAST	--	--	HEAST

a) Safety factors are the products of uncertainty factors and modifying factors. Uncertainty factors used to develop reference doses generally consist of multiples of 10, with each factor representing a specific area of uncertainty in the data available. The standard uncertainty factors include the following:
- a 10 fold factor to account for the variation in sensitivity among the members of the human population;
- a 10-fold factor to account for the uncertainty in extrapolating animal data to the case of humans;
- a 10-fold factor to account for the uncertainty in extrapolating from less than chronic NOAELs to chronic NOAELs; and
- Modifying factors are applied at the discretion of the reviewer to cover other uncertainties in the data.

b) A target organ is the organ most sensitive to a chemical's toxic effect. RfDs are based on toxic effects in the target organ. If an RfD was based on a study in which a target organ was not identified, an organ or system known to be affected by the chemical is listed.

c) PA Weight of Evidence for Carcinogenic Effects:
[A] = Human carcinogen based on adequate evidence from human studies;
[B2] = Probable human carcinogen based on inadequate evidence from human studies and adequate evidence from animal studies;
[C] = Possible human carcinogen based on limited evidence from animal studies in the absence of human studies; and
[D] = Not classified as to human carcinogenicity.

d) The RfD for ammonia is based on a taste threshold rather than on a health effect.

-- IRIS = Integrated Risk Information System - December 1, 1990.
HEAST = Health Effects Assessment Summary Tables - July 1, 1990.
CLEMENT = Number derived by Clement International Corporation.
= No information available.

TABLE 11-15

POTENTIAL RISKS ASSOCIATED WITH HYPOTHETICAL FUTURE INGESTION OF SHALLOW GROUNDWATER AT MICHAELSVILLE LANDFILL (a)

Chemicals Exhibiting Carcinogenic Effects (b)	Estimated Chronic Daily Intake (CDI) (mg/kg-day)	Slope Factor (mg/kg-day)-1	Weight of Evidence Class (c)	Upper Bound Excess Lifetime Cancer Risk
Aldrin (ALDRN)	1.2E-07	1.7E+01	B2	2E-06
Benzene (C6H6)	3.2E-05	2.9E-02	A	9E-07
alpha-BHC (ABHC)	1.2E-07	6.3E+00	B2	8E-07
beta-BHC (BBHC)	1.2E-07	1.8E+00	C	2E-07
Chloroform (CHCL3)	3.3E-05	6.1E-03	B2	2E-07
4,4'-DDD (PPDDD)	1.2E-07	2.4E-01	B2	3E-08
4,4'-DDT (PPDDT)	3.7E-07	3.4E-01	B2	1E-07
1,2-Dichloroethane (12DCLE)	3.3E-05	9.1E-02	B2	3E-06
Dieldrin (DLDRN)	1.2E-07	1.6E+01	B2	2E-06
1,2-Diphenylhydrazine (12DPH)	1.6E-05	8.0E-01	B2	1E-05
bis(2-Ethylhexyl)phthlate (B2EHP)	1.6E-03	1.4E-02	B2	2E-05
Heptachlor (HPCL)	2.4E-07	4.5E+00	B2	1E-06
Heptachlor Epoxide (HPCLE)	1.2E-07	9.1E+00	B2	1E-06
Methylene Chloride (CH2CL2)	3.3E-04	7.5E-03	B2	2E-06
PCBs	2.7E-06	7.7E+00	B2	2E-05
Beryllium (BE)	3.4E-05	4.3E+00	B2	1E-04
TOTAL	--	--	--	2E-04

Chemicals Exhibiting Noncarcinogenic Effects (b)	Estimated Chronic Daily Intake (CDI) (mg/kg-day)	Reference Dose (RfD) (mg/kg-day)	Uncertainty Factor (d)	Target Organ (e)	CDI:RfD Ratio
Acetone (ACET)	1.5E-03	1.0E-01	1,000	Kidney/Liver	1E-02
Aldrin (ALDRN)	2.9E-07	3.0E-05	1,000	Liver	1E-02
Butylbenzylphthalate (BBZP)	1.9E-04	2.0E-01	1,000	Testes/Liver/Kidney	1E-03
Chloroform (CHCL3)	7.7E-05	1.0E-02	1,000	Liver	8E-03
4,4'-DDT (PPDDT)	8.6E-07	5.0E-04	100	Liver	2E-03
Dibutylphthlate (DNPH)	3.7E-04	1.0E-01	1,000	Mortality	4E-03
1,1-Dichloroethane (11DCLE)	1.0E-04	1.0E-01	1,000	Kidney	1E-03
cis-1,2-Dichloroethene (C12DCE)	1.2E-04	1.0E-02	3,000	Blood	1E-02
trans-1,2-Dichloroethene (T12DCE)	7.7E-05	2.0E-02	1,000	Blood	4E-03
Dieldrin (DLDRN)	2.9E-07	5.0E-05	100	Liver	6E-03
Diethylphthlate (DEP)	2.1E-04	8.0E-01	1,000	Body weight	3E-04
2,4-Dimethylphenol (24DMPN)	1.5E-04	2.0E-02	3,000	Blood	8E-03
1,3-Dinitrobenzene (13DNB)	1.1E-04	1.0E-04	3,000	Spleen	1E+00
Di-n-octylphthalate (DNOP)	1.7E-04	2.0E-02	1,000	Liver/Kidney	8E-03
Endosulfans (AENSLF and BENSLF)	5.7E-07	5.0E-05	3,000	Kidney	1E-02
bis(2-Ethylhexyl)phthlate (B2EHP)	3.7E-03	2.0E-02	1,000	Liver	2E-01
Heptachlor (HPCL)	5.7E-07	5.0E-04	300	Liver	1E-03
Heptachlor Epoxide (HPCLE)	2.9E-07	1.3E-05	1,000	Liver	2E-02
Methylene Chloride (CH2CL2)	7.7E-04	6.0E-02	100	Liver	1E-02
PCBs	6.3E-06	1.0E-04	100	Fetus	6E-02
Ammonia (NH3)	2.0E-01 (f)	9.71E-01 (g)	--	--	2E-01
Beryllium (BE)	8.0E-05	5.0E-03	100	Various organs (Tumors)	2E-02
Manganese (MN)	7.1E-02	..0E-01	1	CNS	7E-01
Thallium (TL)	8.3E-05	7.0E-05	3,000	Blood/Hair	1E+00
Zinc (ZN)	1.7E-03	2.0E-01	10	Blood (Anemia)	8E-03
HAZARD INDEX	--	--	--	--	> 1 (4E+00)

(a) Risks are calculated only for chemicals with toxicity criteria. The following chemicals of potential concern are not presented due to lack of toxicity criteria: delta-BHC, chlorethane, endosulfan sulfate, and iron.
(b) USATHAMA chemical codes listed in parentheses.
(c) EPA Weight of Evidence for Carcinogenic Effects:
[A] = Human carcinogen based on adequate evidence from human studies;
[B2] = Probable human carcinogen based on inadequate evidence from human studies and adequate evidence from animal studies; and
[C] = Possible human carcinogen based on limited evidence from animal studies in the absence of human studies.
(d) Factor which reflects the uncertainty in the estimate of the RfD. Larger factors are associated with greater uncertainty.
(e) A target organ is the organ most sensitive to a chemical's toxic effect. RfDs are based on toxic effects in the target organ. If an RfD was based on a study in which a target organ was not identified, an organ or organ system known to be affected by the chemical is listed.
(f) The estimated CDI is based on the concentration of ammonia nitrogen.
(g) The RfD for ammonia, based on a taste threshold, was converted from mg/L to mg/kg-day by assuming that a 70 kg adult drinks 2 liters of water per day.

beryllium. Because of the use of national background groundwater data in the absence of site-specific or regional background concentrations, there is considerable uncertainty associated with the site-relatedness of inorganic chemicals in groundwater. The RME exposure point concentration for beryllium is 2.8 µg/L, which slightly exceeds the national background concentration of 1.0 µg/L. 1,2-Diphenylhydrazine, bis(2-ethylhexyl)phthalate, and PCBs also significantly contributed to the cancer risk with upper-bound excess lifetime cancer risks on the order of 10^{-5}. 1,2-Diphenylhydrazine was detected in 1 of 28 samples. The Hazard Index for ingestion of shallow groundwater by off-post residents under the RME exposure case exceeds 1 (4) due primarily to 1,3-dinitrobenzene, manganese, and thallium. 1,3-Dinitrobenzene was only analyzed for in one sample.

In the future, off-post residents potentially could be exposed via other routes of exposure to chemicals in shallow groundwater at the Michaelsville Landfill during home use of groundwater. For example, residents could be exposed via inhalation to chemicals that have volatilized during activities such as showering, cooking, and washing clothes. Pesticides, PCBs, phthalate esters, 2,4-dimethylphenol, 1,2-diphenylhydrazine, and inorganic chemicals do not volatilize significantly. Volatile organic chemicals including acetone, benzene, chloroform, 1,1-dichloroethane, cis-1,2-dichloroethene, trans-1,2-dichloroethene, and methylene chloride could volatilize from groundwater. Dermal absorption of organic chemicals could result during bathing or washing. Dermal absorption of inorganic chemicals is considered to be negligible. Although exposure via these routes would add to overall exposure and risk, they are not expected to significantly impact risks from ingestion since beryllium, manganese, and thallium are not volatilized or dermally absorbed.

Table 11-16 presents the estimated carcinogenic and noncarcinogenic risks associated with ingestion of chemicals in deep groundwater by future off-post residents using on-site concentrations. The upper-bound excess lifetime cancer risk is 2×10^{-4} under the RME exposure case due primarily to beryllium. Again, there is considerable uncertainty associated with the determination that inorganic chemicals in groundwater at the Michaelsville Landfill are present at above-background concentrations due to the lack of data on site-specific or background groundwater concentrations. The Hazard Index for ingestion of deep groundwater by off-post residents under the RME exposure case is approximately 1 due primarily to acetone.

Other exposures could occur in the future during home use of groundwater, including inhalation of chemicals that have volatilized during showering, cooking, and washing clothes, and dermal absorption during bathing and washing. Acetone and methylene chloride are the only chemicals present in deep groundwater that would be expected to volatilize significantly. Dermal absorption of organic chemicals could occur, but dermal absorption of inorganic chemicals is considered to be negligible. Although, exposure via these pathways would add to overall exposure and risk, the magnitude of the additional risk is expected to be lower than for ingestion of deep groundwater since beryllium, the chemical driving the risk, is not volatilized or dermally absorbed.

11.4 ECOLOGICAL ASSESSMENT

This section assesses potential ecological impacts associated with the chemicals of potential concern at the Michaelsville Landfill in the absence of remediation. The methods used to assess ecological impacts follow those outlined in Chapter 4 and roughly parallel those used in the human health risk assessment. Below, potentially exposed populations (receptors) are identified. Then information on exposure and toxicity is combined to derive estimates of potential impact in these populations. It is emphasized that this ecological assessment is a predictive assessment. Comprehensive field studies of ecological impacts have not been conducted at the Michaelsville Landfill.

TABLE 11-16

POTENTIAL RISKS ASSOCIATED WITH HYPOTHETICAL FUTURE INGESTION OF DEEP GROUNDWATER AT MICHAELSVILLE LANDFILL (a)

Chemicals Exhibiting Carcinogenic Effects (b)	Estimated Chronic Daily Intake (CDI) (mg/kg-day)	Slope Factor (mg/kg-day)-1	Weight of Evidence Class (c)	Upper Bound Excess Lifetime Cancer Risk
4,4'-DDD (PPDDD)	2.4E-07	2.4E-01	B2	6E-08
4,4'-DDT (PPDDT)	8.6E-07	3.4E-01	B2	3E-07
Dieldrin (DLDRN)	1.2E-07	1.6E+01	B2	2E-06
bis(2-Ethylhexyl)phthlate (B2EHP)	8.7E-04	1.4E-02	B2	1E-05
Heptachlor (HPCL)	4.9E-07	4.5E+00	B2	2E-06
Heptachlor Epoxide (HPCLE)	2.4E-07	9.1E+00	B2	2E-06
Methylene Chloride (CH2CL2)	3.5E-04	7.5E-03	B2	3E-06
PCBs	3.3E-06	7.7E+00	B2	3E-05
Beryllium (BE)	3.7E-05	4.3E+00	B2	2E-04
TOTAL	--	--	--	2E-04

Chemicals Exhibiting Noncarcinogenic Effects (b)	Estimated Chronic Daily Intake (CDI) (mg/kg-day)	Reference Dose (RfD) (mg/kg-day)	Uncertainty Factor (d)	Target Organ (e)	CDI:RfD Ratio
Acetone (ACET)	6.6E-02	1.0E-01	1,000	Kidney/Liver	7E-01
Butylbenzylphthalate (BBZP)	2.4E-04	2.0E-01	1,000	Testes/Liver/Kidney	1E-03
4,4'-DDT (PPDDT)	2.0E-06	5.0E-04	100	Liver	4E-03
Dibutylphthlate (DNPH)	5.5E-04	1.0E-01	1,000	Mortality	6E-03
Dieldrin (DLDRN)	2.9E-07	5.0E-05	100	Liver	6E-03
Diethylphthlate (DEP)	2.8E-04	8.0E-01	1,000	Body weight	4E-04
Di-n-octylphthalate (DNOP)	2.3E-04	2.0E-02	1,000	Liver/Kidney	1E-02
Endosulfan I (AENSLF)	2.9E-07	5.0E-05 (f)	3,000	Kidney	6E-03
bis(2-Ethylhexyl)phthlate (B2EHP)	2.0E-03	2.0E-02	1,000	Liver	1E-01
Heptachlor (HPCL)	1.1E-06	5.0E-04	300	Liver	2E-03
Heptachlor Epoxide (HPCLE)	5.7E-07	1.3E-05	1,000	Liver	4E-02
Methylene Chloride (CH2CL2)	8.1E-04	6.0E-02	100	Liver	1E-02
4-Methylphenol (4MP)	1.4E-04	5.0E-02	1,000	Nervous System	3E-03
PCBs	7.7E-06	1.0E-04	100	Fetus	8E-02
Phenol (PHENOL)	3.1E-04	6.0E-01	100	Fetus	5E-04
Ammonia (NH3)	1.1E-01 (g)	9.71E-01 (h)	--	--	1E-01
Beryllium (BE)	8.6E-05	5.0E-03	100	Various organs (Tumors)	2E-02
HAZARD INDEX	--	--	--	--	~ 1 (1E+00)

(a) Risks are calculated only for chemicals with toxicity criteria. The following chemicals of potential concern are not presented due to lack of toxicity criteria: delta-BHC and iron.
(b) USATHAMA chemical codes listed in parentheses.
(c) EPA Weight of Evidence for Carcinogenic Effects:
[A] = Human carcinogen based on adequate evidence from human studies; and
[B2] = Probable human carcinogen based on inadequate evidence from human studies and adequate evidence from animal studies.
(d) Factor which reflects the uncertainty in the estimate of the RfD. Larger factors are associated with greater uncertainty.
(e) A target organ is the organ most sensitive to a chemical's toxic effect. RfDs are based on toxic effects in the target organ. If an RfD was based on a study in which a target organ was not identified, an organ or organ system known to be affected by the chemical is listed.
(f) The RfD reported is for endosulfans (I & II).
(g) The estimated CDI is based on the concentration of ammonia nitrogen.
(h) The RfD for ammonia, based on a taste threshold, was converted from mg/L to mg/kg-day by assuming that a 70 kg adult drinks 2 liters of water per day.

This ecological assessment is divided into four principal sections. Section 11.4.1 describes the habitat of the area and identifies the potential receptor species or species groups selected for evaluation. Section 11.4.2 evaluates and provides estimates of potential exposures for the chemicals and receptors of potential concern. Section 11.4.3 summarizes relevant toxicity information for the chemicals of potential concern, and Section 11.4.4 provides quantitative and qualitative estimates of ecological impact.

11.4.1 RECEPTOR CHARACTERIZATION

The Michaelsville Landfill is approximately 20 acres in size. A plot plan of the Michaelsville Landfill study area is shown in Figure 11-1, and habitat characteristics of the study area are shown in Figure 11-3. The northern part of the site is covered with grass. The southern portion is covered with grass, shrubs, and small trees (1-7 feet high). A pond is located near the southwestern part of the landfill. A drainage ditch runs along the southeastern edge of the landfill and connects with another drainage ditch, which intercepts the seeps from the southern edge of the landfill. Romney Creek is located south and east of the site, and a wetland area is located around Romney Creek.

Terrestrial wildlife in the area of the landfill probably includes song birds, rabbits, and field mice. Small shorebirds may frequent the ditch and the pond. Raccoons may also use these areas. Aquatic invertebrates and amphibians may be present in the drainage ditch along the southern edge of the landfill and in the pond. Fish may also be present in the ditch, but significant fish populations are not expected to be present. Water flow in the seeps is intermittent and dependent on rainfall, thus the diversity and abundance of aquatic life in the seeps is expected to be limited.

As discussed in Chapter 4, it is not feasible to assess potential impacts in each of the species potentially present at the Michaelsville Landfill, and for this reason indicator species or species groups were selected for further evaluation. The selection of indicator species for the Michaelsville Landfill study area is driven by several factors including the potential for exposure, the sensitivity or susceptibility to chemical exposures, the availability of toxicity data, the availability of chemical data for potential exposure media, ecological significance, and societal value. The indicator species or species groups selected for evaluation at the Michaelsville Landfill based on these considerations are a subset of those identified as potential indicators in Chapter 4 and are listed below along with the rationale for their selection.

Aquatic Species

Invertebrates:

- Benthic invertebrates. This species group was selected because they form an important component of the diet of many birds, as well as adult and juvenile fish.

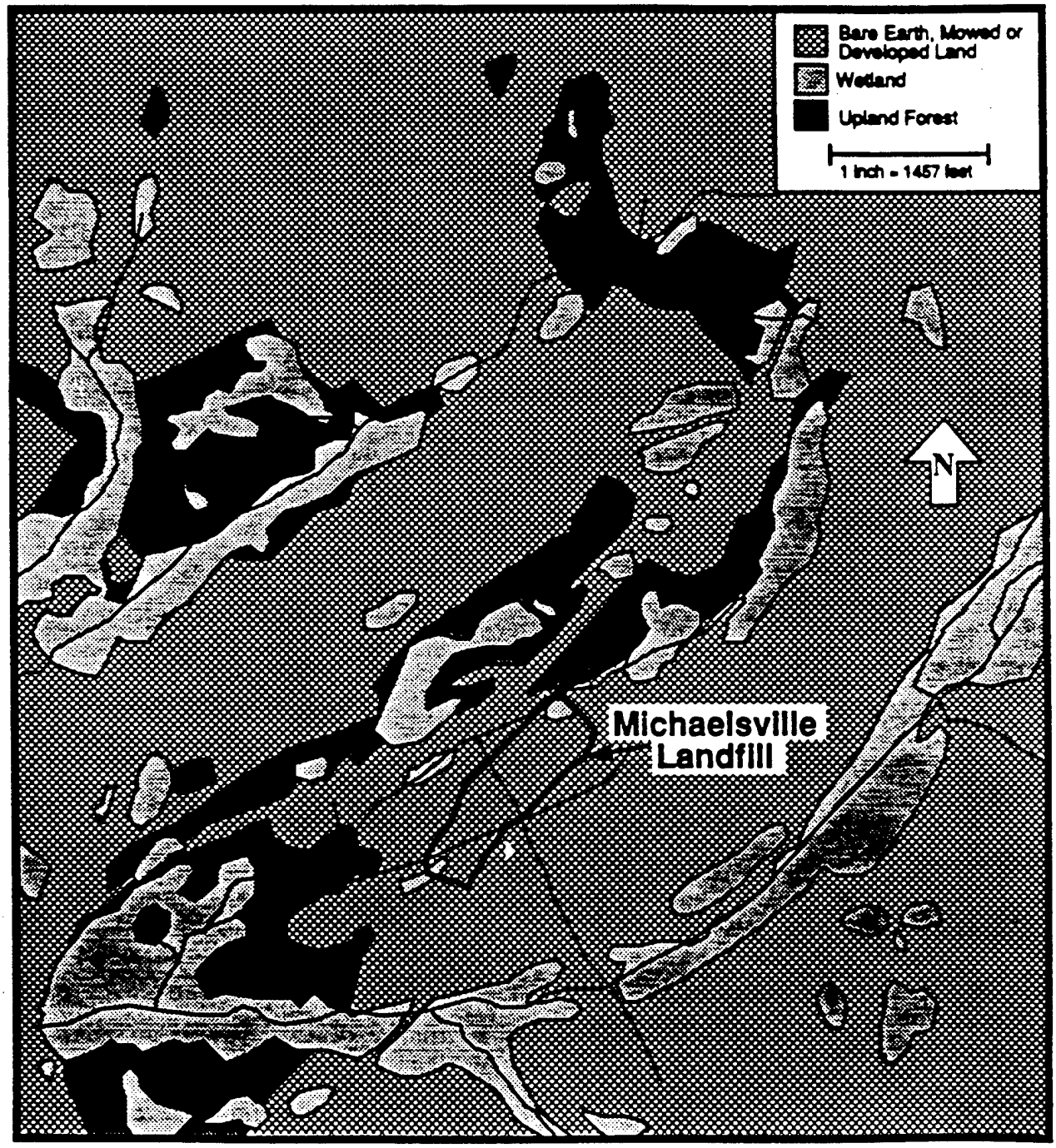
Terrestrial Species

Birds:

- Spotted sandpiper. This species was selected for evaluation because it feeds principally on insects (aquatic larvae and adults). Several of the chemicals of potential concern in the ditch and pond can bioaccumulate in aquatic insects.

Figure 11-3

Habitat Characteristics of the Michaelsville Landfill Study Area At Aberdeen Proving Ground



Mammals:

- Raccoons. This species was selected because it feeds on a variety of aquatic organisms including plants, insects, and other invertebrates.

11.4.2 POTENTIAL EXPOSURE PATHWAYS AND QUANTIFICATION OF EXPOSURE

In this section, the potential pathways by which the selected indicator species and species groups could be exposed to the chemicals of potential concern at the Michaelsville Landfill are discussed, and exposure is quantified for selected exposure pathways. This exposure assessment focuses on potential exposures to chemicals in surface water. No sampling data is available for sediment. No pathways exist by which wildlife could be exposed to chemicals of potential concern in groundwater or subsurface soil.

Potential exposures are presented separately in the following sections for aquatic and terrestrial receptors.

11.4.2.1 Aquatic Life Exposures

As discussed in Chapter 4, aquatic life could be exposed to chemicals in surface water by several pathways. However, most available aquatic toxicity data express toxicity as a function of the concentration in the exposure medium (i.e., surface water or sediment concentration). Thus, in this assessment, exposures to aquatic life were based on concentrations in surface water. Aquatic receptors are expected to include primarily aquatic invertebrates and insects in the pond and the ditch that runs south of the landfill. Measured concentrations of chemicals of potential concern in surface water were used to assess potential aquatic life exposures. The exposure point concentration is the lower value of the 95% upper confidence limit on the arithmetic mean or the maximum detected concentration. The total concentration of BHC was estimated by adding the RME concentration of the BHC isomers. This was done because toxicity and bioconcentration information are not available for each isomer.

11.4.2.2 Terrestrial Wildlife Exposures

As discussed in Chapter 4, terrestrial wildlife could be exposed to chemicals in surface water by a variety of pathways. Because adequate data are not available to assess wildlife exposures via all pathways, however, only exposures via ingestion of surface water and food were selected for consideration in the ecological assessments for the APG study areas.

Water flow in the seeps is intermittent and dependent on rainfall, so use of seeps for drinking water by terrestrial wildlife is expected to be limited. Terrestrial wildlife is more likely to be exposed to chemicals in surface water in the ditch and in the pond. Spotted sandpipers and raccoons, which may feed along the ditch area or shallow areas of the pond, could be exposed to chemicals of potential concern through bioaccumulation in the diet.

Chemical concentrations in wildlife food at the Michaelsville Landfill are estimated in this assessment using bioconcentration factors (BCFs) and chemical concentrations in surface water in the pond and in the ditch. BCFs provide a measure of the extent of chemical partitioning at equilibrium between a

biological medium such as fish or plants and an external medium such as water. For most chemicals and most situations, water is considered to be the predominant source of chemical residues in aquatic organisms (Neff 1979).[3] Therefore use of the BCFs to estimate chemical concentrations in aquatic life is a reasonable approach in the absence of measured tissue concentrations.

Information on the bioconcentration potential of the chemicals of potential concern in surface water was obtained from the available literature. A summary of bioconcentration data for the chemicals of potential concern is presented in the chemical-specific ecological toxicity profiles presented in Appendix C. In selecting BCFs for use in this risk assessment, the following screening procedures were used.

- Data from laboratory studies were used in preference to field data because laboratory studies involve considerably greater control of the parameters affecting bioaccumulation (e.g., chemical concentration, exposure duration).
- Whole-body BCFs were used in preference to muscle or organ-specific BCFs because wildlife typically ingest an entire organism.
- The highest BCF reported in the literature for the particular species of interest was selected for use in this assessment.

Wildlife exposures to chemicals in food are evaluated only for chemicals with BCFs greater than 300. As discussed in Chapter 4, BCFs greater than 300 generally are considered to result in significant bioaccumulation in aquatic life (EPA 1989c). As a result, wildlife food exposures are not evaluated for any volatile chemical, because these chemicals do not bioaccumulate appreciably in aquatic life.

Once BCFs were selected, chemical concentrations in food were estimated using the selected BCF and the measured surface water concentrations in an equilibrium-partitioning model:

$$C_f = C_w * BCF \qquad \text{(Eq. 2)}$$

where:

C_f = chemical concentration in food (mg/kg);

C_w = chemical concentration in the water column (mg/L); and

$BCF_{f:w}$ = food:water BCF (mg/kg food per mg/L water).

For chemical concentrations in water, the total chemical concentration was used because information on dissolved concentrations is not available. This resulted in overestimates of exposure, because chemicals sorbed onto particles are not available for uptake. The RME concentrations reported in Table 11-17 were used to estimate concentrations in aquatic life.

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[3]The principal exceptions to this are highly hydrophobic organic compounds such as PCBs, dioxins, and DDT.

TABLE 11-17

EXPOSURE CONCENTRATIONS OF CHEMICALS OF POTENTIAL CONCERN
IN SURFACE WATER AT MICHAELSVILLE LANDFILL

(Concentrations reported in ug/L)

Chemical (a)	Arithmetic Mean	Upper 95 Percent Confidence Limit on the Arithmetic Mean (b)	Maximum Detected Concentration	RME Concentration (c)
Organic Chemicals:				
alpha-BHC (ABHC)	0.01	NA	0.01	0.01
beta-BHC (BBHC)	0.01	NA	0.01	0.01
delta-BHC (DBHC)	0.02	NA	0.03	0.03
gamma-BHC (GBHC)	0.01	NA	0.01	0.01
bis(2-Ethylhexyl)phthalate (B2EHP)	19	NA	33	33
Inorganic Chemicals:				
Antimony (SB)	0.8	NA	1	1
Iron (FE)	2,300	NA	3,220	3,220
Lead (PB)	2.5	NA	3	3
Nitrate (NO3)	90	NA	103	103
Selenium (SE)	24	NA	28	28
Silver (AG)	11	NA	21	21

(a) USATHAMA chemical codes listed in parentheses.
(b) Value reflects a positively skewed distribution.
(c) Value listed is the lower value of the upper 95 percent confidence limit on the arithmetic mean and the maximum detected value.

NA = Not applicable; two samples only.

Wildlife exposures via ingestion of contaminated food were estimated using the following equation:

$$Dose = C_f * FI_f / BW \qquad \text{(Eq. 3)}$$

where

Dose	=	exposure (mg/kg);
C_f	=	concentration in food (mg chemical/kg food);
FI_f	=	daily food intake by wildlife of contaminated aquatic life (kg); and
BW	=	body weight (kg).

11.4.2.2.1 Estimates of Exposures in Spotted Sandpipers

Spotted sandpipers may feed in the ditch and in the shallow areas of the pond. This species is an aquatic insectivore and feeds predominantly on sediment-dwelling invertebrates. Insects are the principal component of the diet, although marine worms, small crustaceans, and small mollusks also may be eaten. This assessment evaluated sandpiper exposure via ingestion of aquatic insects that have accumulated chemicals from surface water in the ditch.

Chemical concentrations in insects are estimated using the BCF approach outlined above. BCFs for insects were obtained from the available literature, and the highest reported BCF was selected for use in calculating sandpiper exposure. If no BCF was reported for insects, the highest BCF for marine worms, small crustaceans, or small mollusks was selected for use. Insect:water BCFs for BHC, lead, and selenium are presented in Table 11-18. No BCFs are presented for antimony and silver, because these chemicals of potential concern have insect:water BCFs of 300 or less. No BCFs are presented for iron and nitrate, due to the lack of data on bioaccumulation of these chemicals. However, based on physicochemical properties, these chemicals are not expected to accumulate appreciably in aquatic life.

Concentrations of chemicals in insects were estimated by inputting the selected insect:water BCF and the RME surface water concentrations to the equilibrium-partitioning model presented in Equation 2 above.

Sandpiper exposures are quantified using Equation 3. For this assessment, sandpipers were assumed to weigh 43 g (0.043 kg) and ingest 7.5 g (0.0075 kg) of food each day, all of which was assumed to be insects (see Appendix D for source of values). Sandpipers were further assumed to obtain 50% of their daily insect intake, or 3.8 g, from the ditch or the pond. The resulting estimates of exposure are presented in Table 11-19.

TABLE 11-18

INVERTEBRATE BIOCONCENTRATION FACTORS (BCFs) FOR EVALUATION OF SANDPIPER EXPOSURES TO CHEMICALS IN SURFACE WATER AT MICHAELSVILLE LANDFILL

Chemical (a)	BCF	Basis	Reference
BHC	383	Mosquito larvae (Aedes aegypti), 48-hour exposure, gamma-BHC	Matsumura 1977
Lead	1,120	Stonefly (whole body), 28-day exposure	Spehar et al. 1978 in EPA 1985
Selenium	1,100	28-day bioconcentration in daphnids exposed to selenium (IV) in a laboratory closed-system microcosm study (b)	Besser et al. (1989)

(a) Only chemicals of potential concern with measured BCFs greater than 300 are listed here. See text for rationale.
(b) Daphnia are small crustaceans. This information is presented here in the absence of information on bioaccumulation in insects.

TABLE 11-19

ESTIMATED EXPOSURES IN SANDPIPERS INGESTING INSECTS THAT
HAVE ACCUMULATED CHEMICALS FROM SURFACE WATER AT MICHAELSVILLE LANDFILL

Chemical	RME Surface Water Concentration (mg/L) (a)	BCF mg/kg insect per mg/L water) (b)	Estimated Concentration in Insects (mg/kg insect)	Estimated Dose (mg/kg bw) (c)
BHC	0.00006 (d)	383	0.023	0.0020
Lead	0.003	1,120	3.4	0.30
Selenium	0.028	1,100	31	2.7

(a) Reported previously in Table 11-17.
(b) Reported previously in Table 11-18.
(c) Calculated assuming a sandpiper weighs 43 g (0.043 kg) and ingests 3.8 g (0.0038 kg) of insects from Michaelsvill Landfill surface water each day.
(d) Value reported is the sum of BHC isomer RME concentrations.

11.4.2.2.2 Estimates of Exposures to Raccoons

Raccoons may feed in the ditch and the pond in the area of the landfill. This species is an omnivore, and it feeds on aquatic and terrestrial plants, invertebrates, and small vertebrates. This assessment evaluated potential raccoon exposures via ingestion of aquatic macrophytes and invertebrates that have accumulated chemicals from surface waters.

Chemical concentrations in macrophytes and invertebrates were estimated using the BCF approach outlined above. It was assumed that raccoons feed equally on aquatic plants and aquatic invertebrates in the ditch and the pond, so the arithmetic average of the highest BCFs for plants and invertebrates was used to estimate exposures. BCFs for aquatic macrophytes and invertebrates were obtained from the available literature, and the highest reported BCF was selected for use in calculating raccoon exposures. BCFs for BHC, lead, and selenium are presented in Table 11-20. BCFs for aquatic plants are not available for BHC and lead, so only the invertebrate BCFs were used to estimate exposures. No BCFs were presented for the other chemicals of potential concern in surface water, because these chemicals have BCFs of 300 or less for plants and invertebrates. Concentrations of chemicals in plants and invertebrates were estimated by inputting the selected BCF value and the RME surface water concentration into Equation 2. The estimated concentrations in food items are presented in Table 11-21.

Raccoon exposures were quantified using Equation 3. For this assessment, raccoons were assumed to weigh 9.1 kg and ingest 422 g (0.422 kg) of food each day (see Appendix D for sources of values). It was estimated that 75% of the food ingested is from aquatic sources. Raccoons were further assumed to obtain 50% of their daily food intake from the ditch and the pond at the landfill study area. The resulting estimates of exposure are presented in Table 11-21.

11.4.3 TOXICITY ASSESSMENT

The general methodology for the development of toxicity values for the evaluation of ecological impacts has been described in Chapter 4. The toxicity values used to evaluate aquatic life and terrestrial wildlife impacts are presented in this section, along with a brief description of the basis of each value. Freshwater toxicity values were selected over saltwater values, because surface water in the area is freshwater. Tables 11-22 and 11-23 present acute and chronic toxicity values for the assessment of aquatic life impacts from exposure to chemicals of potential concern in surface water. Table 11-24 presents toxicity values for the assessment of impacts in terrestrial wildlife feeding at the Michaelsville Landfill. Note that for BHC, toxicity values for the gamma isomer were used for birds and mammals; toxicity values are not available for the other isomers. Appendix C presents complete ecological toxicity summaries for all chemicals of potential concern for which exposures are being evaluated.

11.4.4 ESTIMATES OF IMPACT

Impacts to aquatic and terrestrial wildlife exposed to chemicals of potential concern at the Michaelsville Landfill are evaluated by comparing estimated exposures with the appropriate toxicity values for the chemical and receptors of concern. Exposures that exceed the selected toxicity value suggest that impacts may be possible in the evaluation species or similar species. Potential impacts to aquatic life are discussed below in Section 11.4.4.1, and those to terrestrial wildlife are discussed in Section 11.4.4.2.

TABLE 11-20

BIOCONCENTRATION FACTORS (BCFs) FOR EVALUATION OF RACCOON EXPOSURES TO CHEMICALS IN SURFACE WATER AT MICHAELSVILLE LANDFILL

Chemical (a)	BCF	Basis	Reference
BHC	383	Mosquito larvae (Aedes aegypti), 48-hour exposure, gamma-BHC (b)	Matsumura (1977)
Lead	1,700	Snail (whole body), 120-day exposure (b)	Borgmann et al. 1978 in EPA (1985)
Selenium	732	Arithmetic average of BCFs for aquatic macrophytes (363) and daphnids (1,100) obtained from a 28-day study of exposures to selenium (IV) in a laboratory closed-system microcosm study	Besser et al. (1989)

(a) Only chemicals of potential concern with measured BCFs greater than 300 are listed here. See text for rationale.
(b) No BCFS for aquatic plants are available.

TABLE 11-21

ESTIMATED EXPOSURES IN RACCOONS INGESTING AQUATIC INVERTEBRATES AND PLANTS THAT HAVE ACCUMULATED CHEMICALS FROM SURFACE WATER AT MICHAELSVILLE LANDFILL

Chemical	RME Surface Water Concentration (mg/L) (a)	BCF (mg/kg in food per mg/L water) (b)	Estimated Concentration in Food Items (mg/kg insect)	Estimated Dose (mg/kg bw) (c)
BHC	0.00006 (d)	383	0.023	0.00040
Lead	0.003	1,700	5.1	0.089
Selenium	0.028	732	20	0.36

(a) Reported previously in Table 11-17.
(b) Reported previously in Table 11-20.
(c) Calculated assuming a raccoon weighs 9,100 g (9.1 kg) and ingests 158 g (0.158 kg) of invertebrates and plants from Michaelsville Landfill surface water each day.
(d) Value reported is the sum of BHC isomer RME concentrations.

TABLE 11-22

ACUTE TOXICITY VALUES FOR ASSESSMENT OF AQUATIC LIFE IMPACTS
FROM EXPOSURE TO CHEMICALS IN SURFACE WATER AT MICHAELSVILLE LANDFILL

Chemical (a)	Acute Toxicity Value (b) (ug/L)	Basis for Value (c)	Reference
Organic Chemicals:			
alpha-BHC (ABHC)	100	LOEC (freshwater)	EPA (1986)
beta-BHC (BBHC)	100	LOEC (freshwater)	EPA (1986)
delta-BHC (DBHC)	100	LOEC (freshwater)	EPA (1986)
gamma-BHC (LIN)	100	LOEC (freshwater)	EPA (1986)
bis(2-Ethylhexyl)phthalate (B2EHP)	133	48-hour EC50 in Daphnia magna	Passino and Smith (1987)
Inorganic Chemicals:			
Antimony (SB)	88	Proposed AWQC (freshwater)	EPA (1988)
Iron (FE)	320	96-hour LC50 in aquatic insects	Warnick and Bell (1969) in EPA (1976)
Lead (PB)	33.8 (d)	AWQC (freshwater)	EPA (1986)
Nitrate (NO3)	1,860,000	96-hr LC50 in bluegills	EPA (1986)
Selenium (SE)	20	AWQC (freshwater)	EPA (1987a)
Silver (AG)	0.92	Proposed AWQC (freshwater)	EPA (1990)

(a) USATHAMA chemical codes listed in parentheses.
(b) Derived using the methodology outlined in Chapter 4.
(c) See Appendix C for more detailed study information.
(d) Hardness dependent criterion (hardness of 50 mg/L as CaCO3 used as lower limit).

AWQC = Ambient Water Quality Criteria.
LOEC = Lowest-observed-effect concentration.

TABLE 11-23

CHRONIC TOXICITY VALUES FOR ASSESSMENT OF AQUATIC LIFE IMPACTS
FROM EXPOSURE TO CHEMICALS IN SURFACE WATER AT MICHAELSVILLE LANDFILL

Chemical (a)	Chronic Toxicity Value (b) (ug/L)	Basis for Value (c)	Reference
Organic Chemicals:			
alpha-BHC (ABHC)	1	Estimated from acute LOEC using an ACR of 100	EPA (1986)
beta-BHC (BBHC)	1	Estimated from acute LOEC using an ACR of 100	EPA (1986)
delta-BHC (DBHC)	1	Estimated from acute LOEC using an ACR of 100	EPA (1986)
gamma-BHC (LIN)	1	Estimated from acute LOEC using an ACR of 100	EPA (1986)
bis(2-Ethylhexyl)phthalate (B2EHP)	360	Proposed AWQC (freshwater)	EPA (1987b)
Inorganic Chemicals:			
Antimony (SB)	30	Proposed AWQC (freshwater)	EPA (1988)
Iron (FE)	1,000	AWQC (freshwater)	EPA (1986)
Lead (PB)	1.3 (d)	AWQC (freshwater)	EPA (1986)
Nitrate (NO3)	400,000	NOEL (freshwater)	EPA (1986)
Selenium (SE)	5	AWQC (freshwater)	EPA (1987a)
Silver (AG)	0.12	Proposed AWQC (freshwater)	EPA (1990)

(a) USATHAMA chemical codes in parentheses.
(b) Derived using the methodology outlined in Chapter 4.
(c) See Appendix C for more detailed study information.
(d) Hardness dependent criterion (hardness of 50 mg/L as CaCO3 used as lower limit).

ACR = Acute to chronic ratio.
AWQC = Ambient Water Quality Criteria.
LOEC = Lowest-observed-effect concentration.

TABLE 11-24

TOXICITY VALUES FOR ASSESSMENT OF TERRESTRIAL WILDLIFE IMPACTS FROM EXPOSURE TO CHEMICALS AT MICHAELSVILLE LANDFILL

Receptor Species/ Chemical (a)	Toxicity Value (mg/kg bw) (b)	Basis for Value (c)					
		Test Species	Exposure Duration	Dose (mg/kg bw)	Effect	Source	Uncertainty Factor
Heron and Sandpiper:							
BHC	0.002	Chicken	3 Months	0.02	NOEL - reduced hatchability	Newell et al. (1987)	10 (to account for interspecies differences)
Lead (PB)	1.2	Kestrel	7 Months	12	NOAEL - eggshell thickness, egg laying	Pattee (1984)	10 (to account for interspecies differences)
Selenium (SE)	0.38	Stilts, avocets	2 Years	3.8 (d)	Reduced survival in young	Williams et al. (1989)	10 (to extrapolate a NOAEL from a LOAEL)
Raccoon:							
BHC	0.03	Dog	4 months	0.3	NOEL, neurotoxicity	Newell et al. (1987)	10 (to account for interspecies differences)
Lead (PB)	0.45	Domestic mammals	Recommended for long-term exposure	0.45 (e)	Recommended safe level	NAS (1980)	None (value derived to protect a variety of domestic mammals)
Selenium (SE)	0.06	Domestic mammals	Recommended for long-term exposure	0.06 (e)	Recommended safe level	NAS (1980)	None (value derived to protect a variety of domestic mammals)

(a) USATHAMA chemical codes listed in parentheses.
(b) Derived using methodology outlined in Chapter 4.
(c) See Appendix C for additional information on the referenced studies.
(d) Estimated dosage. Dietary chemical concentrations reported in the study have been converted to dosages based on average body weights in tested species and estimated food intake rates. See Appendix C for more detail.
(e) Estimated dosage. Dietary chemical concentrations reported in the study have been converted to dosages using the dietary conversion factors reported in Lehman (1954). See Appendix C for more detail.

11.4.4.1 Potential Impacts in Aquatic Life

In this section, aquatic life exposures to chemicals in surface water are evaluated. Potential impacts are discussed below and summarized in Table 11-25.

The exposure concentrations of iron, selenium, and silver exceed both the acute and chronic toxicity values for aquatic life. The chronic toxicity value for lead is also exceeded. Silver may contribute the most to acute and chronic risks since the exposure concentration exceeds the toxicity values by the greatest margin. The silver concentration is more than 20 times greater than the acute toxicity value. Thus, adverse effects to the more sensitive aquatic invertebrates and insects could occur from exposure to silver, iron, selenium, and lead. However, it should be noted that silver was detected only in the ditch, so the pond would not be impacted from silver in surface water.

11.4.4.2 Potential Impacts in Terrestrial Wildlife

Dietary exposures were evaluated for terrestrial wildlife feeding in the surface waters of the ditch and pond at the Michaelsville Landfill. Table 11-26 presents a comparison of selected toxicity values with estimated exposures in sandpipers and raccoons. The results of the comparisons are as follows:

- The estimated dose of BHC to sandpipers is approximately equal to the toxicity value for BHC. No adverse effects would be expected because the toxicity value is based on an estimated NOAEL for birds. However, given the uncertainties of the assessment, the potential for adverse effects cannot be ruled out. It is also important to note that BHC was detected in the pond and not in the ditch. Therefore, sandpipers foraging in the ditch would not be exposed to BHC. The estimated dose of selenium to sandpipers exceeds the toxicity value by a factor of approximately seven. Thus, there is a potential for adverse effects to sandpipers from ingesting aquatic insects from the ditch and the pond at the landfill. Exposure of birds to selenium in the diet has been associated with decreased reproductive success. The estimated dose of lead is less than the lead toxicity value.

- Estimated exposures of raccoons to BHC and lead are less than the toxicity values for these chemicals. The estimated dose of selenium exceeds the toxicity value by a factor of 6. Thus, raccoons could be adversely affected by exposure to selenium in the ditch and the pond. Selenium toxicity in mammals has been associated with decreased growth (NAS 1980).

11.5 UNCERTAINTIES

As in any risk assessment, there is a large degree of uncertainty associated with the estimates of human health and ecological risks. Consequently, these estimates of risks for the Michaelsville Landfill study area should not be regarded as absolute estimates of risk but rather as conditional estimates based on a number of assumptions regarding exposure and toxicity. A complete understanding of the uncertainties associated with the risk estimates is critical to understanding the true nature of the predicted risks and to placing the predicted risks in proper perspective. The principal sources of uncertainty associated with the APG risk assessments were discussed in general in Chapter 4. Some of the key sources of uncertainty associated with the estimates of human health and ecological risk for the Michaelsville Landfill are summarized below.

TABLE 11-25

COMPARISON OF AQUATIC LIFE TOXICITY VALUES WITH EXPOSURE CONCENTRATIONS IN SURFACE WATER AT MICHAELSVILLE LANDFILL

(Concentrations reported in ug/L)

Chemical (a)	Toxicity Value (b) Acute	Toxicity Value (b) Chronic	Exposure Concentrations (c)	Toxicity Value Exceeded
Organic Chemicals:				
BHC	100	1	0.06 (d)	--
bis(2-Ethylhexyl)phthalate (B2EHP)	133	360	33	--
Inorganic Chemicals:				
Antimony (SB)	88	30	1	--
Iron (FE)	320	1,000	3,220	Acute, Chronic
Lead (PB)	33.8	1.3	3	Chronic
Nitrate (NO3)	1,860,000	400,000	103	--
Selenium (SE)	20	5	28	Acute, Chronic
Silver (AG)	0.92	0.12	21	Acute, Chronic

(a) USATHAMA chemical codes listed in parentheses.
(b) Reported previously in Tables 11-22 and 11-23.
(c) Reported previously in Table 11-17.
(d) Value reported is the sum of BHC isomer RME concentrations.

-- No toxicity value exceeded.

TABLE 11-26

COMPARISON OF TOXICITY VALUES WITH ESTIMATED DOSAGES FOR TERRESTRIAL WILDLIFE EXPOSED TO CHEMICALS THAT HAVE ACCUMULATED IN FOOD AT MICHAELSVILLE LANDFILL

Receptor Species/ Chemical (a)	Toxicity Value (mg/kg bw)	Estimated Dosage (b) (mg/kg bw)	Toxicity Value Exceeded ?
Sandpiper:			
BHC	0.002	0.0020	No
Lead (PB)	1.2	0.30	No
Selenium (SE)	0.38	2.7	Yes
Raccoon:			
BHC	0.03	0.00040	No
Lead (PB)	0.45	0.089	No
Selenium (SE)	0.06	0.36	Yes

(a) USATHAMA chemical codes listed in parentheses. Only chemicals with toxicity values are listed. The following chemicals are not presented due to lack of toxicity criteria: bis(2-ethylhexyl)phthalate, antimony, iron, nitrate, and silver.
(b) Reported previously in Tables 11-19 and 11-21.

11.5.1 UNCERTAINTIES RELATED TO SELECTION OF CHEMICALS FOR EVALUATION

There is considerable uncertainty associated with the hydrogeologic assessment data available for this assessment since many chemicals that are not common laboratory contaminants (including pesticides, PCBs, and inorganic chemicals) were detected in the soil, groundwater, and seep blank samples. This raises questions as to the validity of these data.

Because no site-specific background data were available for groundwater and seeps at the Michaelsville Landfill, the site-relatedness of inorganic chemicals in these media was determined by comparing on-site chemical concentrations with national background data. As a result, inorganic chemicals that may not be site-related were selected for evaluation, even though historical information provides no indication that they were associated with past activities at the Michaelsville Landfill. This is particularly critical at the Michaelsville Landfill because inorganic chemicals drive the risk estimates for ingestion of groundwater. Consequently, including chemicals in this risk assessment that are present at natural levels could result in overestimation of risks.

The lack of sediment data and the limited soil data available for this assessment resulted in uncertainty regarding the true nature and extent of contamination. In addition, groundwater samples were not generally analyzed for explosives. An explosive, 1,3-dinitrobenzene, was detected in the one shallow groundwater sample analyzed for explosives.

12.5.2 UNCERTAINTIES ASSOCIATED WITH THE MODELS AND ASSUMPTIONS USED TO ESTIMATE EXPOSURE

Several assumptions were made with respect to the groundwater pathway. Groundwater quality at the landfill was evaluated, not because it is used there, but because there is significant use of upgradient groundwater in the area and the possibility of gradient reversal at the landfill exists. The extent to which this assumption is realistic is not known. With respect to the evaluation of landfill groundwater, the assumptions that chemical concentrations in groundwater will remain constant over the 30 year exposure period assumed could either under- or overestimate risks. However, using concentrations of chemicals detected in on-site wells for the risk evaluation is considered very conservative. This is due to the natural dilution and degradation of chemicals in groundwater originating at Michaelsville Landfill that might reach off-site wells.

There is some uncertainty in the identification of potential terrestrial receptors at the Michaelsville Landfill. It was assumed that sandpipers and raccoons feed in the surface waters of the study area, including the ditch and the pond. Exposures could be more or less, depending on the extent to which these animals utilize these areas. These uncertainties also apply to the representative aquatic receptors.

The estimates of bioconcentration in aquatic life are very uncertain. These estimates were based on a simple partitioning model which assumed equilibrium conditions between the aquatic organism and surface water. The approach also assumed that bioaccumulation in species living in the ditch and the pond were similar to that reported in the literature for other species. The extent to which either of these assumptions is true affects the accuracy of the exposure estimates. Also, total surface water concentrations were used in the model because dissolved concentrations were not available. This results in overestimates of bioaccumulation, because chemicals that are sorbed onto particles are not available for uptake.

12.5.3 UNCERTAINTIES IN THE TOXICITY ASSESSMENT

Toxicological data uncertainties are associated with toxicological data errors such as uncertainty factors of 1,000 for acetone and manganese and 3,000 for thallium and the lack of oral toxicity criteria for chloroethane, delta-BHC, endosulfan sulfate, ammonia nitrogen, and iron.

There is uncertainty in the values used to estimate ecological toxicity. Conservative assumptions were made in order to avoid underestimating toxicity. For hardness-dependent AWQCs (for example, lead) a lower hardness limit of 50 mg/L was used, even though estimated hardness values at the site are less than 50 mg/L. This was done because EPA stated that it is not advisable to extrapolate below 50 mg/L because of limitations of the toxicity database.[4] The effect of hardness on lead toxicity at hardness values of less than 50 mg/L is not known well enough to estimate toxicity values. Lead toxicity may be somewhat greater at a hardness of 37 mg/L (the value at the site) than at 50 mg/L. Thus, potential risks to aquatic receptors could be somewhat underestimated. Nonetheless, acute effects from lead are not expected since the exposure concentration is about 10 times less than the acute toxicity value. There is uncertainty associated with the BHC toxicity values used for birds and mammals. The toxicity values are based on the gamma isomer since no toxicity values are available for the other isomers. It is probable that the isomers are no more toxic than the gamma isomer (Newell et al. [1987] indicate that only the gamma isomer is highly toxic to insects). Thus, the toxicity value based only on the gamma isomer may somewhat overestimate the toxicity of the BHC mixture.

11.6 PRINCIPAL DATA NEEDS

Investigations to date have not provided a complete and exhaustive characterization of the type and degree of contamination at the Michaelsville Landfill. As a result, additional investigation is needed to assess more definitively existing or potential impacts associated with the landfill. The principal data needs are summarized below.

- Additional surface soil, surface water and sediment samples should be collected to fully characterize the extent of surficial contamination at the Michaelsville Landfill. Surface water and sediment samples should be collected from more distant downstream areas.

- Background samples should be collected for analysis for each media sampled. It is recognized that the potential for widespread contamination at APG may prevent the collection of representative background samples from, or close to, the Michaelsville Landfill. However, an attempt should be made to characterize background concentrations as fully as possible. For example, soil background samples should be of the same soil type, and surface water and sediment background samples should be collected from similar creek systems. A sufficient number of background samples should be collected to permit statistical evaluation.

- Groundwater samples should be analyzed for explosives since 1,3-dinitrobenzene was detected in the one groundwater sample analyzed for explosives.

- Information is needed concerning groundwater discharge points so that the current or potential extent of surface water contamination can be defined more accurately.

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[4]Charles Stephen, Environmental Protection Agency, Duluth, Minnesota, personal communication. April 3, 1990.

- It should be determined whether contamination originating from Michaelsville Landfill has any impact upon the City of Aberdeen and Harford County well fields during current periods of maximum pumping of these well fields and in the future when water consumption might increase.

11.7 SUMMARY AND CONCLUSIONS

This baseline risk assessment has addressed potential impacts on human health and the environment associated with the Michaelsville Landfill in the absence of remedial (corrective) actions. The Michaelsville Landfill Hydrogeologic Assessment conducted from May 1987 to April 1990 by the U.S. Army Engineer Waterways Experiment Station (USAEWES 1990) was the primary source of sampling data considered in this assessment. Sampling data were available for surface soil, shallow groundwater, deep groundwater, seeps, and surface water.

A relatively wide range of chemical analyses were performed: volatile organic chemicals, semivolatile organic chemicals, pesticides, PCBs, and inorganic chemicals. Only one groundwater sample was analyzed for explosive-related compounds. Surface soil and surface water had the fewest chemicals of potential concern. However, the two surface soil samples collected at the Michaelsville Landfill are not representative of the contamination in the landfill since they were collected from what is assumed to be "clean" cover. In general, the highest groundwater concentrations were detected south and east of Michaelsville Landfill. Concentrations of organic chemicals in the shallow wells were generally higher than in the deep wells, and concentrations of ammonia nitrogen and chloride were significantly higher in the shallow wells than the deep wells. 1,3-Dinitrobenzene was detected in the one shallow groundwater sample analyzed for explosives[5]. Data from the seeps indicate that contamination from the landfill is being discharged to the surface. Although seeps in the southern portion of the landfill drain into the drainage ditch, there appears to be little impact on these waters, based on the available data. Virtually no organic chemicals were detected in surface water samples. Phthalates, methylene chloride, pesticides, PCBs, and inorganic chemicals were frequently detected in the blanks associated with the hydrogeologic study at the Michaelsville Landfill. These chemicals are not common laboratory contaminants, and their presence in blanks raises the question of the validity of the hydrogeologic assessment data.

11.7.1 HUMAN HEALTH RISK ASSESSMENT SUMMARY

The only potential exposure pathway evaluated in this risk assessment under current land-use conditions is ingestion of game that has accumulated chemicals from the study area. This potential exposure pathway was evaluated qualitatively. No other potential pathways were judged likely to result in significant exposure under current land-use conditions.

The only potential exposure pathway evaluated under future land-use conditions is exposure to shallow and deep groundwater at the study area. This evaluation was performed, not because it is likely that groundwater in the study area will be used in the future, but as a conservative means of evaluating the potential impacts of on-site groundwater on off-site well fields if off-site pumping conditions were such as to cause a gradient reversal at the site. The nearest well field is

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[5]The sample containing 1,3-Dinitrobenzene was collected during well development. Rigorous quality assurance protocols were not followed in the collection of this sample.

approximately 2 miles northwest of the Michaelsville Landfill. The concentrations of chemicals originating from the landfill in off-site groundwater are expected to be lower than on-site concentrations due to dilution during transport. Ingestion of shallow and deep groundwater was evaluated quantitatively, and dermal contact with and inhalation of chemicals in shallow and deep groundwater by off-post residents were evaluated qualitatively.

The estimated human health risks associated with these pathways are as follows:

Current Land-Use

- Since hunting occurs in the area of the landfill, there may potentially be exposure to chemicals originating from the Michaelsville Landfill through ingestion of game in which they have accumulated. Chlorinated pesticides were detected at low levels in surface soil and surface water, and chlorinated pesticides and PCBs were detected at low levels in seep samples; these chemical groups tend to bioaccumulate in organisms. It is difficult to evaluate the relative magnitude and extent of these chemicals and their potential impact on biota at the Michaelsville Landfill since limited sampling data are available for surface soil and surface water, and biota at the Michaelsville Landfill are expected to be exposed to chemicals in seeps only intermittently. The potential for significant exposure from ingestion of game is not expected, since game probably spends only a small portion of its total foraging time at the Michaelsville Landfill.

Future Land-Use

- The upper-bound excess lifetime cancer risk associated with ingestion of chemicals in shallow groundwater by future off-post residents using on-site concentrations is $2x10^{-4}$ primarily due to beryllium[6]. 1,2-Diphenylhydrazine, bis(2-ethylhexyl)phthalate, and PCBs also significantly contributed to the cancer risk with upper-bound excess lifetime cancer risks on the order of 10^{-5}. The Hazard Index for ingestion of shallow groundwater by off-post residents under the RME exposure case exceeds 1 (Hazard Index = 4) due primarily to 1,3-dinitrobenzene, manganese, and thallium. It should be noted that there is considerable uncertainty associated with the selection of inorganic chemicals of potential concern in groundwater at the Michaelsville Landfill due to the absence of site-specific data on background concentrations in groundwater.

  Off-post residents could be exposed via inhalation of volatile chemicals in shallow groundwater and dermal absorption of organic chemicals in shallow groundwater while showering or bathing. Although, exposure via these pathways would add to overall exposure and risk, the magnitude of the additional exposure risk is expected to be lower than for ingestion of groundwater since inorganic chemicals are not volatilized or dermally absorbed.

- The upper-bound excess lifetime cancer risk associated with ingestion of chemicals in deep groundwater by future off-post residents using on-site concentrations is $2x10^{-4}$ due primarily to beryllium. There is considerable uncertainty associated with the selection of inorganic chemicals of potential concern in groundwater at the Michaelsville Landfill due to the lack of specific data on background concentrations in groundwater. The Hazard Index for ingestion

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[6]Again, this is considered a very conservative estimate because on-site concentrations were used to estimate potential exposures from chemicals in off-site wells, with no consideration to dilution and degradation of chemicals during migration.

of deep groundwater by off-post residents under the RME exposure case is approximately 1 due primarily to acetone, a common laboratory contaminant.

Future off-post residents could be exposed via inhalation of volatile chemicals in deep groundwater and dermal absorption of organic chemicals in deep groundwater while showering or bathing. Although, exposure via these pathways would add to overall exposure and risk, the magnitude of the additional risk is expected to be lower than for ingestion of groundwater since inorganic chemicals are not volatilized or dermally absorbed.

11.7.2 ECOLOGICAL ASSESSMENT SUMMARY

Exposures and impacts to aquatic life were evaluated by comparing surface water concentrations with available toxicity values. Surface water concentrations of iron, selenium, and silver exceed these toxicity values and result in adverse acute and chronic effects in more sensitive aquatic invertebrates and insects at the Michaelsville Landfill. Lead may also pose a risk of chronic effects in sensitive aquatic organisms. Exposures and impacts to terrestrial wildlife were also evaluated. Indicator species and pathways were selected for quantitative evaluation. These pathways were: ingestion of aquatic invertebrates that have accumulated chemicals from surface water by spotted sandpipers and ingestion of aquatic plants and invertebrates that have accumulated chemicals from surface water by raccoons. Intakes and dosages estimated for these species were compared with available toxicity values to estimate the potential for adverse effects. Based on this comparison, it was determined that concentrations of selenium in the ditch and the pond could adversely affect birds such as sandpipers through bioaccumulation in the food chain. Raccoons could also be adversely affected by exposure to selenium in food items in the ditch and the pond. However, the degree that the Michaelsville Landfill area is used by the species could be limited given the somewhat limited habitat value of the area.

11.7.3 CONCLUSIONS OF THE RISK ASSESSMENT

It was determined that the only potentially complete exposure pathway under current land-use conditions at the Michaelsville Landfill was ingestion of game that has accumulated chemicals from the study area. It was concluded that significant exposure from ingestion of game is not likely because game is only likely to spend a small portion of its total foraging time at the Michaelsville Landfill.

Potential exposures and risks resulting from use of groundwater were evaluated under future land-use conditions. Although ingestion of groundwater was evaluated, it was recognized that future use of groundwater beneath the Michaelsville Study area is not likely. The evaluation was done strictly to evaluate the resource potential of this groundwater because under some future pumping scenarios of the off-site wells, the potential for groundwater beneath the study area to be within the cone of depression of these wells and ultimately be withdrawn by these wells is not known but is considered to be possible. In evaluating ingestion of groundwater, the upper-bound excess lifetime cancer risk for both shallow and deep groundwater was in excess of 10^{-6}. In both cases this risk was primarily due to beryllium. It should be noted that there is considerable uncertainty associated with the selection of inorganic chemicals of potential concern in groundwater beneath the Michaelsville Landfill due to the absence of site-specific background groundwater data. In evaluating noncarcinogenic risks associated with ingestion of shallow groundwater, the Hazard Index is greater than 1, indicating noncarcinogenic risks are likely. In evaluating ingestion of groundwater from the deep groundwater, the Hazard Index is equal to 1.

Some of the chemicals present in surface water could pose an increased risk of adverse acute and

chronic effects in more sensitive aquatic invertebrates and insects at the Michaelsville Landfill. Additionally, bioaccumulation of selenium through the food chain could adversely affect terrestrial wildlife such as sandpipers and raccoons. However, because these species are not expected to spend large amounts of time in areas such as on-site ditches, the overall impact on the wildlife population is likely to be minimal.

11.8 REFERENCES

BESSER, J.M., HUCKINS, J.N., LITTLE, E.E. and LaPOINT, T.W. 1989. Distribution and bioaccumulation of selenium in aquatic microcosms. Env. Pollut. 62:1-12.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1976. Quality Criteria for Water. Office of Water and Hazardous Materials. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Ambient Water Quality Criteria for Lead-1984. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-84-027.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Quality Criteria for Water 1986. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-86-001.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987a. Ambient Water Quality Criteria for Selenium - 1987. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-87-006. PB88-142237.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987b. Ambient Aquatic Life Water Quality Criteria for Di-n-ethylhexyl phthalate-Draft. Office of Research and Development, Environmental Research Laboratories. Duluth, Minnesota and Narragansett, Rhode Island.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Ambient Water Quality Criteria for Antimony (III)-Draft. Office of Water Regulations and Standards. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989a. Exposure Factors Handbook. Office of Health and Environmental Assessment. Washington, D.C. EPA/600/8-89/043.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989b. Risk Assessment Guidance for Superfund. Volume I: Human Health Evaluation Manual. Interim Final. OSWER Directive 9285.7-01a.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989c. Risk Assessment Guidance for Superfund. Volume II: Environmental Evaluation Manual. Interim Final. EPA/540/1-89-001.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Request for comment on proposed water quality criteria for antimony, phenanthrene, silver, trichlorophenol, others. Washington, D.C. 55:199986 (May 14, 1990).

ENVIRONMENTAL SCIENCE AND ENGINEERING, INC. (ESE). 1981. Installation Assessment of Aberdeen Proving Ground -- Aberdeen Area. Gainesville, Florida. Report No. 301.

LEHMAN, A.J. 1954. Appraisal of the Study of Chemicals in Food, Drugs, and Cosmetics. U.S. Assoc. of Food and Drug Officials. 18:66.

MATSUMURA, F. 1977. Absorption, accumulation, and elimination of pesticides by aquatic organisms. Env. Sci. Res. 10:77-105.

NATIONAL ACADEMY OF SCIENCES (NAS). 1980. Mineral Tolerance of Domestic Animals. National Research Council, Subcommittee on Mineral Toxicity in Animals. Washington, D.C.

NEFF, J.M. 1979. Polycyclic Aromatic Hydrocarbons in the Aquatic Environment: Sources, Fates, and Biological Effects. Applied Science Publishers Ltd. London, England.

NEWELL, A.J., JOHNSON, D.W., and ALLEN, L.K. 1987. Niagara River Biota Contamination Project: Fish Flesh Criteria for Piscivorous Wildlife. New York State Department of Environmental Conservation, Division of Fish and Wildlife, Bureau of Environmental Protection. Albany, New York.

PASSINO, D.R., and SMITH, S.B. 1987. Acute bioassays and hazard evaluation of representative contaminants detected in Great Lake fish. Environ. Toxicol. Chem. 6:901-907.

PATTEE, O.H. 1984. Eggshell thickness and reproduction in American kestrels exposed to chronic dietary lead. Arch. Environ. Contam. Toxicol. 13:29-34.

U.S. ARMY ENGINEER WATERWAYS EXPERIMENT STATION (USAEWES). 1990. Michaelsville Landfill - Hydrogeologic Assessment (Draft Report). Department of the Army, Waterways Experiment Station, Corps of Engineers. Vicksburg, Mississippi.

WALTON, W.C. 1985. Practical Aspects of Groundwater Modeling. National Water Well Association. Worthington, Ohio.

WILLIAMS, M.L., HOTHEM, R.L., and OHLENDORF, H.M. 1989. Recruitment failure in American avocets and black-necked stilts nesting at Kesterson Reservoir, California, 1984-1985. The Condor. 91:797-802.

12.0 PHILLIPS ARMY AIRFIELD LANDFILL ASSESSMENT

This chapter evaluates potential impacts on human health and the environment associated with the Phillips Army Airfield Landfill study area in the absence of remedial (corrective) actions. This assessment is based on the hydrogeologic field investigation conducted by AEHA (1988, 1989), which provides the most recent sampling data in this area. Sampling data are available only for groundwater.

These and other investigations conducted to date have not completely characterized the nature and extent of contamination at the Phillips Army Airfield Landfill study area. Therefore, this risk assessment should be considered largely preliminary and is intended as an initial step in the overall risk assessment of the Phillips Army Airfield Landfill study area.

This assessment follows the general methodology outlined in Chapter 4 of this report, which should be consulted for the rationale and further details of the methods used in this assessment. This assessment is organized into eight primary sections:

- Section 12.1 Background Information
- Section 12.2 Selection of Chemicals of Potential Concern
- Section 12.3 Human Health Risk Assessment
- Section 12.4 Ecological Assessment
- Section 12.5 Uncertainties
- Section 12.6 Principal Data Needs
- Section 12.7 Summary and Conclusions
- Section 12.8 References

12.1 BACKGROUND INFORMATION

The Phillips Army Airfield Landfill study area is located northeast of the Phillips Army Airfield (see Figure 12-1). It is bordered by Boothby Hill Avenue, Combat Road, Airbase Loop, and Bush River Road.

The Phillips Army Airfield Landfill study area has been used since approximately 1950 for the disposal of construction debris, oils and solvents, general refuse, and some unknown materials. There are eight disposal areas: an active debris landfill and an older landfill that partially underlies the active landfill, two inactive landfills, and old borrow pit, an area used for refuse burning and residue disposal, and two grease pits. Figure 12-1 shows the locations of these eight potential source areas. a list of the wastes believed to have been disposed in the area is presented in Table 12-1.

Active Construction Debris Landfill. The active construction debris landfill (active since 1971) reportedly contains old building material, wood, asbestos siding, waste oil, paints, pesticides, herbicides, PCBs, and domestic garbage. It has been partially covered with fill dirt from construction sites at APG.

Older Landfill. The older landfill was in operation from around 1950 to 1971. It contains approximately 260,00 cubic yards of unidentified material and is at least 16 feet deep. It is partially covered by the active construction debris landfill.

Burning Pits. The burning pits were used from the 1960's to 1970. The types and quantities of materials burned or disposed of there are unknown.

Figure 12-1
Phillips Army Airfield Study Area

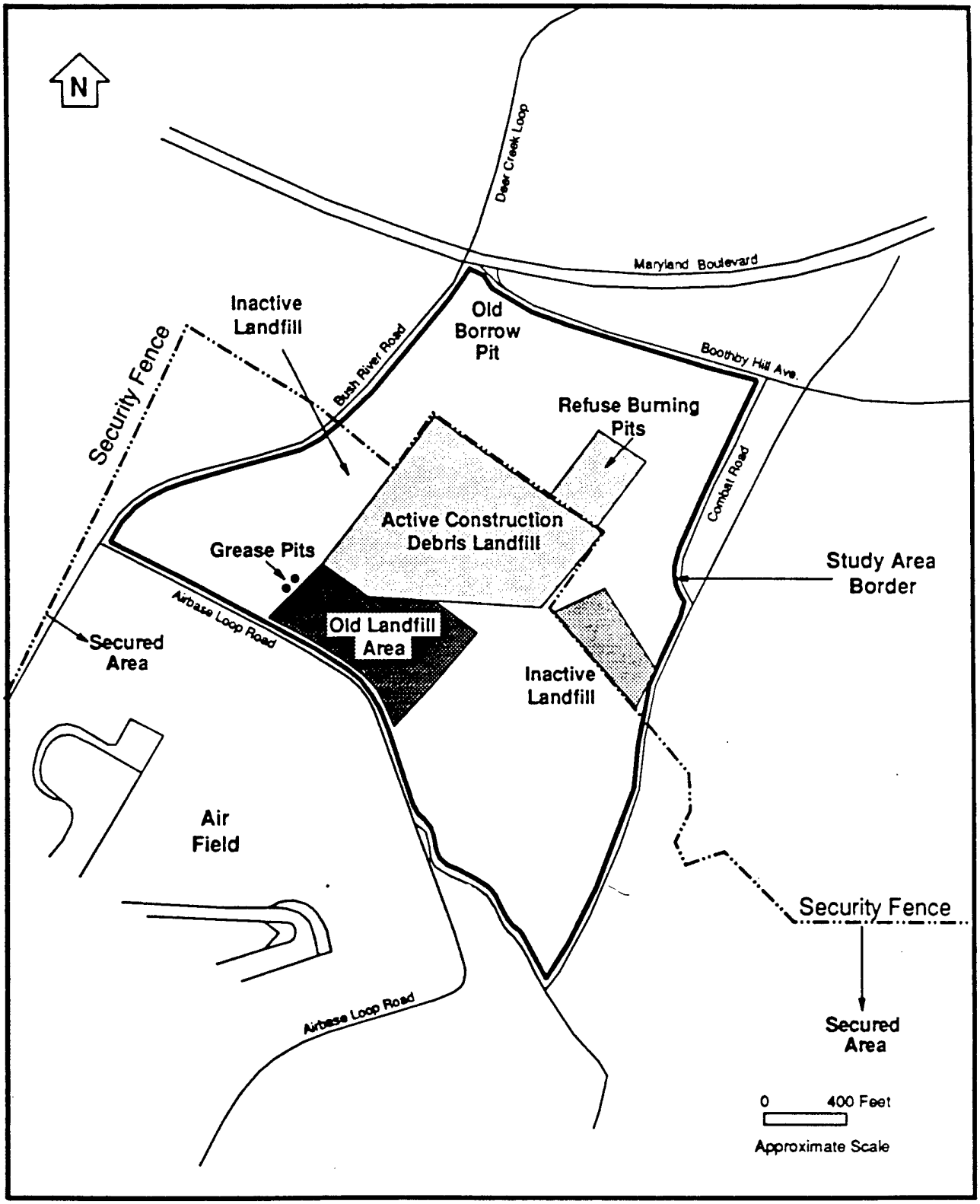

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TABLE 12-1

PRINCIPAL WASTES DISPOSED OF AT
PHILLIPS ARMY AIRFIELD STUDY AREA (a)

Waste oils (various types including motor)
Paints
Pesticides
Herbicides
Solvents
PCBs, PCB transformer oils
Construction debris
Asbestos siding
Domestic garbage
Waste Celotex material

(a) Information obtained primarily from ESE (1981).

Disposal Areas. There are three disposal areas. The disposal area near Boothby Hill Avenue is an old borrow pit that has been intermittently filled since the 1950's. The second disposal area (alandfill),located near the Phillips Field Road guard shack, was operated from 1951 to 1957. The years of use of the third disposal area (another landfill) are unknown. The contents of all three disposal areas are unknown.

Grease Pits. The two 13-foot-deep grease pits slightly west of the old landfill were used until the mid-1980's. The following types of materials were reportedly disposed of in the grease pits: oil and fats from the APG kitchens, used motor oil, solvents, and PCB transformer oil. At some time, absorbent material was added to the pits, and they were then covered with dirt.

12.2 SELECTION OF CHEMICALS OF POTENTIAL CONCERN

In this section, environmental monitoring data collected by AEHA in the Phillips Army Airfield Landfill study area are briefly summarized, and chemicals of potential concern selected for further evaluation are listed. Sampling data are available only for groundwater.

12.2.1 GROUNDWATER

The lithology of the Phillips Army Airfield Landfill study area is a surface clay and silt layer that overlies a sand and gravel layer which, in turn, overlies a clay layer (ESE 1981). The surface clay and silt layer varies in thickness across the site (ESE 1981). The general groundwater gradient at the study area is southeast towards topographically low areas (ESE 1981).

Groundwater samples were collected form 10 monitoring wells during two separate sampling periods in 1988 (AEHA 1988, 1998). The monitoring wells were installed at eight locations within the study area boundaries, and in two locations southeast of the study area. Data from these 10 monitoring wells were combined for the purposes of this assessment. In both sampling periods, samples were analyzed for purgeable organic priority pollutant compounds and for inorganic chemicals. Two trip blanks were analyzed for the April/May 1988 sampling round. The trip blanks associated with the November 1988 sampling round were not analyzed because of mishandling.

Five organic chemicals were detected in the groundwater samples: benzene, trans-1,2-dichloroethene, methylene chloride, trichloroethene, and vinyl chloride. All of the organic chemicals were present at low concentrations (less than 10μg/L) and at relatively low frequencies of detection (1 or 2 out of 10). All of these chemicals were detected in well PW-17, which is located downgradient of the active landfill and the refuse burning pits. Two wells, PW-16 (in the same general area as PW-17) and PW-18 (the furthest downgradient from the study area) each contained one chemical. Methylene chloride, the chemical detected in PW-18 in the November 1988 sampling, is a common laboratory contaminant and is probably not present in groundwater in this area. Methylene chloride was present at similar levels in many samples (including the trip blanks) in the April/May 1988 sampling round and was, therefore, not considered to be site-related in that sampling round. However, as noted above, no blank data are available for the November 1988 sampling round. All five organic chemicals were selected ass chemicals of potential concern, as shown in Table 12-2.

Seventeen inorganic chemicals were detected in the groundwater. Several chemicals with low toxicity (calcium, chloride, potassium, and sodium) were removed from consideration as chemicals of potential concern (see Chapter 4 for a discussion of this step). The concentrations of the remaining chemicals were compared to background concentrations to determine whether or not they were site-related. Because no site-specific or regional background data were available, national background levels were

TABLE 12-2

SUMMARY OF CHEMICALS DETECTED IN GROUNDWATER
AT PHILLIPS ARMY AIRFIELD STUDY AREA (a)

(Concentrations reported in ug/L)

Chemical (b)	Frequency of Detection (c)	Range of Detected Concentrations (d)	Background Concentration (e)
Organic Chemicals:			
* Benzene (C6H6)	1 / 10	3.0	NA
* trans-1,2-Dichloroethene (T12DCE)	2 / 10	3.3 - 8.5	NA
* Methylene Chloride (CH2CL2)	2 / 10	4.0 - 8.0	NA
* Trichloroethene (TRCLE)	2 / 10	3.8 - 4.0	NA
* Vinyl Chloride (C2H3CL)	1 / 10	4.0	NA
Inorganic Chemicals:			
Arsenic (AS)	4 / 10	1.0 - 2.0	100
* Barium (BA)	10 / 10	35.0 - 245	100
Cadmium (CD)	9 / 10	0.8 - 13.9	100
Calcium (CA)	1 / 1	152,000	1,000,000
Chloride (CL)	10 / 10	6,600 - 73,500	1,000,000
Chromium (CR)	4 / 10	0.8 - 1.0	100
Copper (CU)	1 / 1	10.0	100
Fluoride (F)	1 / 10	550	10,000
* Iron (FE)	7 / 10	65.0 - 14,300	10,000
Lead (PB)	10 / 10	2.5 - 44.0	100
* Manganese (MN)	9 / 10	7.5 - 1,850	100
Mercury (HG)	1 / 10	0.3	0.50 (f)
Nitrite/Nitrate (NIT) (g)	10 / 10	60.0 - 2,150	10,000
Potassium (K)	1 / 1	2,300	10,000
Selenium (SE)	2 / 10	0.8 - 2.0	100
Sodium (NA)	1 / 1	5,000	1,000,000
Sulfate (SO4)	10 / 10	1,380 - 43,500	1,000,000

(a) Samples: AA-1, W-8, W-9, W-10, W-11, W-12, W-13, W-16, W-17 and W-18.
(b) USATHAMA chemical codes listed in parentheses.
(c) The number of samples in which the contaminant was detected divided by the total number of samples analyzed for that chemical.
(d) Values reported for metals are dissolved concentrations.
(e) Background concentrations from Walton (1985), except as noted. Values reported are dissolved concentrations, and represent the upper end of the range of typical background concentrations.
(f) Background concentration from EPA (1986).
(g) Concentration is reported as nitrite/nitrate non-specific. The value reported is assumed to represent the total concentration of nitrite/nitrate.

* = Selected as a chemical of potential concern. See text.
NA = Not available.

used for this comparison. Barium, iron, and manganese were present in groundwater at concentrations above national background levels and were, therefore, selected as chemicals of potential concern.

12.2.2 SUMMARY OF CHEMICALS OF POTENTIAL CONCERN

In addition to the chemicals of potential concern selected for groundwater, other chemicals are likely to be present in groundwater at the Phillips Army Airfield Landfill study area, and may be of potential concern regarding possible exposures and impacts./ Table 12-3 summarizes additional chemicals potentially of concern for the study area that either were not included in the chemical analyses, or were not analyzed for in specific media. The chemicals listed are those that have the potential to be present in the greatest quantities based on historical information (ESE 1981). It should be noted, however, that information regarding wastes disposed of in this study area was available only for the active landfill and for the two grease pits. It is unknown what materials are present in the other source areas.

As Table 12-3 shows, PCBs, pesticides, and herbicides were among the principal; wastes in the various source areas. Although these contaminants were not analyzed for in groundwater, based on their physiochemical parameters, PCBs and any of the pesticides or herbicides with low water solubilities would be unlikely to migrate readily from soil to groundwater to any great extent.

12.3 HUMAN HEALTH RISK ASSESSMENT

This section addresses the potential human health risks associated with the Phillips Army Airfield Landfill study area in the absence of remedial actions. This human health risk assessment is divided into three principal sections. Section 12.3.1 evaluates the potential human exposures for the chemicals of potential concern at the site. Section 12.3.2 summarizes relevant toxicity information for the chemicals of potential concern. Section 12.3.3 provides quantitative estimates of human health risks.

12.3.1 EXPOSURE ASSESSMENT

This section identifies the pathways by which human populations may be exposed to chemicals of potential concern at or originating from the Phillips Army Airfield Landfill study area and selected pathways for further evaluation. Only complete pathways were selected for further evaluation (see Chapter 4 for a definition of a complete exposure pathway). Evaluations of exposures were quantitative or qualitative, depending upon several factors, including probability of exposure, the potential magnitude of exposure, and the availability of data to support quantitative evaluations. Exposure point concentrations and daily intakes were estimated for all pathways selected for quantitative evaluation.

This exposure assessment is organized into three principal sections. Section 12.3.1.1 discusses potential exposure pathways under current land-use conditions and Section 12.3.1.2 discusses those potentially occurring under hypothetical future land-use conditions. Section 12.3.1.3 presents estimates of potential human exposures for those pathways selected for quantitative evaluation.

TABLE 12-3

CHEMICALS OF CONCERN POTENTIALLY PRESENT AT
PHILLIPS ARMY AIRFIELD STUDY AREA (a)

Group	Comments
PCBs	PCBs are known to have been disposed in both the active construction landfill as well as the two grease pits. PCBs are very persistent in the environment, and are therefore likely to be present in the subsurface soils if disposed in the area.
Pesticides/herbicides	Since the persistence of herbicides and pesticides depends upon the chemical's structure and properties, it is unknown whether or not the herbicides and pesticides that were disposed of are persistent in the environment, and would be present today.

(a) Based on historical information. Chemicals listed are those not analyzed for and potentially present in the greatest quantities. A number of other chemicals could be present in smaller quantities at the Phillips Army Airfield study area.

12.3.1.1 Potential Exposure Pathways Under Current Land-Use Conditions

A security fence limits access to portions of the study area. The active construction debris landfill, the older landfill, and the two grease pits are all within the secured area and are, thus, inaccessible to unauthorized personnel. One caretaker, working at the active construction debris landfill, and frequent patrols by military police and other security forces preclude trespassing in the secured areas. The remainder of the study area is outside the security fence and is accessible to anyone in the Aberdeen Area.

A physical combat proficiency course is located approximately 600 feet northeast of the construction debris landfill in the same area as the "other landfills," and a vehicle testing area is between th physical combat proficiency course and the construction debris landfill. Two buildings and the fenced area immediately north of Bush River Road are used as a canine training center and a guard shack. Along the fence east of the construction debris landfill there are two deer shacks for hunters. The Ruggles golf course is approximately 1,200 feet west of the construction debris landfill. The area north of Boothby Hill Avenue and east of Combat Drive is semi-industrial with some administrative offices. There are several barracks approximately 1,200 feet east of the vehicle testing area.

Upland game/early migratory bird hunting and bow-and-arrow deer hunting are allowed in the study area and to the west, southeast, and east of the study area. Deer hunting with firearms is allowed southeast and southwest of the study area. Trapping is allowed around tributaries of Romney Creek, approximately 3,000 feet southeast of the study area and 5,000 feet west of the study area. Hunting season for most game animals at APG is in the fall and winter.

The closest on-post housing is approximately 4,400 feet north of the study area. The City of Aberdeen is located approximately 1.5 miles north of the study area, and the City of Perryman is located approximately 2 miles to the west.

The Aberdeen Area obtains drinking water from surface water supplies in Deer Creek at Churchville, Maryland. Ten City of Aberdeen production wells are located approximately 0.4 miles north of the study area, within the Aberdeen Area boundary. These wells are screened from 50 to 72 feet deep in the Talbot formation sediments. Four Harford County production wells are located along the western boundary of the Aberdeen Area, approximately 2 miles west of the construction debris landfill.

12.3.1.1.1 Potential Long-Term Exposure Pathways Under Current Land-Use Conditions

Table 12-4 summarizes the pathways by which humans could be exposed to chemicals at or originating from the Phillips Army Airfield Landfill study area under current land-use conditions. Potential exposure pathways are discussed below by exposure medium.

Soil and Air. Exposure to chemicals at the active construction debris landfill by the caretaker and by workers delivering waste materials to the landfill could occur via contact with soil, and subsequent dermal absorption and/or incidental ingestion of chemicals. Because the active construction debris landfill is an operating unit, it was assumed that workers wear gloves and ar otherwise protected from skin and inhalation exposure to any contaminated soils and wastes. This potential exposure pathway was not selected for further evaluation.

Soldiers and hunters utilizing testing courses and hunting areas at the study area could potentially be exposed to chemicals through direct contact with soils. In addition, soldiers and hunters could be exposed to chemicals in soils and waste through inhalation of dust or vapors. However, no surface soils, subsurface soils, or soil gas samples have been collected in the study area. Thus, potential

Figure 12-2
Wells Located in the Aberdeen Area of APG

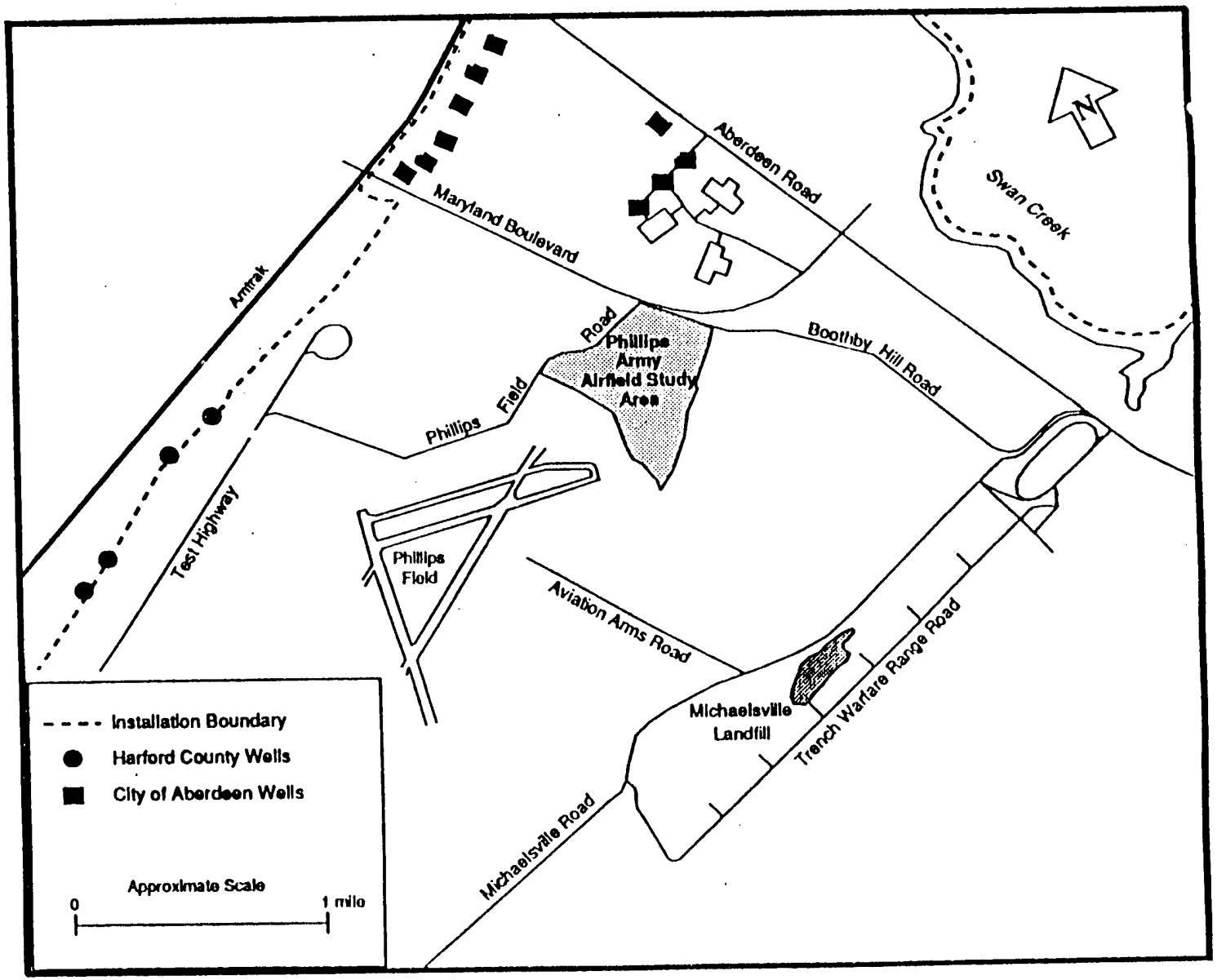

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TABLE 12-4

POTENTIAL PATHWAYS OF HUMAN EXPOSURE UNDER CURRENT LAND-USE CONDITIONS
PHILLIPS ARMY AIRFIELD STUDY AREA

Exposure Medium/ Source Area	Potential Exposure Pathway	Potential for Significant Exposure (a)	Adequacy of Data to Evaluate Pathway	Method of Evaluation
Soil/Air	Direct contact with subsequent dermal absorption and incidental ingestion of soil and inhalation of vapors and dust by workers using the active construction debris landfill.	Negligible. Since the active construction debris landfill is an operating unit, it is assumed that workers there would wear gloves and be otherwise protected from exposure to any contaminated soils and wastes in the area.	Poor. No soil, soil gas, or air samples collected.	None, due to low potential for exposure and lack of data.
Soil/Air	Direct contact with subsequent dermal absorption and incidental ingestion of soil and inhalation of vapors and dust by soldiers training in the study area or by hunters.	Unknown. The impact of the source areas at the Phillips Army Airfield study area on soil is not known. Exposures could occur if the soil is contaminated.	Poor. No soil, soil gas, or air samples collected.	None, due to lack of data.
Groundwater/ On-site	None. Although groundwater in the Phillips Army Airfield study area is contaminated, there are no human uses of groundwater on-site or in the natural downgradient direction of flow (southeast).	No potential for exposure. Pathway not complete.	NA. Pathway not complete.	None. No complete pathway exists.
Groundwater/ Off-site	Ingestion, dermal contact, and inhalation of chemicals in groundwater by off-post residents under conditions of gradient reversal caused by pumping from the City of Aberdeen and Harford County well fields.	Low-to-Moderate, due to the chemicals present in groundwater and the potential for the reversal of groundwater flow through the study area towards the City of Aberdeen wells and the Harford County wells.	Poor. Groundwater only analyzed for volatile organic and inorganic chemicals. Information concerning (1) the possibility of a groundwater flow reversal and for site groundwater to reach these wells, or (2) the concentrations of chemicals in well water if this were to occur, are not available.	On-site groundwater will be used as a conservative estimate of potential impact. The ingestion route will be quantitatively evaluated and the dermal contact and inhalation routes will be evaluated qualitatively.
Game	Ingestion of game, that has accumulated chemicals from the Phillips Army Airfield study area, by hunters.	PCBs, pesticides, and herbicides, the principal wastes that could be present in the study area that were not analyzed for in any media, could bioaccumulate in game.	Poor. No soil, surface water, or sediment samples collected.	Qualitative, with a high degree of uncertainty.

(a) Based on considerations of the types and concentrations of chemicals present, or expected to be present, and on considerations of land use.
NA = Not applicable.

exposure to chemicals in soils cannot be quantified and these exposure pathways were not selected for further evaluation.

Groundwater. There are no groundwater wells at the Phillips Army Airfield Landfill study area itself, so there is no potential for on-site exposure to groundwater beneath the study area under current land-use conditions. Exposure of off-site residents to chemicals in groundwater originating from the Phillips Army Airfield Landfill study area could potentially occur if a sufficient volume of groundwater were pumped by the City of Aberdeen well field and the Harford County well field, resulting in a reversal of the groundwater gradient in the study area. Information concerning the possibility of groundwater flow reversal and off-site groundwater concentrations resulting from this reversal is not available. In the absence of this information, this potential pathway was evaluated using data from the study area itself. This provides a highly conservative estimate since concentrations that might reach off-site wells would be much lower than those detected on-site, given the dilution that would be caused by dispersion during transport to these wells and the fact that supply wells would draw groundwater from all directions. For this pathway, ingestion was quantitatively evaluated, and dermal contact and inhalation were qualitatively evaluated. If pumping by the City of Aberdeen and Harford County well fields does not change the natural direction of flow through the study area, this pathway would not be complete because there are no supply wells southeast of the study area that would be considered downgradient.

Game. Hunters could be at risk of exposure to chemicals by ingestion of game that has bioaccumulated chemicals at or originating from the Phillips Army Airfield Landfill study area. Several chemicals (PCBs, pesticides, and herbicides) that tend to bioaccumulate in wildlife are potentially present at the site (see Table 12-3). No data on concentrations of these chemicals in any media are available. Thus, this pathway was evaluated qualitatively.

12.3.1.1.2 Potential Acute Hazards Under Current Land-Use Conditions

Because it is unknown exactly what types of materials were disposed of in most of the disposal areas, it is not possible to determine whether or not there could be acute hazards to populations such as hunters or workers. There is no indication that unexploded ordnance is in the area, so such acute hazards are not likely. Therefore, exposures from acute hazards were not evaluated.

12.3.1.2 Potential Exposure Pathways Under Future Land-Use Conditions

Land-use under future conditions is not expected to differ from current land-use at the Phillips army Airfield Landfill study area, except that military physical combat proficiency testing could increase as a result of increased military activities at APG (e.g., as a result of relocation of troops from closed bases or in preparation for war). Although exposure frequency might increase, the exposure pathways would be expected to remain the same. Thus, no additional on-site exposure pathways were evaluated under future land-use conditions.

12.3.1.3 Quantification of Exposure

The exposure pathways that were quantitatively or qualitatively evaluated ni this assessment under current land-use conditions are listed below.

- Ingestion of groundwater at the site was quantitatively evaluated, and dermal contact with and inhalation of chemicals in groundwater were evaluated qualitatively as a

conservative estimate of potential off-post exposures.

- Ingestion of game that has accumulated chemicals from the study area was evaluated qualitatively.

The methodology used to quantitatively assess potential exposure by determining the chronic daily intake (CDI) of each chemical of potential concern for each complete exposure pathway being evaluated has been summarized in Chapter 4. In this assessment, exposure pint concentrations were estimated and then combined with other exposure parameters to estimate intake.

Exposure point concentrations for ingestion of groundwater are presented in Table 12-5. Arithmetic mean concentrations and the 95% upper confidence limits on the arithmetic means were calculated for the groundwater in the study area. To evaluate groundwater ingestion exposures, it was assumed that off-post residents drink 2 liters of water for 30 years. Standard assumptions for adult body weight of 70 kg (EPA 1989a) and a lifetime of 70 years (EPA 1989b) were used. Drinking water exposures are calculated using these assumptions and the following equation:

$$CDI = (C_w * IR * EF * ED * Z) / (BW * DY 8 YL) \quad \text{(Eq. 1)}$$

where:

CDI	=	chronic daily intake (mg/kg-day);
C_w	=	exposure point concentration in groundwater (μg/L);
IR	=	ingestion rate (2 liters/day);
EF	=	exposure frequency (365 days/year);
ED	=	exposure duration (30 years);
Z	=	conversion factor (mg/1,000μg);
BW	=	body weight over the period of exposure (70kg);
DY	=	days in a year (365 days/year); and
YL	=	period over which risk is being estimated (a lifetime [70 years] for potential carcinogens and the period of exposure [30 years] for noncarcinogens

CDIs calculated using these exposure assumption are presented in Table 12-6.

12.3.2 TOXICITY ASSESSMENT

The general methodology for the classification of health effects and the development of health effects criteria has been described in Chapter 4 to provide the analytical framework for the characterization of human health impacts. The health effects criteria that were used to derive estimates of risk for off-post residents ingesting groundwater are presented in Table 12-7. An oral toxicity criterion for iron is not available to derive estimates for off-post residents ingesting groundwater. Therefore, potential risks associated with exposure to iron were not quantitatively evaluated. However, a complete toxicity summary of iron is provided in Appendix B.

TABLE 12-5

EXPOSURE CONCENTRATIONS OF CHEMICALS OF POTENTIAL CONCERN AT
PHILLIPS ARMY AIRFIELD STUDY AREA: GROUNDWATER

(Concentrations reported in ug/L)

Chemical (a)	Arithmetic Mean	Upper 95 Percent Confidence Limit on the Arithmetic Mean (b)	Maximum Detected Concentration.	RME Concentration (c)
Organic Chemicals:				
Benzene (C6H6)	1.7	1.9	3.0	1.9
trans-1,2-Dichloroethene (T12DCE)	2.4	3.6	8.5	3.6
Methylene Chloride (CH2CL2)	2.4	3.7	8.0	3.7
Trichloroethene (TRCLE)	2.0	2.6	4.0	2.6
Vinyl Chloride (C2H3CL)	1.8	2.1	4.0	2.1
Inorganic Chemicals:				
Barium (BA)	100	170	245	170
Iron (FE)	2,500	740,000	14,250	14,250
Manganese (MN)	470	43,000	1,850	1,850

(a) USATHAMA chemical codes listed in parentheses.
(b) Values reflect a positively skewed distribution.
(c) Value listed is the lower of the upper 95 percent confidence limit on the arithmetic mean and the maximum detected value.

In addition to the chemicals analyzed for in groundwater, PCBs, pesticides, and herbicides were considered to be potentially of concern at the Phillips Army Airfield Landfill study area (see Table 12-3). A complete toxicity summary of PCBs is provided in Appendix B. Since it is not known exactly which pesticides and herbicides are present at the study area, toxicity summaries cannot be provided for these chemicals.

12.3.3 RISK CHARACTERIZATION

In this section, the human health risks associated with the Phillips Army Airfield Landfill study area under current land-use conditions are described. Risks were evaluated either quantitatively or qualitatively. To assess risks quantitatively, the CDIs calculated in Section 12.3.1 were combined with the toxicity criteria presented in Section 12.3.2.

The only potential exposure pathways evaluated in this risk assessment under current land-use conditions were (1) exposure to groundwater at the study area by off-post residents and (2) ingestion of game from the study area by hunters. As noted previously, exposure to groundwater at the study area by off-post residents was included as a conservative means of evaluating the potential impacts on off-site well fields if pumping conditions caused a gradient reversal at the site. The nearest well field is approximately 0.4 mile north of the Phillips Army Airfield Landfill study area.

Table 12-8 presents the estimated carcinogenic and noncarcinogenic risks associated with the ingestion of chemicals in groundwater from the Phillips Army Airfield Landfill study area by off-post residents. The upper-bound excess lifetime cancer risk is $6x10^{-5}$ under the RME exposure case, from vinyl chloride, a class A carcinogen. The upper-bound excess lifetime cancer risks for the other potential carcinogens range from $3x10^{-7}$ to $7x10^{-7}$. The Hazard Index for ingestion of groundwater by off-post residents under the RME exposure case is less than 1 (0.6). Off-post residents could be exposed via other pathways to the chemicals in groundwater from the Phillips Army Airfield Landfill study area. They could be exposed via inhalation to benzene, trans-1,2-dichloroethene, methylene chloride, trichloroethene, and vinyl chloride that have volatilized during activities such s showering. Dermal absorption of organic chemicals also could result during bathing. Exposure via these pathways would add to overall exposure and risk. The scientific literature on this subject indicates that the risk associated with these activities may be similar in magnitude to that associated with ingestion; the risks calculated for ingestion of these chemicals may, therefore, be doubled to estimate the importance of this effect.

Hunters could be at risk of exposure to chemicals by ingestion of game that has bioaccumulated chemicals at or originating from the Phillips Army Airfield Landfill study area. PCBs, pesticides, and herbicides, which tend to bioaccumulate in wildlife, are potentially present at the study area (see Table 12-3). The potential for significant exposure from ingestion of game is not expected since game hunted in the study area would probably only spend a small portion of its total foraging time at the Phillips Army Airfield Landfill study area.

12.4 ECOLOGICAL ASSESSMENT

This section describes the potential ecological impacts associated with the chemicals of potential concern at the Phillips Army Airfield Landfill study area in the absence of remediation. The methods used to assess ecological impacts follow those outlined in Chapter 4 and roughly parallel those used in the human health risk assessment. In this section, potentially exposed populations (receptors and potential exposure pathways are identified. Steps that normally follow, such as presentation of toxicity information and estimation of ecological impacts, were not included in this assessment due to a lack

TABLE 12-6

EXPOSURE POINT CONCENTRATIONS AND CHRONIC DAILY INTAKES FOR HYPOTHETICAL FUTURE INGESTION OF GROUNDWATER AT THE PHILLIPS ARMY AIRFIELD STUDY AREA (a)

Chemical (b)	RME Concentration (ug/L)	Estimated Chronic Daily Intake (CDI) (mg/kg-day) (c)
Chemicals Exhibiting Carcinogenic Effects		
Benzene (C6H6)	1.9	2.3E-05
Methylene Chloride (CH2CL2)	3.7	4.5E-05
Trichloroethene (TRCLE)	2.6	3.2E-05
Vinyl Chloride (C2H3CL)	2.1	2.6E-05
Chemicals Exhibiting Noncarcinogenic Effects		
trans-1,2-Dichloroethene (T12DCE)	3.6	1.0E-04
Methylene Chloride (CH2CL2)	3.7	1.1E-04
Trichloroethene (TRCLE)	2.6	7.4E-05
Barium (BA)	170	4.9E-03
Manganese (MN)	1,850	5.3E-02

(a) CDIs have been calculated only for those chemicals of potential concern with toxicity criteria. The following chemical of potential concern is not presented due to lack of toxicity criteria: iron.
(b) USATHAMA chemical codes listed in parentheses.
(c) See text for exposure assumptions.

TABLE 12-7

ORAL CRITICAL TOXICITY VALUES FOR CHEMICALS OF POTENTIAL CONCERN
AT PHILLIPS LANDFILL

Chemical	Chronic Reference Dose (mg/kg/day)	Uncertainty Factor (a)	Target Organ (b)	Reference Dose Source	Cancer Slope Factor (mg/kg/day)-1	EPA Weight of Evidence Classification (c)	Slope Factor Source
Organic Chemicals:							
Benzene	--	--	--	IRIS	2.90E-02	A	IRIS
trans-1,2-Dichloroethene	2.00E-02	1,000	Blood	IRIS	--	--	--
Methylene Chloride	6.00E-02	100	Liver	IRIS	7.50E-03	B2	IRIS
Trichloroethene	7.35E-03	1,000	Liver	HA	1.10E-02	B2	HEAST
Vinyl Chloride	--	--	--	--	2.30E+00	A	HEAST
Inorganic Chemicals:							
Barium	7.00E-02	3	Cardiovascular System	IRIS	--	--	IRIS
Iron	--	--	--	HEAST	--	--	--
Manganese	1.00E-01	1	CNS	IRIS	--	--	--

(a) Safety factors are the products of uncertainty factors and modifying factors. Uncertainty factors used to develop reference doses generally consist of multiples of 10, with each factor representing a specific area of uncertainty in the data available. The standard uncertainty factors include the following:
- a 10-fold factor to account for the variation in sensitivity among the members of the human population;
- a 10-fold factor to account for the uncertainty in extrapolating animal data to the case of humans;
- a 10-fold factor to account for the uncertainty in extrapolating from less than chronic NOAELs to chronic NOAELs; and
- a 10-fold factor to account for the uncertainty in extrapolating from LOAELs to NOAELs.

Modifying factors are applied at the discretion of the reviewer to cover other uncertainties in the data.

(b) A target organ is the organ most sensitive to a chemical's toxic effect. RfDs are based on toxic effects in the target organ. If an RfD was based on a study in which a target organ was not identified, an organ or system known to be affected by the chemical is listed.

(c) EPA Weight of Evidence for Carcinogenic Effects:
[A] = Human carcinogen based on adequate evidence from human studies; and
[B2] = Probable human carcinogen based on inadequate evidence from human studies and adequate evidence from animal studies.

NOTE: IRIS = Integrated Risk Information System - December 1, 1990.
HA = Health Advisory.
HEAST = Health Effects Assessment Summary Tables - July 1, 1990.
-- = No information available.

TABLE 12-8

POTENTIAL RISKS ASSOCIATED WITH HYPOTHETICAL FUTURE INGESTION OF GROUNDWATER
AT THE PHILLIPS ARMY AIRFIELD STUDY AREA (a)

Chemicals Exhibiting Carcinogenic Effects (b)	Estimated Chronic Daily Intake (CDI) (mg/kg-day)	Slope Factor (mg/kg-day)-1	Weight of Evidence Class (c)	Upper Bound Excess Lifetime Cancer Risk
Benzene (C6H6)	2.3E-05	2.9E-02	A	7E-07
Methylene Chloride (CH2CL2)	4.5E-05	7.5E-03	B2	3E-07
Trichloroethene (TRCLE)	3.2E-05	1.1E-02	B2	4E-07
Vinyl Chloride (C2H3CL)	2.6E-05	2.3E+00	A	6E-05
TOTAL	--	--	--	6E-05

Chemicals Exhibiting Noncarcinogenic Effects (b)	Estimated Chronic Daily Intake (CDI) (mg/kg-day)	Reference Dose (RfD) (mg/kg-day)	Uncertainty Factor (d)	Target Organ (e)	CDI:RfD Ratio
trans-1,2-Dichloroethene (T12DCE)	1.0E-04	2.0E-02	1,000	Blood	5E-03
Methylene Chloride (Ch2CL2)	1.1E-04	6.0E-02	100	Liver	2E-03
Trichloroethene (TRCLE)	7.4E-05	7.35E-03	1,000	Liver	1E-02
Barium (BA)	4.9E-03	7.0E-02	3	Cardiovasc. Sys.	7E-02
Manganese (MN)	5.3E-02	1.0E-01	1	CNS	5E-01
HAZARD INDEX	--	--	--	--	< 1 (6E-01)

(a) Risks are calculated only for chemicals with toxicity criteria. The following chemical of potential concern is not presented due to lack of toxicity criteria: iron.
(b) USATHAMA chemical codes listed in parentheses.
(c) EPA Weight of Evidence for Carcinogenic Effects:
[A] = Human carcinogen based on adequate evidence from human studies; and
[B2] = Probable human carcinogen based on inadequate evidence from human studies and adequate evidence from animal studies.
(d) Factor which reflects the uncertainty in the estimate of the RfD. Larger factors are associated with greater uncertainty.
(e) A target organ is the organ most sensitive to a chemical's toxic effect. RfDs are based on toxic effects in the target organ. If an RfD was based on a study in which a target organ was not identified, an organ or organ system known to be affected by the chemical is listed.

of applicable data. It is emphasized that the lack of applicable data limits the predictive value of this ecological assessment.

This ecological assessment is divided into two sections. Section 12.4.1 describes the habitat of the area and identifies the potential receptor species. Section 12.4.2 identifies the potential exposures for these species.

12.4.1 RECEPTOR SPECIES

The Phillips Army Airfield Landfill study area consists mainly of bare earth, mowed fields, and developed land. Habitat characteristics of the study area are shown in Figure 12-3. There is a small woodlot adjacent to the southeast side of the older landfill area, and a small non-tidal (palustrine forested) wetland east of the woodlot and southwest of the inactive landfill. The older landfill is covered with vegetation ranging from grasses to small trees.

Comprehensive field studies of ecological receptors and impacts have not yet been conducted in the Phillips Army Airfield Landfill study area, and no information about flora and fauna specific to the study area is available. However, the study area likely supports a variety of wildlife species. Species characteristic of disturbed and developed land are probably the predominant species in the study area, given the prevalence of this habitat type in the area. Probable resident mammalian species of the developed areas include woodchuck (*Marmota norvegicus*), eastern chipmunk (*Tamias striatus*), house mouse (*Mus musculus*), Norway rat (*Rattus norvegicus*), and grey squirrel (*Sciurus carolinenis*). Common bird species in these areas likely include mourning dove (*Zenaida macroura*), house sparrow (*Passer domesticus*), robin (*Turdus migratorius*), mockingbird (*Mimis polyglottos*), cardinal (*Cardinalis cardinalis*), blue jay (*Cyanocitta cristata*), and eastern kingbird (*Tyrannus tyrannus*). Numerous hawks and vultures probably forage in the open fields.

The small size of the woodlot near the Phillips Army Airfield Landfill study area restricts its value as wildlife habitat for larger animals. The woodlot may provide limited habitat for smaller mammalian species including grey squirrel, eastern chipmunk, opossum (*Didelphis virginiana*), striped skunk (*Mephitis mephitis*), and white-footed mouse (*Peromyscus leucopus*). Probable bird species include woodpeckers (*Dendrocopus* spp.), woodcock (*Philohela minor*), wood thrush (*Hylocichla mustelina*), and other songbirds. Salamanders and some amphibians may also be present in the woodlot.

A branch of Romney Creek drains the study area. Although the surface water at the study area is too shallow to support fish populations, a variety of aquatic invertebrates is likely to be present. The wetlands at the site likely support a number of mammal species, including beaver (*Castor canadensis*), muskrat (*Ondatra zibethicus*), and raccoon (*Procyon lotor*). The wetlands may provide foraging habitat for great blue heron (*Ardea herodias*), green heron (*Butorides striatus*), and other wading bird species, mallard (*Anas platyrhynchos*), black duck (*Anas rubripes*), and other waterfowl. Amphibians and aquatic and terrestrial invertebrates likely utilize the wetlands for some portions of their life-cycles.

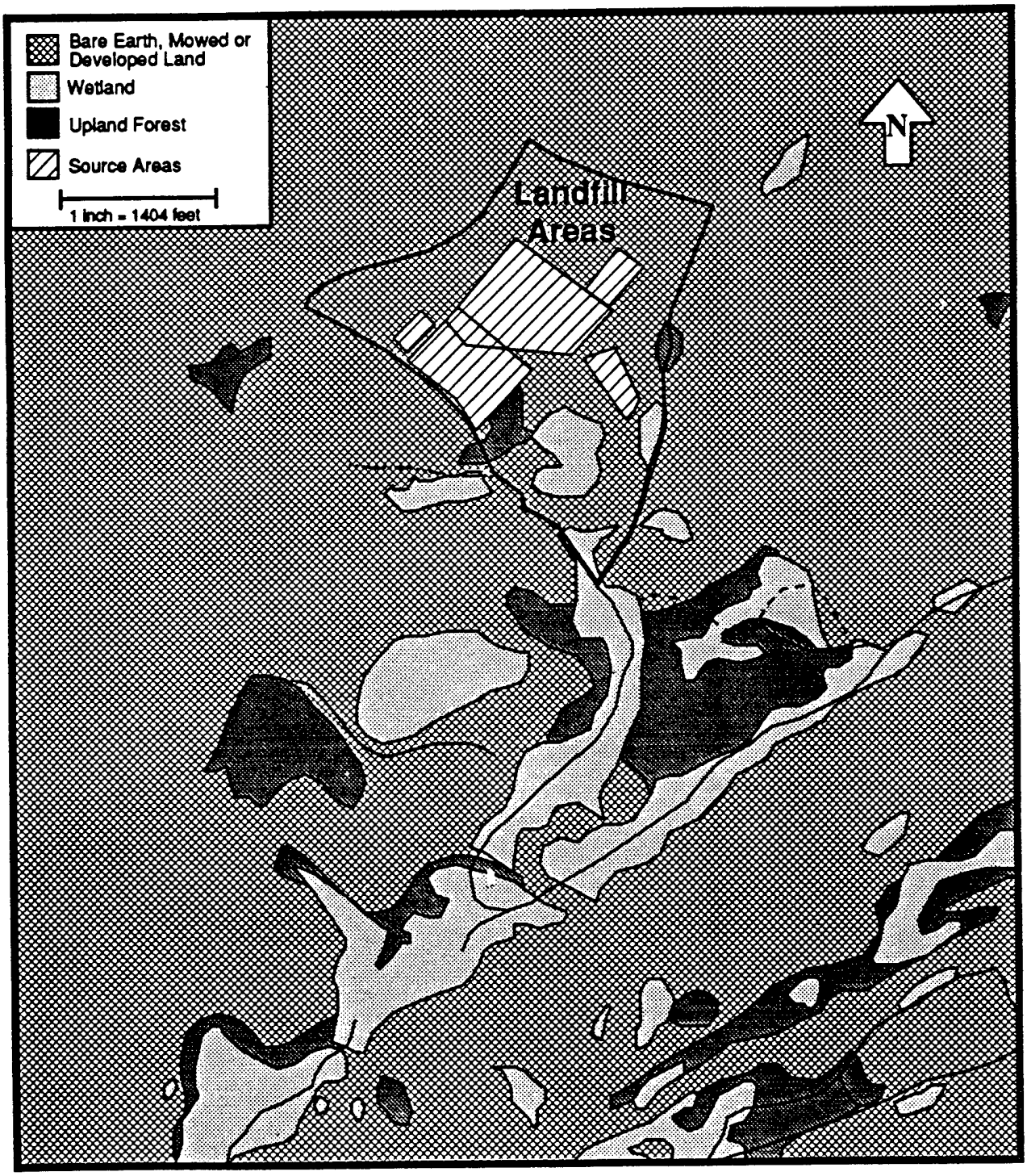
Appendix D provides species profiles for many of the vertebrate species mentioned above. These profiles contain information on the ecology of each listed species.

12.4.2 POTENTIAL EXPOSURE PATHWAYS

No pathways exist by which wildlife could be exposed to chemicals of potential concern in

Figure 12-3

Habitat Characteristics of the Phillips Army Airfield Study Area At Aberdeen Proving Ground



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groundwater, the only media for which sampling data are available[1]. Chemical may have drained from the site into surface water in the nearby wetland and Romney Creek, where aquatic wildlife may be exposed via contact with or ingestion of surface waters and sediments and ingestion of contaminated food. As discussed in Chapter 4, terrestrial wildlife can be exposed to chemicals in surface water, sediment, and surface soils by a variety of pathways. In the Phillips Army Airfield Landfill study area, terrestrial wildlife could be exposed to chemicals in soils by direct ingestion of contaminated soil (while foraging or grooming), sediment, or surface water; or by ingestion of food (plants and animals) that has accumulated chemicals from surface water or sediment.

The extent of exposure via these pathways depends upon the characteristics and concentrations of the chemicals present. Because no sampling data are available for any of these media, the impact of wildlife exposures via any of the potential pathways was not evaluated.

12.5 UNCERTAINTIES

As in any risk assessment, there is a large degree of uncertainty associated with the estimates of human health and ecological risk for the Phillips Army Airfield Landfill study area. Consequently, these estimates of risk should not be regarded as absolute estimates of risk, but rather as conditional estimates based on a number of assumptions regarding exposures and toxicity. A complete understanding of the uncertainties associated with the risk estimates is critical to understanding the true nature of the predicted risks and to placing the predicted risks in proper perspective. The principal sources of uncertainty associated with the APG risk assessments were discussed in general in Chapter 4. Some of the key sources of uncertainty associated with the estimates of risk for the Phillips Army Airfield Landfill study area are summarized below.

12.5.1 UNCERTAINTIES RELATED TO SELECTION OF CHEMICALS FOR EVALUATION

Currently, the limited sampling data is the primary source of uncertainty in this risk assessment. No study area data are available for soil, surface water, sediment, or air and, consequently, human health and/or ecological risks associated with exposure to any contaminants in these media could not be evaluated. Furthermore, groundwater contamination was not fully characterized, given samples were analyzed only for inorganic chemicals and purgeable organic priority pollutant compounds, even though other chemicals could be present. Additionally, samples were collected from only 10 wells, which may not be adequate to characterize the extent of contamination associated with the study area.

Because no site-specific or regional background data were available for groundwater, the site-relatedness of inorganic chemicals in groundwater was determined by comparing on-site chemical concentrations with national background data. As a result, inorganic chemicals that may not be site-related were associated with past activities at the Phillips Army Airfield Landfill study area. Consequently, including chemicals in this risk assessment that are present at natural levels could result in overestimates of impact associated with the study area.

Blank data were not available for the November 1988 data to determine whether the chemicals detected in groundwater in November 1988 were site-related or introduced in the field or lab. Estimating risk without these blank data may result in an overestimation.

---

[1] Groundwater released to surface water was evaluated as surface water exposure.

12.5.2 UNCERTAINTIES ASSOCIATED WITH THE MODELS AND ASSUMPTIONS USED TO ESTIMATE EXPOSURES

It is not possible to determine the impacts of on-site contaminants on groundwater pumped by the City of Aberdeen and Harford County well fields. Therefore, the quality of groundwater in the study area itself was evaluated. This provides a highly conservative estimate of such impacts because concentrations that might reach off-site wells would be much lower than on-site concentrations, given the dilution that would result from dispersion during transport to these wells and the fact that supply wells would draw groundwater from all directions. In addition, chemical concentrations in groundwater are unlikely to remain constant over the 30-year exposure period under current land-use conditions., which could either over- or underestimate risks.

The assumptions used to estimate intake in humans also contribute a great deal of uncertainty to the estimates of exposure and impact. Generally, conservative assumptions were used when estimating exposures, which may result in overestimation of actual exposures at the study area.

12.5.3 UNCERTAINTIES IN THE TOXICITY ASSESSMENT

Toxicological data uncertainties are associated with the lack of an oral toxicity criterion for iron. The overall effect of not evaluating iron would result in an underestimation of risk. However, given the conservative nature of the exposure parameters and the low toxicity of iron, the estimated risk from ingestion of groundwater is still likely to be an overestimation.

12.6 PRINCIPAL DATA NEEDS

Investigations to date have not provided a complete and exhaustive characterization of the type and degree of contamination at the Phillips Army Airfield Landfill study area. As a result, additional investigation is needed to assess more definitively existing or potential impacts associated with the study area. Specific data needs within these two categories are summarized below.

Data on the Nature and Extent of Contamination

- Surface soil, subsurface soil, surface water, and sediment sample should be collected form th study area to characterize the nature and extent of contamination. Soil, surface water, and sediment samples should be, as well as additional groundwater samples, should be analyzed for inorganic chemicals, volatile and semivolatile organic chemicals, pesticides, herbicides, and PCBs. Additional groundwater well should be installed so that the extent of contamination can be evaluated more completely. Total and dissolved concentrations in surface water samples should be measured.

- For each media sampled, background samples should also be collected for analysis. Background samples should be collected from locations with similar characteristics, but which are clearly not influenced by migration of site-related chemicals. In addition, a sufficient number of samples should be collected to permit statistical analysis.

- Information regarding the volume and contents of the older landfill, the burning pits, and the three disposal areas should be provided to determine whether the source areas are a continuing source of chemicals to groundwater and to determine additional chemicals for analysis.

- It should be determined if contamination originating at he Phillips Army Airfield Landfill could impact the City of Aberdeen or the Harford County well fields under conditions of increased pumping. Information is needed concerning groundwater discharge points so that current or potential extent of surface water contamination can be defined more accurately.

12.7 SUMMARY AND CONCLUSIONS

This baseline risk assessment addressed the potential impacts on human health and the environment associated with the Phillips Army Airfield Landfill study area in the absence of remedial actions. This assessment was based mainly on the hydrogeologic field investigation conducted by AEHA (1988, 1989), which provides the most recent sampling data collected in this area. Sampling data are available for groundwater only. Groundwater samples were analyzed for purgeable organic priority pollutant compounds and inorganic chemicals. Five organic chemicals were detected (mainly in one well located downgradient of the active landfill and the refuse burning pits) and selected as chemicals of potential concern: benzene, trans-1,2-dichloroethene, methylene chloride, trichloroethene, and vinyl chloride (although there is some doubt as to the actual presence of methylene chloride in groundwater). All organic chemicals detected were present at low concentrations (less than 10 μg/L) and at low frequencies of detection (1 or 2 out of 10). Barium, iron, and manganese were present in groundwater at concentrations above national background levels and were, therefore, selected as chemicals of potential concern. In addition to the chemicals of potential concern selected for groundwater. other chemicals are likely to be present in the Phillips Army Airfield Landfill study area which may be of potential concern regarding possible exposures and impacts. PCBs, pesticides, and herbicides are the principal wastes in the various source areas that could be present at the site and should be addressed in future analysis.

12.7.1 HUMAN HEALTH RISK ASSESSMENT SUMMARY

The only potential exposure pathways evaluated in this risk assessment under current land-use conditions were (1) exposure of off-post residents to groundwater at the study area and (2) ingestion of game from the study are by hunters. Other potential pathways by which human populations could be exposed to chemicals of potential concern under current land-use conditions at the site were not evaluated due to the lack of data. As noted previously, evaluation of exposure to groundwater at the study area by off-post residents was performed as highly conservative means of evaluating the potential impacts of on-site groundwater to off-site well fields if pumping conditions caused a gradient reversal at the site. (The nearest well field is approximately 0.4 miles north of the study area.) Chemical concentrations in groundwater reaching these off-post wells would be significantly lower than those detected at the site, given that dilution would occur during transport and the water supply wells would draw groundwater from all directions, not just from the vicinity of the study area.

Ingestion of groundwater by off-post residents was evaluated quantitatively and dermal contact and inhalation of chemicals in groundwater by off-post residents were qualitatively evaluated. Ingestion of game by hunters was evaluated qualitatively. Land-use under future conditions is not expected to differ from current land-use at the study area. Thus, no additional exposure pathways were evaluated under future land-use conditions.

The estimated human health risks associated with these pathways under current land-use conditions are as follows:

- The upper-bound excess lifetime cancer risk for ingestion of chemicals in ground

water from the Phillips Army Airfield Landfill study area by off-post residents is $6x10^{-5}$ due to vinyl chloride, and the upper-bound excess lifetime cancer risks for the other potential carcinogens range from $3x10^{-7}$ to $7x10^{-7}$. The Hazard Index for ingestion of groundwater by off-post residents is less than 1 (0.6). Off-post residents could be exposed via inhalation to benzene, trans-1,2-dichloroethene, methylene chloride, trichloroethene, and vinyl chloride that can volatilize during showering. Dermal absorption of these chemicals also could result during bathing. The scientific literature on this subject indicates that, for practical purposes, the risks calculated for ingestion of these chemicals can be doubled to estimate the importance of this effect. Information regarding the volume or contents of most of the source areas was not available.

- Hunters could be exposed to chemicals by ingesting game that has bioaccumulated chemicals at or originating from the Phillips Army Airfield Landfill study area. PCBs, pesticides, and herbicides tend to bioaccumulated in wildlife, and are potentially present at the study are. However, the potential for significant exposure from ingestion of game is not expected since game species hunted at the study area would probably spend only a small portion of their total foraging time on the site.

12.7.2 ECOLOGICAL ASSESSMENT SUMMARY

Potential exposure pathways were identified for aquatic and terrestrial wildlife at the Phillips Army Airfield Landfill study area. Exposures and impacts could not be evaluated due to a lack of appropriate sampling data. surface water, sediment, and surface soil samples should be collected and analyzed for the range of chemicals potentially present at the study area so that a more complete assessment of potential impacts to ecological receptors can be performed.

12.3.7 CONCLUSIONS OF THE RISK ASSESSMENT

Past activities at the Phillips Army Airfield Landfill have resulted in low-level contamination of groundwater with volatile organic chemicals, possibly reducing the value of this groundwater as a domestic water supply source. It is not known if other media in the study area have been similarly contaminated, as no other sampling data are available. However, given the wastes reportedly disposed of in the area, the potential for additional contamination exists. Further investigation is needed to permit a more complete characterization of the potential human health and ecological risks associated with the study area.

12.8 REFERENCES

ARMY ENVIRONMENTAL HYGIENE AGENCY (AEHA). 1988. Geohydrologic Study No. 38-26-0866-88. Phillips Army Airfield Landfill. Aberdeen Proving Ground, Maryland. 28-31 March and 28 April - 4 May 1988.

ARMY ENVIRONMENTAL HYGIENE AGENCY (AEHA). 1989. Phase 2 Geohydrologic Study No. 38-26-0866-88. Phillips Army Airfield Landfill. Aberdeen Proving Ground, Maryland. 15-16 November 1988.

ENVIRONMENTAL SCIENCE AND ENGINEERING, INC (ESE). 1981. Installation Assessment of Aberdeen Proving Ground- Aberdeen Area. Gainsville, Florida. Report No. 301.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Development of Risk Assessment Methodology for Municipal Sludge Landfilling. Environmental Criteria and Assessment Office. ECAO-CIN-485.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989a. Exposure Factors Handbook. Office of Health and Environmental Assessment. Washington, D.C. EPA/600/8-89/043.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989b. Risk Assessment Guidance for Superfund. Volume I: Human Health Evaluation Manual. Interim Final. Office of Emergency and Remedial Response. Washington, D.C. OSWER Directive 9285.7-01a.

WALTON, W.C. 1985. Practical Aspects of Groundwater Modeling. National Water Well Association. Worthington, Ohio.

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13.0 BASE-WIDE RISK ASSESSMENT

Previous chapters of this report have addressed potential risks to human and ecological receptors at eight study areas at APG. The purpose of this chapter is to discuss the potential impacts of these study areas on the overall health of aquatic and terrestrial wildlife populations at APG. Such an evaluation is important because no single study area alone is likely to significantly affect the entire APG population of a particular species. This is because populations of most species at APG are not contained within a given study area, but instead are distributed over significantly larger areas. Thus, the potential for population-level effects depends upon the proportion of the total population that exists in the contaminated area. Generally, the potential for population-level impacts in a given species increases along with the size of the contaminated area. Other factors such as life history and foraging strategies result in differences in the potential for population impacts in different species.

The results of the ecological assessments for the eight study areas being evaluated in this assessment are discussed below in the context of the potential cumulative impact of these areas on APG's wildlife population. Impacts on aquatic and terrestrial wildlife populations are discussed separately, followed by a discussion of potential impacts in the two endangered species living at APG (the bald eagle and peregrine falcon).

13.1 POTENTIAL POPULATION IMPACTS ON AQUATIC LIFE AT APG

The five sites along the Gunpowder River (J-Field, O-Field, Canal Creek, Carroll Island, and Graces Quarters) have the greatest potential for impacts on aquatic populations at APG. These sites run the entire length of the Gunpowder River, and collectively could affect a significant portion of the river and its associated populations. The other study areas (the Nike site, Michaelsville Landfill, and Phillips Army Airfield) investigated in this report are unlikely to contribute to the impacts on the aquatic population associated with the study areas on the Gunpowder River, given their geographical separation from these areas and their location in different drainage basins. Furthermore, the Michaelsville Landfill and Phillips Army Airfield provide minimal aquatic habitat.

Aquatic life impacts were predicted for each of the five study areas along the Gunpowder River. Because of sampling data limitations, the assessments were limited to evaluations of impacts in the waters on or immediately adjacent to each study area, and not in extensive areas of the Gunpowder River. However, each study area supports a proportion of the total aquatic life populations in the river, and therefore, impacts at each study area contributes to cumulative impacts in the river. The degree of impact depends on the proportion of the population that occurs in and adjacent to these study areas. Impacts would be greatest if large areas of the Gunpowder River near these study areas have been contaminated because then, a greater proportion of the aquatic life populations of the river could be impacted. It is possible that large portions of the Gunpowder River near Canal Creek are contaminated as a result of past waste disposal practices in the Canal Creek area, but no sampling data are available.

Potential contamination could be particularly critical to fish species that use the Gunpowder River as a spawning and nursery area, because impacts on survival of larval and juvenile (i.e., young-of-the-year) fish can affect the ability of a population to maintain itself by decreasing the number of individuals that enter the population each year. Fish species that use the Gunpowder River as a spawning and/or nursery area include American shad, alewife, blueback herring, menhaden, bay anchovy, and white perch (Lippson 1985).

Population response to reductions in recruitment depends upon the life history strategy of the species of concern. Barnthouse et al. (1990) demonstrated that long-lived species such as striped bass, which have high variability in annual recruitment, are much more susceptible to impacts on the survival of young-of-the-year than shorter-lived species such as menhaden, which have less variability in annual recruitment. Based on this factor, white perch and American shad may be at a significantly greater risk of population impacts from contaminant exposure than other species that use the Gunpowder River as a spawning and/or nursery area. American shad is probably particularly vulnerable to any additional reductions in recruitment. This species suffered significant population declines in the 1970s, which led to the closing of the fishery in Maryland in 1980 (FWS 1986).

13.2 POTENTIAL POPULATION IMPACTS ON TERRESTRIAL WILDLIFE AT APG

As discussed above, the potential for population-level effects generally depends upon the proportion of the total population that is present in the contaminated area. If contamination is localized, population-level impacts are less likely because a relatively small percentage of the total population is likely to be impacted. The potential for population impacts increases as the size of the contaminated area occupied by a population increases compared to the size of the uncontaminated area.

The six Edgewood Area sites (J-Field, O-Field, Canal Creek, Carroll Island, Graces Quarters, and the Nike site) collectively cover such an extensive area that the potential exists for cumulative impacts on wildlife populations of this area. The two Aberdeen Area sites (the Michaelsville Landfill and Phillips Army Field) are much smaller in area than the Edgewood sites and geographically distant from the Edgewood Area sites. For these reasons, their impacts would not be likely to add significantly to the total population-level impacts on terrestrial wildlife.

Wide-ranging species such as great blue heron would potentially be at greatest risk of population impacts, because, given the larger home range of such species, a significant portion of the populations' range could be contaminated, whereas only small portions may be uncontaminated. Potential impacts in heron were evaluated for the O-Field and Canal Creek study areas. (Heron exposures in other study areas could not be evaluated because too few data were available on contaminant levels in marshes or other surface waters used by this species.) No impacts were predicted for herons spending 10% of their time at O-Field or Canal Creek alone. However, when the chemical exposures for O-Field and Canal Creek are summed, the predicted heron exposures begin to approach the toxicity values estimated for this species. For example, assuming a heron feeds at both O-Field and Canal Creek, the total intake of cadmium approaches the heron toxicity value, with an intake:toxicity value ratio of 0.6. If heron were to feed in any other contaminated area, the total cumulative exposure could exceed the toxicity value. Because a large number of heron could use the Edgewood area, potential population impacts in this species could be possible.

Species with significantly smaller home ranges (e.g., muskrat, sandpipers) are more likely to be impacted by any single study area. However, because most of the remaining population is found in uncontaminated areas, localized reductions are less likely to affect the population as a whole. This prediction, however, is based on the assumption that there are large portions of the Edgewood area that are uncontaminated. If the size of the contaminated area at Edgewood is large, population impacts on these less wide-ranging species could be possible.

13.3 POTENTIAL IMPACTS ON ENDANGERED SPECIES AT APG

The bald eagle and peregrine falcon are the two endangered species living at APG. (Appendix D provides complete species profiles for these two species.) Potential impacts on these species were not evaluated as part of the individual risk assessments, because these species range over extremely large areas and therefore, are unlikely to be impacted by exposures at any single study area. However, exposures to chemical contamination across a much larger area (i.e., base-wide) has a greater potential to be significant.

Of these two species, the bald eagle is most susceptible to potential contamination at APG as this species feeds largely on fish, which often accumulate chemicals to much higher levels than occur in the surrounding environment. Peregrine falcons feed primarily on other birds, and because few chemicals accumulate significantly in the terrestrial food chain[1], impacts on peregrine falcons are less likely.

A large portion of the land area of APG can be classified as optimum breeding habitat for bald eagles. More than 80% (66,000 acres) of the 79,000 total acres of land area on the installation is unimproved (Pottie 1986). Active nests have been recorded at APG since 1936. More recent data suggest that from up to three nests may be active in a given year (Millsap et al. 1983, Buehler et al. 1987). Traditional nest sites are located on the Edgewood peninsula and in the Aberdeen Area near Romney Creek, the Trench Warfare area, and on Spesutie Island. APG also is an important foraging and roosting area for the bald eagle population of the northern Chesapeake Bay, and it may be used by as much as 17% of the bald eagles wintering in the Chesapeake Bay (Millsap et al. 1983, Buehler et al. 1987). High-use areas on APG include Romney Creek, Mosquito Creek, Spesutie Island, the lower half of the Edgewood peninsula, and APG's Chesapeake Bay shoreline (Buehler et al. 1987).

O-Field and J-Field are the two study areas likely to be used by bald eagles. Bald eagles could be exposed to contaminants associated with these areas via the ingestion of fish that have accumulated chemicals.

Endangered species, by definition, are species whose breeding populations are so low or whose population numbers have declined so severely that extinction is possible at some time in the future. Species whose populations are already severely stressed can be expected to have a reduced ability to recover from the impacts of environmental contaminants. Theoretically, impacts on even a single member of a population of endangered species could negatively affect the population as a whole. For this reason, evaluation of potential impacts on endangered species focus on potential impacts on the individual.

Impacts on individual bald eagles from exposure to contaminants at O-Field and J-Field are considered highly unlikely because these areas represent a minute fraction of the total home range area of bald eagles. Home range sizes of eagles radio-tracked at APG ranged from 21 km^2 to 66 km^2 in the late spring and summer and between 63 km^2 to more than 17,000 km^2 in autumn to early spring (Buehler et al. 1987). Impacts could occur if significant portions of the areas of APG used by bald eagles were contaminated. However, data on the breeding success of bald eagles at APG do not indicate that populations are being impacted. In fact, nesting success appears to have increased in recent years. Only two young fledged from 1977 to 1982; nine fledged from 1983 to 1985 (Millsap et al. 1983, Buehler et al., 1987).

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[1]The principal exceptions to this are highly hydrophobic organic compounds such as PCBs, dioxins and DDT, which are not expected to be widespread contaminants at APG.

Based on this analysis, impacts on bald eagles from cumulative exposures to contaminants associated with the study areas being evaluated in this assessment do not appear likely.

13.4 CONCLUSIONS

It is possible that aquatic populations in the Gunpowder River are being impacted by the cumulative exposures to contaminants associated with the five Edgewood Area sites that span the entire length of the Gunpowder River. Impacts would be greatest if large areas of the Gunpowder River near these study areas were contaminated, because a greater proportion of the aquatic life populations of the river could be impacted. However, the extent of contamination in the Gunpowder River is unknown. Contaminant-induced impacts could be particularly critical to fish species that use the Gunpowder River as a spawning and nursery area, e.g., American shad and white perch.

Impacts on terrestrial wildlife populations are possibly associated with all six Edgewood Area sites since collectively they cover such an extensive area. Wide-ranging species such as great blue heron potentially could be at greatest risk of population impacts because a significant portion of the population's range could be present in a contaminated area. Species with significantly smaller home ranges are less likely to suffer population impacts.

Bald eagles and peregrine falcons are unlikely to be impacted from cumulative exposures to contaminants associated with the study areas being evaluated in this assessment.

13.5 REFERENCES

BARNTHOUSE, L.W., SUTER, G.W., and ROSEN, A.E. 1990. Risks of toxic contaminants to exploited fish populations: influence of life history, data uncertainty and exploitation intensity. Envrn. Toxicol. Chem. 9:297-311.

BUEHLER, D.A., FRASER, J.D., and CHASE, J.D. 1987. Bald eagle movements, distribution, and abundance on the northern Chesapeake Bay. Report prepared for the U.S. Army Chemical Research and Development Center, Aberdeen Proving Ground, Maryland, January 15, 1987.

FISH AND WILDLIFE SERVICE (FWS). 1986. Chesapeake Species of Special Emphasis: Shad (Alosa sapidissima). Annapolis, Maryland. FWS-CB-SSE1.

LIPPSON, A.J. 1985. The Chesapeake Bay in Maryland. An Atlas of Natural Resources. Johns Hopkins University Press. Baltimore, Maryland.

MILLSAP, B.A., CLINE, K.W., and GILROY, M.J. 1983. Distribution and movements of bald eagles on Aberdeen Proving Ground, Maryland. Report prepared for the U.S. Army, Aberdeen Proving Ground, Maryland, March, 1983.

POTTIE, J.J. 1986. U.S. Army Aberdeen Proving Ground eagle plan. Report prepared for the U.S. Army, Aberdeen Proving Ground, Maryland, December, 1986.

14.0 SUMMARY AND CONCLUSIONS

This chapter summarizes the principal findings of the risk assessments for each of the eight study areas of APG and provides an overview of the additional data needed to support more complete evaluations of potential human health and environmental impacts at the eight study areas. The chapter concludes by prioritizing the eight study areas for further study.

14.1 OVERVIEW OF THE SCOPE AND LIMITATIONS OF THE RISK ASSESSMENTS

The preceding chapters of this report presented baseline risk assessments for eight priority areas of known or suspected chemical contamination at APG. These risk assessments provided information on potential adverse effects to human and ecological receptors associated with chemical contamination at these sites in the absence of any remediation, including active cleanup measures and any institutional/access controls other than those that are in place at the present time. The risk assessments are intended to meet the requirements of the National Contingency Plan (NCP) (EPA 1990) for the evaluation of baseline conditions at uncontrolled hazardous waste sites and were performed according to the most recent EPA guidance regarding human health and ecological assessments.

As discussed throughout this report, these baseline risk assessments should be considered preliminary. Although they address a wide range of potential adverse effects, they are based in most instances on limited and incomplete chemical data and site characterization information, and are therefore subject to many limitations and uncertainties. The baseline risk assessments are most useful for focusing additional data-gathering efforts on chemicals, exposure pathways, and receptors of greatest potential concern and to provide a preliminary framework for ranking major APG sites using a risk-based approach (rather than perceived threat), so that appropriate decisions regarding further investigation and/or remediation priorities can be made.

14.2 SUMMARY OF FINDINGS FOR EACH STUDY AREA

This section summarizes the principal findings of the risk assessments for the eight individual study areas.

14.2.1 O-FIELD

O-Field is a former test range and ordnance disposal area that includes a chemical munitions/hazardous waste landfill and several open-burning pits that were used for the disposal of chemical agents and other hazardous materials. These past activities have resulted in significant chemical contamination of groundwater, surface water, and sediment in the area. Few data are available on chemical concentrations in soils in the area, but soil contamination is probably significant. Large quantities of wastes are known to be present in subsurface pits and trenches in the area. These wastes are likely to be a continuing source of groundwater contamination.

Under current land-use conditions, ecological populations are the principal receptors of concern; few human health exposure pathways exist. Only the release of lethal chemical agent as a result of an

explosion or spill at O-Field is likely to be associated with increased human health risks. Additional human health risks would result if use restrictions at O-Field were lifted in the future.

It is possible that the aquatic life in Watson Creek and the Gunpowder River and terrestrial wildlife feeding in Watson Creek are being adversely affected by chemical contamination associated with O-Field. Existing contamination in surface water and sediments, as well as continuing discharge of contaminated groundwater to surface water, is contributing to potential impacts.

Acute and chronic toxicity in O-Field surface waters probably has affected the composition and structure of the aquatic communities in Watson Creek and possibly the Gunpowder River near O-Field. Localized reductions in species diversity and number of resident aquatic life (particularly in Watson Creek) are possible, as are impacts on nonresident species that use the area as a nursery. Terrestrial wildlife feeding in Watson Creek appears to be at risk from exposure to heavy metals. The presence of heavy metals in Watson Creek probably has reduced the value of that area as wildlife habitat.

14.2.2 J-FIELD

J-Field is an open-burning/open-detonation area used for disposal of toxic chemical agents, white phosphorus, and organic solvents. Past activities at J-Field may have resulted in significant contamination of soil, groundwater, surface water, and sediment in the area. Few data are available on chemical concentrations in these media, but widespread contamination is likely given the history of activities in the area.

Under current land-use conditions, ecological populations are the principal receptors of concern. Some human exposure pathways exist, but the available data are not adequate to assess potential risks. It is possible that aquatic life in the Gunpowder River and the adjacent Chesapeake Bay is being adversely affected by chemical contamination associated with J-Field. However, too few data are available to assess the extent and magnitude of these impacts. Shorebirds feeding in the Gunpowder River do not appear to be at risk from exposure to heavy metals in the diet. However, chemicals not analyzed for in surface water and chemicals present in surface soil at J-Field could be present at levels which are impacting wildlife.

14.2.3 CANAL CREEK

Canal Creek is a watershed (including extensive wetlands) encompassing the majority of APG's former chemical agent, smoke and incendiary, and protective-clothing manufacturing operations, and includes more than 30 potential contamination source areas. Past activities in the Canal Creek study area have resulted in significant contamination of groundwater, surface water, and sediment. Surface soils in the former manufacturing area also are contaminated. Furthermore, significant contamination of subsurface soils is likely, given past waste disposal practices in the area. Large quantities of wastes possibly still present in subsurface environments could act as a continuing source of groundwater contamination in the area.

Both human and ecological risks may possibly be associated with contamination in the Canal Creek study area. Potential human health risks associated with the Canal Creek study area under current land-use conditions cannot be fully evaluated at this time. However, persons working in the former manufacturing area do not appear to be at significantly increased health risks from contacting surface soil in this area. Other possible long-term exposure pathways such as inhalation exposures of persons living and working in the area and ingestion of fish or game that has accumulated chemicals

cannot be evaluated at this time given the limited data available to support such evaluations. Workers or other individuals involved in activities involving subsurface excavation (such as maintenance of the many sewers in the area) could be exposed to acute hazards if unexploded ordnance or chemical agents were encountered. The potential for such an event is small, however, given the steps that are taken by APG to control exposures during excavation activities. Additional human health risks could result if groundwater were used in the future for drinking water or for industrial purposes.

It is possible that aquatic life in Canal Creek and terrestrial wildlife feeding in Canal Creek could be adversely affected by existing contaminant levels. Acute and chronic toxicity probably has affected the composition and structure of the aquatic communities in Canal Creek. Localized reductions in species diversity and species numbers are possible, as are impacts on nonresident species that use the area as a nursery area. Wildlife feeding in Canal Creek appears to be at risk from exposure to heavy metals in the diet, possibly resulting in localized reductions in population size. The presence of heavy metals in Canal Creek has probably reduced the value of that area as wildlife habitat.

14.2.4 CARROLL ISLAND

Carroll Island is a former test range used for open-air testing of nerve agents, incapacitating agents (e.g., tear gas), and smoke and incendiary munitions. In addition, several former open-burning areas, test sites, and possible disposal pits have been identified in this area. Past activities at Carroll Island have resulted in low-level contamination of the groundwater and surface water of the area. The extent of contamination in other media at the site is currently unknown but some additional contamination is likely given past activities in the area.

Few human health exposure pathways exist under current land-use conditions. Currently, hunters and trappers are the only potentially exposed human populations at Carroll Island. Only acute exposures to chemical agents or large quantities of supertropical bleach are associated with potentially significant human health hazards. Additional human health risks could be possible if human use of Carroll Island changed in the future. In particular, use of groundwater from the area may be associated with human health risks, although the site-relatedness of the chemicals driving these risks (i.e., methylene chloride and thallium) is questionable at the present time.

Aquatic life in seasonal ponded areas and other surface water habitats at Carroll Island could be affected by exposure to metals. Impacts associated with seasonal surface waters are unlikely to significantly affect the seasonal aquatic populations of Carroll Island (e.g, insects, frogs) given the wide availability of more suitable habitat across the island. Similar effects in the marshes of Carroll Island or in adjacent Saltpeter Creek and the Chesapeake Bay could result in more significant ecological impacts. However, this cannot be evaluated because samples that were collected from these waters are not sufficient to characterize potential ecological impacts in these areas.

Sandpipers or other shorebirds feeding near the EPG dump site or in eastern Carroll Island could be affected by dietary exposures to metals. Given the size of the eastern Carroll Island study area, it potentially could support a large number of spotted sandpipers, as this species has a relatively small home range area. Thus, toxic dietary levels of metals across this area could impact a significant number of sandpipers, resulting in local population impacts.

14.2.5 GRACES QUARTERS

Graces Quarters is an open-air testing area for munitions and chemical agents that also contains several potential hazardous waste burial pits and open-burning areas. Past activities at Graces Quarters have resulted in contamination of groundwater and surface water of the area. The extent of contamination in other media is currently unknown.

Few human exposure pathways exist under current land-use conditions, and those that do exist are unlikely to result in significant exposures or risks. Additional human health risks could be possible if human use of Graces Quarters changed in the future. In particular, use of groundwater from the area could be associated with significant human health risks.

Aquatic life in seasonal on-site ponded areas and along the Gunpowder River shoreline could be impacted by chemicals present in these surface waters. Impacts associated with the on-site ponded areas are unlikely to significantly affect seasonal aquatic populations at Graces Quarters (e.g, frogs, insects), given the wide availability of more suitable aquatic habitat across Graces Quarters. The extent of aquatic life impacts on the Gunpowder River cannot be evaluated at this time because the extent of contamination in the river is unknown. Significant impacts could result if contamination extends far out into the river; much smaller impacts are likely if contamination is limited to the shoreline area. Impacts on terrestrial wildlife living or feeding at Graces Quarters could not be evaluated because of insufficient sampling data.

14.2.6 NIKE SITE

The Nike site is the location of a former Nike Ajax and Hercules ballistic missile site (including launch and control areas) that contains areas of suspected waste disposal and leaking fuel storage tanks. The Nike missile site was constructed on some of the school fields used by the U.S. Army Chemical School for training in chemical warfare activities from 1920 to 1951. Past activities at the Nike site have potentially resulted in significant contamination of some environmental media at the site. However, the limited chemical data that have been collected are not adequate to characterize the site.

Few human exposures are expected to occur at the site under current land-use conditions given the infrequent use of most areas. It is possible that groundwater from the site is moving off-post and thus could affect some private drinking water wells in the nearby residential area. However, the hydrogeology of the site has not been characterized sufficiently to determine if contaminated groundwater is migrating off-post. Nevertheless, because the potential exists for off-post transport of contaminants to drinking water wells, the value of the groundwater at the site as a drinking water resource was evaluated. Use of groundwater at the site for drinking water would likely result in adverse health impacts.

Based on the limited data available, no risks are expected to aquatic organisms from exposure to sediments in the launch area drainage ditches. Potential risks to terrestrial wildlife receptors could not be adequately evaluated in this assessment due to the very limited data available.

14.2.7 MICHAELSVILLE LANDFILL

The Michaelsville Landfill is a sanitary landfill suspected also to contain paint sludges, metals, pesticides, PCBs, and other hazardous wastes. Past waste disposal at the site has resulted in groundwater contamination. Subsurface soils also are likely to be contaminated.

There are few pathways by which humans could be exposed to site chemicals under current land-use conditions. Although chemicals have been detected at the landfill (PCBs, pesticides) that could accumulate in game species, ingestion of game caught at the landfill is unlikely to result in significant human health risks. There are no current or expected future uses of groundwater at the landfill. However, its value as a potable water supply source was evaluated because groundwater in nearby off-post areas is used for domestic water supplies. Use of groundwater at the site for drinking water would likely result in adverse health impacts.

Some of the chemicals present in surface water could pose an increased risk of adverse acute and chronic effects in more sensitive aquatic invertebrates and insects at the Michaelsville Landfill. Additionally, bioaccumulation of selenium through the food chain could adversely affect terrestrial wildlife feeding in the area.

14.2.8 PHILLIPS ARMY AIRFIELD

The Phillips Army Airfield study area includes a sanitary/construction-debris landfill and several other potential disposal sites, including open-burning areas and "grease pits" used for disposal of food wastes, petroleum products, and transformer fluids containing PCBs. The contents of several of the disposal sites and the open-burning areas are unknown. Past activities in the Phillips Army Airfield study area have resulted in contamination of the groundwater with low levels of volatile organic chemicals and possibly some inorganic chemicals (the only chemicals analyzed for). It is not known if other media are contaminated as no other sampling data are available. However, given the wastes reportedly disposed of in the area, the potential for additional contamination exists.

Few human exposure pathways exist under current land-use conditions and those that do exist are unlikely to result in significant human health risks. There are no current or expected future uses of groundwater at Phillips Army Airfield. Nevertheless, the value of this groundwater as a potable water supply source was evaluated because groundwater in nearby off-post areas is used for domestic water supplies. Use of groundwater at the site for drinking water would likely result in adverse health impacts. Ecological impacts could not be evaluated because only groundwater data have been collected.

14.3 DATA NEEDS

The baseline risk assessments presented in this report were based entirely on existing data for the eight priority areas. No additional environmental sampling was conducted in support of these risk assessments. The reliance on existing data has limited the baseline risk assessments to some extent in that many complex and potentially complete exposure pathways could not be evaluated based on the available data. As has been discussed in each of the eight risk assessments, additional data are needed to permit a more complete and definitive evaluation of potential human or ecological impacts associated with each study area.

Although each study area has its own set of principal data needs, as outlined in each risk assessment, the types of data needed to enable more complete evaluations of risk fall generally into two categories: (1) chemical data of sufficient quantity and quality to define more fully the nature and extent of contamination and the potential for human health and ecological impacts; and (2) information on aquatic and terrestrial wildlife exposures and/or impacts (i.e., bioassessment). General data needs within these two categories are outlined below.

Data on the Nature and Extent of Contamination

- Chemical data used in risk assessments should be collected and analyzed according to strict quality assurance/quality control (QA/QC) protocols and include analyses of trip blanks, matrix spike, and other QA/QC samples. Data that do not meet strict QA/QC protocols are less reliable for use in risk assessments and are less defensible when supporting decisions on the need for remediation and on the type of remediation called for.

- Background samples are needed for all media to evaluate the site-relatedness of naturally occurring chemicals. This may be a particularly critical data need because inorganic chemicals drive the predictions of ecological risk at many of the study areas investigated in this report. A sufficient number of background samples are needed to permit a statistical evaluation. Background samples should be collected from environments similar to those at the study area of concern.

- A greater number of samples is needed in most study areas and in most media to better characterize the extent of contamination.

- Samples for all environmental media should be analyzed for the full range of contaminants that may be present. This is particularly critical for the Edgewood Area sites, given the wide variety of military-unique compounds that could be present. A complete characterization of the types of contaminants present is needed before human health or ecological impacts can be fully evaluated. Samples from some of the study areas (e.g., O-Field and J-Field) were analyzed for a fairly complete suite of potential contaminants, whereas others (e.g., Phillips Army Airfield) were analyzed only for a limited number of potential contaminants.

- Time-equivalent data sets are needed for the various study areas so that short-term and seasonal changes in chemical concentrations can be understood and impacts associated with these changes properly evaluated. For example, information is needed on daily and seasonal changes in groundwater discharge to surface waters at O-Field and J-Field, and on sediment loadings and chemical concentrations in Canal Creek following storm events.

Data Related to Wildlife Exposures and/or Impacts

For several study areas, additional data related to wildlife exposures and/or impacts (i.e., bioassessments) would result in a more complete evaluation of potential impacts. For the most efficient use of resources, it is recommended that bioassessment work at a given study area not begin until that area has been reasonably well characterized with respect to the type, location, and extent of contamination (e.g., O-Field, Canal Creek, J-Field). In these instances, bioassessment work can be targeted to address the particular concerns at the study area. For some study areas (e.g., Carroll Island, Graces Quarters) it is not possible to recommend particular bioassessment approaches because too little is currently known about contamination in the area. Potentially relevant bioassessment work is outlined below. The risk assessments for each study area should be reviewed for an identification of bioassessment work (if any) that is recommended for each study area.

- Fish and invertebrates (both benthic and water-column species) should be collected from selected areas for whole-body residue analyses. Efforts should be made to collect resident fish species and year-round and seasonal (i.e., aquatic insect larvae) populations of invertebrates. Bottom-feeding and predatory species should be included in the fish samples. Analytes ideally should encompass the range of chemicals that are potentially present in the

area and that have the potential to bioaccumulate in aquatic life. Suitable background samples are needed for evaluation of results.

- Aquatic macrophytes should be collected in selected areas for residue analysis. Again, analytes should include all chemicals potentially present that could accumulate in plants. Suitable background samples are needed for evaluation of these results.

- Surveys of benthic macroinvertebrate species should be conducted in some study areas to provide an indication of the nature of impacts (if any) on sediment-dwelling species. Studies should include benthic species samples from a suitable background location. Possible evaluation endpoints include species number and species diversity.

- Sediment toxicity studies are needed in some study areas to assess impacts associated with chemicals adsorbed onto sediments as well as chemicals discharged from groundwater through sediments and present in sediment pore water. Suitable laboratory and field controls are needed.

- Aquatic toxicity tests are needed in some study areas to assess the toxicity of surface water. Tests should include acute toxicity tests and chronic or early life-stage tests with invertebrates, algae, and fish.

14.4 PRIORITIZATION OF STUDY AREAS FOR FURTHER INVESTIGATION

As discussed in Chapter 1, an important aspect of the baseline risk assessments presented in this report is their identification of the site conditions (chemicals, exposure pathways, receptors) of greatest potential concern, so that subsequent studies, investigations, and cleanup measures can be focused in these areas. This aspect of the risk assessments is considered especially important for APG because more than 700 individual areas of potential contamination sources have been identified, and overall cleanup costs are likely to be extremely high.

At the outset of this project, it was hoped that a determination could be made, at least for some of the eight study areas, that either no remedial action was needed, or if some remediation was needed, the type of remediation needed could be identified. For some sites and some media this was almost possible. However, even for groundwater, the most extensively sampled medium, the estimates of risk in this report are considered only preliminary because of the incomplete nature of the chemical analyses conducted on most samples.

Nevertheless, the risk assessments and other information on the study areas do provide sufficient information to permit a general prioritization of sites with respect to the need for further investigation. The following factors were considered to be relevant for prioritization purposes:

- The potential for human exposures to occur (both on-site and off-site);

- The habitat value of the study area and the sensitivity of potentially impacted ecological receptors;

- The degree of risk posed to human and ecological receptors;

- The size of the potentially contaminated area;

- The amount of hazardous material remaining at the site; and
- The amount of additional data needed to characterize risk.

Consideration of these factors resulted in the following prioritization of the sites (given in decreasing order) for further investigation. In determining this ranking, it was necessary to make value judgments about the importance of the various factors, thus introducing an element of subjectivity to the ordering.

1. Canal Creek: This study area is of great concern because large quantities of wastes have been disposed of in the past and probably still remain in place, thereby acting as a continual source of groundwater contamination. Furthermore, the size of the contaminated area could be large, potentially encompassing a significant portion of the Canal Creek watershed. Also, the potential exists for significant contamination of the Gunpowder River near and downgradient of the mouth of Canal Creek as a result of past disposal of large quantities of wastes in Canal Creek, which flows into the river. Additionally, this is the only study area where long-term human exposures could possibly be significant under current land-use conditions. Finally, significantly more data are needed for this study area (particularly surface water and sediment data for Canal Creek and the Gunpowder River).

2. O-Field: This study area is of great concern because past waste disposal has resulted in significant contamination of the groundwater, surface water, and sediment in the area. Large quantities of wastes still exist and are acting as a continual source of groundwater contamination, thus resulting in continual discharge of contaminants to Watson Creek. Furthermore, the area surrounding O-Field is ecologically rich.

3. J-Field: This area is of great concern because contaminated groundwater may discharge directly to the Chesapeake Bay. Large quantities of solvents and chemical agents were disposed of here in the past, and it is possible that some solvents still exist as free product and are acting as a continual source of contaminants in the groundwater.

4/5. Carroll Island and Graces Quarters (equal): These areas are of moderate concern because prior testing of chemical agent could have resulted in contamination across significant portions of the study area. However, contaminant levels are probably low. Few potential sources of concentrated wastes likely remain. Both areas are ecologically rich.

6. Nike Site: This area is of moderate concern because it is located near the APG boundary and is adjacent to off-post residential areas. However, this study area probably has resulted only in low-level contamination of the area. Furthermore, large quantities of waste probably do not remain in place in this area, although some localized contaminant sources are possible (e.g., abandoned underground storage tanks).

7. Phillips Army Airfield: This area is of low to moderate concern, primarily because of its location relative to off-post drinking water supply wells. Habitat value of the area immediately surrounding the landfill is limited given that most of it has been developed and/or is regularly mowed.

8. Michaelsville Landfill: This area is of low concern relative to the other study areas. It is has limited human use and is relatively isolated from off-post drinking water sources. Habitat value of the immediate surrounding area is low relative to most other study areas. This study area has been relatively well characterized with respect to potential contamination.

14.5 REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. National oil and hazardous substances pollution contingency plan. Fed. Reg. 55:8666-8865 (March 8, 1990).

APPENDIX A

FATE AND TRANSPORT MODELS USED IN THE O-FIELD RISK ASSESSMENT

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INFORMATION IN THIS SECTION (PAGES A-13 THROUGH A-32) REGARDING ACUTE EXPOSURES TO CHEMICAL AGENTS RESULTING FROM EXPLOSIONS OR SPILLS HAS BEEN REMOVED BECAUSE OF INACCURACIES ASSOCIATED WITH THE ORIGINAL DATA. RECENT INFORMATION REGARDING ACUTE EXPOSURES FOR SPECIFIC CHEMICAL AGENTS AT O-FIELD CAN BE FOUND IN THE "DESIGN REPORT FOR THE OLD O-FIELD SOURCE AREA - FINAL DOCUMENT" (FEBRUARY 1995), AND FOR A SIMILAR SCENARIO IN "INSTALLATION RESTORATION PROGRAM - CLUSTER I: FORMER NIKE SITE - ABERDEEN PROVING GROUND REMEDIAL INVESTIGATION/FEASIBILITY STUDY, VOLUME I: REMEDIAL INVESTIGATION REPORT, APPENDIX O."

A.1 SIMPLIFIED MODEL FOR ESTIMATING ORGANIC CHEMICAL CONCENTRATIONS IN WATSON CREEK

A simplified model based on observed groundwater flow conditions and groundwater-surface water discharge relationships in the shallow aquifers at Old O-Field was used to predict (1) mass loading of volatile organic compounds (VOCs) and other organic contaminants to Watson Creek (the discharge point for the contaminated groundwater plume migrating from the site) and (2) the average surface water concentration of these contaminants between discharge events. This model is described in the following paragraphs.

A representative groundwater discharge rate for the contaminated groundwater plume discharging from the shallow aquifers at Old O-Field (i.e., water-table and upper-confined aquifers) to Watson Creek was estimated from information gathered during USGS studies performed at the site from 1985 to 1988 (Vroblesky et al. 1990). The discharge rate was estimated from a simple Darcy's Law relationship:

$$Q = KiA \qquad \text{(Eq. 1)}$$

where:

Q = groundwater discharge rate (L3/T)

K = hydraulic conductivity (L/T)

i = hydraulic gradient (DIM)

A = cross-sectional area of discharge zone (L2)

In calculating the actual estimated discharge rate this equation was modified somewhat to account for transient conditions that exist in the shallow aquifers at Old O-Field, namely that (1) hydraulic gradients in both aquifers vary considerably in response to diurnal tidal cycles as well as seasonal variations in precipitation; and (2) hydraulic conductivities appear to vary significantly within the aquifers, especially in different areas of the water-table aquifer. To account for these conditions, we used an average groundwater flow velocity range of 130 to 330 feet/year (estimated by Vroblesky et al. 1990) to approximate the Ki term in Equation 1 above. However, in using this factor, it is very important to note that transient conditions (mainly related to tidal effects) greatly influence the discharge rate on a short-term basis, resulting in pulse-type or "slug" releases of contaminated groundwater to the creek. These "discharge events" are taken into account in a later stage of this model, described below.

The cross-sectional area of the plume discharge zone was also estimated from information contained in Vroblesky et al. (1990). The lateral extent (or width of the plume) was estimated at approximately 650 feet, with the discharge zone for the shallow aquifers extending approximately 60 to 120 feet from the Watson Creek shoreline, resulting in an estimated discharge zone of 39,000 to 78,000 square feet.

Taking into account variability in estimates of groundwater flow rates and discharge area, "low estimate" and "high estimate" values for groundwater discharge (Q) were developed, using the range of flow velocities and discharge zone cross-sectional areas noted above. These values result in an estimated range for Q of approximately 14,000 to 70,000 cubic feet/day (5 to 25 million cubic

feet/year).

To estimate initial dilution and mixing of contaminated groundwater containing VOCs and other organic chemicals in the near-shore area of Watson Creek, it is necessary to evaluate not only groundwater discharge rates, but also the initial dilution provided by the creek as well as contaminant concentrations in the discharging groundwater. Although detailed information on the bathymetry and hydraulic conditions in Watson Creek are not available, limited information from Vroblesky et al. 1990) indicates that (1) the creek is quite shallow in the suspected plume discharge zone; and (2) mixing conditions depend greatly on the tidal stage at the time the contaminated groundwater "pulse" enters the stream (which in the area near Old O-Field is really more a tidal pond than a creek). To approximate this dilution volume, we estimated an average water depth of 2.5 feet, and assumed no initial mixing outside the area of the plume discharge zone (previously estimated as extending from 60 to 120 feet east of the shoreline. This estimation yields a range of initial dilution volumes of approximately 97,000 to 195,000 cubic feet (i.e., an initial surface water:groundwater dilution ratio of about 3:1 to 5:1, depending on the use of "high estimate" or "low estimate" values).

To predict contaminant concentrations in surface water, we used information from a USGS study (Vroblesky et al. 1990) that showed that VOC concentrations from creek-bottom vapor samplers within the discharge zones were very similar to VOC concentrations measured in groundwater monitoring wells near the Watson Creek shoreline. Because the creek-bottom sampler data represented single point measurements with limited quality assurance compared to the groundwater data (several rounds of groundwater sampling conducted according to USATHAMA QA protocols), we considered the groundwater data more reliable for estimating average initial loading to the creek, and used these data in our model. A summary of this information, including the specific wells used and statistical average concentrations for the contaminants of concern, is presented in Table 5-4 in Chapter 5 of this report.

Initial surface water concentrations, resulting from dilution of contaminated groundwater in Watson Creek but not accounting for any other process (e.g., volatilization), were estimated using a standard mass balance approach:

$$C3 = (C1V1 + C2V2)/(V1 + V2) \qquad \text{(Eq. 2)}$$

where:

C3 = initial surface water chemical concentration as a result of groundwater discharge;

C1 = chemical concentration in groundwater;

C2 = background chemical concentration in surface water;

V1 = daily groundwater discharge volume; and

V2 = creek dilution volume.

For the purpose of the baseline risk assessment, lower-bound and upper-bound dilution volumes were used for creek dilution volume (V2) to account for uncertainty regarding the discharge area and likely initial mixing volume of surface water. In addition, the background chemical concentration (C2) (before groundwater discharge) was assumed to be zero for all VOCs, under the assumption that volatilization and dilution between discharge events would be sufficient to result in negligible residual

concentrations in surface water (this assumption is consistent with measured levels of VOCs in Watson Creek, which are generally quite low). Finally, the assumption was made that contaminants discharged from groundwater upward through bottom sediments to surface water were not attenuated or altered to a significant degree by sediment-contaminant interactions. This assumption is considered appropriate for most of the highly soluble compounds that represent contaminants of concern at O-Field and for sandy, low-organic bottom sediment conditions that exist in some portions of Watson Creek, but will result in conservatively high concentration estimates for more hydrophobic compounds and/or areas of fine-grained bottom sediment with high organic content.

Based on the ranges of values for groundwater discharge and dilution volume presented previously, "low estimate" initial surface water concentrations (i.e., based on a minimum groundwater discharge volume and a maximum surface water dilution volume) and "high estimate" initial surface water concentrations (i.e., based on a maximum groundwater discharge volume and a minimum surface water dilution volume) were generated. These values are presented in Table 5-16 in Chapter 5 of this report.

Using these estimated initial surface water concentrations (C3), average surface water concentrations between discharge events are estimated by integrating volatilization loss over time between discharge periods. Concentrations of volatile chemicals in surface water at any time between discharge periods is given by:

$$C(t) = C_o * e^{-k_v t} \qquad \text{(Eq. 2)}$$

where:

- $C(t)$ = concentration in the surface water at any time between discharge periods (µg/L);
- C_o = initial surface water concentration (µg/L);
- k_v = volatilization rate constant (hrs^{-1}); and
- t = time after groundwater discharge (hrs).The average surface water concentration between discharge periods is the integral of Eq. 2 divided by the time between groundwater discharge events as follows:

$$C_{avg} = \frac{1}{t}\int_0^t C_o e^{-k_v t} dt \qquad \text{(Eq. 3)}$$

where:

C_{avg} = average surface water concentration between groundwater discharge events (µg/L);

C_o = initial surface water concentration (µg/L);

k_v = volatilization rate constant (hrs^{-1}); and

t = time between discharge events (24 hrs).

A.1.1 REFERENCES

VROBLESKY, D.A., LORAH, M.M., and TRIMBLE, S.P. 1990. Mapping Zones of Contaminated Ground Water Discharge Using Creek Bottom Sediment Vapor Samplers. Aberdeen Proving Ground, Maryland. Accepted for publication.

A.2 SURFACE WATER VOLATILIZATION AND AIR DISPERSION MODELING USED TO EVALUATE CHRONIC INHALATION EXPOSURES AT O-FIELD

A.2.1 EMISSIONS MODEL

As discussed in Section A.1, chemical concentrations in groundwater discharged to Watson Creek were modeled to estimate resulting concentrations in the surface waters of Watson Creek. Inhalation of volatile organic chemicals emitted from the surface waters of the creek create a potential exposure pathway to nearby receptors. Therefore, the emissions of volatile organic chemicals from Watson Creek were estimated using a water volatilization model presented by Liss and Slater (1974). This volatilization model estimates the steady-state mass transfer from the liquid-phase to the gas-phase which occurs across a small layer at the air-water interface. This layer is composed of two smaller layers or films above and below the water surface. Above the water surface is a gas-phase film and a second film, a liquid-phase film, extends downward from the water surface. A separate concentration gradient exists within each of these films. These concentration gradients drive the flux of chemicals from the water body and into the air. Above and below this layer it is assumed that the chemicals within the air and water are well mixed.

Both the gas and liquid-phase films are assumed to be stagnant. Thus the mass transfer across these films is due to molecular diffusion. Chemical-specific mass transfer coefficients describe the rate of molecular diffusion for each phase. Liss and Slater (1974) suggest the following semi-empirical equations for calculating the chemical-specific mass transfer coefficients for the liquid- and gas-phases.

$$k_l = 20\sqrt{44/M}$$

where:

k_l = liquid phase exchange coefficient (cm/hr)

M = molecular weight (g/mol)

and

$$k_g = 3000\sqrt{18/M}$$

where:

k_g = gas phase exchange coefficient (cm/hr)

M = molecular weight (g/mol)

The two-layer film model assumes that the flux rate of chemicals from the water to the air is determined by the total resistance to mass transport from both the liquid-phase and gas-phase films. The reciprocal of the chemical-specific mass transfer coefficients describes the resistance to mass transport within each phase. The interface between the gas and liquid-phase films is assumed to offer negligible resistance to mass transport.

The chemical-specific resistances to mass transport for the liquid and gas-phases are summed to determine the total resistance to mass transfer. The total resistance to mass transfer is the overall mass transfer coefficient for the flux of a chemical from the water body into the overlying air. According to Liss and Slater (1974) the overall mass transfer coefficient (K_L) is given by:

$$K_L = \frac{H' k_g k_l}{H' k_g + k_l}$$

where:

H' = dimensionless Henry's Law Constant

k_g = gas phase exchange coefficient (cm/hr)

k_l = liquid phase exchange coefficient (cm/hr)

The emission rate of volatile organic chemicals from the surface water of Watson Creek is then calculated using the following equation:

$$ER = C_o * K_L * Area * CF$$

where:

ER = emission rate of volatile organics (g/sec)

C_o = initial surface water concentration (µg/L)

K_L = overall mass transfer coefficient (cm/hr)

Area = size of surface water body (m^2); the area size under the low estimate scenario was approximately 7,250 m^2 and the area size under the high estimate scenario was approximately 3,620 m^2

CF = 2.78×10^{-9}; conversion factor
= (µg/L) x ($g/10^6$ µg) x ($L/10^{-3}$ m^3) x (cm/hr) x (m/100 cm) x (hr/3600 s)

Two sets of emissions rates were calculated using the methodology described above and the same input parameters, with the exception of the initial surface water concentrations. The low estimate emission rates were calculated using the low estimate surface water concentrations, while the high estimate emission rates were calculated using the high estimate surface water concentrations. Both sets of emission rates are listed in Table A-1 and were linked to an air dispersion model (described in the following section) to estimate ambient air concentrations for those receptors nearest to Watson Creek.

TABLE A-1

Input Parameters and Emission Rate Calculations for Surface Water Volatilization Model of Watson Creek

Chemical	Surface Water Concentration (ug/L)		Molecular Weight (g/mole)	Henry's Law Constant (non-dim.)	Liquid-Phase Exchange Coefficient k_l (cm/hr)	Gas-Phase Exchange Coefficient k_g (cm/hr)	Overall Mass Transfer Coefficient K_l (cm/hr)	Emission Rate (g/sec)	
	Low Estimate	High Estimate						Low Estimate	High Estimate
1,1,2,2-Tetrachloroethane	290.0	46.0	167.85	1.96E-02	10.24	982.42	6.68E+00	8.14E-03	2.58E-03
1,1,2-Trichloroethane	12.0	2.0	133.41	5.00E-02	11.49	1101.95	9.50E+00	3.64E-04	1.21E-04
1,2-Dichloroethane	80.0	13.0	98.96	4.58E-02	13.34	1279.46	1.09E+01	2.47E-03	8.04E-04
1,2-Dichloroethene	250.0	40.0	96.94	6.25E-01	13.47	1292.73	1.33E+01	7.87E-03	2.52E-03
1,3-Dinitrobenzene	10.0	2.0	168.11	2.27E-06	10.23	981.66	2.22E-03	2.24E-07	8.95E-08
Benzene	84.0	13.0	78.00	2.28E-01	15.02	1441.15	1.44E+01	2.66E-03	8.22E-04
Carbon Tetrachloride	20.0	3.0	154.00	9.58E-01	10.69	1025.65	1.06E+01	6.16E-04	1.85E-04
Chlorobenzene	18.0	3.0	112.56	1.42E-01	12.50	1199.68	1.16E+01	5.61E-04	1.87E-04
Chloroform	130.0	20.0	119.38	1.58E-01	12.14	1164.91	1.14E+01	4.04E-03	1.24E-03
Methylene Chloride	29.0	5.0	84.93	1.08E-01	14.39	1381.11	1.31E+01	9.12E-04	3.14E-04
Tetrachloroethene	39.0	6.0	165.85	9.58E-01	10.30	988.33	1.02E+01	1.20E-03	3.68E-04
Toluene	5.0	1.0	92.15	2.75E-01	13.82	1325.90	1.33E+01	1.57E-04	6.29E-05
Trichloroethene	220.0	35.0	131.29	3.71E-01	11.58	1110.82	1.13E+01	6.83E-03	2.17E-03
Vinyl Chloride	84.0	13.0	62.50	2.88E+01	16.78	1609.97	1.68E+01	2.67E-03	8.27E-04

A.2.2 AIR DISPERSION MODEL

EPA's Industrial Source Complex Long-Term (ISCLT) dispersion model (EPA 1987) was used to estimate annual average concentrations of volatile organic compounds released from Watson Creek. The ISCLT model is part of EPA's UNAMAP family of models which are considered to be EPA's preferred group of air models. It is a steady-state Gaussian plume model which can be used to assess pollutant concentrations from a wide variety of sources (EPA 1986). ISCLT estimates annual average ground-level concentrations in all directions around an emission source out to 50 km.

The first step in the process was to link the appropriate STAR meteorological data with the ISCLT model. STAR data represents summaries of the observed joint frequency of occurrence of wind speed and direction for a range of atmospheric stabilities. STAR data compiled for the years 1955-1957 at Phillips Field at Aberdeen Proving Ground was used in this assessment.

ISCLT allows for the selection of atmospheric dispersion coefficients representative of a rural or more turbulent urban environment. Due to the location of Watson Creek, the rural setting was chosen for this assessment.

Emissions of volatile organic chemicals occur along a southwest portion of Watson Creek, and can be characterized as a rectangular line source with finite dimensions. ISCLT can be used to estimate dispersion from a finite line source such as that defined by the discharge area of groundwater along Watson Creek. A line source is approximated in the model by a series of square volume sources placed at equal intervals along the line. The user must input to the model initial lateral (σ_{yo}) and vertical (σ_{zo}) dimensions for each volume source. When characterizing a line source by a group of adjacent volume sources, the initial lateral dimensions (σ_{yo}) for each volume source is defined by the length of the square source divided by 2.15. Similarly, the initial vertical dimensions (σ_{zo}) is defined by the height of the emission source divided by 2.15. The height of the modeled source (groundwater discharge area along Watson Creek) is negligible, however, ISCLT requires that the source have some non-zero vertical dimension. Therefore, the height of the source was assumed to be 4.75×10^{-3} m which is representative of a stagnant air layer (boundary layer) above a smooth surface in which a concentration gradient drives chemical transport via molecular diffusion (Jury et al. 1983). This layer is analogous to the two-film layer at the air-water interface in the Liss and Slater (1974) surface water volatilization model used to estimate emissions from Watson Creek. The concentration gradient carries the volatile chemicals through the boundary layer and into the overlying atmosphere where they are transported and dispersed. Therefore the top of this boundary layer can be considered the height of emissions from Watson Creek. Table A-2 lists the initial source parameters used in the volume source option of ISCLT.

To be consistent with the modeling conducted to determine the surface water concentrations in Watson Creek resulting from groundwater discharge and for the surface water volatilization modeling, two scenarios were developed for modeling in ISCLT. For the low estimate scenario, the line source was assumed to be 720 feet long and 120 feet wide, which was represented in the model by 6 square volume sources situated adjacent to each other along the shoreline. This source was assigned the low estimate emission rates calculated in the surface water volatilization modeling. For the high estimate scenario the line source was assumed to be 720 feet long and 60 feet wide, which was simulated in the model by 12 square volume sources situated adjacent to each other along the shoreline.

Although the groundwater discharge area along the shore of Watson Creek was estimated to be 650 feet, the line source input into the ISCLT model was 720 feet in length. Because of model restrictions, the north-south and east-west dimensions of each volume source used in the model must be the same. A value of 720 was selected for use in the modeling because it results in an integer number of

Table A-2

Initial Source Parameters for Volume Sources Used in ISCLT
to Characterize Surface Water Volatilization from Watson Creek

	HIGH ESTIMATE SCENARIO	LOW ESTIMATE SCENARIO
Number of Volume Sources Used	12	6
Initial Lateral Dimensions (σ_y) (m)	8.5	17
Initial Vertical Dimensions (σ_z) (m)	2.21×10^{-3}	2.21×10^{-3}
Volume Source Coordinates (m,m)	(0,0) (18.3,0) (36.6,0) (54.9,0) (73.2,0) (91.4,0) (109.7,0) (128,0) (146.3,0) (164.6,0) (182.9,0) (201.2,0)	(0,0) (36.6,0) (73.2,0) (109.7,0) (146.3,0) (182.9,0)

equal size volume sources to approximate the line source for both the 60 foot and 120 foot wide scenarios.

A total emission rate (representing the sum of emissions from all volume sources) of 1 g/sec was used in the ISCLT model. Thus for the low estimate scenario, each of the 6 volume sources was assigned an emission rate of 1/6 g/sec, and the 12 sources in the high estimate scenario was assigned a 1/12 g/sec emission rate. For both scenarios the air concentrations at a given receptor were calculated for the emissions from all sources modeled. This resulted in unit air concentrations (in $\mu g/m^3$ per 1 g/sec) which can be multiplied by chemical-specific emission rates (in g/sec) to estimate chemical-specific air concentrations at the receptor points.

The modeled line source along the shoreline of Watson Creek is situated approximately 40 degrees from a true east-west orientation. Due to model limitations the source to receptor relationship can be simulated much more accurately if the series of volume sources are orientated along a model grid axis. To accomplish this, the line source was rotated 40 degrees in a counterclockwise direction and the receptor grid and discrete receptors were defined relative to the rotated source. An option in the ISCLT model allows the user to rotate wind direction for the input STAR data. Therefore, the input winds were rotated 40 degrees in a counterclockwise direction to account for the rotated source and receptors. Thus, the proper directional relationship between the source, receptors and the observed wind patterns was maintained.

A 4 km by 4 km rectangular generic receptor grid with a 200 meter spacing was used in the model, resulting in 441 receptor locations. Additionally, three discrete receptors were identified at H-Field, M-Field, and new O-Field to estimate workers' potential exposure to volatile chemicals emitted from Watson Creek. The origin (0,0) of the receptor grid was located in the center of the western most volume source. The location of all other volume sources in the line are relative to this origin. Air concentrations at the three discrete receptors were used in this assessment. The unit air concentrations for the low estimate scenario are $2.34x10^{-3}$ mg/m^3, $1.66x10^{-3}$ mg/m^3, and $6.45x10^{-3}$ mg/m^3 for the receptors at H-Field, M-Field, and New O-Field, respectively. The unit air concentrations for the high estimate scenario are $2.32x10^{-3}$ mg/m^3, $1.69x10^{-3}$ mg/m^3, and $6.33x10^{-3}$ mg/m^3 for the receptors at H-Field, M-Field, and New O-Field, respectively. These unit air concentrations (in mg/m^3 per g/sec) were then multiplied by the chemical-specific emission rates (in g/sec) shown in Table A-1 to obtain chemicals-specific air concentrations at the three receptors. Table A-3 lists the chemical-specific air concentrations for both scenarios evaluated.

A.2.3 REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Guideline On Air Quality Models (Revised). Office of Air Quality Planning and Standards. Research Triangle Park, North Carolina. EPA-450/2-78-027R.

ENVIRONM[illegible]AL PROTECTION AGENCY (EPA). 1987. Industrial Source Complex (ISC) Dispersion Model U[illegible] Guide - Second Edition (Revised). Volume I. Office of Air Quality and Planning and Standard[illegible] Research Triangle Park, North Carolina. EPA-450/4-88-002a.

JURY, W.A., SPENCER, W.F., and FARMER, W.J. 1983. Behavior assessment model for trace organics in soil: I. model description. J. Environmental Quality. 12(4).

LISS, P.S[illegible]SLATER, P.G. 1974. Flux of gases across the air-sea interface. Nature (London) 247:18[illegible]

TABLE A-3

AMBIENT AIR CONCENTRATIONS ASSOCIATED WITH VOLATILZATION FROM WATSON CREEK

(mg/m^3)

CHEMICAL	Low Estimate Scenario			High Estimate Scenario		
	H-FIELD	M-FIELD	NEW O-FIELD	H-FIELD	M-FIELD	NEW O-FIELD
1,1,2,2-Tetrachloroethane	6.04E-06	4.27E-06	1.66E-05	1.89E-05	1.37E-05	5.15E-05
1,1,2-Trichloroethane	2.84E-07	2.01E-07	7.83E-07	8.44E-07	6.14E-07	2.31E-06
1,2-Dichloroethane	1.88E-06	1.33E-06	5.18E-06	5.73E-06	4.17E-06	1.57E-05
1,2-Dichloroethene	5.89E-06	4.17E-06	1.62E-05	1.82E-05	1.33E-05	4.98E-05
1,3-Dinitrobenzene	2.09E-10	1.48E-10	5.77E-10	5.19E-10	3.78E-10	1.42E-09
Benzene	1.92E-06	1.36E-06	5.30E-06	6.15E-06	4.48E-06	1.68E-05
Carbon Tetrachloride	4.32E-07	3.06E-07	1.19E-06	1.43E-06	1.04E-06	3.90E-06
Chlorobenzene	4.37E-07	3.09E-07	1.20E-06	1.30E-06	9.45E-07	3.55E-06
Chloroform	2.91E-06	2.06E-06	8.01E-06	9.36E-06	6.81E-06	2.56E-05
Methylene Chloride	7.36E-07	5.21E-07	2.03E-06	2.11E-06	1.54E-06	5.77E-06
Tetrachloroethene	8.61E-07	6.09E-07	2.37E-06	2.77E-06	2.02E-06	7.57E-06
Toluene	1.47E-07	1.04E-07	4.06E-07	3.65E-07	2.65E-07	9.96E-07
Trichloroethene	5.08E-06	3.60E-06	1.40E-05	1.58E-05	1.15E-05	4.32E-05
Vinyl Chloride	1.93E-06	1.37E-06	5.33E-06	6.19E-06	4.50E-06	1.69E-05

APPENDIX B

HUMAN HEALTH TOXICITY PROFILES

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INTRODUCTION

Appendix B presents human health toxicity profiles for chemicals discussed in this baseline risk assessment. Profiles for all chemicals of potential concern are included, along with summaries for many of the military-unique chemicals potentially present at APG. Also presented are brief profiles for chemicals not evaluated in this risk assessment due to their low toxicities. Where available, EPA-approved carcinogenic and noncarcinogenic toxicity criteria are included for chemicals which were evaluated quantitatively. These toxicity criteria, for oral and inhalation exposures, are presented in Table B-1 and Table B-2, respectively.

TABLE B-1

ORAL CRITICAL TOXICITY VALUES FOR CHEMICALS OF POTENTIAL CONCERN

Chemical	Chronic Reference Dose (mg/kg/day)	Uncertainty Factor (a)	Target Organ (b)	Reference Dose Source	Cancer Slope Factor (mg/kg/day)-1	EPA Weight of Evidence Classification (c)	Slope Factor Source
Organic Chemicals:							
Acetone	1.00E-01	1,000	Kidney/Liver	IRIS	--	D	IRIS
Aldrin	3.00E-05	1,000	Liver	IRIS	1.70E+01	B2	IRIS
Aroclor-1221	--	--	--	--	--	--	--
Aroclor-1254	--	--	--	--	--	--	--
Benzene	--	--	--	IRIS	2.90E-02	A	IRIS
alpha-BHC (alpha-HCCH)	--	--	--	IRIS	6.30E+00	B2	IRIS
beta-BHC (beta-HCCH)	--	--	--	IRIS	1.80E+00	C	IRIS
delta-BHC (delta-HCCH)	--	--	--	IRIS	--	D	IRIS
gamma-BHC (gamma-HCCH)	3.00E-04	1,000	Liver/Kidney	IRIS	1.30E+00	B2/C	HEAST
4-Bromofluorobenzene	--	--	--	--	--	--	--
Butylbenzylphthalate	2.00E-01	1,000	Testes/Liver/ Kidney	IRIS	--	C	IRIS
Carbon Disulfide	1.00E-01	100	Fetus	IRIS	--	--	IRIS
Carbon Tetrachloride	7.00E-04	1,000	Liver	IRIS	1.30E-01	B2	IRIS
p-Chloro-m-cresol	--	--	--	HEAST	--	--	--
4-Chloroaniline (p-chloroaniline)	4.00E-03	3,000	Spleen	IRIS	--	--	IRIS
Chlorobenzene	2.00E-02	1,000	Kidney/Liver	IRIS	--	--	IRIS
Chloroethane	--	--	--	--	--	--	--
bis(2-Chloroethyl)ether	--	--	--	IRIS	1.10E+00	B2	IRIS
Chloroform	1.00E-02	1,000	Liver	IRIS	6.10E-03	B2	IRIS
Chloromethane	--	--	--	--	1.30E-02	C	HEAST
4,4'-DDD	--	--	--	IRIS	2.40E-01	B2	IRIS
4,4'-DDE	--	--	--	IRIS	3.40E-01	B2	IRIS
4,4'-DDT	5.00E-04	100	Liver	IRIS	3.40E-01	B2	IRIS
Di-n-butylphthalate	1.00E-01	1,000	Mortality	IRIS	--	--	IRIS
1,2-Dichlorobenzene	9.00E-02	1,000	Liver	IRIS	--	D	IRIS
1,3-Dichlorobenzene	8.90E-02	1,000	Liver	HA	--	D	IRIS
1,1-Dichloroethane	1.00E-01	1,000	Kidney	HEAST	--	C	IRIS
1,2-Dichloroethane	--	--	--	IRIS	9.10E-02	B2	IRIS
1,1-Dichloroethene	9.00E-03	1,000	Liver	IRIS	6.00E-01	C	IRIS
cis-1,2-Dichloroethene	1.00E-02	3,000	Blood	HEAST	--	--	--
trans-1,2-Dichloroethene	2.00E-02	1,000	Blood	IRIS	--	--	--
1,2-Dichloropropane	--	--	--	HEAST	6.80E-02	B2	HEAST
1,3-Dichloropropene (Telone II)	3.00E-04	10,000	Kidney	IRIS	1.80E-01	B2	HEAST
Dieldrin	5.00E-05	100	Liver	IRIS	1.60E+01	B2	IRIS
Diethylphthalate	8.00E-01	1,000	Body weight	IRIS	--	--	IRIS
1,3-Dinitrobenzene	1.00E-04	3,000	Spleen	IRIS	--	--	IRIS
Di-n-octylphthalate	2.00E-02	1,000	Liver/Kidney	HEAST	--	--	HEAST
1,2-Diphenylhydrazine	--	--	--	IRIS	8.00E-01	B2	IRIS
Dithiane	1.00E-01	1,000	Nasal epithelium	CLEMENT	--	--	--
Endosulfans	5.00E-05	3,000	Kidney	IRIS	--	--	IRIS
Endosulfan Sulfate	--	--	--	IRIS	--	--	--
Endrin Aldehyde	--	--	--	--	--	--	--
Ethyl Benzene	1.00E-01	1,000	Liver/Kidney	IRIS	--	D	IRIS
2-Ethyl-1-hexanol	--	--	--	--	--	--	--
bis(2-Ethylhexyl)phthalate	2.00E-02	1,000	Liver	IRIS	1.40E-02	B2	IRIS
Heptachlor	5.00E-04	300	Liver	IRIS	4.50E+00	B2	IRIS
Heptachlor Epoxide	1.30E-05	1,000	Liver	IRIS	9.10E+00	B2	IRIS
Hexanedioic Acid, Dioctyl Ester	--	--	--	--	--	--	--
Methoxychlor	5.00E-03	1,000	Fetus	IRIS	--	D	IRIS
3-Methyl-4-chlorophenol	--	--	--	--	--	--	--
Methylene Chloride	6.00E-02	100	Liver	IRIS	7.50E-03	B2	IRIS
4-Methylphenol (p-cresol)	5.00E-02	1,000	Nervous System	IRIS	--	--	IRIS
Methylphosphonic Acid	--	--	--	--	--	--	--
Nitrobenzene	5.00E-04	10,000	Kidney/Liver	IRIS	--	D	IRIS
4-Nitrophenol	--	--	--	HEAST	--	--	--
1,4-Oxathiane	--	--	--	--	--	--	--
PAHs	4.00E-03 (d)	10,000	Eye	HEAST	1.15E+01 (e)	B2	HEA
PCBs (Total)	1.00E-04	100	Fetus	CLEMENT	7.70E+00	B2	IRIS
Pentachlorophenol	3.00E-02	100	Liver/Kidney	IRIS	--	--	IRIS
Phenol	6.00E-01	100	Fetus	IRIS	--	D	IRIS
1,1,2,2-Tetrachloroethane	4.60E-04	1,000	Liver/White Blood Cells	(f)	2.00E-01	C	IRIS
Tetrachloroethene	1.00E-02	1,000	Liver	IRIS	5.10E-02	B2	HEAST
Thiodiglycol	--	--	--	--	--	--	--
Toluene	2.00E-01	1,000	Liver/Kidney	IRIS	--	D	IRIS

See footnotes on page B-4.

TABLE B-1 (Continued)

ORAL CRITICAL TOXICITY VALUES FOR CHEMICALS OF POTENTIAL CONCERN

Chemical	Chronic Reference Dose (mg/kg/day)	Uncertainty Factor (a)	Target Organ (b)	Reference Dose Source	Cancer Slope Factor (mg/kg/day)-1	EPA Weight of Evidence Classification (c)	Slope Factor Source
Organic Chemicals (continued):							
1,2,3-Trichlorobenzene	--	--	--	--	--	--	--
1,2,4-Trichlorobenzene	2.00E-02	1,000	Kidney	HEAST	--	D	IRIS
1,1,1-Trichloroethane	9.00E-02	1,000	Liver	IRIS	--	D	IRIS
1,1,2-Trichloroethane	4.00E-03	1,000	Clinical chem.	IRIS	5.70E-02	C	IRIS
Trichloroethene	7.35E-03	1,000	Liver	HA	1.10E-02	B2	HEAST
Trichlorofluoromethane	3.00E-01	1,000	Mortality	IRIS	--	--	--
bis(2,4,6-Trichlorophenyl)urea	--	--	--	--	--	--	--
1,1,2-Trichloro-1,2,2-trifluoroethane	3.00E+01	10	CNS	IRIS	--	--	IRIS
2,4,6-Trinitrotoluene	5.00E-04	1,000	Liver	IRIS	3.00E-02	C	IRIS
Vinyl Chloride	--	--	--	--	2.30E+00	A	HEAST
Xylenes (total)	2.00E+00	100	Nervous System/ Mortality	IRIS	--	D	IRIS
Inorganic Chemicals:							
Aluminum	--	--	--	--	--	--	--
Ammonia	3.40E+01 mg/l	--	(g)	HEAST	--	--	--
Ammonia Nitrogen	--	--	--	--	--	--	--
Antimony	4.00E-04	1,000	Blood	IRIS	--		IRIS
Arsenic	1.00E-03	1	Skin	HEAST	2.00E+00 (h)	A	IRIS
Barium	7.00E-02	3	Cardiovascular System	IRIS	--	--	IRIS
Beryllium	5.00E-03	100	Various Organs (Tumors)	IRIS	4.3E+00	B2	--
Boron	9.00E-02	100	Testes	IRIS	--	--	--
[illegible]romide	--	--	--	--	--	--	--
[illegible]amine	--	--	--	--	--	--	--
[illegible]dmium	5.00E-04	10	Kidney	IRIS	--	--	IRIS
Calcium	--	--	--	--	--	--	--
Chloride	--	--	--	--	--	--	--
Chromium III and compounds	1.00E+00	1,000	Liver	IRIS	--	--	--
Chromium VI and compounds	5.00E-03	500	CNS	IRIS	--	--	IRIS
Copper	3.71E-02 (i)	1	Gastrointestinal Tract	HEAST	--	D	IRIS
Cyanide	2.00E-02	500	Nervous System	IRIS	--	D	IRIS
Fluoride	6.00E-02	1	Teeth	HEAST	--	--	--
Iron	--	--	--	HEAST	--	--	--
Lead	--	--	--	IRIS	--	B2	IRIS
Magnesium	--	--	--	--	--	--	--
Manganese	1.00E-01	1	CNS	IRIS	--	--	--
Mercury, inorganic	3.00E-04	1,000	Kidney	HEAST	--	D	IRIS
Nickel	2.00E-02	300	Body weight	IRIS	--	--	IRIS
Nickel refinery dust	--	--	--	IRIS	--	--	IRIS
Nickel subsulfide	--	--	--	IRIS	--	--	IRIS
Nitrate	--	--	--	IRIS	--	--	IRIS
Nitrite	1.00E-01	10	Blood	IRIS	--	--	IRIS
Potassium	--	--	--	--	--	--	--
Selenious Acid/Selenium	3.00E-03	15	Skin	IRIS	--	--	IRIS
Silver	3.00E-03	2	Skin (Argyria)	IRIS	--	D	IRIS
Sodium	--	--	--	--	--	--	--
Sulfate	--	--	--	--	--	--	--
Sulfide	--	--	--	--	--	--	--
Thallium	7.00E-05	3,000	Blood/Hair	HEAST	--	--	HEAST
White Phosphorus	2.00E-05	1,000	Reproductive System/Mortality/Hair	IRIS	--	--	--
Zinc	2.00E-01	10	Blood (Anemia)	HEAST	--	--	HEAST
Radiological Parameters:							
Gross Alpha	--	--	--	--	--	--	--
Gross Beta	--	--	--	--	--	--	--
Potassium 40	--	--	--	--	1.10E-11 (pCi/L)-1	A	HEAST
[illegible]edium 226	--	--	--	--	1.20E-10 (pCi/L)-1	A	HEAST

See footnotes on page B-4.

TABLE B-1 (Continued)

ORAL CRITICAL TOXICITY VALUES FOR CHEMICALS OF POTENTIAL CONCERN

(a) Safety factors are the products of uncertainty factors and modifying factors. Uncertainty factors used to develop reference doses generally consist of multiples of 10, with each factor representing a specific area of uncertainty in the data available. The standard uncertainty factors include the following:
- a 10-fold factor to account for the variation in sensitivity among the members of the human population;
- a 10-fold factor to account for the uncertainty in extrapolating animal data to the case of humans;
- a 10-fold factor to account for the uncertainty in extrapolating from less than chronic NOAELs to chronic NOAELs; and
- a 10-fold factor to account for the uncertainty in extrapolating from LOAELs to NOAELs.

Modifying factors are applied at the discretion of the reviewer to cover other uncertainties in the data.

(b) A target organ is the organ most sensitive to a chemical's toxic effect. RfDs are based on toxic effects in the target organ. If an RfD was based on a study in which a target organ was not identified, an organ or system known to be affected by the chemical is listed.

(c) EPA Weight of Evidence for Carcinogenic Effects:
[A] = Human carcinogen based on adequate evidence from human studies;
[B2] = Probable human carcinogen based on inadequate evidence from human studies and adequate evidence from animal studies;
[C] = Possible human carcinogen based on limited evidence from animal studies in the absence of human studies;
[D] = Not classified as to human carcinogenicity; and
[E] = Evidence of noncarcinogenicity.

(d) The RfD for PAHs is for naphthalene, which was chosen as a surrogate for all noncarcinogenic PAHs.

(e) The slope factor for PAHs is for benzo[a]pyrene, which was chosen as a surrogate for all carcinogenic PAHs.

(f) Interim RfD approved by ECAO, Cincinnati.

(g) The RfD for ammonia is based on a taste threshold rather than on a health effect.

(h) EPA 1988. Special report on ingested inorganic arsenic skin cancer; nutritional essentiality. Risk assessment forum. EPA, Washington, D.C. EPA/625/3-87/013F. July 1988.

(i) Drinking water standard reported in mg/L was converted to mg/kg-day by assuming a 70 kg adult drinks 2 liters of water per day.

NOTE: IRIS = Integrated Risk Information System - December 1, 1990.
HA = Health Advisory.
HEA = Health Effects Assessment Document.
HEAST = Health Effects Assessment Summary Tables - July 1, 1990.
CLEMENT = Number derived by Clement International Corporation.
-- = No information available.

TABLE B-2

INHALATION CRITICAL TOXICITY VALUES FOR CHEMICALS OF POTENTIAL CONCERN

Chemical	Chronic Reference Dose (mg/kg-day)	Uncertainty Factor (a)	Target Organ (b)	Reference Dose Source	Cancer Slope Factor (mg/kg-day)-1	EPA Weight of Evidence Classification (c)	Slope Factor Source
Organic Chemicals:							
Acetone	--	--	--	IRIS	--	D	IRIS
Aldrin	--	--	--	IRIS	1.70E+01	B2	IRIS
Aroclor-1221	--	--	--	--	--	--	IRIS
Aroclor-1254	--	--	--	--	--	--	IRIS
Benzene	--	--	--	IRIS	2.90E-02	A	IRIS
alpha-BHC (alpha-HCCH)	--	--	--	IRIS	6.30E+00	B2	IRIS
beta-BHC (beta-HCCH)	--	--	--	IRIS	1.80E+00	C	IRIS
delta-BHC (delta-HCCH)	--	--	--	IRIS	--	D	IRIS
gamma-BHC (gamma-HCCH)	--	--	--	IRIS	--	B2/C	HEAST
4-Bromofluorobenzene	--	--	--	--	--	--	--
Butylbenzylphthalate	--	--	--	IRIS	--	C	IRIS
Carbon Disulfide	2.86E-03 (d)	100	Fetus	HEAST	--	--	IRIS
Carbon Tetrachloride	--	--	--	IRIS	1.30E-01	B2	IRIS
p-Chloro-m-cresol	--	--	--	HEAST	--	--	IRIS
4-Chloroaniline (p-chloroaniline)	--	--	--	IRIS	--	--	IRIS
Chlorobenzene	5.00E-03	10,000	Liver/Kidney	HEAST	--	D	HA
Chloroethane	--	--	--	--	--	--	--
bis(2-Chloroethyl)ether	--	--	--	IRIS	1.10E+00	B2	IRIS
Chloroform	--	--	--	IRIS	8.10E-02	B2	IRIS
Chloromethane	--	--	--	--	6.30E-03	C	HEAST
4,4'-DDD	--	--	--	IRIS	--	B2	IRIS
4,4'-DDE	--	--	--	IRIS	--	B2	IRIS
4,4'-DDT	--	--	--	IRIS	3.40E-01	B2	IRIS
Di-n-butylphthalate	--	--	--	IRIS	--	--	IRIS
1,2-Dichlorobenzene	4.00E-02	1,000	Body weight	HEAST	--	D	HEAST
1,3-Dichlorobenzene	--	--	--	--	--	D	IRIS
1,1-Dichloroethane	1.00E-01	1,000	Kidney	HEAST	--	C	IRIS
1,2-Dichloroethane	--	--	--	IRIS	9.10E-02	B2	IRIS
1,1-Dichloroethene	--	--	--	IRIS	1.20E+00	C	IRIS
cis-1,2-Dichloroethene	--	--	--	HEAST	--	--	--
trans-1,2-Dichloroethene	--	--	--	IRIS	--	--	--
1,2-Dichloropropane	--	--	--	HEAST	--	B2	HEAST
1,3-Dichloropropene (Telone II)	2.86E-03 (e)	100	Nasal Epithelium	HEAST	1.30E-01	B2	HEAST
Dieldrin	--	--	--	IRIS	1.60E+01	B2	IRIS
Diethylphthalate	--	--	--	IRIS	--	D	IRIS
1,3-Dinitrobenzene	--	--	--	IRIS	--	--	IRIS
Di-n-octylphthalate	--	--	--	HEAST	--	--	IRIS
1,2-Diphenylhydrazine	--	--	--	IRIS	8.00E-01	B2	IRIS
Dithiane	--	--	--	--	--	--	--
Endosulfans	--	--	--	IRIS	--	--	IRIS
Endosulfan Sulfate	--	--	--	IRIS	--	--	--
Endrin Aldehyde	--	--	--	--	--	--	--
Ethyl Benzene	--	--	--	IRIS	--	--	IRIS
2-Ethyl-1-hexanol	--	--	--	--	--	--	--
bis(2-Ethylhexyl)phthalate	--	--	--	IRIS	--	B2	IRIS
Heptachlor	--	--	--	IRIS	4.50E+00	B2	IRIS
Heptachlor Epoxide	--	--	--	IRIS	9.10E+00	B2	IRIS
Hexanedioic Acid, Dioctyl Ester	--	--	--	--	--	--	--
Methoxychlor	--	--	--	IRIS	--	D	IRIS
3-Methyl-4-chlorophenol	--	--	--	--	--	--	--
Methylene Chloride	3.00E+00	100	Liver	HEAST	1.40E-02	B2	IRIS
4-Methylphenol (p-cresol)	--	--	--	--	--	--	IRIS
Methylphosphonic Acid	--	--	--	--	--	--	--
Nitrobenzene	5.71E-04 (f)	3,000	Kidney/Liver	HEAST	--	D	IRIS
4-Nitrophenol	--	--	--	HEAST	--	--	--
1,4-Oxathiane	--	--	--	--	--	--	--
PAHs	--	--	--	--	6.1E+00 (g)	B2	IRIS
PCBs (Total)	--	--	--	--	--	B2	IRIS
Pentachlorophenol	--	--	--	IRIS	--	--	IRIS
Phenol	--	--	--	IRIS	--	D	IRIS
1,1,2,2-Tetrachloroethane	--	--	--	--	2.00E-01	C	IRIS

See footnotes on page B-7.

TABLE B-2 (Continued)

INHALATION CRITICAL TOXICITY VALUES FOR CHEMICALS OF POTENTIAL CONCERN

Chemical	Chronic Reference Dose (mg/kg-day)	Uncertainty Factor (a)	Target Organ (b)	Reference Dose Source	Cancer Slope Factor (mg/kg-day)-1	EPA Weight of Evidence Classification (c)	Slope Factor Source
Organic Chemicals (continued):							
Tetrachloroethene	--	--	--	IRIS	3.30E-03	B2	HEAST
Thiodiglycol	--	--	--	--	--	--	--
Toluene	5.71E-01 (h)	100	CNS	HEAST	--	D	IRIS
1,2,3-Trichlorobenzene	--	--	--	--	--	--	--
1,2,4-Trichlorobenzene	3.00E-03	1,000	Liver	HEAST	--	D	IRIS
1,1,1-Trichloroethane	3.00E-01	1,000	Liver	HEAST	--	D	IRIS
1,1,2-Trichloroethane	--	--	--	IRIS	5.70E-02	C	IRIS
Trichloroethene	--	--	--	IRIS	1.70E-02	B2	HEAST
Trichlorofluoromethane	2.00E-01	10,000	Lung	HEAST	--	--	--
bis(2,4,6-Trichlorophenyl)urea	--	--	--	--	--	--	--
1,1,2-Trichloro-1,2,2-trifluoroethane	--	--	--	IRIS	--	--	IRIS
2,4,6-Trinitrotoluene	--	--	--	IRIS	--	C	IRIS
Vinyl Chloride	--	--	--	--	2.95E-01	A	HEAST
Xylenes (total)	8.57E-02 (i)	100	CNS/Respiratory System	HEAST	--	D	IRIS
Inorganic Chemicals:							
Aluminum	--	--	--	HEAST	--	--	IRIS
Ammonia	3.60E-01 mg/m3	--	(j)	HEAST	--	--	IRIS
Ammonia Nitrogen	--	--	--	--	--	--	--
Antimony	--	--	--	IRIS	--	--	
Arsenic	--	--	--	IRIS	5.00E+01	A	IRIS
Barium	1.00E-04	1,000	Fetus	HEAST	--	--	IRIS
Beryllium	--	--	--	IRIS	8.40E+00	B2	IRIS
Boron	--	--	--	HEAST	--	--	IRIS
Cyanide	--	--	--	--	--	--	--
Cyanide	--	--	--	--	--	--	--
Cadmium	--	--	--	IRIS	6.10E+00	B1	IRIS
Calcium	--	--	--	--	--	--	--
Chloride	--	--	--	--	--	--	--
Chromium III and compounds	--	--	--	IRIS	--	--	--
Chromium VI and compounds	--	--	--	IRIS	4.10E+01	A	IRIS
Copper	--	--	--	IRIS	--	--	IRIS
Cyanide	--	--	--	IRIS	--	--	IRIS
Fluoride	--	--	--	--	--	--	--
Iron	--	--	--	HEAST	--	--	--
Lead	--	--	--	IRIS	--	B2	HEAST
Magnesium	--	--	--	--	--	--	--
Manganese	3.00E-04	100	CNS	HEAST	--	--	--
Mercury, inorganic	8.57E-05 (k)	30	CNS	HEAST	--	D	IRIS
Nickel	--	--	--	IRIS	--	--	IRIS
Nickel refinery dust	--	--	--	IRIS	8.40E-01	A	IRIS
Nickel subsulfide	--	--	--	IRIS	1.70E+00	A	IRIS
Nitrate	--	--	--	IRIS	--	--	IRIS
Nitrite	--	--	--	IRIS	--	--	IRIS
Potassium	--	--	--	--	--	--	--
Selenious Acid/Selenium	--	--	--	IRIS	--	--	IRIS
Sodium	--	--	--	--	--	--	--
Silver	--	--	--	IRIS	--	D	IRIS
Sulfate	--	--	--	--	--	--	--
Sulfide	--	--	--	--	--	--	--
Thallium	--	--	--	HEAST	--	--	HEAST
White Phosphorus	--	--	--	--	--	--	--
Zinc	--	--	--	HEAST	--	--	HEAST
Radiological Parameters:							
Gross Alpha	--	--	--	--	--	--	--
Gross Beta	--	--	--	--	--	--	--
Potassium 40	--	--	--	--	7.60E-12 (pCi/L)	A	HEAST
Radium 226	--	--	--	--	3.00E-09 (pCi/L)	A	HEAST

See footnotes on page B-7.

TABLE B-2 (Continued)

INHALATION CRITICAL TOXICITY VALUES FOR CHEMICALS OF POTENTIAL CONCERN

(a) Safety factors are the products of uncertainty factors and modifying factors. Uncertainty factors used to develop reference doses generally consist of multiples of 10, with each factor representing a specific area of uncertainty in the data available. The standard uncertainty factors include the following:
- a 10-fold factor to account for the variation in sensitivity among the members of the human population;
- a 10-fold factor to account for the uncertainty in extrapolating animal data to the case of humans;
- a 10-fold factor to account for the uncertainty in extrapolating from less than chronic NOAELs to chronic NOAELs; and
- a 10-fold factor to account for the uncertainty in extrapolating from LOAELs to NOAELs.

Modifying factors are applied at the discretion of the reviewer to cover other uncertainties in the data.
(b) A target organ is the organ most sensitive to a chemical's toxic effect. RfDs are based on toxic effects in the target organ. If an RfD was based on a study in which a target organ was not identified, an organ or system known to be affected by the chemical is listed.
(c) EPA Weight of Evidence for Carcinogenic Effects:
[A] = Human carcinogen based on adequate evidence from human studies;
[B2] = Probable human carcinogen based on inadequate evidence from human studies and adequate evidence from animal studies;
[C] = Possible human carcinogen based on limited evidence from animal studies in the absence of human studies;
[D] = Not classified as to human carcinogenicity; and
[E] = Evidence of noncarcinogenicity.
(d) Reported RfD of 1.00E-2 mg/m3 was converted to mg/kg-day by assuming a 70 kg adult inhales 20 m3 of air per day.
(e) Reported RfD of 1.00E-2 mg/m3 was converted to mg/kg-day by assuming a 70 kg adult inhales 20 m3 of air per day.
(f) Reported RfD of 2.00E-3 mg/m3 was converted to mg/kg-day by assuming a 70 kg adult inhales 20 m3 of air per day.
(g) The slope factor for PAHs is for benzo[a]pyrene, which was selected as a surrogate for all carcinogenic PAHs.
(h) Reported RfD of 2.00 mg/m3 was converted to mg/kg-day by assuming a 70 kg adult inhales 20 m3 of air per day.
(i) Reported RfD of 3.00E-1 mg/m3 was converted to mg/kg-day by assuming a 70 kg adult inhales 20 m3 of air per day.
(j) The RfD for ammonia is based on an odor threshold rather than on a health effect.
(k) Reported RfD of 3.00E-4 mg/m3 was converted to mg/kg-day by assuming a 70 kg adult inhales 20 cm3 of air per day.

NOTE: IRIS = Integrated Risk Information System - December 1, 1990.
HA = Health Advisory.
HEA = Health Effects Assessment Document.
HEAST = Health Effects Assessment Summary Tables - July 1, 1990.
HA = Health Advisory - March 1, 1987.
-- = No information available.

ACETONE

Acetone is absorbed in humans and animals following oral or inhalation exposure (EPA 1984). Approximately 75 percent of inhaled vapor is absorbed by the pulmonary route (Kagan 1924). Acute exposure to acetone vapors of 500 ppm produce irritation of the mucosal membranes in humans (EPA 1984, Nelson et al. 1943). Prolonged or repeated dermal contact may defat the skin and produce dermatitis (Krasavage et al. 1981). Rats acutely exposed to acetone vapors showed behavioral changes as demonstrated by an inability to pole climb following stimulation (Goldberg et al. 1964). In rats, slight increases in organ weights, decreases in body weights, and nephrotoxicity have been observed following long-term oral exposure to acetone (EPA 1986). Humans chronically exposed to atmospheric concentrations in excess of 10,000 ppm are likely to experience central nervous system depression and narcotic effects (Krasavage et al. 1981).

EPA (1990a) derived an oral reference dose (RfD) for acetone of 0.1 mg/kg-day based on a study sponsored by the EPA Office of Solid Waste (EPA 1986) in which increased liver and kidney weights and nephrotoxicity were observed in rats exposed orally to acetone; an uncertainty factor of 1,000 was used to derive the RfD. EPA (1990b) derived a subchronic oral RfD of 1 mg/kg-day using an uncertainty factor of 100, based on the same study and effect of concern.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Acetone. Environmental Criteria and Assessment Office. Cincinnati, Ohio. EPA 540/1-86-016.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Ninety-Day Gavage Study in Albino Rats Using Acetone. Office of Solid Waste. Washington, DC. (As cited in EPA 1989).

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office. Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response. Washington, D.C.

GOLDBERG, M.E., JOHNSON, H.E., POZZANI, N.C., and SMYTH, H.F. 1964. Effects of repeated inhalation of vapors of industrial solvents on animal behavior. Am. Ind. Hyg. Assoc. J. 25:369-375.

KAGAN, E. 1924. Arch Hyg. 94:41. (As cited in Krasavage et al. 1981).

KRASAVAGE, W.J., O'DONOGHUE, J.L., and DIVENCENZO, C.D. 1981. Ketones. In: Clayton, G.D., and Clayton, P.B. (eds.). Patty's Industrial Hygiene and Toxicology. 3rd edition. John Wiley and Sons. New York, New York. Vol. 2B

NELSON, K.W., EGE, J.F., ROSS, N., WOODMAN, L.E., and SILVERMAN, L. 1943. Sensory response to industrial solvent vapors. J. Ind. Hyg. Toxicol. 25:282-285.

ADAMSITE

INTRODUCTION

Adamsite (DM) has the chemical name diphenylamine chloroarsine (Army 1975).

TOXICOKINETICS

The absorption, distribution, metabolism and excretion of adamsite are unknown at present. Small quantities of admasite are rapidly detoxified. Incapacitating amounts lose their effects after about 30 minutes (Army 1975).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

DM is classified as a vomiting agent, an incapacitant, and as an irritant (RCRA Facility Assessment n.d.). Adamsite is rapidly hydrolyzed when in aerosl form to diphenylarsenious oxide and hydrogen chloride. The oxide is extremely poisonous when ingested or inhaled (Army 1975). Adamsite is a rapily acting agent; temporary incapacitation results following exposure to vapor concentrations of 22 mg/m^3 (Army 1975). The minimum detectable concentration which may cause irritation to the throat is 0.22 mg/m^3 (RCRA Facility Assessment n.d.); irritation to the skin and eyes are also common (Army 1975). Much higher vapor concentrations of 30 gm/m^3 and 54 ppm for a 30 minute exposure are lethal to humans (RTECS 1987). The lowest toxic concentration (TClo) of 19 mg/m^3 for a 3-minute exposure had induced gastrointestinal effects, and changes in structure and function of the salivary glands (RTECS 1987). Some of the common symptoms following high concentrations of exposure to adamsite include irritation of the eyes and mucous membranes, lacrimation, nasal secretion, excessive salivation, sneezing, coughing, severe headache, tightness and acute pain in the chest, and nausea and vomiting (Army 1975). The severity and persistence of effects is dose-dependent. Exposure to relatively low concentrations of adamsite may result in mild symptoms resembling a cold (RCRA Facility Assessment n.d.). Tolerance to eye and nose irritation will develop with chronic low-level exposure, however, serious chonic effects include dermatitis and respiratory tract damage (RCRA Facility Assessment n.d.).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

No regulated standards or criteria have been promulgated for admasite. OSHA (1989) has recommended a 8-hour time-weighted-average (TWA) permissible exposure limit (PEL) exposure of 0.5 $mg(AS)/m^3$ for occupational exposure to adamsite.

REFERENCES

ARMY. 1975. Military Chemistry and Chemical Compounds Field Manual, Oct. 1975, Department of The Army and The Air Force. Pp. 3-19 & 3-20.

ICF Feasibility Study. 1987.

OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION (OSHA). 1989. Federal Register 54(12):2923.

RCRA Facility Assessment. n.d. Edgewood Area, AP6, MD.

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1987. National Institute for Occupational Safety and Health (NIOSH). U.S. Department of Health and Human Services. Pp. 3194.

Aldrin is absorbed following inhalation exposure; between 20-50% of the inhaled vapor is absorbed and retained (Beyermann and Eckrich 1973, Shell 1984). Absorption also occurs following ingestion (Farb et al. 1973, Heath and Vandekar 1964, Hunter and Robinson 1967, 1969, Iatropoulos et al. 1975) and dermal exposure (Feldmann and Maibach 1974, Sundaram et al. 1978a,b). It is metabolically converted to dieldrin in fatty tissues (ACGIH 1986) and these two insecticides are considered to have similar chemical and toxic effects (EPA 1988). Acute symptoms of aldrin intoxication in humans and animals following ingestion or inhalation indicate CNS stimulation manifested primarily as hyperexcitability, muscle twitching, convulsions, and depression (Borgmann et al. 1952a,b, Hayes 1982, Hodge et al. 1967, Hoogendam et al. 1962, Jager 1970). Experimental studies indicate that dogs exposed for longer periods of time to levels as low as 1 mg/kg aldrin developed hepatic and renal toxicity (Fitzhugh et al. 1964, Treon and Cleveland 1955, Walker et al. 1969). Rats fed aldrin for 2 years developed hepatic lesions and nephritis at doses of 0.5 and 50 ppm, respectively (Fitzhugh et al. 1964). Aldrin produced fetotoxic and/or teratogenic effects in hamsters fed a single oral dose of 50 mg/kg (approximately 84 ppm) and in mice fed a single oral dose of 25 mg/kg (approximately 6 ppm) (Ottolenghi et al. 1974). Aldrin produced marked effects on fertility, gestation, viability, and lactation in mice given 25 mg/kg-day in a six-generation study (Deichmann 1972). Aldrin produces chromosomal aberrations in mouse, rat, and human cells (Georgian 1974) and unscheduled DNA synthesis in rats (Probst et al. 1981) and humans (Rocchi et al. 1980). Chronic oral exposure to aldrin has produced an increase in hepatocellular tumors in mice (Davis 1965, Epstein 1975, NCI 1978). In contrast, chronic feeding studies with aldrin in rats indicate that exposure was associated with nonneoplastic changes in the liver (NCI 1978, Fitzhugh et al. 1964).

EPA (1990) has classified aldrin as a group B2 agent (probable human carcinogen) and has developed an oral and inhalation cancer potency factor of $1.70 \times 10^{+1}$ $(mg/kg\text{-}day)^{-1}$ based on the increased incidence of liver carcinoma observed in male and female C3H mice (Davis 1965, Epstein 1975) and in male B6C3F1 mice (NCI 1978). EPA (1990) derived an oral reference dose (RfD) for aldrin of 3.0×10^{-5} mg/kg-day based on a study in which rats were fed aldrin for 2 years and displayed liver lesions at dose levels of 0.025 mg/kg-day (0.5 ppm) and greater (Fitzhugh et al. 1964). An uncertainty factor of 1,000 was used to calculate the oral RfD.

REFERENCES

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1987. Toxicological profile for Aldrin/Dieldrin. Draft. USPHS. Atlanta, Georgia.

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, Ohio. Pp. 17, 196. (As cited in ATSDR 1987).

BEYERMANN, K. and ECKRICH, W. 1973. Gas-chromatographische Bestimmung von Insecticid-Spuren in Luft. Z. Anal. Chem. 265:4-7. (As cited in ATSDR 1987).

BORGMANN, A., KITSELMAN, C., DAHM, P., PANKASKIE, J., and DUTRA, F. 1952a. Toxicological studies of aldrin on small laboratory animals. Unpublished report of Kansas State College. (As cited in ATSDR 1987).

IATROPOULOS, M., MILLING, A., MILLER, W., NOHYNEK, G., ROZMAN, K., COULSTON, F., and KORTE, F. 1975. Absorption, transport, and organotropism of dichlorobiphenyl (DCB), dieldrin, and hexachlorobenzene (HCB) in rats. Environ. Res. 10:384-389 (As cited in ATSDR 1987)

JAGER, K. 1970. Aldrin, Dieldrin, Endrin, and Telodrin: An epidemiological and toxicological study of long-term occupational exposure. Elsevier Publ. Co., New York. Pp. 121-131 (As cited in ATSDR 1987)

NATIONAL CANCER INSTITUTE (NCI). 1978. Bioassay of aldrin and dieldrin for possible carcinogenicity. DHEW Publication No. (NIH) 78-821. NCI Carcinogenesis Tech. Rep. Ser. No. 21 NCI-C6-TR-21 (As cited in EPA 1988c)

OTTOLENGHI, A., HASEMAN, J., and SUGGS, F. 1974. Teratogenic effects of aldrin, dieldrin, and endrin in hamsters and mice. Teratology 9:11-16 (As cited in ATSDR 1987)

PROBST, G., MCMAHON, R., HILL, L., THOMPSON, D., EPP, J., and NEAL, S. 1981. Chemically-induced unscheduled DNA synthesis in primary rat hepatocyte cultures; A comparison with bacterial mutagenicity using 218 chemicals. Environ. Mutagenesis 3:11-32 (As cited in ATSDR 1987)

ROCCHI et al. 1980. (As cited in EPA 1988c)

SHELL. 1984. Review of mammalian and human toxicology, aldrin and dieldrin. Review series HSE 84.003. Shell Internationale Petroleum Maatschappij. B.V. The Hague. (As cited in ATSDR 1987)

SUNDARAM, K., DAMODARAN, V., VENKITASUBRAMANIAN, T. 1978a. Absorption of dieldrin through monkey and dog skin. Indian J. Exp. Biol. 16:101-103 (As cited in ATSDR 1987)

SUNDARAM, K., DAMODARAN, V., and VENKITASUBRAMANIAN, T. 1978b. Absorption of dieldrin through skin. Indian J. Exp. Biol. 16:1004-1007 (As cited in ATSDR 1987)

TREON, J. and CLEVELAND, F. 1955. Toxicity of certain chlorinated hydrogen insecticides for laboratory animals, with special reference to aldrin and dieldrin. Agric. Food Chem. 3:402-408 (As cited in ATSDR 1987)

WALKER, A., STEVENSON, D., ROBINSON, J., THORPE, E., ROBERTS, M. 1969. The toxicology and pharmacodynamics of dieldrin (HEOD): Two-year oral exposures of rats and dogs. Toxicol. Appl. Pharmacol. 15:345-373 (As cited in ATSDR 1987)

ALUMINUM

Little is known about the absorption of aluminum following oral exposure, although there are reports that this inorganic metal is absorbed from the gastrointestinal tract to some extent (NAS 1982). Aluminum has low acute toxicity following oral exposure. Oral LD_{50}s in several animal species range from 380 to 780 mg/kg (EPA 1985). Neurofibrillar degeneration has been-observed in experimental animals given subcutaneous injections of aluminum (Goyer 1986). Intratracheal and intraperitoneal administration of aluminum compounds have been associated in experimental animals with pulmonary fibrosis and fibrotic peritonitis, respectively (NAS 1982). Evidence to date suggests that aluminum is not teratogenic or fetotoxic; however, rats exposed by gavage to 2.5 mg/kg aluminum for 6 months had reduced sperm counts and reduced sperm motility (EPA 1985). Studies to evaluate the potential of aluminum or its salts to induce mutagenic or carcinogenic effects have yielded negative results (EPA 1985). In humans, pulmonary fibrosis has been observed following inhalation of aluminum fumes. Aluminum has been presumed to be a potential etiological factor in two neurological disorders: Alzheimer's disease and chronic renal failure accompanied by senile dementia (Goyer 1986). However, the importance of aluminum in these disorders had not been fully established. Because there are inadequate dose-response data from which to estimate an acceptable daily intake level for ionic aluminum. no health-based criteria have been established by EPA.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. National primary drinking water regulation; synthetic organic chemicals, inorganic chemicals and microorganisms. Fed. Reg. 50:46937-47025 (November 13, 1985)

GOYER, R.A. 1986. Toxic effects of metals. In Doull, J. Klaassen, C.D., and Amdur, M.D., eds. Casarett and Doull's Toxicology. 3rd ed. MacMillan Publishing Co., New York

NATIONAL ACADEMY OF SCIENCE (NAS). 1982. Drinking Water and Health. 4 vols. National Academy Press, Washington, D.C.

AMMONIA

Ammonia is absorbed by inhalation and ingestion; absorption by dermal or ocular exposure is likely at concentrations high enough to cause tissue injury (Wands 1982). Acute dermal exposure may result in second degree skin burns (Levy et al. 1964). Acute exposure to ammonia vapors results in a burning sensation of the nose and throat, laryngitis, pharyngitis and rhinitis in humans (NIOSH 1974). Long-term symptoms such as pulmonary edema, bronchopneumonia, and pneumonitis have been reported in individuals accidentally exposed to ammonia gas (NIOSH 1974). Accidental poisoning or intentional ingestion of ammonia solutions by humans, frequently results in severe burning pain in the mouth, throat, and stomach (NIOSH 1974). In experimental animals, acute inhalation exposure has been found to produce eye and respiratory tract irritation (Propkopieva et al. 1973) and to reduce ciliary activity in rat tracheas (Dalhamn 1956). Subchronic inhalation exposures in animals can produce congestion of the spleen, liver, and kidney, degenerative changes in the adrenal gland, and nonspecific lung inflammation (Weatherby 1952, Coon et al. 1970).

EPA (1990) reported a value of 34 mg/liter as a chronic oral reference dose (RfD). This value is equivalent to 0.97 mg/kg-day assuming a 70-kg individual consumes 2 liters of water per day. The oral RfD was based upon the taste threshold (EPA 1981, WHO 1986). A safe concentration may be higher, but the data are inadequate to assess. EPA (1990) has also derived an inhalation RfD of 0.36 mg/m^3 based upon the odor threshold and potential for respiratory tract irritation (Carson et al. 1981, Campbell et al. 1958). No safety factors were used to develop the RfDs.

REFERENCES

CAMPBELL, C.L., DAWES, R.K., DEOLALKA, S., and MERRITT, M.C. 1958. Effect of certain chemicals in water on the flavor of brewed coffee. Food Res. 23:575-579 (As cited in EPA 1981)

CARSON, B.L., BEALL, C.M., ELLIS, H.V., III, and BAKER, L.H. 1981. Ammonia Health Effects. Prepared by Midwest Research Institute for Environmental Protection Agency, Office of Mobile Source Air Pollution Control, Emission Control Technology Division, Ann Arbor, Michigan. EPA 460/3-81-027

COON, R.A., JONES, R.A., JENKINS, L.L., Jr., and SIEGEL, J. 1970. Animal inhalation studies on ammonia, ethylene glycol, formaldehyde, dimethylamine, and ethanol. Toxicol. Appl. Pharmacol. 16:646-655

DAHLMANN, T. 1956. Mucus flow and ciliary activity in the trachea of healthy rats and rats exposed to respiratory irritant gases. Acta Physiol. Scand. 36 (Suppl. 123):93-97 (As cited in NIOSH 1974)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1981. Ambient Water Quality Criterion for the Protection of Human Health: Ammonia. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria Assessment Office, Cincinnati, Ohio, for the Office of Water Regulations and Standards, Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

LEVY, D.M., DIVERTIE, M.B., LITZOW, T.J., and HENDERSON, J.W. 1964. Ammonia burns of the face and respiratory tract. JAMA 190:873-876

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1974. Criteria for a Recommended Standard—Occupational Exposure to Ammonia. U.S. DHEW (NIOSH) Publication No. 74-136

PROPKOPIEVA, A.S., YUSHOV., G.G., and UBASHEEV, I.O. 1973. [Materials for a toxicological characteristics of the one-term effect of ammonia on the organism of animals after brief exposure] Gig. Tr. Prof. Zabol. 6:56-57 (as cited in WHO 1976)

WANDS, R.C. 1982. Alkaline Materials. In Clayton, G.D., and Clayton, F.E., eds. Pattys Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York

WEATHERBY, J.H. 1952. Chronic toxicity of ammonia fumes by inhalation (1985). Proc. Soc. Exp. Biol. Med. 81:300-301

WORLD HEALTH ORGANIZATION (WHO). 1986. Environmental Health Criteria. 54. Ammonia. WHO, Geneva, Switzerland

In animals, aniline has been found to be rapidly absorbed following oral administration, application to the skin, or inhalation exposure (IARC 1982). The most prominent effect of acute exposure to aniline hydrochloride is methemoglobinemia and accompanying cyanosis, headache, vertigo, and mental confusion (NCI 1978). In humans, chronic aniline hydrochloride exposure can result in anemia, anorexia, weight loss, and cutaneous lesions (NCI 1978). Long-term oral exposure to aniline has produced decreased body weight and chronic inflammatory biliary lesions in mice; fatty metamorphosis, fibrosis, and papillary hyperplasia of the spleen, liver and kidney hemosiderosis, and endometrial polyps have been observed in rats (NCI 1978). Rats exposed to aniline hydrochloride in the diet for 2 years have exhibited increased incidences of hemangiosarcoma in the spleen, fibrosarcoma and sarcoma in the body cavity and malignant pheochromocytoma (NCI 1978, CIIT 1982).

EPA (1990) has derived an inhalation RfC for aniline of 1.00×10^{-3} mg/m^3 with an uncertainty factor of 3,000 based on the absence of toxicity to rats, guinea pigs, and mice after subchronic inhalation exposure to 3.4 mg/m^3 aniline vapors (Oberst et al. 1956). In another study in which rats were subchronically exposed to aniline vapors, a LOAEL (lowest-observed-effects-level) of 3.4 mg/m^3 was derived based on mild splenic effects (duPont deNemours 1982). EPA (1990) has developed an oral cancer potency factor for aniline of 5.7×10^{-3} (mg aniline hydrochloride/kg-day)$^{-1}$ based on the incidence of splenic sarcomas in rats (CIIT 1982). Aniline has a weight-of-evidence classification of Group B2--Probable Human Carcinogen.

REFERENCES

CIIT (Chemical Industry Institute of Toxicology). 1982. 104-Week Chronic Toxicity Study in Rats: Aniline Hydrochloride. Final Report

DUPONT DENEMOURS AND COMPANY, INC. 1982. Subacute inhalation toxicity study of aniline in rats. OTS No. 0215025.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Health and Environmental Effects Profile for Aniline. Environmental Criteria and Assessment Office, Cincinnati, Ohio. EPA 600/X-85/356

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1982. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Some Aromatic Amines, Anthraquinones and Nitroso Compounds, and Inorganic Fluorides Used in Drinking Water and Dental Preparations. IARC, Lyon, France. Vol. 27

NATIONAL CANCER INSTITUTE (NCI). 1978. Bioassay of Aniline Hydrochloride for Possible Carcinogenicity. NCI Carcinogenesis Technical Report Series No. 130. DHEW (NIH) Publication No. 78-1318.

OBERST FW, HACKLEY E, COMSTOCK C. 1956. Chronic toxicity of aniline vapor (5 ppm) by inhalation. Arch Ind Health 13:379-384.

ANTIMONY

Antimony is a metal which occurs both in the trivalent and pentavalent oxidation states (EPA 1980). Absorption of this metal via oral and inhalation routes of exposure is low (EPA 1980). Humans and animals exposed acutely by oral or inhalation exposures to either the trivalent or pentavalent forms of antimony displayed electrocardiogram (ECG) changes and myocardial lesions (EPA 1980). Pneumoconiosis has been observed in humans exposed by acute inhalation and dermatitis has occurred in individuals exposed either orally or dermally. Oral administration of therapeutic doses in humans has been associated with nausea, vomiting, and hepatic necrosis (EPA 1980). Chronic exposure by inhalation of antimony has led to respiratory effects including macrophage proliferation and activity, fibrosis and pneumonia in animals (EPA 1980). Chronic oral exposure in rats has resulted in altered blood glucose and blood cholesterol levels and decreased lifespan (Schroeder et al. 1970). A single report (Balyeava 1967) noted an increase in spontaneous abortions, premature births, and gynecological problems in 318 female workers exposed to a mixture of antimony metal, antimony trioxide, and antimony pentasulfide dusts.

EPA (1990) derived an oral reference dose (RfD) of $4x10^{-4}$ mg/kg-day for antimony based on a chronic oral study (Schroeder et al. 1970) in which rats given the metal in drinking water had altered blood glucose and blood cholesterol levels and decreased lifespan. An uncertainty factor of 1,000 and a LOAEL of 0.35 mg/kg-day were used to derive the oral RfD.

REFERENCES

BALYAEVA, A.P. 1967. The effects of antimony on reproduction. Gig. Truda Prof. Zabol. 11:32

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Antimony. Office of Water Regulations and Standards, Washington, D.C. EPA 440/5-80-020

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

SCHROEDER, H.A., MITCHNER, M., and NASOR, A.P. 1970. Zirconium, niobium, antimony, vanadium, and lead in rats: Life-term studies. J. Nutr. 100:59-66

ARSENIC

Both inorganic and organic forms of arsenic are readily absorbed via the oral and inhalation routes. Soluble forms are more readily absorbed than insoluble forms (EPA 1984). Approximately 95% of soluble inorganic arsenic administered to rats is absorbed from the gastrointestinal tract (Coulson et al. 1935, Ray-Bettley and O'Shea 1975). Approximately 70%-80% of arsenic deposited in the respiratory tract of humans has been shown to be absorbed (Holland et al. 1959). Dermal absorption is not significant (EPA 1984). Acute exposure of humans to metallic arsenic has been associated with gastrointestinal effects, hemolysis, and neuropathy (EPA 1984). Chronic exposure of humans to this metal can produce toxic effects on both the peripheral and central nervous systems, keratosis, hyperpigmentation, precancerous dermal lesions, and cardiovascular damage (EPA 1984, Tseng 1977). Arsenic is embryotoxic, fetotoxic, and teratogenic in several animal species (EPA 1984). Arsenic is a known human carcinogen. Epidemiological studies of workers in smelters and in plants manufacturing arsenical pesticides have shown that inhalation of arsenic is strongly associated with lung cancer and perhaps with hepatic angiosarcoma (EPA 1984). Ingestion of arsenic has been linked to a form of skin cancer and more recently to bladder, liver, and lung cancer (Tseng 1977, Tseng et al. 1968, Chen et al. 1986).

EPA has classified arsenic in Group A—Human Carcinogen—and has developed inhalation (EPA 1990) and oral (EPA 1988) slope factors of 50 $(mg/kg\text{-}day)^{-1}$ and 2.0 $(mg/kg\text{-}day)^{-1}$, respectively. The inhalation potency factor is the geometric mean value of potency factors derived from four occupational exposure studies on two different exposure populations (EPA 1984). The oral cancer potency factor was based on an epidemiological study in Taiwan which indicated an increased incidence of skin cancer in individuals exposed to arsenic in drinking water (Tseng 1977). A risk assessment for noncarcinogenic effects of arsenic is currently under review by EPA (1990). An oral reference dose (RfD) of $1x10^{-3}$ mg/kg-day was calculated for arsenic based on the same oral epidemiological study (Tseng 1977) which also showed greater incidence of keratosis and hyperpigmentation in humans (EPA 1990). An uncertainty factor of 1 was used to derive the oral RfD. This RfD is presently being reconsidered by the RfD workgroup.

REFERENCES

CHEN, C., CHUANG, Y., YOU, S., LIN, T., and WU, H. 1986. A retrospective study on malignant neoplasms of bladder, lung, and liver in blackfoot disease endemic area in Taiwan. Br. J. Cancer 53:399-405

COULSON, E.J., REMINGTON, R.E., and LYNCH, K.M. 1935. Metabolism in the rat of the naturally occurring arsenic of shrimp as compared with arsenic trioxide. J. Nutr. 10:255-270

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Assessment Document for Inorganic Arsenic. Office of Health and Environmental Assessment, Washington D.C. EPA 600/8-83-021F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Special Report on Ingested Inorganic Arsenic Skin Cancer: Nutritional Essentiality. Risk Assessment Forum. U.S. Environmental Protection Agency, Washington, D.C. EPA/625/3-87/013F. July 1988

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Health Criteria and Assessment Office, Cincinnati, Ohio.

HOLLAND, R.H., McCALL, M.S., and LANZ, H.C. 1959. A study of inhaled arsenic-74 in man. Cancer Res. 19:1154-1156

RAY-BETTLEY, F., and O'SHEA, J.A. 1975. The absorption of arsenic and its relation to carcinoma. Br. J. Dermatol. 92:563-568

TSENG, W.P., CHU, H.M., HOW, S.W., FONG, J.M., LIN, C.S., and YEH, S. 1968. Prevalence of skin cancer in an endemic area of chronic arsenicism in Taiwan. J. Natl. Cancer Inst. 40:453-463

TSENG, W.P. 1977. Effects and dose-response relationships of skin cancer and blackfoot disease with arsenic. Environ. Health Perspect. 19:109-119

BARIUM

Adverse effects in humans following oral exposure to soluble barium compounds include gastroenteritis, muscular paralysis, hypertension, ventricular fibrillation, and central nervous system damage (EPA 1984). Inhalation of barium sulfate or barium carbonate in occupationally exposed workers has been associated with baritosis, a benign pneumoconiosis (Goyer 1986). Human epidemiologic studies have shown that chronic ingestion of drinking water containing high levels of barium induces high blood pressure that results in a prevalance of hypertension, stroke, heart and renal disease (Brenniman and Levy 1984, Wones et al. 1990). Chronic oral exposure of experimental animals to barium in drinking water also increases blood pressure (EPA 1984, Perry et al. 1983). Inhalation of barium carbonate dust by experimental animals has been associated with reduced sperm count, increased fetal mortality, atresia of the ovarian follicles, decreased body weight, and alterations in liver function (EPA 1984, Tarasenko et al. 1977).

EPA (1990a) derived an oral reference dose (RfD) based on two human epidemiologic studies which did not observe any adverse effects following consumption of drinking water containing barium (Brenniman and Levy 1984, Wones et al. 1990). Although no LOAEL was identified, the effect of concern was high blood pressure. Using a NOAEL of 0.21 mg/kg-day and an uncertainty factor of 3, an oral RfD of $7x10^{-2}$ mg/kg-day was calculated. A subchronic RfD of $5x10^{-2}$ mg/kg-day has been established by EPA (1990b) based on increased blood pressure in rats chronically exposed to 5.1 mg barium/kg-day in their drinking water (Perry et al. 1983). EPA (1990b) has also developed a chronic and subchronic inhalation RfD of $1.0x10^{-4}$ mg/kg-day and $1.0x10^{-3}$ mg/kg-day for barium based on a study by Tarasenko et al. (1977). In this study rats were exposed to barium carbonate dust at airborne concentrations of up to 5.2 mg/m^3 for 4-6 months. Adverse effects noted at this concentration included decreased body weight, alterations in liver function, and increased fetal mortality. Uncertainty factors of 1,000 and 100 were used in developing the RfDs.

REFERENCES

BRENNIMAN GR, AND LEVY PS. 1984. High barium levels in public drinking water and its association with elevated blood pressure. In: Advances in Modern Toxicology IX, E.J., Calabrese, Ed. Princeton Scientific Publication, Princeton, NJ. p 231-249.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Barium. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-021

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office. Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

GOYER, R.A. 1986. Toxic effects of metals. In Klaasen, C.D., Amdur, M.D., and Doull, J., eds. Casarett and Doull's Toxicology: The Basic Science of Poisons. 3rd ed. Macmillan Publishing Co., New York. Pp. 623-624

PERRY, H.M., KOPP, S.J., ERLANGER, M.W., and PERRY, E.G. 1983. XVII. Cardiovascular effects of chronic barium ingestion. In Hemphill, D.D., ed. Proceedings of he University of Missouri's 17th Annual Conference of Trace Substances in Environmental Health. University of Missouri Press, Columbia, Missouri (As cited in EPA 1984)

TARASENKO, M., PROMIN, O., and SILAYEV, A. 1977. Barium compounds as industrial poisons (an experimental study). J. Hyg. Epiderm. Microbial. Immunol. 21:361 (As cited in EPA 1984)

WONES RG, STADLER BL, FROHMAN LA. 1990. Lack of effect of drinking water barium on cardiovascular risk factor. Environ Health Perspective. 85:1-13

BENZENE

Benzene is readily absorbed following oral and inhalation exposure (EPA 1985). The toxic effects of benzene in humans and other animals following exposure by inhalation include central nervous system effects, hematological effects, and immune system depression. In humans, acute exposures to high concentrations of benzene vapors have been associated with dizziness, nausea, vomiting, headache, drowsiness, narcosis, coma, and death (NAS 1976). Chronic exposure to benzene vapors can produce reduced leukocyte, platelet, and red blood cell counts (EPA 1985). Benzene induced both solid tumors and leukemias in rats exposed by gavage (Maltoni et al. 1985). Many studies have also described a causal relationship between exposure to benzene by inhalation (either alone or in combination with other chemicals) and leukemia in humans (IARC 1982, Rinsky et al. 1981, Ott et al. 1978, Wong et al. 1983).

Applying EPA's criteria for evaluating the overall evidence of carcinogenicity to humans, benzene is classified in Group A (Human Carcinogen) based on adequate evidence of carcinogenicity from epidemiological studies. EPA (1990) derived both an oral and an inhalation cancer potency factor for benzene of 2.9×10^{-2} $(mg/kg\text{-}day)^{-1}$. This value was based on several studies in which increased incidences of nonlymphocytic leukemia were observed in humans occupationally exposed to benzene principally by inhalation (Rinsky et al. 1981, Ott et al. 1978, Wong et al. 1983). EPA (1990) is currently reviewing both oral and inhalation RfDs for benzene, for which the status is pending.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Drinking Water Criteria document for Benzene (Final Draft). Office of Drinking Water, Washington, D.C. April 1985

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1982. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Some Aromatic Amines, Anthraquinones and Nitroso Compounds, and Inorganic Fluorides Used in Drinking-Water and Dental Preparations. World Health Organization, Lyon, France. Vol. 27

MALTONI, C., CONTI, B., COTTI, G., and BELPOGGI, F. 1985. Experimental studies on benzene carcinogenicity at the Bologna Institute of Oncology: Current results and ongoing research. Am. J. Ind. Med. 7:415-446

NATIONAL ACADEMY OF SCIENCE (NAS). 1976. Health Effects of Benzene: A Review Committee on Toxicology, Assembly of Life Sciences. National Research Council, Washington, D.C.

OTT, M.G., TOWNSEND, J.C., FISHBECK, W.A., and LANGNER, R.A. 1978. Mortality among individuals occupationally exposed to benzene. Arch. Environ. Health 33:3-10

RINSKY, R.A., YOUNG, R.J., and SMITH, A.B. 1981. Leukemia in benzene workers. Am. J. Ind. Med. 3:217-245.

WONG, O., MORGAN, R.W., and WHORTON, M.D. 1983. Comments on the NIOSH Study of Leukemia in Benzene Workers. Technical report submitted to Gulf Canada, Ltd., by Environmental Health Associates.

BERYLLIUM

Beryllium is not readily absorbed by any route of exposure. Occupational exposure to beryllium results in bone, liver, and kidney depositions (EPA 1986). In humans, acute respiratory effects due to beryllium exposure include rhinitis, pharyngitis, tracheobronchitis, and acute pneumonitis. Dermal exposure to soluble beryllium compounds can cause contact dermatitis, ulceration, and granulomas (Hammond and Beliles 1980). Ocular effects include conjunctivitis and corneal ulceration from splash burns. The most common clinical symptom caused by chronic beryllium exposure is granulomatous lung inflammation (IARC 1980, EPA 1986). Chronic skin lesions sometimes appear after a long latent period in conjunction with the pulmonary effects. Systemic effects from beryllium exposure may include right heart enlargement with accompanying cardiac failure, liver and spleen enlargement, cyanosis, digital clubbing, and kidney stone development (EPA 1986, Schroeder and Mitchner 1975). Beryllium has been shown to be carcinogenic in experimental animals resulting primarily in lung and/or bone tumors when given by injection, intratracheal administration, or inhalation (EPA 1986). Chronic oral administration to rats resulted in an increased occurrence of gross tumors of all sites combined (Schroeder and Mitchner 1975). Several epidemiological studies have suggested that occupational exposure to beryllium may result in an increased lung cancer risk although the data are inconclusive (EPA 1986, Wagoner et al. 1980).

Beryllium has been classified by EPA in Group B2--(Probable Human Carcinogen) based on increased incidences of lung cancer and osteosarcomas in animals (EPA 1990). EPA (1990) has calculated an inhalation cancer potency factor of 8.4 $(mg/kg\text{-}day)^{-1}$ based on the relative risk for lung cancer, estimated from an epidemiological study by Wagoner et al. (1980). EPA (1990) established an oral cancer potency factor of 4.3 $(mg/kg\text{-}day)^{-1}$ based on the induction of tumors (type and site unspecified) in rats chronically administered beryllium sulfate in their drinking water (Schroeder and Mitchner 1975). EPA (1990) has also developed an oral reference dose (RfD) for beryllium of 5.0×10^{-3} mg/kg-day based on a study by Schroeder and Mitchner (1975) in which rats exposed to 0.54 mg/kg-day beryllium sulfate (the highest dose tested) in drinking water for a lifetime did not exhibit adverse effects. An uncertainty factor of 100 was used to develop the RfD.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Health Assessment Document for Beryllium. Review Draft. Office of Health and Environmental Assessment, Washington, D.C. EPA 600/8-84-026B. April 1986

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

HAMMOND, P.B., and BELILES, R.P. 1980. Metals. In Doull, J., Klaassen, C.D., and Amdur, M.O., eds. Casarett and Doull's Toxicology: The Basic Science of Poisons. 2nd ed. Macmillan Publishing Co., New York. Pp. 438-439

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1980. Some Metals and Metallic Compounds. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. World Health Organization, Lyon, France. Vol. 23, pp. 143-204

SCHROEDER, H.A., AND MITCHNER, M. 1975. Life-term studies in rats: Effects of aluminum, barium, beryllium, and tungsten. J. Nutr. 105:421-421

WAGONER, J.K., INFANTE, P.F., and BAYLISS, D.L. 1980. Beryllium: An etiologic agent in the induction of lung cancer, nonneoplastic respiratory disease, and heart disease among industrially exposed workers. Environ. Res. 21:15-34

BORON

Boron is absorbed in both humans and animals following oral and inhalation exposure as inferred by the toxicity associated with these routes of exposure. Boron is a nonmetallic element that is most commonly found in nature as boric acid and sodium borates (borax) (ATSDR 1990). Short-term ingestion of boron can be lethal to humans and animals. The minimal lethal doses of ingested boron (as boric acid) are reported to be 2-3g in infants, 5-6g in children, and 15-20g in adults (Locatelli et al. 1987, Wong et al. 1964); death has been attributed to respiratory failure. In rabbits, acute dermal exposure to boron oxide results in conjuctivitis and erythema (Wilding et al. 1959). In humans, acute and subchronic oral exposure can cause degenerative neurological effects, biochemical changes of the liver and kidney, and gastrointestinal disorders including vomiting and diarrhea (Wong et al. 1964, Linden et al. 1986). Infants are particularly susceptible to boron toxicity (Wong et al. 1964). Subchronic exposure to boron oxide vapors cause irritation of the nasal cavity in rats (Wilding et al. 1959). Workers occupationally exposed to boron as boron oxide and boric acid have experienced irritation of the upper respiratory tract (Garabrant et al. 1984, 1985). The testes of animals are particularly sensitive to subchronic and chronic ingestion of boron (borax and boric acid) as evidenced by testicular atrophy and impaired spermatogenesis (Seal and Weeth 1980, NTP 1987, Weir and Fisher 1972).

EPA (1990) derived an oral reference dose (RfD) for boron of $9x10^{-2}$ mg/kg-day based on a study by Weir and Fisher (1972) in which severe testicular atrophy and spermatogenic arrest were observed in dogs exposed orally to borax and boric acid; an uncertainty factor of 100 was used to derive the RfD.

REFERENCES

ATSDR. 1990. Agency for Toxic Substances and Disease Registry. Toxicological profile for boron and compounds. U.S. Public Health Service.

EPA. 1990. ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

GARABRANT, D.H., BERNSTEIN, L., PETERS, J.M., et al. 1984. Respiratory and eye irritation from boron oxide and boric acid dusts. J. Occup. Med. 26:584-586.

GARABRANT, D.H., BERNSTEIN, L., PETERS, J.M., et al. 1985. Respiratory effects of borax dust. Br. J. Ind. Med. 42:831-837.

LINDEN, C.H., HALL, A.H., KULIG, K.W., et al. 1986. Acute ingestions of boric acid. Clin. Toxicol. 24:269-279.

LOCATELLI, C., MINOIA, C., TONINI, M., et al. 1987. Human toxicology of boron with special reference to boric acid poisoning. G. Ital. Med. Lav. 9:141-146.

NTP. 1987. NTP Technical Report on the Toxicology and Carcinogenesis of Boric Acid in $B6C_3F_1$ Mice (Feed Studies). NTP TR324.

SEAL, B.S., WEETH, H.J. 1980. Effect of boron in drinking water on the male laboratory rat. Bull. Environ. Contam. Toxicol. 25:782-789.

WEIR, R.J., JR., FISHER, R.S. 1972. Toxicologic studies on borax and boric acid. Toxicol Appl. Pharmacol 23:351-364.

WILDING, J.L., SMITH, W.J., YEVICH, P., et al. 1959. The toxicity of boron oxide. Am. Ind. Hyg. Assoc. J. 20:284-289.

WONG, L.C., HEIMBACH, M.D., TRUSCOTT, D.R., et al. 1964. Boric acid poisoning: Report of 11 cases. Can. Med. Assoc. J. 90:1018-1023.

BROMIDE

Bromide (Br^-) is a negatively-charged, reactive ion frequently found as bromide-containing compounds. Bromide is readily absorbed via the gastrointestinal and respiratory tracts and is rapidly distributed. Its biological half-life is 12 days (NRC 1980). Physiological effects of the soluble bromides, sodium bromide, potassium bromide, and ammonium bromide, are similar and are all attributable to the bromide ion (Stokinger 1981). In humans, acute overdoses of bromide salts such as those used in oral sedatives, diuretics, and antiepileptics can cause vomiting, profound stupor, or death (Gosselin et al. 1984, Stokinger 1981). Systemic effects of acute bromide salt ingestion are primarily neurological manifestations: drowsiness, irritability, ataxia, vertigo, confusion, mania, hallucinations, sensory disturbances, and coma; other effects include skin rashes, increased spinal fluid pressures, and gastrointestinal disturbances (NRC 1980, Gosselin et al. 1984). Acute exposure to hydrogen bromide vapors can cause nasal irritation in humans (Stokinger 1981). Subchronic oral exposure to sodium bromide produces different effects in dogs, horses, rabbits, and rats (NRC 1980). Dogs experience gastrointestinal and nervous system toxicity; horses show weakness, ataxia, and paralysis; rabbits have decreased serum ascorbate; and, rats exhibit increased thyroid weights, hyperplasia of the thyroid, increased relative adrenal and prostate weights, decreased spermatogenesis, hair loss, decreased serum ascorbate, and a shortened latent period. Chronic ingestion of bromide salts as therapeutic agents can produce depression, ataxia, and psychoses in humans (Stokinger 1981). No health-based criteria have been established for bromide by EPA.

REFERENCES

GOSSELIN, R.E., SMITH, R.P., HODGE, H.C. 1984. Clinical Toxicology of Commercial Products. 5th ed. Williams and Wilkins, Baltimore. p.II-112.

NATIONAL RESEARCH COUNCIL (NRC). 1980. Drinking Water and Health. Vol 3. Safe Drinking Water Committee, Washington, D.C., pp.181-187.

STOKINGER, H.E. 1981. The Halogens and the Nonmetals Boron and Silicon. In Clayton, G. and Clayton, F., eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York. Vol 2B. pp.2965-2972.

BROMINE

Bromine (Br_2) is absorbed in humans and animals following inhalation, oral, and dermal exposure (NIOSH/OSHA 1981). Elemental bromine toxicity is attributed to its extreme chemical reactivity. Liquid bromine and its vapors are highly corrosive to the eyes, nose, throat, skin, and respiratory tract (Stokinger 1981). In humans, acute inhalation of bromine in small amounts causes coughing, a feeling of oppression, epistaxis, dizziness, and headache, followed by abdominal pain and diarrhea, and sometimes by a measle-like eruption on the face, trunk, and extremities (Stokinger 1981, NIOSH/OSHA 1981). Acute inhalation exposure to high concentrations of bromine can produce pulmonary edema, edema of the glottis, and pneumonia (NIOSH/OSHA 1981, ACGIH 1986), which can lead to death due to excessive choking (Proctor et al. 1988). Guinea pigs and rabbits acutely exposed to bromine vapors experienced lung edemas, pseudomembranous deposits on the trachea and bronchi, hemorrhages of the gastric mucosa, and functional disturbances of the central nervous system (Stokinger 1981). Brief dermal exposure to liquid bromine leads to the formation of vesicles and pustules which induce deep, painful, slow-healing ulcers if not removed at once (Stokinger 1981, NIOSH/OSHA 1981). Since bromine exposure is most common in the workplace, where bromine exists in the gaseous form, little information is available on the effects of oral exposure to bromine. ACGIH (1986) has recommended a Time-Weighted Average Threshold Limit Value (TLV-TWA) of 0.1 ppm (0.7 mg/m^3) and a Short-Term Exposure Limit (TLV-STEL) of 0.3 ppm (2 mg/m^3) based on irritation to the eyes and upper airways. EPA had not developed health-based criteria values for bromine.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS INC. (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, OH.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH/OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION (NIOSH/OSHA). Occupational Health Guideline for Bromine. U.S. Department of Health and Human Services. p. 1-5.

PROCTOR, N.H., HUGHES, J.P., FISCHMAN, M.L. 1988. Bromine. In Chemical Hazards of the Workplace. 2nd ed. J.B. Lippincott Company. Philadelphia, PA. p. 101-102.

STOKINGER, H.E. 1981. The Halogens and the Nonmetals Boron and Silicon. In Clayton, G. and Clayton, F., eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York. Vol 2B. pp.2965-2972.

BROMOBENZYL CYANIDE

Bromobenzyl cyanide is classified by NIOSH as an experimental pesticide (RTECS 1990). It can be inferred from available animal toxicity studies that bromobenzyl cyanide is readily absorbed following oral and inhalation exposure. Humans exposed to bromobenzyl cyanide vapors may experience respiratory tract irritation (at concentrations as low as 0.15 mg/m^3), bronchitis, or pneumonitis; oral exposure may cause hypotension, seizures or coma (HSDB 1990). If inhaled, a concentration of 0.90 mg/L bromobenzyl cyanide would be fatal to humans in 30 minutes (HSDB 1990); the LC_{50} for humans has been reported to be 3,500 mg/m^3 (RTECS 1990). Bromobenzyl cyanide is a severe ocular irritant in humans and rabbits causing pain, swelling, lacrimation, and photophobia; corneal corpuscles become swollen and brownish followed by macrophage infiltration, loss of normal endothelial reflex and the appearance of fine irregularities in the endothelium (HSDB 1990). Workers who are exposed daily to cyanide solutions may develop "cyanide rash," characterized by itching, and by macular, papular, and vesicular eruptions (HSDB 1990). Sixty-one percent of the rats exposed to 500 ppm bromobenzyl cyanide in their drining water for 4 weeks developed papillary or nodular hyperplasia of the urinary bladder after 32 weeks of dietary exposure (Hiasa et al. 1985). The lowest oral dose that caused death (LDLo) in rats has been determined to be 100 mg/kg (RTECS 1990). No health-based criteria have been derived by EPA.

REFERENCES

HIASA Y., Y. KITAHORI, N. KONISHI, T. SHIMOYAMA, A. MIYASHIRO. 1985. Trisodium Nitriloacetate Monohydrate: promoting effect in urinary bladder carcinogenesis in rats treated with n-butyl-n-(4-hydroxybutyl)nitrosamine. J Natl Cancer Institute. 74(1):235-239.

HAZARDOUS SUBSTANCES DATABANK (HSDB). 1990. The National Library of Medicine Database. The National Institutes of Health. U.S. Department of Health and Human Services.

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1990. National Institute for Occupational Safety and Health (NIOSH). U.S. Department of Health and Human Services.

TOXLINE. 1990. The National Library of Medicine Database. The National Institutes of Health. U.S. Department of Health and Human Services. 1981.

BROMOFLUOROBENZENE

No information is available on the toxicological properties and health effects of 4-bromofluorobenzene. However, studies in rabbits indicate that 2-bromofluorobenzene is a mild skin irritant at 500 mg/24 hours, and a moderate eye irritant at 100 mg/24 hours (RTECS 1990). The isomer 3-bromofluorobenzene is a more potent irritant, inducing moderate skin irritation at 20 mg/24 hours, and eye irritation at 20 mg/24 hours (RTECS 1990). No health-based criteria have been established by EPA.

REFERENCES

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES. 1990. Department of Health and Human Services.

BUTYL BENZYL PHTHALATE

Butyl benzyl phthalate is absorbed following oral exposure. Butyl benzyl phthalate is not especially toxic. Acute oral doses of 50,000 or 100,000 mg/kg administered to male rats resulted in testicular degeneration. Thymic atrophy was reported in both male and female rats given 100,000 mg/kg for 14 days (NTP 1982). Depressed body weight gain, testicular degeneration, and liver and kidney effects have been observed in animals subchronically administered benzyl butyl phthalate in the diet (NTP 1982, NTP 1985). Butyl benzyl phthalate has been tested for carcinogenicity in chronic feeding studies using mice and female rats, and via intraperitoneal injection in male mice (NTP 1982). In female rats, an increased incidence of myelomonocytic leukemia was observed in the high exposure group. No increased tumor incidence was noted for mice (NTP 1982).

EPA has classified butyl benzyl phthalate in Group C--Possible Human Carcinogen. EPA (1990) derived an oral RfD of $2x10^{-1}$ mg/kg-day for butyl benzyl phthalate based on a subchronic study in rats in which effects on body weight gain, testes, liver, and kidney were observed (NTP 1985). An uncertainty factor of 1,000 was used to derive the oral RfD. No inhalation criteria have been developed for butyl benzyl phthalate.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

NATIONAL TOXICOLOGY PROGRAM (NTP). 1982. Bioassay of Butyl Benzyl Phthalate for Possible Carcinogenicity. U.S. Department of Health and Human Services, Washington, D.C. DHHS (NIH) Publication No. 82-1769. NTP Technical Report Series No. 213

NATIONAL TOXICOLOGY PROGRAM (NTP). 1985. Project #12307-02,-3. Hazelton Laboratories of America, Inc. Unpublished Study. (As cited in EPA 1989)

CADMIUM

Gastrointestinal absorption of cadmium in humans ranges from 5-6% (EPA 1985a). Pulmonary absorption of cadmium in humans is reported to range from 10% to 50% (CDHS 1986). Cadmium bioaccumulates in humans, particularly in the kidney and liver (EPA 1985a,b). Chronic oral or inhalation exposure of humans to cadmium has been associated with renal dysfunction, itai-itai disease (bone damage), hypertension, anemia, endocrine alterations, and immunosuppression. Renal toxicity occurs in humans at a renal cortex concentration of cadmium of 200 μg/g (EPA 1985b). Epidemiological studies have demonstrated a strong association between inhalation exposure to cadmium and cancers of the lung, kidney, and prostate (EPA 1985b, Thun et al. 1985). In experimental animals, cadmium induces injection-site sarcomas and testicular tumors. When administered by inhalation, cadmium chloride is a potent pulmonary carcinogen in rats. Cadmium is a well-documented animal teratogen (EPA 1985b).

EPA (1990a,b) classified cadmium as a Group B1 agent (Probable Human Carcinogen) by inhalation. This classification applies to agents for which there is limited evidence of carcinogenicity in humans from epidemiologic studies. EPA (1990a,b) derived an inhalation cancer potency factor of 6.1 $(mg/kg\text{-}day)^{-1}$ for cadmium based on epidemiologic studies in which respiratory tract tumors were observed (Thun et al. 1985, EPA 1985b). Using renal toxicity as an endpoint, and a safety factor of 10, EPA (1990a,b) has derived two separate oral reference doses (RfD). The RfD associated with oral exposure to drinking water is $5x10^{-4}$ mg/kg-day, and is based upon the lowest-observed-adverse-effect level (LOAEL) of 0.005 mg/kg in humans (EPA 1985a, Friberg et al. 1974). The RfD associated with exposure to cadmium in food is $1x10^{-3}$ mg/kg-day.

REFERENCES

CALIFORNIA DEPARTMENT OF HEALTH SERVICES (CDHS). 1986. Report to the Scientific Review Panel on Cadmium. Part B. Health Effects of Cadmium. Revised. Prepared by the Epidemiological Studies and Surveillance Section, Berkeley, California. September 19, 1986

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria Document for Cadmium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, Ohio for the Office of Water Regulations and Standards, Washington, D.C. EPA 440/5-80-025

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Drinking Water Criteria Document for Cadmium. Final Draft. Office of Drinking Water, Washington, D.C. April 1985. PB86-117934

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. Updated Mutagenicity and Carcinogenicity Assessment of Cadmium. Addendum to the Health Assessment Document for Cadmium (May 1981; EPA/600/8-81/023). Office of Health and Environmental Assessment, Washington, D.C. June 1985. EPA 600/8-83-025F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Health Advisory for Cadmium. Office of Drinking Water, Washington, D.C. March 31, 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

FRIBERG, L., PISCATOR, M., NORDBERG, G.F., and KJELLSTROM, T. 1974. Cadmium in the Environment. 2nd ed. CRC Press, Inc., Boca Raton, Florida

THUN, M.J., SCHNORR, T.M., SMITH, A.B., HALPERIN, W.E., and LEMEN, B.A. 1985. Mortality among a cohort of U.S. cadmium production workers—an update. JNCI 74:325-333

CALCIUM

Calcium is an essential nutrient which comprises a major portion (90%) of the bone and is necessary for the functional integrity of the nerve and muscle fibers where it influences the excitability and relase of neurotransmitters (Haynes and Murad 1985). The recommended daily allowance of calcium is 800 mg/day for adults. Approximately thirty percent of ingested calcium is absorbed primarily in the proximal segments of the small intestine (Haynes and Murad 1985). Prolonged deficiency may result in osteoporosis and in newborn children, tetany (twitches and spasms) is prevalent. Calcium is relatively non-toxic when administered orally. Calcium salts may have toxic effects depending on the toxicity of the anion constituent. Examples of potentially toxic calcium salts include calcium chloride, flouride, bromide, and phosphate. Diets that are high in calcium have produced symptoms of zinc deficiency in rats, chickens and pigs after prolonged feeding (Hedsted 1957). Peach (1975) indicated that calcium intakes in excess of 1,000 mg/L when coupled with high vitamin D intake raise blood levels of calcium and can depress serum magnesium levels following prolonged periods (NRC 1980). Kidney stones in humans have been associated with high calcium intakes. Hypercalciemia, a pathological condition of retaining excess calcium in the body, results in increased nerve and muscle excitation. This is manifested clinically by muscle weakness, lethargy, and eventually coma. Kidney and renal dysfunction are also associated with excess calcium levels, with prominent pathological changes in the collecting ducts and distal tubules (Haynes and Murad 1985). There is no evidence that calcium is mutagenic, carcinogenic or teratogenic. No health based-criteria have been established by EPA.

REFERENCES

HAYNES RC., AND MURAD F. 1985. Agents affecting Calcification: Calcium, parathyroid hormone, calcitonin, vitamin D, and other compounds. in Gilman AG., Goodman LS., Rall TW., and Murad F, eds. The Pharmacological Basis of Therapeutics. MacMillan Publishing Co., New York, p. 1517-1643.

HEGSTED DM. 1957. Calcium requirements. Nutrition Review. 15:257-258 (As cited in NRC 1980)

NATIONAL RESEARCH COUNCIL (NRC). 1980. Drinking Water and Health. Washington DC. National Academy Press. Vol. 3. 269-270.

PEACH MJ. 1975. Cations: Calcium, Magnesium, Barium, Lithium and Ammonium. in Goodman LS., and Gilman A., eds. The Pharmacological Basis of Therapeutics. 5th ed. MacMillan Publishing Co. Inc. New York.

CARBON DISULFIDE

Carbon disulfide is absorbed largely through the lungs but toxic quantities can also be absorbed through the skin. Carbon disulfide can be lethal in humans exposed to high oral doses or concentrated vapors. The nonlethal acute effect of carbon disulfide is narcosis (Sax 1984). Neurotoxicity is the primary effect of carbon disulfide exposure and manifests as neurophysiological and behavioral changes in the structure and function of the peripheral nervous system (ATSDR 1990). Adverse neurological effects of human exposure to prolonged, high levels of carbon disulfide include organic brain damage, peripheral nervous system decrements, neurobehavioral dysfunction, and ocular and auditory effects (Andrews and Snyder 1986). Symptoms include nervousness, irritability, indigestion, excessive fatigue, loss of appetite and headaches (ACGIH 1986). Repeated exposure to carbon disulfide may also cause cardiovascular and gastrointestinal effects in humans (ATSDR 1990). Longer-term exposure of animals to vapor concentrations of carbon disulfide include inreased serum lipids, histopathological changes in the myocardium, visual impairment, hindlimb paralysis, and lethargy (ATSDR 1990). Developmental effects in rats exposed in utero include behavioral and learning deficits; fetal malformations were evident at vapor concentrations which elicited maternal toxicity (Tabacova et al. 1983). Carbon disulfide has also been reported to increase the incidence of fetal resorption in rabbits exposed to 25 mg/kg-day carbon disulfide in water (Price et al. 1984). Inhalation exposure of rabbits to concentrations of 11 mg/kg-day did not induce fetal toxicity or malformations (Hardin et al. 1981).

EPA (1990a) derived an oral RfD for carbon disulfide of 0.1 mg/kg-day based on a study in which no fetal toxicity or malformations were observed in rabbits following inhalation exposure to 11 mg/kg-day carbon disulfide (Hardin et al. 1981). An uncertainty factor of 100 was used to develop the RfD. EPA (1990b) established an inhalation RfC of $1x10^{-2}$ mg/m^3 for carbon disulfide based upon fetotoxicity and developmental effects in rabbits exposed via inhalation (Hardin et al. 1981).

REFERENCES

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1990. Toxicological profile for Carbon Disulfide. Draft for Public Comment. October 1990.

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of Threshold Limit Values and Biological Exposure Indices. 5th ed. ACGIH, Cincinnati, Ohio

ANDREWS L.S. AND SNYDER R. 1986. Toxic Effects of Solvents and Vapors. In Casarett and Doull's Toxicology. Klaassen C.D., Amdur M.O., and Doull J., eds. 3rd ed. MacMillian Publishing Co., New York. pp. 348

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

HARDIN, B.D., BOND, G.P., SIKOV, M.R., ANDREW, F.D., BELILES, R.P., and NIEMEIR, R.W. 1981. Testing of selected work place chemicals for teratogenic potential. Scand. J. Work Environ. Health 7(Suppl. 4):66-75

PRICE, et al. 1984. Teratologic Evaluation of Carbon Disulfide Administered to Rabbits on GEstation Days 6-19. NTIS PB84-192350

SAX, N.I. 1984. Dangerous Properties of Industrial Materials. Van Nostrand Reinhold Co., New York

TABACOVA, S., B. Nikiforov, L. Balabaeva. 1983. Carbon disulfide intrauterine sensitization. J. Appl. Toxicol. 3:223-229

CARBON TETRACHLORIDE

Carbon tetrachloride (CCl_4) is readily absorbed following oral and inhalation exposure. About 60% of an oral dose was absorbed within 6 hours, and up to 86% was absorbed by 24 hours. Absorption from the lung has been estimated at about 30% (EPA 1984). CCl_4, like many other chlorinated hydrocarbons, acts as a central nervous system depressant (ACGIH 1986). The toxic effects of oral and inhalation exposure to CCl_4 in humans and animals include damage to the liver, kidney and lung, although the liver is the most sensitive tissue (EPA 1985, Bruckner et al 1986). In animals, acute oral administration produces fatty infiltration and histological alterations in the liver. High doses produce irreversible liver damage and necrosis while the effects observed following lower doses are largely reversible (EPA 1985). Humans occupationally exposed to 5-15 ppm of CCl_4 experience biochemical alterations, nausea, headaches and in more severe cases, liver dysfunction (jaundice, enlargement and fatty infiltration) (ACGIH 1986, EPA 1984). Animals chronically exposed to CCl_4 exhibit similar effects to those observed following acute exposure. Prenatal toxicity has been demonstrated in mammalian fetuses and neonates after inhalation exposure in pregnant rats (EPA 1984), although CCl_4 has not been shown to be teratogenic (EPA 1985). Carbon tetrachloride is a carcinogen in animals producing mainly hepatic neoplasms. Oral administration of 30 mg/kg-day or higher for 6 months has been found to produce an increased frequency of hepatomas, hepatocellular adenomas and hepatocellular carcinomas in mice, rats and hamsters (EPA 1985).

EPA (1990a) has classified CCl_4 as a B2 agent (probable human carcinogen). The cancer potency factor for both oral and inhalation exposure is 1.3×10^{-1} $(mg/kg\text{-}day)^{-1}$ (EPA 1990a) and is based on several gavage studies in which hepatocellular carcinomas and hepatomas were observed in rats, mice and hamsters (Della Porta et al. 1961, Edwards et al. 1942, NCI 1976a, 1976b, 1977). EPA (1990a) has derived an oral reference dose (RfD) of 7×10^{-4} mg/kg-day based on a subchronic rat gavage study in which liver lesions were the most sensitive effect (Bruckner et al. 1986). A no-observed-adverse-effect level (NOAEL) of 0.71 mg/kg-day and an uncertainty factor of 1,000 were used to derive the RfD. EPA (1990b) derived a subchronic oral RfD of 7×10^{-3} mg/kg-day, using an uncertainty factor of 100, based on the same study and effect of concern.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. ACGIH, Inc. Cincinnati, Ohio

BRUCKNER, J.V., MACKENZIE, W.F., MURALIDHARA, S., et al. 1986. Oral toxicity of carbon tetrachloride: Acute, subacute and chronic studies in rats. Fund. Appl. Toxicol. 6(1):16-34

DELLA PORTA, G.B., TERRACINI, B., and SHUBIK, K.P. 1961. Induction with carbon tetrachloride of liver cell carcinomas in hamsters. J. Natl. Cancer Inst. 26:855-863

EDWARDS, J., HESTON, W.E., and DALTON, H.A. 1942. Induction of carbon tetrachloride hepatoma in strain L mice. J. Natl. Cancer Inst. 3:297-301

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Assessment Document for Carbon Tetrachloride. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA-600/8-82-001F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Drinking Water Criteria Document for Carbon Tetrachloride (Final Draft). Office of Drinking Water, Washington, D.C. January 1985

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

NATIONAL CANCER INSTITUTE (NCI). 1976a. Report on the Carcinogenesis Bioassay of Chloroform. Carcinogenesis Program, Division of Cancer Cause and Prevention, Washington, D.C.

NATIONAL CANCER INSTITUTE (NCI). 1976b. Carcinogenesis Bioassay of Trichloroethylene. Carcinogenesis Technical Report Series No. 2, Washington, D.C.

NATIONAL CANCER INSTITUTE (NCI). 1977. Bioassay of 1,1,1-Trichloroethane for Possible Carcinogenicity. Carcinogenesis Technical Report Series No. *, Washington, D.C.

CHLORIDE COMPOUNDS

Chloride (Cl^-) is a negatively charged, reactive ionic element of the halogen class, which is usually detected as chloride-containing compounds. There is a paucity of toxicologic information for the chloride ion however, abundant information is available on chloride-containing compounds such as hydrogen chloride, sodium chloride, and ammonium chloride. These chlorides are absorbed following inhalation, oral, and dermal exposure as inferred by the toxicity associated with these routes of exposure. Human inhalation exposure to hydrogen chloride gas at high concentrations causes necrosis of tracheal and bronchial epithelium, pulmonary edema, atelectasis, emphysema, and damage to the pulmonary blood vessels (Machle et al. 1942). Dermal exposure to gaseous hydrogen chloride can produce severe burns in workers (White 1934). Acute inhalation of high concentrations of hydrogen chloride mist can be lethal to small laboratory animals (NIOSH n.d.). Rabbits, guinea pigs, and pigeons subchronically exposed to hydrogen chloride experience slight unrest and irritation of eyes and nose. Chronic inhalation of hydrogen chloride at low concentrations causes erosion of the teeth, bleeding of the nose and gums, and ulceration of the nasal and oral mucosa in humans (Miller et al. 1956). Workers exposed to sodium chloride particles experience mild nasal irritation. The main systemic effect of excess sodium chloride absorption is on blood pressure elevation (NIOSH n.d.). Ammonium chloride is a mild irritant to the skin and respiratory passage (ACGIH 1986). No health-based criteria have been established for chloride by EPA.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS INC. (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, OH.

JONES F.L., JR. 1972. Journal of the American Medical Association. Vol. 222. (As cited in Stokinger 1981).

MACHLE W., ET AL. 1942. Journal of Industrial Hygiene and Toxicology. Vol. 24. p. 222. (As cited in Stokinger 1981).

MILLER F., et al. 1956. American Industrial Hygiene Association. Vol. 17. (As cited in Stokinger 1981).

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). No date. NIOSH Registry of Toxic Effects of Chemical Substances for 1976. Rockville, MD. (As cited in Stokinger 1981).

STOKINGER, H.E. 1981. The Halogens and the Nonmetals Boron and Silicon. In Clayton, G. and Clayton, F., eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York. Vol 2B. pp. 2954-2965.

WHITE, R.P. 1934. The Dermatergoses. 4th ed. Lewis, London. (As cited in Stokinger 1981).

CHLOROACETOPHENONE

INTRODUCTION

Chloroacetophenone (CN), or phenacyl chloride, is a powerful lacrimator (tearing agent) and a potent irritant to the upper respiratory tract (ACGIH 1986, RCRA ND). Because of its strong lacrimating capacity, CN is used as a riot-control agent under the name of Chemical Mace or "Tear gas" (ACGIH 1986).

TOXICOKINETICS

It can be inferred from the results of toxicity studies that CN is absorbed following inhalation or dermal exposure.

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

CN stimulates sensory nerve receptors in the skin and mucosa of the eyes and respiratory tract to induce a burning sensation of the eyes, nose, throat, and skin, and to cause excess lacrimation, blepharospasm, salivation, rhinorrhea, sneezing, coughing, labored breathing, and reduced respiration (NTP 1990). These symptoms occur almost instantaneously on exposure to CN and generally resolve within about 20 minutes after exposure has ceased; however, severe and permanent corneal injury has been demonstrated in laboratory animals following direct application of CN to the eye (NTP 1990, Gosselin et al. 1984). Acute dermal exposure to CN has been shown to cause marked and persistent allergic contact dermatitis in humans (NTP 1990). At higher concentrations, aerosol or liquid CN will cause skin irritation characterized by purpura, erythema, edema, desquamation, and vesication (NTP 1990, RCRA ND). Estimates from animal data indicate that the respiratory LC_{50} for CN in humans is 8,000-11,000 mg-min/m^3 (ACGIH 1986, NTP 1990). Human volunteers exposed to concentration time (Ct) doses of 350 mg-min/m^3 CN, which are extremely irritating to the eyes and respiratory tract, were relatively free from systemic toxicity (ACGIH 1986).

Chronic inhalation exposure of rodents to CN induced minimal to mild suppurative inflammation of the nasal mucosa in male rats, and increased incidences of hyperplasia and squamous metaplasia of nasal respiratory epithelium in male and female rats (NTP 1990). Female rats also had increases in inflammation, ulcers and squamous hyperplasia of the forestomach.

In a 2-year inhalation study of CN (85% pure) carcinogenicity there was no evidence of carcinogenic activity in mice or male rats exposed to 2 mg/m^3 (0.3 ppm) CN or mice exposed to 4 mg/m^3 (0.6 ppm); there was equivocal evidence of carcinogenic activity for female F344/N rats, based on a marginal increase in fibroadenomas of the mammary gland (NTP 1990). CN did not appear to be carcinogenic in a 5-month dermal bioassay conducted with mice (ACGIH 1986).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

ACGIH has recommended a time-weighted average Threshold-Limit-Value (TLV) of 0.05 ppm (0.3 mg/m^3) for repeated exposure to CN to prevent irritation and sensitization (ACGIH 1986). No health-based criteria have been derived by EPA. For the purposes of this risk assessment Clement has derived an inhalation reference concentration (RfC) based on nasal histopathology in the 2-year inhalation bioassay conducted by NTP (1990). Increased incidences of hyperplasia and squamous metaplasia of the nasal respiratory epithelium were statistically elevated ($p<0.05$) in female rats at 1 mg/m^3; male rats also had elevated ($p<0.05$) incidences of squamous metaplasia at this concentration. Thus, 1 mg/m^3 was designated as a LOAEL. Adjustments were made for intermittent

exposure and to obtain the human equivalent concentration using the EPA (1990) methodology for derivation of Inhalation Reference Concentrations (RfC). Using a safety factor of 1,000, the inhalation RfC derived for chloroacetophenone is $4x10^{-5}$ mg/cu.m. Derivations are provided in Appendix A.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the threshold limit values and biological exposure indices. 5th ed. Cincinnati, OH, Pp. 121.

ENVIRONMENTAL PROTECTION AGENCY. 1990. Interim Methods for Developement of Inhalation Reference Concentrations. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment. Office of Research and Development. Researc Triangle Park, NC. EPA 600/8-90/066A. August 1990. Review Draft.

GOSSELIN, R.E., SMITH, R.P., HODGE, H.C. 1984. Clinical toxicology of commercial products. 5th ed. Williams and Wilkins, Baltimore. Pp 96-97.

ICF FEASIBILITY STUDY. 1987.

MILITARY CHEMISTRY OF CHEMICAL COMPOUNDS FIELD MANUAL. 1975

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1985. NIOSH pocket guide to chemical hazards. US Department of Health and Human Services (DHHS). DHHS Publication No. 85-114.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1990. NTP Technical report on the toxicology and carcinogenesis studies of 2-chloroacetophenone in F334/N rats and B6C3F1 mice (inhalation studies). NTP TR 379.

RCRA FACILITY ASSESSMENT, EDGEWOOD AREA, APG, MD.

APPENDIX A FOR CHLOROACETOPHENONE

Conversion Factors: 6 hours/day, 5 days/7 day, inhalation rate (chronic, female F344 rat) of 0.24 cu. m/day and 20 cu. m/day for human; extrathoracic respiratory surface areas of 11.6 and 177 sq. cm for rats and human, respectively. This a respiratory effect of a soluble vapor.

$LOAEL_{[HEC]}$ (mg/m^3) = $LOAEL_{[ADJ]}$(mg/m^3) x RGDR

RGDR = (animal ventilation rate/ extrathoracic surface area) x (human extrathoracic surface area/human ventilation rate)
= (0.24 mg/cu.m/11.6 sq/cm) x (177 sq.cm/20 mg/cu.m) = 0.24

$LOAEL_{[HEC]}$ (mg/cu.m) = 1 mg/m^3 x (6hr/24 hr) x (5 days/7 days) x 0.24
= 0.04 mg/cu.m

$LOAEL_{[ADJ]}$ = 0.04

$LOAEL_{[HEC]}$ = 0.04 mg/cu.m

UNCERTAINTY FACTORS (UF):

Animal to Human (A) 10

LOAEL to NOAEL (L) 10

Human sensitivity (H) 10

$LOAEL_{[HEC]}$ = 0.04 mg/cu.m/ UF = RfC

RfC = $4x10^{-5}$ mg/cu.m

CHLOROACETOPHENONE (CN) BREAKDOWN PRODUCTS

INTRODUCTION

Chloroacetophenone (CN), or phenacyl chloride, is a powerful lacrimator (tearing agent) and a potent irritant to the upper respiratory tract (ACGIH 1986, RCRA ND). CN-breakdown products include acetophenone, benzophenone, and dichlorobenzophenone which are likely to exhibit milder yet similar toxicity to the parent compound CN.

TOXICOKINETICS

The primary routes of potential exposure to the breakdown products (such as benzophenone, acetophenone, and dichlorobenzophenone) are oral and percutaneous absorption. The low volatility of acetophenone makes significant exposure by the pulmonary route unlikely (Krasavage et al. 1981). Acetophenone is metabolized to methyl phenyl carbonal and benzoic acid; benzoic acid is conjugated with glycine and excreted in the urine as hippuric acid. Other minor urinary metabolites of acetophenone include o-, m-, and p-hydroxyacetophenone and mandelic acid (Krasavage et al. 1981).

P-hydroxybenzophenone is a urinary metabolite of benzophenone in rats (Stocklinski et al. 1980). No toxicokinetic information was found for dichlorobenzophenone.

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

Acetophenone has a low to moderate toxicity via inhalation exposure or ingestion and is the most toxic CN-breakdown product as indicated by the acute lethal doses in animals. The oral rat and mouse LD_{50}'s for acetophenone are 815 and 740 mg/kg, respectively (RTECS 1987). The oral rat and mouse LD_{50}'s for benzophenone are >10,000 and 2,895 mg/kg, respectively (RTECS 1990, Weiss 1986). The mouse intraperitoneal LD_{50}'s have been reported to be 200, 727 and 200 mg/kg for acetophenone, benzophenone and dichlorobenzophenone, respectively (RTECS 1987, 1990).

Liquid acetophenone and benzophenone are ocular and skin irritants on contact (Weiss 1986). Transient corneal injury may result from prolonged eye contact (Krasavage et al. 1981). Acetophenone can cause severe eye irritation in rabbits at 771 µg, and skin irritation at doses of 10 mg for 24 hours (RTECS 1987). Ingestion of acetophenone may cause CNS depression (slight narcotic effect). No adverse effects were noted in rats fed 102 mg/kg-day for 30 days, or in rats fed 10,000 ppm (423 mg/kg-day) in the diet for 17 weeks (Hagen et al. 1967). Dermal application of acetophenone to the skin of pregnant rats did not alter the gestation period, size of litter, weight of offspring, time for appearance of teeth or hair, opening of eyes, or appearance of reflexes (Krasavage et al. 1981). There is evidence that acetophenone is mutagenic in S. cerevisiae, and in the lungs of hamsters (RTECS 1987).

Benzophenone is not especially toxic, as indicated by acute lethal doses in rodents. Ingestion causes gastrointestinal disturbances such as nausea or vomiting. There is evidence to suggest that benzophenone, like its parent compound, CN, induces allergic reactions manifested as urticaria and contact sensitivities (Ramsey et al. 1972).

No toxicological information was available for dichlorobenzophenone.

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

No regulated standards or criteria have been promulgated for benzophenone or dichlorobenzophenone. However, EPA (1990) has derived an oral RfD of $1x10^{-1}$ mg/kg-day for acetophenone based on the appearance of general toxicity in rats (Hagen et al. 1967). A no-observed-adverse-effect level (NOAEL) of 10,000 ppm (423 mg/kg-day) and an uncertainty factor of 3,000 were used to calculate the RfD. No toxic effects were noted in this study.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the threshold limit values and biological exposure indices. 5th ed. Cincinnati, OH, Pp. 121.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office. Cincinnati, OH.

KRASAVAGE, W.J, J.L. O'DONOGHUE, G.D. DIVINCENZO. 1981. Ketones. In Clayton and Clayton, ed. Patty's Industrial Hygiene and Toxicology. pp. 4784-85.

RAMSEY D.L., H.J. COHEN, R.L. BAER. 1972. Allergic reaction to benzophenone. Simultaneous occurrence of urticarial and contact sensitivities. Arch Dermatol. 105(6):906-8.

RCRA FACILITY ASSESSMENT, EDGEWOOD AREA, APG, MD.

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1987. Vol. 3A. National Institute for Occupational Safety and Health (NIOSH). U.S. Department of Health and Human Services.

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1990. National Institute for Occupational Safety and Health (NIOSH). U.S. Department of Health and Human Services.

STOCKLINSKI, A.W., O.B. WARE, T.J OBERST. 1980. Benzophenone metabolism. I. Isolation of p-hydroxybenzophenone from rat urine. Life Sci. Feb 4; 26(5):365-9.

p-Chloroaniline can be absorbed following inhalation and oral exposure as inferred by the toxicity associated with these routes of exposure; liquid p-chloroaniline can be rapidly absorbed through the skin (Weiss 1986). p-Chloroaniline is a mild skin irritant and a severe eye irritant (RTECS 1987); dermal exposure has caused primary irritation to the skin and appendages of rabbits (RTECS 1987). Inhalation, ingestion and dermal exposure causes headache, drowsiness, and nausea, followed by unconsciousness; a bluish tint may be observed in the fingernails, lips, and ears that is indicative of cyanosis (Weiss 1986). Dermal contact with p-chloroaniline has been reported to cause more intense methemoglobinemia than by ingestion, and cases of hematuria have been attributed to hemorrhagic cystitis resulting from p-chloroaniline exposure (Gosselin et al 1984). Data indicate that p-chloroaniline is mutagenic inducing unscheduled DNA synthesis, microbial mutations without activation, mutations in microsomal assays and oncogenic transformation in hamster and rat embyro cells (RTECS 1987). p-Chloroaniline appears to be an equivocal tumorigenic agent that causes blood and vascular tumors in rats and mice following oral exposure (RTECS 1987). A chronic dietary bioassay conducted by the National Cancer Institute (NCI) observed nonneoplastic proliferative lesions in the spleen of rats treated with 12.5 mg p-chloroaniline/kg-day (NCI 1979). Confidence in this bioassay is low because a no-observable-effect level (NOEL) was not defined (EPA 1990).

EPA (1990) has derived an oral RfD of $4x10^{-3}$ mg/kg-day for p-chloroaniline based on nonneoplastic proliferative lesions of the splenic capsule in rats at the lowest dose tested (NCI 1979). A safety factor of 3,000 was applied to the lowest-adverse-effect level (LOAEL) of 12.5 mg/kg-day to calculate the RfD.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental criteria and assessment office, Cincinnati, Ohio.

GOSSELIN, R.E., SMITH, R.P., HODGE, H.C. 1984. Clinical toxicology of commercial products. 5th ed. Williams and Wilkins, Baltimore. Pp 208.

NATIONAL CANCER INSTITUTE (NCI). 1979. Bioassay of p-chloroaniline for possible carcinogenicity. NCI carcinogenesis Tech Rep Ser No. 189. NTIS PB 295896.

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1987. Vol. 3A. National Institute for Occupational Safety and Health (NIOSH). U.S. Department of Health and Human Services.

WEISS, G. 1986. Hazardous chemicals data book. 2nd ed. Noyes data corporation, Park Ridge, New Jersey. Pp 257.

CHLOROBENZENE

Evidence from toxicity studies suggests that chlorobenzene is absorbed following oral, inhalation, and dermal exposures (EPA 1985). In humans, acute and chronic exposures to chlorobenzene via inhalation or ingestion have been associated with central nervous system (CNS) effects (EPA 1985). In animals, acute exposure by inhalation causes sensory irritation, respiratory distress, narcosis and CNS depression which can result in death (EPA 1985). Subchronic oral or inhalation exposure can elicit neurotoxicity, liver and kidney lesions and adverse hematological effects (EPA 1984, EPA 1985, Dilly 1977, Monsanto 1967, NTP 1983). Results of reproductive studies with rats and dogs also indicate that chlorobenzene induces testicular lesions (EPA 1985).

EPA (1990a) derived an oral chronic RfD for chlorobenzene of $2x10^{-2}$ mg/kg-day based on a study by Monsanto (1967) in which dogs administered chlorobenzene in capsules for 90 days exhibited liver and kidney effects; an uncertainty factor of 1,000 was used to develop the RfD. EPA (1990b) also reported inhalation chronic and subchronic RfDs for chlorobenzene of $5x10^{-3}$ mg/kg-day and $5x10^{-2}$ mg/kg-day, respectvely, based on a study by Dilley (1977) in which rats exposed to chlorobenzene for 120 days exhibited liver and kidney effects; an uncertainty factor of 10,000 and 1,000 were used to develop the RfDs for chronic and subchronic exposures, respectively. EPA (1990b) derived a subchronic oral RfD of $2x10^{-1}$ mg/kg-day with an uncertainty factor of 100 based on the same study and effects of concern.

REFERENCES

DILLEY, J.V. 1977. Toxic Evaluation of Inhaled Chlorobenzene. NIOSH, Cincinnati, Ohio. Contract 210-76-0126

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Health Assessment Document for Chlorinated Benzenes. Final Report. Office of Health and Environmental Assessment, Washington, D.C. EPA/600/8-84/015F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Chlorobenzene. Office of Emergency and Remedial Response, Washington, D.C. EPA/540/1-86-040

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment. Environmental Assessment and Criteria Office, Cincinnati, Ohio for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

MONSANTO COMPANY. 1967. 13-Week Oral Administration--Dogs, Monochlorobenzene. EPA, OPTS, Washington, D.C. TSCA Sec 8(d) submission 8DHQ-1078-0202(2)

NATIONAL TOXICOLOGY PROGRAM (NTP). 1983. Carcinogenesis bioassay of chlorobenzene in F344/N rats and B6C3F$_1$ mice (gavage study) NTP-82-090 NIH Publ. No. 83-2517 (Draft)

o-CHLOROBENZYLIDENE MALONONITRILE

INTRODUCTION

o-Chlorobenzylidene malononitrile (CS) is a condensation product of chlorobenzaldehyde with malononitrile; it is a potent lacrimator and sternutator (sneezing agent) and is a peripheral sensory irritant (NTP 1990).

TOXICOKINETICS

CS is absorbed following inhalation exposure as indicated by the presence of its two metabolites, 2-chlorobenzyl malononitrile and 2-chlorobenzaldehyde, in the blood of rats (NTP 1990). CS is a potent toxin in animals via the intraperitoneal or intravenous routes due to its rapid metabolism, which leads to high levels of cyanide and thiocyanate in the urine; autopsies revealed marked congestion and hemorrhage of the respiratory system (NTP 1990).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

Symptoms of acute exposure to CS in humans include headache, eye irritation, excess lacrimation, blepharospasm, coughing, sneezing, excess salivation, constricting sensations in the chest, and burning sensations of the nose, throat, and exposed skin (NTP 1990, ACGIH 1986). Heavy concentrations will cause nausea and vomiting and may also cause intense irritation of mucous membranes and the central nervous system (ICF Feasibility Study 1987, Army 1975). Humans acutely exposed to CS could barely tolerate concentrations ranging from 4.3 to 6.7 mg/m^3 when reached gradually over a period of 30 minutes; a burning sensation and deep pain in the eyes persisted for 2 to 5 minutes following cessation of exposure, and erythema of the eyelids with some blepharospasm for 1 hour; there was a burning sensation in the throat with cough, followed by a constricting sensation in the chest; no therapy other than removal from exposure was necessary (NIOSH 1978).

Acute exposure to CS vapors causes irritation in rats and mice as evidenced by excessive lacrimation, spasm and closure of the eyelids, nasal discharge, attempts at mouth breathing, and erythema of the extremities (NTP 1990). Rats exposed to CS vapors for 13 weeks and two years had lesions in the upper respiratory tract, primarily in tissues of the nasal passage, but not in the lung (NTP 1990). These lesions were characterized by focal erosion with regenerative hyperplasia and squamous metaplasia of the respiratory epithelium and suppurative inflammation; the larynx and trachea of some exposed rats also showed acute inflammation and hyperplasia of the respiratory epithelium (NTP 1990). Short term animal exposure tests involving high exposures to CS did not reveal this compound to be embryolethal, or teratogenic (ACGIH 1986). A two-year inhalation study of CS carcinogenicity found no evidence of carcinogenic activity in mice or rats (NTP 1990).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

ACGIH recommends a ceiling limit of 0.05 ppm (4 mg/m^3) to prevent acute effects from handling CS. No health-based criteria have been derived by EPA. For the purposes of this risk assessment Clement has derived an inhalation reference concentration (RfC) based on nasal histopathology in male and female F344 rats in the two year inhalation study (NTP 1990). The incidence of hyperplasia and focal squamous metaplasia of the respiratory epithelium in the nasal passage were statistically elevated in both sexes at 0.75 mg/m^3. Thus, 0.25 mg/m^3 was designated as a NOAEL. Adjustments were made for intermittent exposure and to obtain the human equivalent concentration using the EPA

(1990) methodology for derivation of Inhalation Reference Concentrations (RfC). Using a safety factor of 100, the inhalation RfC derived for CS is $1x10^{-4}$ mg/cu.m. Derivations are provided in Appendix A.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the threshold limit values and biological exposure indices. 5th ed. Cincinnati, Ohio. Pp. 124.

ENVIRONMENTAL PROTECTION AGENCY. 1990. Interim Methods for Development of Inhalation Reference Concentrations. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment. Office of Research and Development. Research Triangle Park, NC. EPA 600/8-90/066A. August 1990. Review Draft.

ICF FEASIBILITY STUDY. 1987.

Army. 1975. MILITARY CHEMISTRY OF CHEMICAL COMPOUNDS FIELD MANUAL. U.S. Department of the Army. Washington, D.C.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1978. Occupational health guideline for o-chlorobenzylidene malonitrile. U.S. Department of Health and Human Services.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1990. NTP Technical report on the toxicology and carcinogenesis studies of CS2 (o-Chlorobenzal malononitrile) in F334/N rats and B6C3F1 mice (inhalation studies). NTP TR 377.

APPENDIX A

Conversion Factors: 6 hours/day, 5 days/7 day, inhalation rate (chronic, female F344 rat) of 0.24 cu. m/day and 20 cu. m/day for human; extrathoracic respiratory surface areas of 11.6 and 177 sq. cm for rats and human, respectively. This a respiratory effect of a soluble vapor.

$NOAEL_{[HEC]}$ (mg/m^3) = $NOAEL_{[ADJ]}$(mg/m^3) x RGDR

RGDR = (animal ventilation rate/ extrathoracic surface area) x (human extrathoracic surface area/human ventilation rate)
= (0.24 mg/cu.m/11.6 sq/cm) x (177 sq.cm/20 mg/cu.m) = 0.24

$NOAEL_{[HEC]}$ (mg/cu.m) = 0.25 mg/m^3 x (6hr/24 hr) x (5 days/7 days) x 0.24
= 0.01 mg/cu.m

$NOAEL_{[ADJ]}$ = 0.01

$NOAEL_{[HEC]}$ = 0.01 mg/cu.m

UNCERTAINTY FACTORS (UF):

Animal to Human (A) 10

Human sensitivity (H) 10

$NOAEL_{[HEC]}$ = 0.01 mg/cu.m/ 100 = RfC

RfC = $1x10^{-4}$ mg/cu.m

o-CHLOROBENZYLIDENE MALONONITRILE (CS) BREAKDOWN PRODUCTS

INTRODUCTION

o-Chlorobenzealdehyde and malononitrile are the two major breakdown products of o-Chlorobenzylidene malononitrile (CS). Malononitrile is a white, colorless solid used as a lubricating oil additive, and in anti-cancer agent synthesis (HSDB 1990). In the late 1940's malononitrile was used experimentally in the treatment of schizophrenia and depression (HSDB 1990). o-Chlorobenzealdehyde is a colorless to yellowish liquid used in synthesis of alcohols, acids, and in the rubber, tanning and paper industries (HSDB 1990).

TOXICOKINETICS

Studies of tissue homogenates exposed to malononitrile showed that cyanide and thiocyanate are produced (Clayton and Clayton 1981). Malononitrile possesses little if any acute toxicity in the absence of normal hepatic function in mice. It is activated by hepatic mechanisms to release cyanide which can account for major acute toxic effects (Willhite and Smith 1981). In rats 6.8% of intraperitoneally injected o-chlorobenzealdehyde was excreted in the urine as mercapturic acids (Rietveld et al. 1983).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

Malononitrile is a severe dermal and eye irritant in rabbits (RTECS 1990). The rat dermal LD_{50} is 350 mg/kg; convulsions and seizures were apparent prior to death (RTECS 1990). The mouse LD_{50}'s for oral and intraperitoneal routes are 19 and 13 mg/kg, respectively (RTECS 1990). In adults, the lethal dose is estimated to be 50 to 300 mg (HSDB 1990). o-Chlorobenzealdehyde has a mouse intraperitoneal LD_{50} of 10 mg/kg (RTECS 1990).

In humans, malononitrile is poisonous if swallowed, or inhaled. It causes a burning sensation in the mouth and throat, and red retinal arteries and veins. Acute effects include hypertension and tachycardia followed by bradycardia and hypotension; cardiac arrhythmias are also common. Respiratory effects include tachypnea and hyperpnea followed by respiratory depression and possibly pulmonary edema. Neurologic manifestations include headache, vertigo and agitation followed by combative behavior, coma, convulsions and death (HSDB 1990). Malononitrile used experimentally in the treatment of schizophrenia and depression induced redness, nausea, vomiting, headache shivering, muscle spasms and numbness (HSDB 1990).

In animals, acute subcutaneous exposure to 0.5% malononitrile caused inflammatory reaction on the skin of rats, guinea pigs and rabbits within 24 hours. Enlargement of the liver, kidney and spleen and histopathologic changes of the liver parenchymal cells and kidney epithelial cells were observed following subacute administration of malononitrile to rabbits (Krysiak et al. 1976). Mice acutely exposed to malononitrile vapors developed signs of respiratory distress, and subsequently became cyanotic, uncoordinated and were followed by tremors and convulsions leading to death (Clayton and Clayton 1981).

In humans, o-chlorobenzealdehyde can cause ocular, dermal or respiratory tract irritation, bronchitis, pneumonitis, pain and swelling of the eyes, lacrimation and photophobia (HSDB 1990). o-Chlorobenzealdehyde tested negative for mutagenic activity in yeast cell strains D7 and XV185-14C without metabolic activation (Nestmann and Less 1983).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

NIOSH has recommended an occupational exposure limit (REL) for nitriles of 8 mg/m^3 for a 10 hour workday (RTECS 1990). No regulated standards or criteria have been promulgated for o-chlorobenzealdehyde or malononitrile by EPA.

REFERENCES

CLAYTON AND CLAYTON eds. 1981-2. Patty's Industrial Hygiene and Toxicology. p. 4886.

HAZARDOUS SUBSTANCE DATA BANK (HSDB). 1990. National Library of Medicine, Toxicology Information Program, Bethesda, MD. January 1991.

KYRSIAK G. ET AL. 1976. Acute and subacute toxic action of malononitrile. Med. Pr. 27 (1):1-19.

NESTMANN E.R., E.G-H., LEE. 1983. Mutagenicity of constituents of pulp and paper mill effluent in growing cells of saccharomyces cervisiae. Mutat Res. 119:273.

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1990. National Institute for Occupational Safety and Health (NIOSH). U.S. Department of Health and Human Services.

RIETVELD E.C. ET AL. 1983. Mechanism of formation of mercapturic acids from aromatic aldehydes in vivo. Arch Toxicol 52(3):199.

WILLHITE, C.C., R.P. SMITH. 1981. The role of cyanide liberation in the acute toxicity of aliphatic nitriles. Toxicol Appl Pharmacol. 59(3):589-602.

CHLOROETHANE (ETHYL CHLORIDE)

Chloroethane is primarily absorbed through the lungs, although some dermal absorption may occur. Absorption and excretion of chloroethane occurs rapidly via the lungs; it is not metabolized to a significant degree (Clayton and Clayton 1981). Severe acute inhalation of chloroethane by humans produces minor neurological effects that are manifested as stupor and lack of coordination, and in some incidences as cardiac arrhythmia produced by the potentiation of adrenalin (Clayton and Clayton 1981). Acute inhalation of chloroethane by animals has produced histological or pathological changes in the liver, brain, and lungs (Troshina 1964). Chronic exposure of animals to chloroethane produced kidney damage and fatty changes in the liver, and at high levels has upset cardiac rhythm (EPA 1985). Studies assessing the mutagenicity and carcinogenicity of chloroethane are currently being conducted (EPA 1985). No health-based criteria have been established by EPA.

REFERENCES

CLAYTON, G. and CLAYTON, F., ed. 1981. Patty's Industrial Hygiene and Toxicology: Volume 2B-Toxicology. 3rd ed. John Wiley and Sons, New York, New York

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Chemical, Physical, and Biological Properties of Compounds Present at Hazardous Waste Sites. Final Report: Methyl Chloride. Prepared for EPA by Clement Associates, Inc., Arlington , Va. September 27

TROSHINA, M. 1964. Toksikol. Novykk. Prom. Khim. Veshchestv. 6: 45 (through Chem. Abstr.) (As cited in Clayton and Clayton 1981)

BIS (2-CHLOROETHYL) ETHER

Bis(2-Chloroethyl)ether (BCEE) is absorbed following oral, dermal and inhalation exposure (Symth & Carpenter 1948, Schrenk et al. 1933, Innes et al. 1969). Volunteers experienced eye and nasal irritation at vapor concentrations of 100 to 260 ppm; no noticeable irritation was observed at 35 ppm (Schrenk et al. 1933). Acute inhalation exposure of guinea pigs to 500 ppm BCEE resulted in eye, skin and respiratory irritation with lesser effects on liver, kidney and brian (Schrenk et al. 1933). Hepatomas were significantly increased in male mice administered 100 mg/kg BCEE by intubation for 14 days followed by 300 mg/kg in their diet for 80 weeks (Innes et al. 1969).

BCEE is classifeid in Group B2--Probable Human Carcinogen. EPA (1990) established an oral cancer potency factor of 1.1 $(mg/kg\text{-}day)^{-1}$ based on the chronic oral study by Innes et al. (1969) in which hepatomas were observed in male mice administered 100-300 mg/kg BCEE.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

INNES, J.R., ULLAND, B.M., VALERIO, M.G., PETRUCELLI, L., FISHBEIN, L., HART, E., PALLOTTA, A.J., BATES, R.R., FALK, H.L., GART, J.J., KLEIN, M., MITCHELL, I. and PETERS, J. 1969. Bioassay of pesticides and industrial chemicals for tumorigenicity in mice: a preliminary report. J. Nat. Cancer Inst. 42:1101-1114.

SCHRENK, H., PATTY, F.A., and YANT, W.P. 1933. Acute response of guinea pigs to vapors of osme new commercial organic compounds. Publ. Hlth. Rep. (Wash.) 48:1389-1397.

SYMTH, H.F. and CARPENTER, C.P. 1948. Further experience with the range finding test in the industrial toxicology laboratory. J. Industr. Hyg. Toxicol. 30:63-68.

CHLOROFORM

Chloroform, a trihalomethane, is rapidly absorbed through the respiratory and gastrointestinal tracts in humans and experimental animals; dermal absorption from contact of the skin with liquid chloroform can also occur (EPA 1985). In humans, acute exposures to chloroform may result in depression of the central nervous system, hepatic and renal damage, and death caused by ventricular fibrillation following an acute ingested dose of 10 ml (EPA 1984). Acute exposure to chloroform may also cause irritation to the skin, eyes, and gastrointestinal tract (EPA 1984, 1985). In experimental animals, chronic exposure may lead to fatty cyst formation in the liver (Heywood et al. 1979), renal and cardiac effects, and central nervous system depression (EPA 1985). Chloroform has been reported to induce renal epithelial tumors in rats (Jorgenson et al. 1985) and hepatocellular carcinomas in mice (NCI 1976). Suggestive evidence from human epidemiological studies indicates that long-term exposure to chloroform and other trihalomethanes in contaminated water supplies may be associated with an increased incidence of bladder tumors (EPA 1985). Chloroform is embryotoxic in pregnant rats and has retarded fetal development and increased the incidences of fetal resorption, absence of tail, imperforate anus, missing ribs and delayed ossification of sternebrae (Schwetz et al. 1974).

Chloroform has been classified by EPA (1990a) as a Group B2 Carcinogen (Probable Human Carcinogen). EPA (1990a) developed an oral cancer potency factor for chloroform of 6.1×10^{-3} $(mg/kg\text{-}day)^{-1}$ based on a study in which kidney tumors were observed in rats exposed to chloroform in drinking water (Jorgenson et al. 1985). An inhalation cancer potency factor of 8.1×10^{-2} $(mg/kg\text{-}day)^{-1}$ has been developed by EPA (1990a) based on an NCI (1976) bioassay in which liver tumors were observed in mice. EPA also derived an oral reference dose (RfD) of 0.01 mg/kg-day for both chronic (EPA 1990a) and subchronic (EPA 1990b) exposure to chloroform based on a chronic bioassay in dogs in which liver effects were observed at 12.9 mg/kg-day (Heywood et al. 1979); an uncertainty factor of 1,000 was used to derive the RfD for both exposure levels.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Chloroform. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-010

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Health Assessment Document for Chloroform. Environmental Criteria and Assessment Office, Research Triangle Park, North Carolina. September 1985. EPA 600/8-84-004F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

HEYWOOD, R., SORTWELL, R.J. NOEL, P.R.B., et al. 1979. Safety evaluation of toothpaste containing chloroform. III. Long-term study in beagle dogs. J. Environ. Pathol. Toxicol. 2:835-851

JORGENSON, T.A., MEIERHENRY, E.F., RUSHBROOK, C.J., et al. 1985. Carcinogenicity of chloroform in drinking water to male Osborne-Mendel rats and female B6C3F1 mice. Fund. Appl. Toxicol. 5:760-769

NATIONAL CANCER INSTITUTE (NCI). 1976. Carcinogenesis Bioassay of Chloroform. CAS No. 67-66-3. NCI Carcinogenesis Technical Report Series No. 0. Bethesda, M.D. DHEW (NIH) Publication No. 76

SCHWETZ, B.A., LEONG, B.K.J., GEHRING, P.J. 1974. Embryo- and fetotoxicity of inhaled chloroform in rats. Toxicol. Appl. Pharmacol. 28:442-451

p-Chloro-m-cresol is also known as 3-methyl-4-chlorophenol and 4-chloro-m-cresol. Very little toxicological information is available on this compound. 4-chloro-m-cresol is an allergen, antiseptic, and disinfectant and is more active in acidic than alkaline solutions (Merck Index 1983, Von Oettingen 1949, Sax and Lewis 1989). Studies indicate that this compound is moderately toxic when ingested (Sax and Lewis, 1989), and this is supported by the rat oral LD_{50} of 1,830 mg/kg (RTECS 1987). In humans, chlorocresol is an occasional skin contact sensitizer (Anderson and Hamann 1984). In addition, animal studies demonstrate that dermal exposure to p-chloro-m-cresol is not especially irritating to the skin at concentrations of 0.5 to 1.0% in alcohol (Von Oettingen, 1949). In male Wistar rats sacrificed 60 hours after a single oral or subcutaneous dose of 400 mg/kg, hepatocellular abnormalities were found including an increase in mitochondria, membrane-surrounded vacuoles, alterations in the rough endoplasmic reticulum and alterations in the intercellular space. Moreover, the bile canaliculi were reported to be dilated and the tight junctional complexes were present on the plasmalemma--away from their usual location (Robenek et al. 1980). Similar ultrastructural changes have also been reported in mouse hepatocytes (Meiss et al. 1981). No health-based criteria have been derived by EPA.

REFERENCES

ANDERSON, K.E., AND K. HAMANN. 1984. How sensitizing is chlorocresol? Allergy tests in guinea pigs versus the clinical experience. Contact Dermatitis 11:11-20.

MEISS, R., ET AL. 1981. New aspects of the origin of hepatocellular vacuoles. An ultrastructural study on rat and mice liver after different intoxications. Exp Pathol 19:239-246.

ROBENEK, H., ET AL. 1980. Alterations in the rat liver induced by p-chloro-m-cresol with emphasis on the intercellular junctions. A thin-section and freeze-fracture study. J Submicrosc. Cytol. 12:635-646.

SAX, N.I. AND LEWIS, R.J., 1989. Dangerous Properties of Industrial Materials, Seventh Edition, Volume II, VAn Nostrand Reinhold, New York, p. 791.

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1987. Vol. 3A. National Institute for Occupational Safety and Health (NIOSH). U.S. Department of Health and Human Services.

VON OETTINGEN, W.F., POWELL, C.C., SHARPLESS, N.E., ET AL., 1949. Relation Between the Toxic Action of Chlorinated Methanes and Their Chemical and Physiochemical Properties, Natl. Insts. Health Bull. No. 191, 1949. In: Gosselin, R.E., Smith, R.P., and Hodge, H.C., Clinical Toxicology of Commercial Products, fifth edition, Williams and Wilkins, Baltimore, MD, p. 193.

CHLOROMETHANE (METHYL CHLORIDE)

Chloromethane is primarily absorbed by inhalation, although some is absorbed through the skin (NIOSH 1977). The compound is widely distributed in the body and rapidly metabolized and excreted within 24 hours of exposure. Acute exposure to humans produced primarily central nervous system (CNS) effects including headache, drowsiness, giddiness, ataxia, convulsions, hepatic and renal effects, depression of bone marrow activity, coma, and respiratory failure (ACGIH 1986). Symptoms may develop a few hours after exposure to chloromethane. Chronic effects of chloromethane exposure include blurred vision, dizziness, weakness, gastrointestinal disturbances with prolonged vomiting, sleep disturbances, muscular incoordination, and tachycardia (Hansen et al. 1953). Chronic exposure in animals was reported to produce neuromuscular, liver, kidney, and testicular damage and death (Evtushenko 1966, Mitchell et al. 1979, Smith and von Oettingen 1947). Chloromethane has produced teratogenic effects in the form of heart defects in the offspring of exposed mice (CIIT 1981a). Chloromethane was found to be carcinogenic in male mice exposed by inhalation for 24 months, producing tumors of the kidney and liver (CIIT 1981b).

EPA (1990) has classified chloromethane in Group C (Possible Human Carcinogen) and has developed cancer potency factors for chloromethane based on kidney tumor data in male mice obtained from the CIIT (1981b) study. An inhalation cancer potency factor of 6.3×10^{-3} $(mg/kg\text{-}day)^{-1}$ was calculated. EPA (1990) also calculated an oral cancer potency factor of 1.3×10^{-2} $(mg/kg\text{-}day)^{-1}$ based on an oral extrapolation from data obtained in the CIIT (1981b) study.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. ACGIH, Inc., Cincinnati, Ohio. p. 380

CHEMICAL INDUSTRY INSTITUTE OF TOXICOLOGY (CIIT). 1981a. Final Report on Structural Teratogenicity Evaluations of Methyl Chloride in Rats and Mice after Inhalation. Prepared by Battelle Columbus Laboratories. April 30. (As cited in EPA 1985)

CHEMICAL INDUSTRY INSTITUTE OF TOXICOLOGY (CIIT). 1981b. Final Report on 24 Month Inhalation Study on Methyl Chloride. Prepared by Battelle Columbus Laboratories. December 31. (As cited in EPA 1985)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Chemical, Physical, and Biological Properties of Compounds Present at Hazardous Waste Sites. Final Report: Methyl Chloride. Prepared for EPA by Clement Associates, Inc., Arlington , Va. September 27

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

EVTUSHENKO, G. 1966. Gig. Tr. Prof. Zabol. 10:20 (As cited in Clayton and Clayton 1981)

HANSEN, H., WEAVER, K., and VENABLE, F. 1953. Arch. Ind. Hyg. Occup. Med. 8:328-334 (As cited in ACGIH 1986)

MITCHELL, R., PAVKOV, K., EVERETT, R., and HOLZWORTH, D. 1979. CIIT Docket No. 63059. Research Triangle Park, North Carolina (As cited in Clayton and Clayton 1981)

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1977. Occupational Diseases, A Guide to their Recognition. p. 208 DHEW (NIOSH) Pub. No. 77-181 (June 1977). (As cited in ACGIH 1986)

SMITH, W. and VON OETTINGEN, W. 1947. J. Ind. Hyg. Toxicol. 29:47 (As cited in Clayton and Clayton 1981)

CHLOROPICRIN

INTRODUCTION

Chloropicrin (PS), or trichloronitromethane, is a severe irritant of the eyes, skin, and respiratory tract that was used as a chemical agent during World War I (Hawley 1981, Stokinger 1982, NIOSH 1978). Chloropicrin has been used by industry in organic synthesis, and has been produced for use in fumigants, fungicides, insecticides, and rodenticides (Hawley 1981).

TOXICOKINETICS

It can be inferred from the results of toxicity studies that chloropicrin is absorbed following inhalation exposure.

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

In humans, chloropicrin causes lacrimation, vomiting, bronchitis, and pulmonary edema (Hawley 1981, Stokinger 1982, ACGIH 1986). Humans acutely exposed (3 to 30 seconds) to vapor concentrations of 0.3 to 0.37 ppm chloropicrin experienced painful irritation and tearing of the eyes (Flury and Zernik 1931, RCRA ND), while exposure to higher concentrations (15 ppm) for only a few seconds resulted in respiratory tract damage (Flury and Zernik 1931). A lethal exposure of chloropicrin for humans is 119 ppm for 30 minutes, with death usually resulting from pulmonary edema; particular injury occurs in the meduim and small bronchi (NIOSH 1978). In rodents, chloropicrin levels of 350 ppm in air are lethal in one minute, and levels of 110 ppm are lethal in twenty minutes (ACGIH 1986). The acute oral LD_{50} in rats is 250 mg/kg (RCRA ND, ACGIH 1986). The major pathological manifestations of chloropicrin exposure are congestion, hemmorrhage, edema, and infiltration of the lung tissue in early stages; chronic stages show marked necrosis of the kidney, liver and skeletal muscles (ACGIH 1986). Other symptoms reported include vertigo, fatigue, orthostatic hypotension, colic, diarrhea, and severe irritation of upper respiratory passages (ACGIH 1986). Chloropicrin has also been found to react with SH-groups in hemoglobin, interfering with oxygen transport (RCRA ND). Chronic oral gavage exposure of rats and mice to chloropicrin resulted in no significant increase of tumor incidence, although two carcinomas and one papiloma were found in the squamous epithelium of the forestomach of dosed mice (NCI 1978). These results are inconclusive due to the high mortality among exposed rats and to the lack of statistical significance in mice (NCI 1978).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

A time-weighted average Threshold-Limit-Value (TLV) of 0.1 ppm (0.7 mg/m^3) is recommended by ACGIH for repeated exposure to chloropicrin to protect against eye irritation and pulmonary effects (ACGIH 1986). No health-based criteria have been derived by EPA.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the threshold limit values and biological exposure indices. 5th ed. Cincinnati, OH, Pp. 134.

FLURY, F. AND ZERNIK, K. 1931. (As cited in ACGIH, 1986; and in Stokinger, 1982).

HAWLEY. 1981. (As cited in RCRA, ND).

ICF FEASIBILITY STUDY. 1987.

NATIONAL CANCER INSTITUTE (NCI). 1978. Bioassay of chloropicrin for possible carcinogenicity. DHEW Publication No. (NIH) 78-1315. NCI Carcinogenesis Tech. Rep. Ser. No. 65 NCI-CG-TR-65.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1978. Occupational health guideline for chloropicrin. U.S. Department of Health and Human Services.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1985. NIOSH pocket guide to chemical hazards. US Department of Health and Human Services (DHHS). DHHS Publication No. 85-114.

STOKINGER, H.E. 1982. Aliphatic nitro compounds, nitrates, nitrites. In Patty's Industrial Hygiene and Toxicology. Clayton, G.D. and Clayton, P.B., eds. 3rd ed. John Wiley and Sons, New York. Vol. 2C.

RCRA FACILITY ASSESSMENT, EDGEWOOD AREA, APG, MD.

beta-CHLOROVINYLARSONIC ACID

beta-Chlorovinylarsonic acid is an oxidation product of Lewisite oxide where arsenic is in the +5 oxidation state. It can be inferred from the available animal toxicity studies that beta-chlorovinylarsonic acid is absorbed following acute exposure. Human toxicity data on beta-chlorovinylarsonic acid is severely limited, however the lowest oral dose that caused death (LDLo) in rats has been determined to be 50 mg/kg (RTECS 1990). OSHA (1989) has reccommended an 8 hour Time-Weighted-Average (TWA) Permissible Exposure Limit (PEL) of 0.5 mg(As)/m^3 for occupational exposure to beta-chlorovinylarsonic acid. No health-based criteria have been derived by EPA.

REFERENCES

OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION (OSHA). 1989. The Federal Register. Vol. 54. Pp 2923.

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1987. National Institute for Occupational Safety and Health (NIOSH). U.S. Department of Health and Human Services.

CHROMIUM

Chromium exists in two states, as chromium (III) and as chromium (VI). Following oral exposure, absorption of chromium (III) is low while absorption of chromium (VI) is high (EPA 1987). Chromium is an essential micronutrient and is not toxic in trace quantities (EPA 1980). High levels of soluble chromium (VI) and chromium (III) can produce kidney and liver damage following acute oral exposure. CNS effects including hypoactivity have been reported in rats when exposed to acute and subchronic levels of chromium VI in drinking water (Diaz-Mayans et al. 1986). Chronic inhalation exposure may cause respiratory system damage (EPA 1984) . Further, epidemiological studies of worker populations have clearly established that inhaled chromium (VI) is a human carcinogen; the respiratory passages and the lungs are the target organs (Mancuso 1975, EPA 1984). Inhalation of chromium (III) or ingestion of chromium (VI) or (III) has not been associated with carcinogenicity in humans or experimental animals (EPA 1984). Certain chromium salts have been shown to be teratogenic and embryotoxic in mice and hamsters following intravenous or intraperitoneal injection (EPA 1984).

EPA has classified inhaled chromium (VI) in Group A--Probable Human Carcinogen by the inhalation route (EPA 1990a). Inhaled chromium (III) and ingested chromium (III) and (VI) have not been classified with respect to carcinogenicity (EPA 1990a). EPA (1990a) developed an inhalation slope factor of 41 $(mg/kg\text{-}day)^{-1}$ for chromium (VI) based on an increased incidence of lung cancer in workers exposed to chromium over a 6 year period, and followed for approximately 40 years (Mancuso 1975). EPA (1990a) derived a chronic oral reference dose (RfD) of 5.0×10^{-3} mg/kg-day for chromium (VI) based on a study by MacKenzie et al. (1958) in which no adverse effects were observed in rats exposed to 2.4 mg chromium (VI)/kg-day in drinking water for 1 year. A safety factor of 500 was used to derive the RfD. EPA (1990b) calculated a subchronic RfD for chromium VI of 2×10^{-2} mg/kg-day based on the same study, effect of concern and using a safety factor of 100. EPA (1990a) developed an oral RfD of 1 mg/kg-day for chromium (III) based on a study in which rats were exposed to chromic oxide baked in bread; no effects due to chromic oxide treatment were observed at any dose level (Ivankovic and Preussman 1975), however hepatotoxicity was the effect of concern. EPA (1990b) established a subchronic RfD of 10 mg/kg-day for chromium III based on the same study and endpoint. Safety factors of 1,000 and 100 were used to calculate the chronic and subchronic oral RfDs, respectively.

REFERENCES

DIAZ-MAYANS, J., LABORDA, R., and NUNEZ, A. 1986. Hexavalent chromium effects on motor activity and some metabolic aspects of Wistar albino rats. Comp Biochem Physiol 83(1):191-195.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Chromium. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-80-035

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Assessment Document for Chromium. Environmental Criteria and Assessment Office, Research Triangle Park, N.C. EPA 600/8-83-014F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Draft Health Advisory for Chromium. Office of Drinking Water, Washington, D.C. March 31, 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

IVANKOVIC, S., and PREUSSMAN, R. 1975. Absence of toxic and carcinogenic effects after administration of high doses of chromic oxide pigment in subacute and long-term feeding experiments in rats. Fd. Cosmet. Toxicol. 13:347-351

MANCUSO, T.F. 1975. International Conference on Heavy Metals in the Environment. Toronto, Canada

MACKENZIE, R.D., BYERRUM, R.V., DECKER, C.F., HOPPERT, C.A., AND LONGHAM, F.L. 1958. Chronic toxicity studies II. Hexavalent and trivalent chromium administered in drinking water to rats. Arch. Ind. Health 18:232-234

CNS

CNS is a mixture of 23% chloroacetophenone (CN), 38.4% chloropicrin (PS), and 38.4% chloroform (Army 1975). Therefore, in addition to the powerful lacrimating capability of CN, CNS also exhibits long-lasting irritation of the eyes and skin, coupled with vomiting, bronchitis, and pulmonary edema, characteristic of PS (Army 1975). Nausea, vomiting, colic, and diarrhea induced by CNS may persist for several weeks (Army 1975). The chloroform content of CNS allows it to be rapidly absorbed following dermal exposure, in addition to its potent absorption ability through the respiratory and gastrointestinal tracts of humans and animals (Army 1975). While CN has caused permanent corneal injury to the eyes of laboratory animals (NTP 1990), the effects of CNS on the eyes and skin are irritating but not toxic (Army 1975). In humans, acute exposure to the chloroform present in CNS may cause depression of the central nervous system, damage to the hepatic and renal systems, and death resulting from ventricular fibrillation (EPA 1984). Death can also result from pulmonary edema caused by an acute exposure to PS (119 ppm for 30 minutes) (NIOSH 1978). PS also interferes with oxygen transport by reacting with the SH-groups in hemoglobin (ICF Feasibility Study 1987). PS carcinogenicity studies involving rats and mice have been inconclusive, and the only evidence suggesting CN carcinogenicity is a marginal increase in fibroadenomas of the mammary gland of female rats during a 2-year inhalation study (NTP 1990). Chloroform, however, causes renal epithelial tumors in rats (Jorgenson et al. 1985) and hepatocellular carcinomas in mice (NCI 1976). Long-term exposure to chloroform in contaminated water supplies may be associated with an increased incidence of bladder tumors in humans (EPA 1985). In addition, chloroform is embryotoxic in pregnant rats causing retarded fetal development, increased incidence of fetal resorption, absence of tail, imperforate anus, missing ribs, and delayed ossification of sternebrae (Schwetz et al. 1974).

No health-based criteria for CNS have been derived by EPA. Chloroform, however, has been classified by EPA (1990a) as a Group B2 Carcinogen (Probable Human Carcinogen). EPA (1990a) has developed an oral cancer potency factor for chloroform of 6.1×10^{-3} $(mg/kg\text{-}day)^{-1}$ based on a study in which kidney tumors were observed in rats exposed to chloroform in drinking water (Jorgenson et al. 1985). An inhalation cancer potency factor of 8.1×10^{-2} $(mg/kg\text{-}day)^{-1}$ has been developed for chloroform by EPA (1990a) based on an NCI (1976) bioassay in which liver tumors were observed in mice. EPA also derived an oral reference dose (RfD) of 0.01 mg/kg-day for both chronic (EPA 1990a) and subchronic (EPA 1990b) exposure to chloroform based on a chronic bioassay in dogs in which liver effects were observed at 12.9 mg/kg-day (Heywood et al. 1979); an uncertainty factor of 1,000 was used to derive the RfD for both exposure levels. No health-based criteria for PS or CN have been derived by EPA, however, ACGIH (1986) has recommended time-weighted average Threshold-Limit-Values (TLVs) of 0.1 ppm (0.7 mg/m^3) and 0.05 ppm (0.3 mg/m^3), respectively, for repeated exposure to PS and CN (ACGIH 1986).

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the threshold limit values and biological exposure indices. 5th ed. Cincinnati, OH, Pp. 121.

ARMY. 1975. Military Chemistry and Chemical Compounds Field Manual. Department of the Army and Air Force, Washington, D.C. October 1975. FM 3-9, AFR 355-7.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Chloroform. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-010.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Health Assessment Document for Chloroform. Environmental Criteria and Assessment Office, Research Triangle Park, North Carolina. September 1985. EPA 600/8-84-004F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of /solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990.

HEYWOOD, R., SORTWELL, R.J., NOEL P.R.B., et al. 1979. Safety evaluation of toothpaste containing chloroform. III. Long-term study in beagle dogs. J. Environ. Pathol. Toxicol. 2:835-851

ICF FEASIBILITY STUDY. 1987. Toxicity of contaminants suspected to be present at old "O" field. December 1987.

JORGENSON, T.A., MEIERHENRY, E.F., RUSHBROOK, C.J., et al. 1985. Carcinogenicity of chloroform in drinking water to male Osborne-Mendel rats and female B6C3F1 mice. Fund. Appl. Toxicol. 5:760-769.

NATIONAL CANCER INSTITUTE (NCI). 1976. Carcinogenesis Bioassay of Chloroform. CAS No. 67-66-3. NCI Carcinogenesis Technical Report Series No. O. Bethesda, MD. DHEW (NIH) Publication No. 76.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1978. Occupational health guideline for chloropicrin. U.S. Department of Health and Human Services.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1990. NTP Technical report on the toxicology and carcinogenesis studies of 2-Chloroacetophenone in F334/N rats and B6C3F1 mice (inhalation studies). NTP TR 379.

SCHWETZ, B.A., LEONG, B.K.J., GEHRING, P.J. 1974. Embryo- and fetotoxicity of inhaled chloroform in rats. Toxicol. Appl. Pharmacol. 28:442-451.

COPPER

Copper is an essential element. A daily copper intake of 2 mg is considered to be adequate for normal health and nutrition; the minimum daily requirement is 10 µg/kg (EPA 1985). In humans, absorption of copper following oral exposure is approximately 60% and is influenced by competition with other metals and the level of dietary protein and ascorbic acid in both humans and animals (EPA 1984). Copper is absorbed following inhalation exposures, although quantitative data on the extent of absorption are unavailable (EPA 1984). Adverse effects in humans resulting from acute exposure to copper at concentrations that exceed these recommended levels by ingestion include salivation, gastrointestinal irritation, nausea, vomiting, hemorrhagic gastritis, and diarrhea (ACGIH 1986). Dermal or ocular exposure of humans to copper salts can produce irritation (ACGIH 1986). Acute inhalation of dusts or mists of copper salts by humans may produce irritation of the mucous membranes and pharynx, ulceration of the nasal septum, and metal fume fever. The latter condition is characterized by chills, fever, headache, and muscle pain. Limited data are available on the chronic toxicity of copper; however, chronic over-exposure to copper by humans has been associated with anemia (ACGIH 1986) and local gastrointestinal irritation (EPA 1987). Results of several animal bioassays suggest that copper compounds are not carcinogenic by oral administration; however, some copper compounds can induce injection-site tumors in mice (EPA 1985).

EPA (1990) has reported the drinking water standard of 1.3 mg/liter as an oral reference dose (RfD) for both chronic and subchronic exposures based on local gastrointestinal irritation (EPA 1987). Assuming a 70-kg adult ingests 2 liters of water per day, this concentration is equivalent to a dose of 3.7×10^{-2} mg/kg-day. However, EPA (1987) concluded toxicity data were inadequate for the calculation of a reference dose (RfD) for copper. An uncertainty factor of 1 was used to derive the oral RfD.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Copper. Environmental Criteria and Assessment Office, Cincinnati, Ohio. EPA/540-1-86-025

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. National primary drinking water regulations; synthetic organic chemicals, inorganic chemicals and microorganisms. Fed. Reg. 50:46937-47025 (November 13, 1985)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Drinking Water Criteria Document for Copper. Prepared by the Office of Health and Environmental Assessment. Environmental Criteria and Assessment Office, Cincinnati, Ohio, for the Office of Drinking Water, Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment. Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

CYANIDES

The toxicity of cyanides is strongly dependent on their chemical speciation. Free cyanides are readily absorbed from the gastrointestinal tract, lungs, and skin and, once absorbed, are rapidly distributed throughout the body (EPA 1985). The toxic effects in humans following acute oral exposure to free cyanides include hyperventilation, vomiting, unconsciousness, convulsions, vascular collapse and cyanosis, and death (EPA 1985). Inhalation of high concentrations of hydrogen cyanide (HCN) gas can result in almost immediate collapse, respiratory arrest, and death within minutes (DiPalma 1971). The hydrogen cyanide LC_{lo} for humans is 200 ppm/5 minutes, or 132 ppm/hr (120 mg/m^3/hour) (RTECS 1988). The rat LC_{50} is 484 ppm/5 minutes for hydrogen cyanide (RTECS 1988). Airborne hydrogen cyanide concentrations between 109–582 ppm (99 and 528 mg/m^3) are fatal within 30-60 minutes (NIOSH 1976). For a summary of man's physiological response to various concentrations of hydrogen cyanide, see Table 1. There is limited data on chronic exposures of cyanide in humans, although the following effects have been identified in chronic occupationally exposed workers in some epidemiologic studies: neurological dysfunction, lacrimation, abdominal pain, muscular weakness, and shortness of breath (NIOSH 1976). Cyanide appears to be less toxic to animals following chronic exposures than following acute exposures. In animals, chronic oral exposure has produced weight loss, thyroid effects and myelin degeneration (Howard and Hanzal 1955). Cyanide can cause teratogenic effects when subcutaneously administered to hamsters; this teratogenic effect has not been observed in other species although some reproductive toxicity has been noted (EPA 1985).

EPA calculated an oral reference dose (RfD) for cyanide of $2x10^{-2}$ mg/kg-day for both chronic (EPA 1990a) and subchronic (EPA 1990b) exposures based on a chronic study by Howard and Hanzal (1955) in which rats were maintained on a diet fumigated with hydrogen cyanide and exhibited weight loss, thyroid effects and myelin degeneration. No observed adverse effects (NOAEL) were noted at the highest dose administered (10.8 mg/kg-day). A NOAEL of 10.8 mg/kg-day and a safety factor of 500 were used to derive the RfD (EPA 1990a). Hydrogen cyanide has a ceiling limit of 10 ppm (11 mg/m^3) which should not be exceeded during any time of the workday. This value contains a two-fold margin of safety against mild symptoms of HCN toxicity, and a seven or eight-fold margin against lethal effects (ACGIH 1986). OSHA has established a short term exposure limit (STEL) of 4.7 ppm (5 mg/m^3), which is the employee's 15-minute time weighted average exposure that shall not be exceeded at any time during a work day (OSHA 1989).

TABLE 1

Physiological Response to Various Concentrations of Hydrogen Cyanide in Air - Man

Response	Concentration mg/L	ppm
Immediately fatal	0.3	270
Fatal after 10 minutes	0.2	181
Fatal after 30 minutes	0.15	135
Fatal after 0.5 to 1 hour without immediate or late effects	0.12-0.15	110-135
Tolerated for 0.5 to 1 hour without immediate or late effects	0.05-0.06	45-54
Slight symptoms after several hours	0.02-0.04	18-36

Source: ACGIH 1986

REFERENCES

ACGIH. 1986. American Conference of Governmental Industrial Hygienists Inc. Documentation of the Threshold Limit Values and Biological Exposure Indices. Fifth Edition. Cincinnati, Ohio.

DIPALMA, J.R., ed. 1971. Noxious gases and vapors: I. Carbon monoxide, cyanides, methemoglobin, and sulfhemoglobin. In Drill's Pharmacology in Medicine. McGraw-Hill Book Co., New York. Pp. 1189-1205

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Drinking Water Criteria Document for Cyanide (Final Draft). Environmental Criteria and Assessment Office, Cincinnati, Ohio. EPA 600/X-84-192-1. PB86-117793

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D,C. July 1990

HOWARD, J.W. and HANZAL, R.F. 1955. Chronic toxicity for rats of food treated with hydrogen cyanide. J. Agric. Food Chem. 3:325-329

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1976. Criteria for a Recommended Standard—Occupational Exposure to Hydrogen Cyanide and Cyanide Salts (NsCN, KCN, and $Ca(CN)_2$). Washington, D.C. DHEW (NIOSH) Publication No. 77-108

OSHA. 1989. Occupational Safety and Health Administration. 29 CFR 1910.1000.

RTECS. 1988. Registry of the Toxic Effects of Chemicals Database.

DDT is absorbed by humans and experimental animals from the gastrointestinal tract (EPA 1984, 1980). Jenson et al. (1957) reported that 95% of ingested DDT in rats is absorbed from the gastrointestinal tract. Absorption of DDT through the skin is minimal (EPA 1980). In humans, DDT and its metabolites, DDD and DDE are stored primarily in adipose tissue; storage of DDT in human tissues can last up to 20 years (NIOSH 1978). Acute oral exposure to DDT in humans and animals may cause dizziness, confusion, tremors, convulsions, and parasthesia of the extremities. Allergic reactions in humans following dermal exposure to DDT have also been reported (EPA 1980). Long-term occupational exposure to DDT results in increased activity in hepatic microsomal enzymes, increased serum concentrations of LDH, SGOT and cholesterol, decreased serum concentrations of creatinine phosphokinase, increased blood pressure, and increased frequency of miscarriages (NIOSH 1978). Liver effects, neurological effects, immunosuppression, reduced fertility, embryotoxicity, and fetotoxicity have also been reported in animals following subchronic and chronic exposure to DDT (Laug et al. 1950, NIOSH 1978, McLachlan and Dixon 1972, Schmidt 1973). DDT has been shown to be carcinogenic in mice and rats at several dose levels or dosage regimens. The principal site of action is the liver, but an increased incidence of tumors of the lung and lymphatic system have also been reported in several investigations (NIOSH 1978, Tomatis et al. 1974, NCI 1978). In animals, DDD and DDE are typically less toxic and than DDT. The oral rat LD_{50}'s are 113 and 1000 mg/kg for DDD and DDE, respectively (RTECS 1987). Exposure to DDD can produce lethargy and convulsions, although studies reveal that this occurs less frequently than DDT exposure (Gosselin et al. 1984). Exposure to DDD dust is irritating to the eyes, nose and throat; ingestion causes vomiting and delayed symptoms similar to those caused following ingestion of DDT (Weiss 1986). Symptoms of dermal exposure to DDD include convulsions, excitement and reduced seizure threshold (RTECS 1987). There is evidence to suggest that DDD is mutagenic and tumorigenic inducing thorax, respiratory, liver and thyroid tumors in rodents (RTECS 1987). DDE has been shown to cause DNA inhibition (RTECS 1987).

DDD, DDE, and DDT are classified by EPA's Carcinogen Assessment Group in Group B2 (Probable Human Carcinogen) based on inadequate evidence of carcinogenicity from human studies and sufficient evidence of carcinogenicity from animal studies (EPA 1990). EPA (1990) developed an inhalation and oral cancer potency factor for DDT of 0.34 $(mg/kg\text{-}day)^{-1}$ based on the geometric mean of a number of carcinogenicity studies. EPA (1990) also developed an oral RfD for DDT of $5x10^{-4}$ mg/kg-day based on a study in which liver lesions were observed in rats fed 5 ppm but not in those fed 1 ppm (0.05 mg/kg-day) DDT for 27 weeks (Laug et al. 1950); an uncertainty factor of 100 was used to derive the RfD. EPA (1990) has reported an oral cancer potency factor of 0.24 $(mg/kg\text{-}day)^{-1}$ for DDD based on a study in which an increased incidence of lung tumors in males and lung and liver tumors in females was observed in mice fed 250 ppm (TWA) DDD for 13 weeks (Tomatis et al. 1974). EPA (1990) has reported an oral cancer potency factor of 0.34 $(mg/kg\text{-}day)^{-1}$ for DDE based on feeding studies that reported increased incidences of hepatomas in hamsters (Rossi et al. 1983) and in CF-1 mice (Tomatis et al. 1974), and an increased incidence of hepatocellular carcinomas in $B6C3F_1$ mice (NCI 1978).

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria Document for DDT. Office of Water Regulations and Standards, Washington, D.C. EPA 440/5-80-038

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment Document for DDT. Office of Emergency and Remedial Response, Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

JENSON, J.A., et al. 1957. DDT metabolites in feces and bile of rats. Agric. Food Chem. 5:919

LAUG, E.P., NELSON, A.A., FITZHUGH, O.G., and KUNZE, F.M. 1950. Liver cell alteration and DDT storage in the rat of the rat induced by dietary levels of 1-50 ppm DDT. J. Pharmacol. Exp. Ther. 98:268-273

MCLACHLAN, J.A., and DIXON, R.L. 1972. Gonadal function in mice exposed prenatally to p,p'-DDT. Toxicol. Appl. Pharmacol. 22:327

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1978. Special Occupational Hazard Review: DDT. DHEW Publication No. 78-200

NATIONAL CANCER INSTITUTE (NCI). 1978. Bioasssays of DDT, TDE, and p,p'-DDE for Possible Carcinogenicity. NCI-CG-TR-1321

ROSSI, L., BARBIERI, O., SANGUINETI, M., CABRAL, J., BRUZZI, P., and SANTI, L. 1983. Carcinogenicity study with technical-grade DDT and DDE in hamsters. Cancer Res 43:776-781

GOSSELIN R.E., SMITH R.P, AND HODGE H.C. 1984. Clinical Toxicology of Commercial Products. 5th ed. Williams and Wilkins. Baltimore.

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1987. Volume 5. U.S. Department of Health and Human Services.

SCHMIDT, R. 1973. [Effects of 1,1,1-trichloro-2,2-bis(p-chlorophenol)-ethane (DDT) on the prenatal development of the mouse.] Biol. Rundsch. 11:316-317 (German)

TOMATIS, L., TURUSOV, V., CHARLES, R.T., DiOCCHI, M., and GATI, E. 1974. Liver tumors in CF-1 mice exposed for limited periods to technical DDT. Z. Krebsforsch. 82:25-35

WEISS G. 1986. Hazardous Chemicals Data Book. Noyes Data Corporation. Park Ridge, New Jersey. 2nd ed.

1,2-DICHLOROBENZENE

1,2-Dichlorobenzene is readily absorbed through the lungs, skin, and gastrointestinal tract (EPA 1987). The principal toxic effects of this compound in humans and experimental animals from acute and longer-term exposure include central nervous system depression, blood dyscrasias, and lung, kidney, and liver damage (EPA 1985, NTP 1985). Subchronic gavage administration to mice and rats has resulted in liver pathology, lymphocyte depletion in the spleen and thymus, renal tubular degeneration (male rats only) and slight decreases in hemoglobin, hematocrit and red blood cell counts (rats only) (Hollingsworth et al. 1958, NTP 1985). Guinea pigs exposed to 1,2-dichlorobenzene via inhalation for 6 to 7 months developed reduced spleen weight (Hollingsworth et al. 1958). 1,2-Dichlorobenzene does not appear to be teratogenic in rabbits or rats (Hayes et al. 1985). Chromosome breaks also have been observed in exposed humans (EPA 1987).

EPA (1990b) derived chronic and subchronic inhalation RfDs of 0.04 mg/kg-day and 0.4 mg/kg-day, respectively, for 1,2-dichlorobenzene based on a study in which decreased body weight gain was observed in rats administered 1,2-dichlorobenzene, 7 hours/day, 5 days/week for up to 7 months (Hollingsworth et al. 1958); uncertainty factors of 1,000 and 100 were used to derive the chronic and subchronic RfDs, respectively. EPA (1990a) also reported an oral reference dose for 1,2-dichlorobenzene of $9x10^{-2}$ mg/kg-day based on an NTP (1985) study in which no adverse effects were observed in rats exposed to 1,2-dichlorobenzene by gavage 5 days/week for 103 weeks; an uncertainty factor of 1,000 was used to develop the RfD. EPA (1990b) derived a subchronic oral RfD of $9x10^{-1}$ mg/kg-day with an uncertainty factor of 100 based on the same study and effect of concern.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Drinking Water Criteria Document for *ortho*-Dichlorobenzene, *meta*-Dichlorobenzene, and *para*-Dichlorobenzene (Draft). Office of Drinking Water, Washington, D.C. June 1985. PB86-117918

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Health Advisory for *ortho-*, *meta-*, and *para*-Dichlorobenzene. Office of Drinking Water, Washington, D.C. March 31, 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

HAYES, W.C., T.R. HANLEY, JR., T.S. GUSHOW, K.A. JOHNSON, AND J.A. JOHN. 1985. Teratogenic potential of inhaled dichlorobenzene in rats and rabbits. Fund. Appl. Toxicol. 5:190-202.

HOLLINGSWORTH, R.L., ROWE, V.K., OYEN, F., TORKELSON, and ADAMS, E.M. 1958. Toxicity of *o*-dichlorobenzene. Am. Med. Assoc. Arch. Ind. Health 17:180-187 (As cited in EPA 1985)

NATIONAL TOXICOLOGY PROGRAM (NTP). 1985. Toxicology and Carcinogenesis Studies of 1,2-Dichlorobenzene in F344/N Rats and B6C3F1 Mice. U.S. DHHS NIH Technical Report Series No. 255 No. 86-2511 and PB86-144888

1,3-DICHLOROBENZENE

1,3-Dichlorobenzene (1,3-DCB or *meta*-dichlorobenzene) is absorbed following oral and inhalation exposure (EPA 1985a). Results of pharmacokinetic studies indicate that dichlorobenzenes are readily distributed after absorption regardless of exposure route and that some tissues preferentially accumulate these compounds, particularly the kidney, liver, lung, and adipose tissue (EPA 1985b). 1,3-DCB is metabolized to arene oxide intermediates via action by epoxidase enzymes. Although no studies were available on the acute or chronic effects of 1,3-DCB exposure, these effects are expected to be similar to those associated with 1,2-DCB or 1,4-DCB. Acute exposure to high doses of 1,2-DCB or 1,4-DCB primarily affects the respiratory tract, central nervous system, and hematologic systems. Subchronic and chronic exposure to 1,2-DCB or 1,4-DCB has been associated with damage to the reticuloendothelial and hematopoietic systems, as well as the central nervous system, liver, and kidneys (NJDWQI 1987, Battelle-Columbus 1978a,b). No data are available on either the carcinogenic or reproductive/teratogenic potential of 1,3-DCB.

EPA (1987) calculated an oral RfD for 1,3-DCB based on subchronic data for 1,2-dichlorobenzene. In two separate unpublished studies (Battelle-Columbus 1978a,b), rats and mice were administered 1,2-dichlorobenzene in corn oil by gavage at doses of 30, 60, 125, 250, or 500 mg/kg-day 5 days/week for 13 weeks. A NOAEL of 125 mg/kg-day was identified from these studies; at higher doses (188 mg/kg-day, 5 days/week) (Hollingsworth et al. 1958) kidney and liver weights increased in rats. Applying a safety factor of 1,000 to the NOAEL and adjusting for exposure for 5 days a week, EPA (1987) derived an oral RfD for 1,3-DCB of 8.9×10^{-2} mg/kg-day.

REFERENCES

BATTELLE'S COLUMBUS LABORATORIES. 1978a. Subchronic Toxicity Study: *ortho*-Dichlorobenzene (C54944), B6C3F1 mice. Subcontract No. 76-34-106002. December 21, 1978 (Unpublished report) (As cited in EPA 1987)

BATTELLE'S COLUMBUS LABORATORIES. 1978b. Subchronic Toxicity Study: *ortho*-Dichlorobenzene (C54944), Fischer 344 Rats. Subcontract No. 76-34-106002. (Unpublished report) (As cited in EPA 1987)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Drinking Water Criteria Document for *ortho*-Dichlorobenzene, *meta*-Dichlorobenzene, and *para*-Dichlorobenzene (Draft). Office of Drinking Water, Washington, D.C. PB86-117918

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. Health Assessment Document for Chlorinated Benzenes. Final Report. Office of Health and Environmental Assessment. Washington, D.C. EPA/600/8-84/015F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Health Advisory for *ortho*-, *meta*-, and *para*-Dichlorobenzenes. Office of Drinking Water, Washington, D.C. March 31, 1987

HOLLINGSWORTH, R.L., ROWE, V.K., OYEN, F., TORKELSON, T.R., and ADAMS, E.M. 1958. Toxicity of 0-dichlorobenzene: Studies on animals and industrial experience. AMA Arch. Ind. Health 17(1):180-187

NEW JERSEY DRINKING WATER QUALITY INSTITUTE (NJDWQI). 1987. Maximum Contaminant Level Recommendations for Hazardous Contaminants in Drinking Water. Appendix B: Health-Based Maximum Contaminant Level Support Documents. Submitted to New Jersey Department of Environmental Protection, Trenton, New Jersey. March 26, 1987

1,1-DICHLOROETHANE

1,1-DCA is probably less toxic than the 1,2-isomer (EPA 1980). At one time, the compound was used as an anesthetic, but it induced cardiac arrhythmias and its use was discontinued. It is probable that human exposure to sufficiently high levels of 1,1-DCA would cause central nervous system depression and respiratory tract and skin irritation, since many of the chlorinated aliphatics cause these effects (Parker at al. 1979). However, no dose-response data concerning these effects are available. Renal damage was observed in cats exposed by inhalation in a subchronic study (Hofmann et al. 1971). Inhalation exposure of pregnant rats to high doses of 1,1-DCA (6,000 ppm) retarded fetal development (Schwetz et al. 1974). A carcinogenicity bioassay of 1,1-DCA was limited by poor survival of both treatment and control groups, and the physical conditions of the treated animals were markedly stressed. Dose-related marginal increases in mammary gland adenocarcinomas and in hemangiosarcomas were seen in female rats, and a statistically significant increase in endometrial stromal polyps was seen in female mice; however, these data were not interpreted as providing conclusive evidence for the carcinogenicity of 1,1-DCA because of the previously mentioned limitations of the bioassay (NCI 1978).

EPA (1990) has classified 1,1-DCA as a Group C agent (Possible Human Carcinogen) based on the occurrence of hemangiosarcomas in rats administered 1,1-DCA via gavage (NCI 1978). EPA (1990) developed an oral and inhalation reference dose of 0.1 mg/kg-day based on adverse renal effects seen in cats following subchronic inhalation exposure (Hofmann et al. 1971). A safety factor of 1000 was used to develop the RfD. Subchronic oral and inhalation RfDs are both 1 mg/kg-day and were calculated using a safety factor of 100, based on the same study and same effects of concern (EPA 1990).

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for 1,1-Dichloroethane. Office of Water Regulations and Standards, Washington, D.C. EPA 440/5-80-029

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables Prepared by Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati Ohio for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

HOFMANN, H.T., BIRNSTIEL, H., and JOBST, P. 1971. The inhalation toxicity of 1,1- and 1,2-dichloroethane. Arch. Toxikol. 27:248-265

NATIONAL CANCER INSTITUTE (NCI). 1978. Bioassay of 1,1-Dichloroethane for Possible Carcinogenicity. CAS No. 75-34-3. NCI Carcinogenesis Technical Report Series No. 66, Washington, D.C. DHEW Publication No. (NIH) 78-1316

PARKER, J.C., CASEY, G.E., and BAHLNON, L.J. 1979. NIOSH current intelligence bulletin No. 27. Chloroethanes: Review of toxicity. Am. Ind. Hyg. Assoc. J. 40:A46-A60

SCHWETZ, B.A., LEONG, B.K.J., and GEHRING, P.J. 1974. Embryo and fetotoxicity of inhaled carbon tetrachloride, 1,1-dichloroethane, and methyl ethyl ketone. Toxicol. Appl. Pharmacol. 28:452-464

1,2-DICHLOROETHANE

Data on the toxicokinetic properties of 1,2-dichloroethane (1,2-DCA) in humans are limited, but data from animal studies suggest that the chemical is rapidly absorbed following oral and inhalation exposure and after dermal contact with the liquid form of the compound (EPA 1985). Effects of acute inhalation exposure in humans include irritation of mucous membranes in the respiratory tract and central nervous system depression (EPA 1985). Death may occur as a result of respiratory and circulatory failure. Pathological examinations typically show congestion, degeneration, necrosis, and hemorrhagic lesions of the respiratory and gastrointestinal tracts, liver, kidney, spleen, and lungs (EPA 1985). Adverse effects caused by less extreme exposures are generally associated with the gastrointestinal and nervous systems. Occupational exposures to 1,2-DCA vapors result in anorexia, nausea, vomiting, fatigue, nervousness, epigastric pain, irritation of the eyes and respiratory tract, and gastrointestinal, liver, and gallbladder disease (EPA 1984, 1985). Chronic inhalation studies in animals also have revealed toxic effects including degeneration of the liver (EPA 1985). Available data suggest that 1,2-DCA does not adversely affect reproductive or developmental processes in experimental animals except at maternally toxic levels (EPA 1985). In long-term oral bioassays sponsored by the National Cancer Institute (NCI 1978), increased incidences of squamous-cell carcinomas of the forestomach, mammary gland adenocarcinomas, and hemangiosarcomas have been observed in rats exposed to 1,2-DCA; pulmonary adenomas, mammary adenocarcinomas, and uterine endometrial tumors have been observed in mice exposed to this chemical.

EPA (1990) has classified 1,2-DCA in Group B2 (Probable Human Carcinogen) based on inadequate evidence of carcinogenicity from human studies and sufficient evidence of carcinogenicity from animal studies. EPA (1990) derived an oral and an inhalation cancer potency factor (q_1^*) of 9.1×10^{-2} $(mg/kg\text{-}day)^{-1}$ for 1,2-DCA based on the incidences of hemangiosarcomas in Osborne-Mendel male rats observed in the NCI (1978) gavage study.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for 1,2-Dichloroethane. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-002

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Health Assessment Document for 1,2-Dichloroethane. Office of Health and Environmental Assessment, Washington, D.C. September 1985. EPA 600/8-84-006F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

NATIONAL CANCER INSTITUTE (NCI). 1978. Bioassay of 1,2-Dichloroethane for Possible Carcinogenicity. NCI Carcinogenesis Technical Report Series No. 55. Washington, D.C. DHEW (NIH) Publication No. 78-1361

1,1-DICHLOROETHENE

1,1-Dichloroethene (1,1-DCE) is rapidly absorbed after oral and inhalation exposure (EPA 1984, 1987). Humans acutely exposed to 1,1-DCE vapors exhibit central nervous system depression. In animals, the liver is the principal target organ of 1,1-DCE toxicity. Acute exposures may result in liver damage which ranges from fatty infiltration to necrosis (EPA 1987). Workers chronically exposed to 1,1-DCE in combination with other vinyl compounds exhibit liver dysfunction, headaches, vision problems, weakness, fatigue and neurological sensory disturbances (EPA 1987). Chronic oral administration of 1,1-DCE to experimental animals results in both hepatic and renal toxicity (EPA 1984, Quast et al. 1983). Inhalation or oral exposure of rats and rabbits has produced fetotoxicity and minor skeletal abnormalities, but only at maternally toxic doses. 1,1-DCE vapors produced kidney tumors and leukemia in a single study of mice exposed by inhalation, but the results of other studies were equivocal or negative (EPA 1987, Maltoni et al. 1985).

EPA has classified 1,1-DCE as a Group C agent (Possible Human Carcinogen) and has developed inhalation and oral cancer potency factors of 1.2 $(mg/kg\text{-}day)^{-1}$ and 0.6 $(mg/kg\text{-}day)^{-1}$, respectively (EPA 1985, 1990a). The inhalation potency factor was based on the increased incidence of renal adenocarcinomas in male mice exposed to 1,1-DCE via inhalation for 52 weeks and observed for a total of 121 weeks (Maltoni et al. 1985). The oral potency factor was derived by estimating an upper-limit value from negative bioassay data and assuming that a carcinogenic response occurs via ingestion, although there is no direct evidence that this is true. EPA developed an oral reference dose (RfD) of $9x10^{-3}$ mg/kg-day for both chronic (EPA 1990a) and subchronic (EPA 1990b) exposure based on the occurrence of hepatic lesions in rats chronically exposed to 1,1-DCE in drinking water (Quast et al. 1983). A safety factor of 1000 was applied to the lowest-observed-adverse-effect level (LOAEL) of 9 mg/kg-day to derive the chronic and subchronic oral RfDs.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Health Assessment Document for Vinylidene Chloride. Final Report. Environmental Criteria and Assessment Office, Research Triangle Park, North Carolina. August 1985. EPA 600/8-83/031F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Health Advisory for 1,1-Dichloroethene. Office of Drinking Water, Washington, D.C. March 31, 1987.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office. Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for 1,1-Dichloroethene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-051

MALTONI, C., LEFEMINE, G., COTTI, G., CHIECO, P., and PATELLA, V. 1985. Experimental Research on Vinylidine Chloride Carcinogenesis. In Archives of Research on Industrial Carcinogenesis. Princeton Scientific Publishers, Princeton, New Jersey. 3 vols.

QUAST, J.F., HUMISTON, C.G., WADE, C.E., BALLARD, J., BEYER, J.E., SCHWETZ, R.W., and NORRIS, J.M. 1983. A chronic toxicity and oncogenicity study in rats and subchronic toxicity study in dogs on ingested vinylidene chloride. Fund. Appl. Toxicol. 3:55-62

trans- and *cis*-1,2-DICHLOROETHENE

Both *trans*-1,2-Dichloroethene (1,2-DCE) and *cis*-1,2-DCE are expected to be absorbed by any route of exposure (EPA 1987). Information on the health effects of both *trans*-1,2-DCE and *cis*-1,2-DCE is limited. In humans, *trans*-1,2-DCE and *cis*-1,2-DCE are central nervous system depressants, and exposure to high concentrations can result in anesthetic effects (Irish 1963). Acute exposure to higher dose levels of the trans-isomer can cause narcosis and death in rats (Torkelson and Rowe 1981). In animals, *cis*-1,2-DCE also has narcotic effects at high doses. Inhalation exposure of rats to 200 ppm *trans*-1,2-DCE has been associated with pneumonic infiltration of the lungs and progressive fatty degeneration of the liver (Freundt et al. 1977). Exposure of rats to 200 ppm *trans*- or *cis*-1,2-DCE by inhalation results in inhibition of the mixed function oxidase enzyme system; the cis-isomer was more potent (Freundt and Macholz 1978). Subchronic gavage exposure to *cis*-1,2-DCE resulted in decreased hematocrit and hemoglobin in rats (McCauley et al. n.d.). Rats chronically administered *cis*-1,2-DCE in their diet have exhibited hepatocellular swelling and fatty changes (Quast et al. 1983). Chronic oral exposure of rats to *trans*-1,2-DCE has resulted in increased serum alkaline phosphatase (Barnes et al. 1985). *cis*-1,2-DCE was reported to induce mutations using a host-mediated assay and chromosomal aberrations in mouse bone marrow cells (Cerna and Kypenova 1977).

EPA (1990a) has derived an oral reference dose (RfD) of $2x10^{-2}$ mg/kg-day for *trans*-1,2-DCE based on a 90-day drinking water study conducted in mice (Barnes et al. 1985). A no-observed-adverse-effect level (NOAEL) of 17 mg/kg-day for increased serum alkaline phosphatase and an uncertainty factor of 1,000 were used to derive the RfD. EPA (1990b) derived a subchronic oral RfD for trans-1,2-DCE of $2x10^{-1}$ mg/kg-day, using a safety factor of 100, based on the above study, and the same effect of concern. EPA (1990b) has derived an oral RfD of $1x10^{-2}$ mg/kg-day for cis-1,2-DCE based on results of a 90-day gavage study in which rats exhibited decreased hamatological and hemoglobin levels (McCauley et al. n.d.). The RfD was calculated using a lowest-observed-effect level (LOAEL) of 32 mg/kg-day and an uncertainty factor of 3,000.

REFERENCES

BARNES, D.W., SANDERS, V.M., WHITE, K.L., Jr., et al. 1985. Toxicology of *trans*-1,2-dichloroethene in the mouse. Drug Chem. Toxicol. 8:373-392

CERNA, M., KYPENOVA, H. 1977. Mutagenic activity of chloroethenes analyzed by screening system tests. Mutat. Res. 46:214-215

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Health Advisory for *cis*-1,2-Dichloroethene. Office of Drinking Water. Washington, D.C. March 31, 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

FREUNDT, K.J., LIEBALDT, G.P., and LIEBERWIRTH, E. 1977. Toxicity studies on *trans*-1,2-dichloroethene. Toxicology 7:141-153

FREUNDT, K.J., MACHOLZ, J. 1978. Inhibition of mixed function oxidases in rat liver by *trans*- and *cis*-1,2-Dichloroethene. Toxicology 10:131-139. (As cited in EPA 1987)

IRISH, D.D. 1963. Vinylidene chloride. In Patty, F.A., ed. Industrial Hygiene and Toxicology. 2nd ed. John Wiley and Sons, New York. Vol. II, pp. 1305-1309

MCCAULEY, P.T., M. ROBINSON, L.W. CONDIE, M. PARVELL. n.d. The effects of subacute and subchronic oral exposure to cis-1,2-dichloroethene in rats. Health Effects Research Laboratory, U.S. EPA, Cincinnati, OH.

QUAST, J.F., HUMISTON, C.G., WADE, L.E., BALLARD, J., BEYER, J.E., SCHWETZ, R.W., and NORRIS, J.M. 1983. A chronic toxicity and oncogenicity study in rats and subchronic toxicity study in dogs on ingested vinylidene chloride. Fund. Appl. Toxicol. 3:55-62. (As cited in EPA 1987)

TORKELSON, T.R., and ROWE, V.K. 1981. Halogenated aliphatic hydrocarbons. In Clayton, G.D., and Clayton, P.B., eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York. Vol. 2B, pp. 3550-3555

1,2-DICHLOROPROPANE

1,2-Dichloropropane is absorbed following ingestion and inhalation exposure. Adverse effects associated with acute exposure in humans have included CNS depression, narcosis, headache, and mucous membrane irritation. In animals, effects associated with acute exposure have included histopathological changes in the liver, kidney, and adrenals. Altered serum enzyme activities were also reported (EPA 1985). Chronic or subchronic exposure of experimental animals to 1,2-dichloropropane has been associated with reduced body weight, liver necrosis, centrilobular congestion, altered CNS function, and altered serum enzyme activities (EPA 1985). DeLorenzo et al. (1977) reported that 1,2-dichloropropane induced reverse and forward mutations in some test species. The compound also induced sister chromatid exchanges and chromosomal aberrations in Chinese hamster ovary cells *in vitro* (NTP 1986). 1,2-Dichloropropane caused a dose-related increased incidence of hepatocellular adenomas in rats (NTP 1986).

1,2-Dichloropropane is categorized in Group B2 (Probable Human Carcinogen) (EPA 1990). This chemical has an oral cancer potency factor of 6.8×10^{-2} $(mg/kg\text{-}day)^{-1}$ based on an increased incidence of hepatocellular adenomas in mice in the gavage study conducted by NTP (1986) (EPA 1990).

REFERENCES

DELORENZO, F., DEGL'LNNOCENTI, S., RUOCCO, A., SILENGO, L., CORTESE, R. 1977. Mutagenicity of pesticides containing 1,2-dichloropropane. Cancer Res. 37:1915-1917

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Drinking Water Criteria Document for 1,2-Dichloropropane. Environmental Criteria and Assessment Office, Cincinnati, Ohio EPA 600/X-84-162

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

NATIONAL TOXICOLOGY PROGRAM (NTP). 1986. NTP Technical report on the carcinogenicity bioassay of 1,2-dichloropropane (CAS No. 78-87-5) in F344/N rats and B6C3F$_1$ mice (gavage study). NTP-82-092, NIH Publ. No. 84-2519, NTP TR 263. U.S. DHHS, PHS, NIH. August 1986

1,3-DICHLOROPROPENE

1,3-Dichloropropene, otherwise known as Telone II, is absorbed by experimental animals following oral, inhalation, or dermal exposure (ACGIH 1986, IARC 1986, NTP 1985). Accidental exposure of humans to 1,3-dichloropropene fumes has been associated with malaise, headache, abdominal and chest discomfort, mucous membrane irritation, dizziness, nausea, vomiting, weakness, breathing difficulty, and elevated levels of serum transaminases. Ocular and dermal injuries have been associated with accidental exposure to pesticides containing 1,3-dichloropropene (IARC 1986). Acute or subchronic oral exposure has been associated with injury to the liver and kidneys, increased renal weights, and depression of renal organic ion transport (IARC 1986). Acute or subchronic inhalation exposure of experimental animals to 1,3-dichloropropene has been associated with lesions of the liver, kidney, lung, and nasal epithelium; hepatic and renal necrosis; hepatocellular enlargement; reduced body weight gain; and increased organ-to-body-weight ratios (IARC 1986, Stott et al. 1982). Edema and necrosis of the skin have been observed in rabbits following dermal exposure (IARC 1986). NTP (1985) evaluated the carcinogenicity of technical grade 1,3-dichloropropene in rats and mice following oral administration. Dose-related increases in the incidence of tumors in the bladder, alveoli, forestomach, liver, adrenal, and thyroid were observed. A two-year inhalation bioassay observed an increased incidence of benign lung tumors in mice (Lomax et al. 1989).

EPA (1990a) classifies 1,3-dichloropropene as a Group B2 agent (Probable Human Carcinogen). This weight of evidence applies to those substances for which there is sufficient evidence of carcinogenicity in animals and inadequate evidence of carcinogenicity in humans. EPA (1990b) developed inhalation and oral cancer potency factors of 1.3×10^{-1} $(mg/kg\text{-}day)^{-1}$ and 1.8×10^{-1} $(mg/kg\text{-}day)^{-1}$, respectively. The inhalation value was based on the development of benign lung tumors in mice exposed to 1,3-dichloropropene vapors for 2 years (Lomax et al. 1989). The oral slope factor was derived from the observation of forestomach, liver, adrenal and thyroid tumors in rats chronically exposed by gavage (NTP 1985). EPA (1990a) reported an oral reference dose (RfD) of 3×10^{-4} mg/kg-day based on a subchronic feeding study in rats in which increased organ weights (kidney weights) were observed (Dow Chemical 1973). An uncertainty factor of 10,000 was used to derive the RfD. EPA (1990b) has derived an inhalation RfD of 1×10^{-2} mg/m^3 for 1,3-Dichloropropene based on degenerative changes in the nasal mucosa in rats (Stott et al. 1982). A safety factor of 100 was applied to a no-observed-adverse effect level (NOAEL) of 45.4 mg/m^3 to derive the inhalation RfD.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices: 1,3-Dichloropropene. 5th ed. Cincinnati, Ohio

DOW CHEMICAL CO. 1973. FOI, Environmental Protection Agency, Washington, D.C. MRID Nos. 00067977, 00039684

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1986. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Some Halogenated Hydrocarbon and Pesticide Exposures. World Health Organization, Lyon, France. Vol. 41, pp. 113-130

LOMAX, L.G., W.T. STOTT, K.A. JOHNSON, L.L. CALHOUN, B.L. YANO AND J.F. QUAST. 1989. The chronic toxicity and oncogenicity of inhaled technical grade 1,3-dichloropropene in rats and mice. Fund. Appl. Toxicol. 12:418-431.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1985. Toxicology and Carcinogenesis Studies of Telone II in F344/N Rats and B6C3F$_1$ Mice. Gavage Studies. Technical Grade 1,3-Dichloropropene (CAS No. 542-75-6) Containing 1.0% Epichlorohydrin as a Stabilizer. Technical Report Series No. 269. DHHS (NIH) Publication No. 85-75-6. May 1985

STOTT, W.T., J.T. YOUNG, L.L. CALHOUN, AND J.E. BATTJES. 1988. Subchronic toxicity of inhaled technical grade 1,3-dichloropropene in rats and mice. Fund. Appl. Toxicol. 11:207-220.

Dieldrin can be absorbed by humans from the gastrointestinal tract following ingestion of the pesticide (NIOSH 1978), and absorbed through human skin following percutaneous exposure (Feldmann and Maibach 1974). NIOSH (1978) reported that another possible route of absorption by humans is through inhalation (NIOSH 1978). Reported effects in humans following acute exposure to dieldrin include malaise, incoordination, headache, dizziness, gastrointestinal disturbances, and major motor convulsions (NRC 1982). Dieldrin is acutely toxic to laboratory animals by the oral, dermal, and inhalation routes. It is mildly irritating to the eye and skin. Dieldrin affects the central nervous system, producing irritability, tremors, and convulsions (Health and Vandekar 1964). In experimental animals chronic oral administration of dieldrin is associated with liver and kidney damage (Walker et al. 1969, Treon and Cleveland 1955, Murphy and Korschgen 1970). Oral administration of dieldrin is reported to result in reproductive toxicity, fetotoxicity, and teratogenicity in mice and hamsters (Diechmann 1972, Ottolenghi et al. 1974). Dieldrin is reported to cause a significant dose-related increase in the incidence of hepatocellular carcinoma in mice exposed in the diet (NCI 1978, Davis and Fitzhugh 1962, Walker et al. 1972).

EPA has classified dieldrin in Group B2 (Probable Human Carcinogen) based on inadequate evidence of carcinogenicity from human studies and sufficient evidence of carcinogenicity from animal studies (EPA 1990). EPA (1990) reported a cancer potency factor of 1.6×10^{1} $(mg/kg\text{-}day)^{-1}$ for both oral and inhalation exposures based on several studies in which hepatocellular carcinomas were observed in mice administered dieldrin in the diet (Walker et al. 1972, Thorpe and Walker 1973, NCI 1978, Tennekes et al. 1981). EPA (1990) has established an oral reference dose (RfD) of 5.0×10^{-5} mg/kg-day for dieldrin based on liver lesions observed in rats (Walker et al. 1969). The RfD was derived using a no-observed-effect level (NOEL) of 0.005 mg/kg-day and an uncertainty factor of 100.

REFERENCES

DAVIS, K.J., and FITZHUGH, O.G. 1962. Tumorigenic potential of aldrin and dieldrin for mice. Toxicol. Appl. Pharmacol. 4:187-189

DEICHMANN, W.R. 1972. Toxicology of DDT and related chlorinated hydrocarbon pesticides. J. Occup. Med. 14:285-292

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Aldrin/Dieldrin. Office of Water Regulations and Standards. October 1980. NTIS Publication No. PB81-117301

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

FELDMANN, R.J., and MAIBACH, H.I. 1974. Percutaneous penetration of some pesticides and herbicides in man. Toxicol. Appl. Pharmacol. 28:126-132

HEALTH, D.F., and VANDEKAR, M. 1964. Toxicity and metabolism of dieldrin in rats. Br. J. Ind. Med. 21:269-279

MURPHY,D.A., and KORSCHGEN, L.F. 1970. Reproduction growth and tissue residues of deer fed dieldrin. Wildlife Mgmt. 34:887-903

NATIONAL CANCER INSTITUTE (NCI). 1978. Bioassays of Aldrin and Dieldrin for Possible Carcinogenicity. DHEW Publication No. (NIH) 78-821. National Cancer Institute Carcinogenesis Technical Report Series No. 21. NCI-CG-TR-21. 184 pp.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1978. Special Occupational Hazard Review. Aldrin/Dieldrin. U.S. DHEW Publication No. 78-201

NATIONAL RESEARCH COUNCIL (NRC). 1982. An Assessment of the Health Risks of Seven Pesticides Used for Termite Control. August 1982. NRC-P901. NTIS Publication PB83-136374

OTTOLENGHI, A.D., HASEMAN, J.K., and SUGGS, F. 1974. Teratogenic effects of aldrin, dieldrin, and endrin in hamsters and mice. Teratology 9:11-16

TENNEKES, H.A., WRIGHT, A.S., DIX, K.M., KOEMAN, J.H. 1981. Effects of dieldrin, diet and bedding on enzyme function and tumor incidence in livers of male CF-1 mice. Cancer Res. 41:3615-3620

THORPE, E., WALKER, A.I.T. 1973. The toxicology of dieldrin (HEOD). Part II. Comparative long-term oral toxicology studies in mice with dieldrin, DDT, phenobarbitone, beta-BHC and gamma-BHC. Food Cosmet. Toxicol. 11:433-441

TREON, J., and CLEVELAND, F.D. 1955. Toxicity of certain chlorinated hydrocarbon insecticides for laboratory animals with special reference to aldrin and dieldrin. Agric. Food Chem. J. 3:402

WALKER, A.I.T., STEVENSON, D.E., ROBINSON, J., THORPE, E., and ROBERTS, M. 1969. The toxicology and pharmacodynamics of dieldrin (HEOD): Two-year oral exposures of rats and dogs. Toxicol. Appl. Pharmacol. 15:345-373

WALKER, A.I.T., THORPE, E., STEVENSON, D.E. 1972. The toxicology of dieldrin (HEOD). I. Long-term oral toxicity studies in mice. Food. Cosmet. Toxicol. 11:415-432

DIETHYLPHTHALATE

Diethylphthalate (DEP) is absorbed following ingestion and inhalation exposures. Its acute toxicity for laboratory animals by most routes of administration is very low (NIOSH 1986). Exposure of humans to the heated vapor may cause respiratory irritation (ACGIH 1986). No specific lesions were observed in subchronic or chronic feeding studies of DEP to rats and dogs. However, decreased consumption of food and increased relative organ weights were observed in some of the animals (EPA 1980, EPA 1986, Brown et al. 1978). Workers chronically exposed to DEP experienced pain, numbness, and spasms in the upper and lower extremities (ACGIH 1986). Reduced fetal weight, resorptions and dose-related musculoskeletal abnormalities were observed among fetuses from rats exposed to DEP intraperitoneally during gestation (EPA 1980). Studies indicate that DEP is mutagenic in bacterial test systems (EPA 1986, Seed 1982). Currently, no information is available on the carcinogenic potential of DEP in humans or animals.

EPA (1990) calculated an oral reference dose (RfD) of $8x10^{-1}$ mg/kg-day based on a subchronic rat study in which decreased growth rate, food consumption, and altered organ weights were the observed effects (Brown et al. 1978). The oral RfD was derived using a no-observed-adverse-effect level (NOAEL) of 750 mg/kg-day and an uncertainty factor of 1,000.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed.. AGCIH, Cincinnati, Ohio

BROWN, D., BUTTERWORTH, K.R., GAUNT, I.F., GRASSO, P., and GANGOLLI, S. 1978. Short-term toxicity study of diethyl phthalate in the rat. Food Cosmet. Toxicol. 16:415-422

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Phthalate Esters. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-067

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Verified Reference Doses (RfDs) of the USEPA. The ADI Work Group of the Risk Assessment Forum. Cincinnati, Ohio. January 1986. ECAO-CIN-475

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1986. Data base. Registry of Toxic Effects of Chemical Substances. Washington, D.C.

SEED, J.L. 1982. Mutagenic activity of phthalate esters in bacterial liquid suspension assays. Environ. Health Perspect. 45:111-114

2,4-DIMETHYLPHENOL

Little information is available on the health effects from exposure to 2,4-dimethylphenol (2,4-DMP). Exposure to 2,4-DMP is usually as a component of a complex mixture (EPA 1980). It is readily absorbed through the skin of animals and has been reported to be an adenosine triphosphate (ATP) blocking agent (EPA 1980). In rats, dermal and oral LD_{50}s for 2,4-dimethylphenol of 1,040 mg/kg and 3,200 mg/kg, respectively, have been reported (Uzhdovini et al. 1974). Also, 2,4-dimethylphenol has been reported to act as a cancer promoting agent in skin painting studies in rats (Boutwell & Bosch 1959).

EPA (1990) has derived an oral RfD for 2,4-DMP of 2.00×10^{-2} mg/kg-day with an uncertainty factor of 3,000 based on clinical signs (lethargy, prostration, and ataxia) and hematological changes in mice which were exposed to 250 mg 2,4-DMP/kg-day for 90 days by gavage. The NOAEL (no-observed-adverse-effects-level) was 50 mg 2,4-DMP/kg-day (EPA 1989). 2,4-DMP has not been evaluated by the U.S. EPA for evidence of human carcinogenic potential.

REFERENCES

BOUTWELL, R.K., and BOSCH, D.K. 1959. The tumor-producing action of phenol and related compounds for mouse skin. Cancer Res. 19:413

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for 2,4-Dimethylphenol. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-044

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989. Ninety-day gavage study in Albino mice using 2,4-dimethylphenol. Office of Solid Waste and Emergency Response, Washington, D.C. Study No. 410-2831.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Office of Health and Environmental Assessment Office, Cincinnati, Ohio.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1983. Data base. Registry of Toxic Effects of Chemical Substances. Washington, D.C. October 1983

UZHDOVINI, E.R., et al. 1974. Acute toxicity of lower phenols. Gig. Tb. Prof. Zabol. 2:58. (as cited by EPA 1980)

DINITROBENZENES

Absorption of the dinitrobenzene (DNB) isomers (1,2-DNB, 1,3-DNB, and 1,4-DNB) have not been well characterized, however ready absorption through the skin is a major factor in its toxicity (ACGIH 1986). Occupational exposures to the DNB isomers have been associated with methemoglobinemia and respiratory tract irritation. Prolonged exposures of humans to dinitrobenzene may result in anemia, liver damage and cyanosis (Beard and Noe 1981). In animals, subchronic oral exposures have resulted in retarded growth, decreased hemoglobin concentrations, splenic enlargement and hemosiderin deposits. Testicular atrophy and decreased spermatogenesis have also been observed in male rats following oral exposures (Cody et al. 1981).

EPA (1990) has developed an oral reference dose (RfD) of 1.0×10^{-4} mg/kg-day for 1,3-DNB based on a subchronic drinking water study in rats. This study identified a lowest-observed-effect-level (LOEL) of 8 ppm for increased splenic weight and a no-observed-effect-level (NOAEL) of 3 ppm (0.40 mg/kg-day) (Cody et al. 1981). The RfD was calculated using the NOAEL and an uncertainty factor of 3,000.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENIST (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices 5th ed. ACGIH Inc. Cincinnati, Ohio

BEARD, R., NOE, J.T. 1981. Aromatic Nitro and Amino Compounds. In Clayton, G.D., and Clayton, P.B. eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York. Vol 2A

CODY, T.E., WINTHERUP, S., HASTINGS, L., STEMMER, K., and CHRISTIAN, R.T. 1981. 1,3-Dinitrobenzene: Toxic effect in vivo and in vitro. J. Toxicol. Environ. Health 7(5):829-847

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria Assessment Office, Cincinnati, Ohio

2,4- AND 2,6- DINITROTOLUENE

Although five isomers of dinitrotoluene (DNT) exist, only two (2,4-and 2,6-) will be discussed. Dinitrotoluene is rapidly absorbed following inhalation, oral and dermal exposure. The blood, liver, and neuromuscular systems are the primary target organs for DNT toxicity in both humans and experimental animals. In humans, exposure symptoms include moderate cyanosis, anemia, dyspnea, dizziness, sleepiness, and methemoglobinemia (Hamblin 1963; von Oettingen 1941). Long term occupational exposures also have been correlated with an increase in ischemic heart disease (Levine et al. 1986). In rats, chronic exposure to pure 2,6-DNT and mixtures of 2,4- and 2,6-DNT in the diet have significantly increased the incidence of liver tumors (Ellis et al. 1979; Leonard et al. 1987).

EPA (1989) has classified 2,4- and 2,6-dinitrotoluene in Group B2 --Probable Human Carcinogen for oral and inhalation routes. Both isomers have an oral cancer potency factor of 0.68 $(mg/kg\text{-}day)^{-1}$ based on a 2-year study in which rats developed liver and mammary gland tumors when fed a mixture of 2,4- and 2,6- dinitrotoluene isomers (Ellis et al. 1979). The oral cancer potency factor for 2,4-DNT is pending by EPA (1990).

REFERENCES

ELLIS, H.V., HAGEUSEN, S.H. HODGSON, J.R., ET AL. 1979. Mammalian Toxicity of Munition Compounds. Phase III. Effects of Lifetime Exposure. Part I. 2,4-Dinitrotoluene. NTIS AD A077692. 281pp.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, OH.

ETNIER, E.L. 1987. Water Quality Criteria for 2,4-Dinitrotoluene and 2,6-Dinitrotoluene. Final Report. Oak Ridge National Laboratory, Oak Ridge, Tennessee.

HAMBLIN, D.O. 1963. Industrial Hygiene and Toxicology, 2nd ed. Vol. II, pp. 2105-2127. Interscience, New York.

LEONARD, T.B., GRAICHEN, M.E., AND POPP, J.A. 1987. Dinitrotoluene isomer-specific hepatocarcinogenesis in Fischer-344 rats. J. Nat. Cancer Inst.

LEVINE, R.J., ANDJELKOVICH, D.A., KERSTETER, S.L., ARP, E.W., BALOGH, S.A., BLUNDEN, P.B., AND STANLEY, J.S. 1986. Heart disease in workers exposed to dinitrotoluene. J. Occup. Me. 28(9):811-816.

VON OETTINGEN, W.F. 1941. The Aromatic Amino and Nitro Compounds, Their Toxicity and Potential Dangers. pp. 108-111. U.S. Public Health Service Bull. No 271.

1,2-DIPHENYLHYDRAZINE

1,2-Diphenylhydrazine is more commonly called hydrazobenzene. Absorption of 1,2-diphenylhydrazine following inhalation, oral and dermal exposure can be inferred from the toxicity associated with these routes of exposure. There is a paucity of toxicologic information for this compound in humans. There is evidence in experimental animals that 1,2-diphenylhydrazine is carcinogenic following oral exposure. In a 78-week dietary study low-dose male rats fed 1,2-diphenylhydrazine (0.008%) showed significant increased incidences of hepatocellular carcinoma, while incidences of neoplastic liver nodules and mammary adenocarcinomas were significantly elevated in female rats (NCI 1979). High-dose male rats (0.03%) also had an increase in combined incidence of squamous-cell carcinomas or papillomas of the Zymbal's gland, the ear canal and the skin of the ear (NCI 1979). In addition, a significant increase of hepatocellular carcinoma was observed among treated female, but not male mice. Another study found tumor incidences of approximately 22%-50% in mice treated with 1,2-diphenylhydrazine via subcutaneous, topical application to the skin and in the diet and a tumor incidence of 22% in rats treated by all routes (Pliss 1974). Additional studies suggest that 1,2-diphenylhydrazine is an equivocal tumorigenic agent that causes tumors in the liver, skin, and appendages of rats and mice (RTECS 1987). 1,2-diphenylhydrazine was also shown to depress testicular DNA synthesis in mice when administered intraperitoneally at a dose of 100 mg/kg (Seiler 1979).

1,2-Diphenylhydrazine has been given a B2 classification by EPA as a probable human carcinogen based on data in both rats and mice (EPA 1990). EPA (1990) has estimated and oral and inhalation cancer potency factor of 0.8 $(mg/kg\text{-}day)^{-1}$ based on an increased incidence of hepatocellular carcinomas and neoplastic liver nodules in male F344 rats fed dietary concentrations of 1,2-diphenylhydrazine (NCI 1979). Inhalation risk estimates were calculated from the oral exposure data.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Office of Health and Environmental Assessment, Cincinnati, Ohio.

NATIONAL CANCER INSTITUTE (NCI). 1979. Bioassay of hydraazobenzene for possible carcinogenicity. U.S. DHEW Publ. No. NIH 78-1342.

PLISS 1974. (as cited in EPA 1990).

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1987. Vol. 3A. National Institute for Occupational Safety and Health (NIOSH). U.S. Department of Health and Human Services.

SEILER 1977. (as cited in EPA 1990).

1,4-DITHIANE

INTRODUCTION

1,4-Dithiane is an organo-sulfur chemical associated with production and storage of munitions.

TOXICOKINETICS

The absorption, distribution, metabolism and excretion of 1,4-dithiane are unknown at present.

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

There is a paucity of toxicological information on 1,4-Dithiane. 1,4-Dithiane vapors are irritating to the nasal cavity and upper respiratory tract of humans (Fairfield Chemical Co. 1985). 1,4-Dithiane does not appear to be extremely toxic as noted by the rat oral LD_{50} of 2,768 mg/kg. Tremors, ataxia and dyspnea were noted in animals prior to the onset of death (RTECS 1990).

In a 90-day subchronic study, CD strain rats (30 rats/sex/dose) were dosed with 1,4-dithiane by daily gavage at concentrations of 0, 105, 210 and 420 mg/kg-day (Schieferstein et al. 1987). No overt toxicity, treatment-related mortality or ophthalmologic changes were found. No clear dose-related relationships were evident for serum enzyme activities. Treatment-related increases were found in female liver ($p<0.003$) and in male kidney ($p<0.02$) and thymus weight ($p<0.02$); a treatment-related decrease in female brain weight ($p<0.02$) was also evident. At 105 mg/kg-day, significant differences were noted in organ weight of exposed animals compared to controls in the spleen of both sexes, female brain and the male kidneys. The nose, liver and kidney showed microscopic lesions in the high-dose group. The nasal lesion consisted of anisotrophic crystals of undetermined chemical composition (not 1,4-dithiane) were deposited in the olfactory nasal mucosa of both sexes, (and were in greater concentrations in the low-dose females), but were not present in controls. The nasal lesions, crystals and granulomatous inflammation was dose-related; in the low-dose at 2.30 (6.7%) in males, and 24/29 (82.8%) in females. This tissue is capable of metabolizing xenobiotics, therefore it is assumed that these crystals caused ganulomatous inflammatory reaction. Other treatment-related anatomic abnormalities were eosinophilic cytoplasmic granulation of the convoluted renal tubule cells in the high-dose males (26/28) and minimal hypertrophy of the centrilobular region of the liver in the high-dose females (26/30). The most significant pathological finding was the lesions of the nasal cavity. The no-observed-effect level (NOAEL) was 105 mg/kg-day, based in a novel form of toxicity (depostion of anisotrophic crystals in the olfactory mucosa) and granulomatous inflammation.

1,4-Dithiane was not mutagenic in the Ames Salmonella/mammalian microsome mutagenicity assay tester strains TA98, TA100, TA1535, TA1537, and TA1538 (Sano and Korte 1985).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

No regulated standards or criteria have been promulgated for 1,4-dithiane, however, a drinking water criterion of 2.45 mg 1,4-dithiane/liter of water has been suggested (Schieferstein et al. 1987). For the purposes of this risk assessment, Clement has derived an oral RfD of 0.105 mg/kg-day based on the 90-day gavage study (Schieferstein et al. 1987). A no-observed-effect level (NOEL) of 105 mg/kg-day was identified for deposition of anisotrophic crystals in the olfactory mucosa and granulomatous inflammation. An uncertainty factor of 1,000 was applied, (10 to account for animal to human extrapolation, 10 to adjust from subchronic to lifetime exposure and 10 to protect sensitive members of the human population).

DI-n-BUTYL PHTHALATE

Di-n-butyl phthalate is readily absorbed following oral and inhalation exposure (EPA 1980). Acute exposures of di-n-butyl phthalate aerosol in mice have produced irritation of the eyes and upper respiratory tract mucous membranes. Extreme exposures can result in labored breathing, ataxia, paresis, convulsions, and death from paralysis of the respiratory system (ACGIH 1986). Workers chronically exposed to di-n-butyl phthalate in combination with other phthalate plasticizers have exhibited pain, numbness, and spasms in the upper and lower extremities. Further evaluation revealed vestibular dysfunction and polyneuritis (ACGIH 1986). Reduced fetal weight, increased numbers of resorptions, and dose-related musculoskeletal abnormalities have been observed among fetuses from rats and mice exposed to very high doses of di-n-butyl phthalate during gestation (Shiota and Nishimura 1982).

EPA (1990a) calculated an oral reference dose (RfD) for di-n-butyl phthalate based on a study by Smith (1953) in which male Sprague-Dawley rats were fed a diet containing dibutyl phthalate for a period of 1 year. One-half of all rats receiving the highest di-n-butyl phthalate concentration (1.25% of diet, or 600 mg/kg-day) died during the first week of exposure. The remaining animals survived the study with no apparent adverse effects. Using a NOAEL of 125 mg/kg-day (0.25% di-n-butyl phthalate in diet) and an uncertainty factor of 1,000, an oral reference dose (RfD) of 0.1 mg/kg-day was derived; a LOAEL of 600 mg/kg-day (1.25% dibutyl phthalate in diet) was observed in this study. EPA (1990b) derived a subchronic oral RfD of 1 mg/kg-day using an uncertainty factor of 100, based on the same study, and effect of concern.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, OH.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Phthalate Esters. Office of Water Regulations and Standards, Washington, D.C. October 1980. EPA 440/5-80-067

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment summary Tables. Prepared by the Office of Health and Envrionmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

SHIOTA, K., and NISHIMURA, H. 1982. Teratogenicity of di(2-ethylhexyl) phthalate (DEHP) and di-n-butyl phthalate (DBP) in mice. Environ. Health Perspect. 45:65-70

SMITH, C.C. 1953. Toxicity of butyl stearate, dibutyl sebacate, dibutyl phthalate, and methoxyethyl oleate. Arch. Ind. Hyg. 7:310

DI-*n*-OCTYL PHTHALATE

Di-*n*-octyl phthalate is not especially toxic. It is a severe eye and a mild skin irritant in rabbits (NIOSH 1985, NTP/IRLG 1982, EPA 1980). Subchronic dietary exposure to rodents has resulted in elevated kidney and liver weights, in addition to increased SGOT and SGPT (Piekacz 1971). Fetotoxicity and developmental abnormalities were observed in the offspring of rats administered 5 g/kg intraperitoneal injections on days 6 to 15 of gestation (NTP/IRLG 1982, EPA 1980).

EPA (1990) has derived an oral RfD of $2x10^{-2}$ mg/kg-day for di-n-octyl phthalate based on elevated kidney and liver weights and increased SGOT and SGPT in rats exposed to dietary concentrations of 175 mg/kg-day for 7-12 months (Piekacz 1971). A saftey factor of 1,000 was used to calculate the RfD.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Phthalate Esters. Office of Water Regulations and Standards, Washington, D.C. October 1980. EPA 440/5-80-067

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment summary Tables. Prepared by the Office of Health and Envrionmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1985. Registry of Toxic Effects of Chemical Substances Data Base. Washington, D.C. July 1985

NATIONAL TOXICOLOGY PROGRAM AND THE INTERAGENCY REGULATORY LIAISON GROUP (NTP/IRLG). 1982. The Conference on Phthalates. Environ. Health Perspect. 45:1-153

PIEKACZ, H. 1971. Effect of dioctyl and dibutyl phthalates on the organism of rats after oral administration in prolonged experiment. II. Subacute and chronic toxicity. Rocz. Panstw. Zakl. Hig. 22(3):295-307.

ENDOSULFAN

Technical grade endosulfan is composed of two isomers, endosulfan I (α) and endosulfan II (β) in approximately a 7:3 ratio, respectively (Hayes 1982). Absorption of the β-isomer exceeds that of the α-isomer and occurs in mammals following both oral and dermal exposure (EPA 1980). Absorption is enhanced by alcohols, oils, and emulsifiers (Maier-Bode 1968). Substantial absorption following inhalation exposure to endosulfan is not expected to occur, due to the substance's low vapor pressure (EPA 1980). Following ingestion endosulfan is distributed initially to the liver and then subsequently to the brain, heart, kidney, lungs, spleen, testes, thymus gland and other tissues and organs (EPA 1980). Acute endosulfan poisoning in humans produces symptoms which include gagging, vomiting, diarrhea, agitation, tonic-clonic convulsions, dyspnea, apnea, cyanosis, loss of consciousness, and death in some cases (Hayes 1982). Acute exposure in animals causes signs of CNS toxicity including hyperactivity, tremors, and convulsions followed by death (WHO 1984). Subchronic oral exposure to rats has resulted in adverse renal effects (Hoeschst Aktiengesellschaft 1984). Chronic exposure results in reduced survival, enlarged kidneys and signs of renal tubular damage with interstitial nephritis and hepatocellular changes in rats (WHO 1984). Diets deficient in protein are reported to increase the toxicity of technical grade endosulfan in rats (EPA 1980, Hayes 1982). Adverse reproductive effects including testicular degeneration and atrophy have been reported in mice and rats following chronic exposure (EPA 1980).

EPA (1990) has derived an oral risk reference dose (RfD) for endosulfan of $5x10^{-5}$ mg/kg-day based on an unpublished reproduction study (Hoeschst Aktiengesellschaft 1984). In this study, rats were administered endosulfan at dietary concentrations of 0, 3, 15, or 75 ppm for two generations. Renal toxicity was observed at an endosulfan concentration of 3 ppm (0.15 mg/kg-day); an uncertainty factor of 3,000 was used to derive the RfD.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Endosulfan. Office of Water Regulations and Standards. Washington, D.C. October 1980. EPA 440/5-80-046

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

GUPTA, P, CHANDRA, S., and SAXENA, D. 1978. Teratogenic and embryonic effects of endosulfan in rats. Acta Pharmacol. Toxicol 42:150-152

HAYES, W., Jr. 1982. Pesticides Studied in Man. Williams and Wilkins. Baltimore, Maryland

HOESCHST AKTIENGESELLSCHAFT. 1984. Unpublished two-generation reproduction study in rats. Acc. Nos. 256127, 257727 (As cited in EPA 1987)

NATIONAL CANCER INSTITUTE (NCI). 1978. Bioassay of endosulfan for possible carcinogenicity. Natl. Cancer Inst. Div. Cancer Cause and Prevention. Bethesda, Maryland. DHEW Pub. No. (NIH) 78-1312

WORLD HEALTH ORGANIZATION (WHO). 1984. Endosulfan. Environmental Health Criteria 40. World Health Organization. Geneva

ENDOSULFAN SULFATE

Endosulfan sulfate is a metabolite of endosulfan in mammals and therefore is expected to be toxicologically similar to endosulfan (Worthing 1987). Endosulfan produces gastrointestinal, renal, CNS, and reproductive toxicity (Hayes 1982, EPA 1980, WHO 1984). No information concerning the health effects of endosulfan sulfate was located in the available literature and no health based criteria have been established by EPA for endosulfan sulfate.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Endosulfan. Office of Water Regulations and Standards. Washington, D.C. October 1980. EPA 440/5-80-046

HAYES, W., Jr. 1982. Pesticides Studied in Man. Williams and Wilkins. Baltimore, Maryland. pp 252-253

WORLD HEALTH ORGANIZATION (WHO). 1984. Endosulfan. Environmental Health Criteria 40. World Health Organization. Geneva

WORTHING, C.R. (Ed.) 1987. The Pesticide Manual, a world compendium. The British Crop Protection Council. 8th Ed. pp.335-336

ENDRIN ALDEHYDE

Endrin aldehyde occurs as an impurity of endrin and as a degradation product (ATSDR 1990). No information was available concerning the health effects of endrin aldehyde. However, because endrin aldehyde is structurally similar to endrin, the aldehyde degradation product is assumed to have similar toxicity as endrin. The physical and chemical properties of aldehydes (elevated soubility in both water and organics; elevated reactivity relative to ketones) suggests that the aldehyde may be more readily absorbed and more active that endrin. In humans, the central nervous system is the predominant target organ of endrin exposure (ATSDR 1990). The primary manifestations of repeated oral and inhalation exposure to endrin in experimental are renal, hepatic and neurological effects (Treon et al. 1955, ATSDR 1990). No health-based criteria have been established by EPA for endrin aldehyde.

REFERENCES

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1990. Toxicological profile for endrin. Prepared by Life Systems, Inc. under subcontract to Clement associates, Inc. Contract No. 205-88-0608.

TREON, J., F.P. CLEVELAND, J. CAPPEL. 1955. Toxicity of endrin for laboratory animals. J Agric Food Chem 3:402-408.

2-ETHYL-1-HEXANOL

2-Ethyl-1-hexanol, also known as 2-ethylhexanol, and 2-ethylhexyl alchol is a colorless liquid. It is absorbed via the gastrointestinal and respiratory tract but is not readily absorbed through the skin in toxic amounts (Rowe and McCollister 1981). Ninety percent of an ingested dose was excreted in the urine of rabbits (Rowe and McCollister 1981). No toxicologic information is available for humans. 2-Ethyl-1-hexanol is a dermal and ocular irritant in rabbits following inhalation and dermal exposure (RTECS 1987, Smyth et al. 1969, Scala and Burtis 1973). One drop applied to the eyes of rabbits produces reddening and swelling; severe irritation progressing to corneal opacity may occur following application of 0.1 ml of undiluted 2-ethyl-1-hexanol (Scala and Burtis 1973, Schmidt et al. 1973). This compound is not especially toxic in experimental animals following ingestion as indicated by the rat oral LD_{50} of 2.0 gm/kg; the oral LD_{50}'s in mice, guinea pigs and rabbits are 2.5, 1.86 and 1.18 gm/kg, respectively (RTECS 1987). Acute inhalation exposures can cause irritation of the respiratory tract in animals but are of a low order of toxicity (Rowe and McCollister 1981). Vapors of 2-ethyl-1-hexanol increase the central nervous system excitability of rats and rabbits (Weiss 1986) and cause CNS depression including labored respiration (Rowe and McCollister 1981). Repeated dermal exposures for 12 days to rabbits resulted in changes in the liver, kidney and heart, inflammation of the lungs, and impaired spermatogenesis (Schmidt et al. 1973). Subchronic dietary exposures produced liver and kidney effects in rats characterized by mild and reversible histopathologic changes (Rowe and McCollister 1981). 2-Ethyl-1-hexanol adversely affects reproduction as indicated by decreased viability index, growth statistics and effects on newborn in mice orally exposed. Developmental abnormalities of the musculoskeletal, cardiovascular and urogenital system were evident in rats exposed in utero (RTECS 1987). There is some evidence that this compound is mutagenic in S. typhimurium without activation (RTECS 1987). No health-based criteria have been derived by EPA.

REFERENCES

GOSSELIN, R.E., SMITH, R.P., and HODGE, H.C., 1984. Clinical Toxicology of Commerical Products. Fifth Edition, Williams & Wilkins, Baltimore, MD. p. 174

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1987. Vol. 3A. National Institute for Occupational Safety and Health (NIOSH). U.S. Department of Health and Human Services.

MELLAN, I. 1950. Industrial Solvents, Reinhold, New York, pp. 512 (as cited in Rowe and McCollister 1981).

ROWE, V.K. and McCOLLISTER, S.B. 1981. Alcohols. In Clayton and Clayton eds., Patty's Industrial Hygiene and Toxicology, Volume IIc, pp. 4620-4704.

SAX, N.I. AND LEWIS, R.J. 1989. Dangerous Properties of Industrial Materials. Seventh Edition, Volume II, Van Nostrand Reinhold Co., New York, p. 1640.

SCALA, R.A. AND BURTIS,E.G., 1973. American Industrial Hygiene Association Journal, Volume 34 (11), p. 493. as cited in ROWE, V.K. AND McCOLLISTER, S.B. 1981, Alcohols in Clayton and Clayton eds., Patty's Industrial Hygiene and Toxicology, Volume IIc, pp. 4620-4604.

SCHMIDT, P., GOHLKE, R., AND ROTHE, R., 1973. Ges Hyg. Grenzeb, Volume 19, No. 7, p.485, as cited in ROWE, V.K. AND McCOLLISTER, S.B. 1981, Alcohols in Clayton and Clayton eds., Patty's Industrial Hygiene and Toxicology, Volume IIc, pp. 4620-4704.

SMYTH, H.F., Jr., CARPENTER, C.P., WEIL, C.S., ET. AL., 1969. American Industrial Hygiene Association Journal, Volume 30, Number 5, p. 470. Alcohols in Clayton and Clayton eds., Patty's Industrial Hygiene and Toxicology, Volume IIc, pp. 4620-4704.

ETHYLBENZENE

Ethylbenzene is absorbed via inhalation and distributed throughout the body in rats; the highest levels were detected in the kidney, lung, adipose tissue, digestive tract, and liver (Chin et al. 1980). In humans, short-term inhalation exposure to 435 mg/m^3 ethylbenzene for 8 hours can result in sleepiness, fatigue, headache, and mild eye and respiratory irritation (Bardodej and Bardodejova 1970); eye irritation has also been observed in experimental animals exposed to ethylbenzene (EPA 1987). Increased weights and cloudy swelling were observed in the liver and kidney of rats exposed to ethylbenzene by gavage at a dose of 408 mg/kg-day for 182 days (Wolf et al. 1956). A single oral dose of ethylbenzene administered to male and female Wistar-derived rats was reported to have an LD_{50} of 3,500 mg/kg body weight, with systemic effects occurring primarily in the liver and kidney (Wolf et al. 1956). Maternal toxicity was observed in rats exposed by inhalation to 4,348 mg/m^3 ethylbenzene for 6-7 hours/day during the first 19 days of gestation (Hardin et al. 1981).

EPA (1990a) derived an oral reference dose of 0.1 mg/kg-day for ethylbenzene based on the chronic study by Wolf et al. (1956) in which no liver or kidney effects were observed in rats exposed to 136 mg/kg-day. An uncertainty factor of 1,000 was applied to the no-observed-effect-level to derive the reference dose. EPA (1990b) derived a subchronic oral RfD of 1 mg/kg-day using an uncertainty factor of 100 based on the same study and same effects of concern.

REFERENCES

BARDODEJ, Z., and BARDODEJOVA, E. 1970. Biotransformation of ethylbenzene, styrene, and alpha-methylstyrene in man. Am. Ind. Hyg. Assoc. J. 31:206-209

CHIN, B.H., McKELVEY, J.A., TYLER, T.R., CALISTI, L.J., KOZBELT, S.J., and SULLIVAN, L.J. 1980. Absorption, distribution and excretion of ethylbenzene, ethylcyclohexane and methyl ethylbenzene isomers in rats. Bull. Environ. Contam. Toxicol. 24:477-483

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Health Advisory for Ethylbenzene. Office of Drinking Water, Washington, D.C. March 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

HARDIN, B.D., BOND, G.P., SIKOV, M.R., ANDREW, F.D., BELILES, R.P., and NIEMEIER, R.W. 1981. Testing of selected workplace chemicals for teratogenic potential. Scand. J. Work Environ. Health 7:66-75

WOLF, M.A., ROWE, V.K., McCOLLISTER, D.D., HOLLINGSWORTH, R.L., and OYEN, F. 1956. Toxicological studies of certain alkylated benzenes and benzene. Arch. Ind. Health 14:387-398

bis(2-ETHYLHEXYL)PHTHALATE

Bis(2-Ethylhexyl)phthalate, also known as di-ethylhexyl phthalate (DEHP), is readily absorbed following oral or inhalation exposure (EPA 1980). Chronic exposure to relatively high concentrations of DEHP in the diet can cause retardation of growth and increased liver and kidney weights in laboratory animals (NTP 1982, EPA 1980, Carpenter et al. 1953). Reduced fetal weight and increased number of resorptions have been observed in rats exposed orally to DEHP (EPA 1980). DEHP is reported to be carcinogenic in rats and mice, causing increased incidences of hepatocellular carcinomas or neoplastic nodules following oral administration (NTP 1982).

DEHP has been classified in Group B2--Probable Human Carcinogen (EPA 1986, 1990a). EPA (1990a) calculated an oral cancer potency factor for DEHP of 1.4×10^{-2} $(mg/kg\text{-}day)^{-1}$ based on data from the NTP (1982) study in which liver tumors were noted in mice. EPA has recommended an oral reference dose (RfD) for DEHP of 2×10^{-2} mg/kg-day for both chronic (EPA 1990a) and subchronic (EPA 1990b) exposures based on a study by Carpenter et al. (1953) in which increased liver weight was observed in female guinea pigs exposed to 19 mg/kg bw/day in the diet for 1 year (EPA 1990a); an uncertainty factor of 1,000 was used to develop both RfDs.

REFERENCES

CARPENTER, C.P., WEIL, C.S., and SMYTH, H.F. 1953. Chronic oral toxicity of di(2-ethylhexyl)phthalate for rats, guinea pigs, and dogs. Arch. Indust. Hyg. Occup. Med. 8:219-226

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Phthalate Esters. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 40/5-80-067

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Superfund Public Health Evaluation Manual. Office of Emergency and Remedial Response, Washington, D.C. EPA 540/1-86-060

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

NATIONAL TOXICOLOGY PROGRAM (NTP). 1982. Carcinogenesis Bioassay of Di(2-ethylhexyl)phthalate in F344 Rats and B6C3F$_1$ Mice. Feed Study. NTP Technical Report Series No. 217, U.S. Department of Health and Human Services. NIH Publication No. 82-1773. NTP-80-37

ETHYLMETHYLPHOSPHONIC ACID (EMPA)

INTRODUCTION

Ethylmethylphosphonic acid (EMPA) is a breakdown product of VX (agent GB) (Chemical Stockpile Disposal Program 1988). Hydrolysis products, such as EMPA are smaller, less complex molecules and are usually less potent than the parent agent (Chemical Stockpile Disposal Program 1988).

TOXICOKINETICS

The primary routes of potential exposure to the hydrolysis products of chemical agents are oral and percutaneous absorption. Extensive dilutions of hydrolysis products has usually occurred (Chemical Stockpile Disposal Program 1988). Little is known about the absorption, distribution, metabolism and excretion of EMPA.

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

No toxicological information was available for EMPA. However, like methylphosphonic acid (MPA), it is assumed to be relatively nontoxic (Chemical Stockpile Disposal Program 1988).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

No regulated standards or criteria have been promulgated for EMPA.

REFERENCES

CHEMICAL STOCKPILE DISPOSAL PROGRAM. 1988. Final Programmatic Environmental Impact Statement. Program Executive Officer--Program Manager for Chemical Demilitarization. Aberdeen Proving Ground MD.

The gastrointestinal absorption of the fluoride anion has been estimated to be 97% in human volunteers following administration of 1 mg fluoride, as sodium fluoride in 250 mg of water (Calson et al. 1960). Calcium fluoride administered as solids in food to volunteers were substantially less absorbed than fluoride in aqueous solution (McClure et al. 1945). Fluoride is absorbed via the lungs as evidenced by biological monitoring of workers involved in mining and manufacturing operations (NIOSH 1975). In humans, the primary effects observed following acute high-level oral exposures to sodium fluoride are hypocalcemia (resulting in tetany and ventricular fibrillation) (Eichler et al. 1982) and gastrointestinal pain (Waldbott 1981, Hoffman et al. 1980). The predominant effects observed in rats following acute high-level inhalation exposure include marked nasal irritation, respiratory distress, lacrimation and reddened conjunctivae, depression and sluggishness (Rosenholtz et al. 1963). Chronic oral and occupational exposures have resulted in skeletal fluorosis, a clinical syndrome characterized by mottled tooth enamel, osteosclerosis (increased bone density), brittle bones, and increased bone x-ray opacity (Czerwinski et al. 1988, Chan-Yeung et al. 1983, Kaltreider et al. 1972, Mann et al, 1987). Crippling skeletal fluorosis affects all bones but particularly cancellous bones. Lesions consist of thickening of bones, exostoses, periosteal proliferation, and calcification of ligaments particularly of the neck and vertebral column (EPA 1985a,b). Hodge and Smith (1965) reviewed a number of chronic studies of fluoride ingestion of various species and concluded that histological and functional changes in the kidneys can develop if the drinking water contains more than 100 mg/liter fluoride. Leone et al. (1956) reported hypertension, electrocardiogram irregularities, and slowing of the heart in dogs following oral doses of 9 mg/kg fluoride.

EPA (1990) has derived an oral RfD of 0.06 mg/kg-day for fluorine (soluble fluoride) based on the epidemiological database for dental fluorosis in children. Using the studies of Hodge (1950) and Underwood (1977), a no-observable-effect level for fluoride in drinking water of 1.0 ppm (1 mg/liter) was identified. Assuming that children consume 1 liter of water per day, and weigh 20 kg, total fluoride consumption is equivalent to approximately 0.06 mg/kg-day. A safety factor of 1 was used to derive the RfD. A lowest-observed-effect level (LOAEL) of 2 ppm was identified from this database.

REFERENCES

CARLSON, CH, ARMSTRONG WD, and SINGER L. 1960. Distribution and excretion of radiofluoride in the human. Proc Soc Exp Biol Med. 104:235-239

CHAN-YEUNG M, WONG R, TAN F, et al. 1983. Epidemiologic health study of workers in an aluminum smelter in Kitimat, B.C. II. Effects on musculoskeletal and other systems. Arch Environ Health. 38:34-40.

CZERWINSKI E, NOWAK J, DABROWKSA D, et al. 1988. Bone and joint pathology in fluoride-exposed workers. Arch Environ Health 43:340-343.

EICHLER HG, LENZ K, FUHRMANN M, et al. 1982. Accidental ingestion of NaF tablets by children. Int J Clin Pharmacol Ther Toxicol 20:334-338.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980a. Drinking water Criteria Document for Fluoride. Office of Drinking Water, Washington, D.C. PB118163. Draft

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980b. National primary drinking water regulations; fluoride. Fed. Reg. 50:47156-47171

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

HODGE HC. 1950. The concentration of fluorides in drinking water to give the point of minimum caries with maximum safety. J Am Dent Assoc. 40:436.

HODGE HC, and SMITH FA. 1965. Biological properties of inorganic fluorides. In Simons, JH., ed. Fluorine Chemistry, Vol IV. Academic Press, New York. Pp. 3-7, 10-13, 29-33, 89-113, 518.

HOFFMAN R, MANN J, CALDERONE J, et al. 1980. Acute fluoride poisoning in a New Mexico elementary school. Pediatrics 65:897-900.

KALTREIDER NL, ELDER MJ, CRALLEY LV, et al. 1972. Health survey of aluminum workers with special reference to fluoride exposure. J Occup Med 14:531-541.

LEONE NC, GEEVER EF, MORAN NC. 1956. Acute and subacute toxicity studies of sodium fluoride in animals. Publ. Health Rep. (Wash) 71:459-467.

MANN J, TIBI M, SGAN-COHEN HD. 1987. Fluorosis and caries prevalence in a community drinking above-optimal fluoridated water. Community Dent Oral Epidemiol. 15:293-295.

McCLURE FJ, MITCHELL HH, HAMILTON TS, and KINSER CA. 1945. Balances of fluoride ingested from various sources in food and water by five young men. J Ind Hyg Toxicol 27:159-170.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1975. Criteria for a Recommended Standard. Occupational Exposure to Inorganic Fluorides. Cincinnati, Ohio.

ROSENHOLTZ MJ, CARSON TR, WEEKS MH, et al. 1963. A toxicopathologic study in animals after brief single exposures to hydrogen fluoride. Am Ind Hyg Assoc J. 24:253-261.

UNDERWOOD EJ. 1977. Trace Elements in Human and Animal Nutrition. Academic Press, N.Y.

WALDBOTT GL. 1981. Mass intoxication from accidental overfluoridation of drinking water. Clin Toxicol. 18:531-541.

Gamma-BHC is readily absorbed by humans and animals following oral and dermal exposures (EPA 1985). Turner and Shanks (1980) showed that in male rats approximately 48% of an oral dose of gamma-BHC appeared in the blood 30 minutes following administration. Humans acutely exposed to gamma-BHC via inhalation or topical application have exhibited adverse hematological effects (Morgan et al. 1980, Vodopick 1975). Seizures and convulsions have been observed in individuals who have acutely ingested or applied gamma-BHC to their skin (Davies et al. 1983, Matsuoka 1981). In animals, the major toxic effects associated with acute and longer-term oral exposures to gamma-BHC include immunosuppression (Desi et al. 1978, Dewan et al. 1980), central nervous system effects (Tilson et al. 1987), and adverse kidney and liver effects (Fitzhugh et al. 1950). Chronic occupational exposures to gamma-BHC have resulted in hematological abnormalities (Samuels and Milby 1971). Various reproductive effects from exposure to gamma-BHC have been demonstrated in rodents (Shivanandappa and Krishnakumari 1982). Hepatocellular carcinomas have been observed in mice exposed to gamma-BHC in the diet (Thorpe and Walker 1973, Wolff et al. 1987).

EPA (1990b) has classified gamma-BHC in group B2-C (Probable/Possible Human Carcinogen), however this weight of evidence is currently under review by EPA. EPA (1990b) estimated an oral cancer potency factor for gamma-BHC of 1.3 $(mg/kg\text{-}day)^{-1}$ based on the incidence of hepatocellular carcinomas observed in mice administered gamma-BHC in their diet (Thorpe and Walker 1973). An oral reference dose (RfD) of 3.0×10^{-4} mg/kg-day has been derived by EPA (1990a) based on an unpublished study in which rats were administered gamma-BHC in their diet for 12 weeks (Zoecon Corporation 1983). In this study liver and kidney toxicity were observed at 20 ppm (1.55 mg/kg-day) but not at 4 ppm (0.3 mg/kg-day). An uncertainty factor of 1,000 was used to derive the RfD.

REFERENCES

DAVIES, J.E., DEDHIA, H., MORGADE, C., et al. 1983. Lindane poisonings. Arch. Dermatol. 119:142-144

DESI, I., VARGA, L., and FARKAS, I. 1978. Studies on the immunosuppressive effect of organochlorine and organophosphoric pesticides in subacute experiments. J. Hyg. Epidemiol. Microbiol. Immunol (Praha) 22:115-122

DEWAN, A., GUPTA, S.K., JAIN, J.P., et al. 1980. Effect of lindane on antibody response to typhoid vaccine in weanling rats. J. Environ. Sci. Health. B15(3):395-402

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Lindane. Office of Emergency and Remedial Response. Washington, D.C. EPA 540/1-8-056. September 1984

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Drinking Water Criteria Document for Lindane. Final Draft. Environmental Criteria and Assessment Office. ECAO-CIN-402. March 1984

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by the Office of Health and Envrionmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

FITZHUGH, O.G., NELSON, A.A., and FRAWLEY, J.P. 1950. The chronic toxicities of technical benzene hexachloride and its alpha, beta, and gamma-isomers. J. Pharmacol. Exp. Ther. 100:59-66

MATSUOKA, L.M. 1981. Convulsions following application of gamma-benzene hexachloride (letter). J. Am. Acad. Dermatol. 5(1):98-99

MORGAN, D.P., ROBERTS, R.J., WALTER, A.W., et al. 1980. Anemia associated with exposure to lindane. Arch. Environ. HEalth 35:307-310

SAMUELS, A.J., and MILBY, T.H. 1971. Human exposure to lindane: Clinical, hematological, and biochemical effects. J. Occup. Med 13:147-151

SHIVANANDAPPA, T., and KRISHNAKUMARI, M.K. 1983. Hexachlorocyclohexane-induced testicular dysfunction in rats. Acta. Pharmacol. Toxicol. 52:12-17

THORPE, E., and WALKER, A.I.T. 1973. Toxicology of dieldrin (HEDD). II. Comparative long-term oral toxicity studies in mice with dieldrin, DDT, phenobarbitone, beta-HCH and gamma-HCH. Food Cosmet. Toxicol. 11:433-442

TILSON, H.A., SHAW, S., MCLAMB, and R.L. 1987. The effects of lindane, DDT and chlordane on avoidance responding and seizure activity. Toxicol. Appl. Pharmacol. 88(1):57-65

TURNER, J.C., and SHANKS, V. 1980. Absorption of some organochlorine compounds by the rat small intestine—*in vivo*. Bull. Environ. Contam. Toxicol. 24(5):652-655

VODOPICK, H. 1975. Erythropoietic hypoplasia after exposure to gamma-benzene hexachloride. JAMA 24:850-851

WOLFF, G., ROBERTS, D., MORRISSEY, R., et al. 1987. Tumorigenic responses to lindane in mice: Potentiation by a dominant mutation. Carcinogenesis 8:1889-1897

ZOECON CORPORATION. 1983. MRID No. 00128356. (As cited in EPA 1988b). Available from EPA

GROSS ALPHA

Decay of alpha-emitting radionuclides (e.g., uranium, plutonium, radium and thorium) may result in the emission of several different alpha particles, each with its own discrete energy. Alpha particles are identical to the helium nucleus consisting of two neutrons and two protons. Because of their double positive charge, alpha particles have great ionizing power but their large size results in very little penetrating power. Their range in tissues is measured in micrometers and they are unable to penetrate clothes, or the skin layer. However, once inside the body, alpha particles may deposit energy in a short distance to severely affect the region of impact. Alpha particles, charged and by virtue of their mass and motion, produce ionizations along their path as a result of impulses imparted to orbital electrons.

The cells which frequently undergo mitosis are the most radiosensitive to ionizing radiation from alpha particles and include hematopoietic stem cells, bone marrow cells, dividing cells in the intestinal glands, and germ cells (spermatocytes and oocytes). Depending on the size and distribution of the absorbed dose, the clinical manifestations of the acute radiation syndrome include hematopoietic depression, gastrointestinal denudation and central nervous system (CNS) disruption. Destruction of the gastrointestinal mucosa results in nausea, vomiting, diarrhea and may lead to ulceration and hemorrhage especially in the small intestine following severe exposures. Damage to the bone marrow cells is reflected by changes in the circulating blood (e.g., drastic fall in the number of lymphocytes). The germ cells of both males and females are radiosensitive. In males, sperm counts can become depressed temporarily, but, exposure to high doses may result in permanent sterility in both males and females due to a lack of stem cells. Furthermore, ionizing radiation produces gene mutations and chromosome aberrations in both somatic and germ cells. Chromosomal aberrations in the lymphocytes of human blood is the most sensitive indicator of irradiation. The most important late somatic effect of low-dose ionizing radiation is the induction of cancer.

REFERENCES

HOBBS, C.H. AND R.O. MCCLELLAN. 1986. Toxic effects of Radiation and Radioactive Materials. In Casarett and Doull's Toxicology the Basic Science of Poisons. 3rd Ed. Klaassen, C.D, Amdur, M.O., and Doull J. eds. pp. 669-705.

GROSS BETA

Beta emission is the most frequent decay process of radionuclides. Beta particles (B-) are electrons resulting from the conversion of a neutron to a proton in the nucleus of an atom while the positron (B+) is a particle with the same mass as an electron but possesses a single positive charge. Beta particles are not emitted with discrete energy levels, but rather exhibit a continuous spectrum of energy levels. Beta particles possess the energy to produce ionizations along their path as a result of impulses imparted to orbital electrons. Beta particles have a greater range and penetrating power but much less ionizing power than alpha particles.

The cells most affected by ionizing radiation include rapidly dividing cells such as hematopoietic stem cells, bone marrow cells, dividing cells in the intestinal glands, and germ cells (spermatocytes and oocytes). Exposure of the lens of the eye to beta particles may cause cataracts. The early effects of exposure to ionizing radiation result primarily from cell death. Depending on the size and distribution of the absorbed dose, the clinical manifestations of the acute radiation syndrome include bone marrow suppression (drastic fall in the number of lymphocytes), gastrointestinal effects (nausea, vomiting, anorexia, diarrhea and in more severe exposures ulceration and hemorrhage), and central nervous system disruption. The severity of bone marrow depression and the latent period between exposure and appearance of the symptoms are related to the magnitude of dose. The germ cells of both males and females are radiosensitive. In males, acute doses of 10 to 100 rads (unit of radiation-absorbed dose) will cause a dose-related depression of the sperm count, which recovers slowly. With doses in the range of 500 rads, permanent sterility is likely. In females, destruction of oocytes results in permanent sterility due to a lack of stem cells in the adult ovary. Also, destruction of germinal epithelium of the ovary involves interruption of the production of the sex hormones. Tissues tolerate larger total doses of ionizing radiation when the dose is fractionated. Ionizing radiation produces gene mutations and chromosome aberrations in both somatic and germ cells. Chromosomal aberrations in the lymphocytes of human blood is the most sensitive indicator of irradiation. Chronic, low-level exposure to ionizing radiation can result in cancer induction.

REFERENCES

HOBBS, C.H. AND R.O. MCCLELLAN. 1986. Toxic effects of Radiation andRadioactive Materials. In Casarett and Doull's Toxicology the Basic Science of Poisons. 3rd Ed. Klaassen, C.D, Amdur, M.O., and Doull J. eds. pp. 669-705.

HEPTACHLOR/HEPTACHLOR EPOXIDE

Heptachlor epoxide is a contaminant and metabolite of the insecticide heptachlor. Heptachlor is readily absorbed from the gastrointestinal tract following oral exposure (ATSDR 1987). Acute symptoms due to heptachlor exposure in humans include irritability, excessive salivation, labored respiration, muscle tremors, and convulsions (EPA 1987). Acute exposure of animals to heptachlor and heptachlor epoxide produced tremors, convultions, paralysis, and hypothermia (EPA 1985). Chronic exposure of experimental animals to dietary concentrations of heptachlor or heptachlor epoxide has been associated with increased liver weight and hepatocellular carcinoma; heptachlor also induced hepatic lesions (EPA 1987, Velsicol Chemical Corp 1955, Dow Chemical 1958, 1977, Davis 1965, Epstein 1976, Velsicol 1973). In the presence of metabolic activation, both heptachlor and heptachlor epoxide induced unscheduled DNA synthesis in transformed human fibroblasts (Ahmed et al. 1977). Hepataclor also increased the frequency of chromosomal aberrations in bone marrow cells of mice (Markarjan 1966). Results of studies with rodents also indicate that heptachlor epoxide induces reproductive and developmental effects (EPA 1987).

Heptachlor and heptachlor epoxide are classified as Group B2 agents (Probable Human Carcinogens) (EPA 1990). This classification applies to those agents for which there is sufficient evidence of carcinogenicity in animal studies and inadequate evidence of carcinogenicity in humans. Using the geometric mean of potency factors from four separate experiments in which mice exposed to dietary concentrations of heptachlor or heptachlor epoxide exhibited hepatocellular carcinomas (Davis 1965, NCI 1977, Velsicol 1973), EPA (1990) estimated an oral and inhalation cancer potency factor for heptachlor of 4.5 $(mg/kg\text{-}day)^{-1}$ and for heptachlor epoxide of 9.1 $(mg/kg\text{-}day)^{-1}$. Oral reference doses (RfDs), based on chronic systemic toxicity, have also been calculated for both heptachlor and heptachlor epoxide (EPA 1990). Male rats fed heptachlor in the diet for two years developed increases in liver weight at doses of 5 ppm (0.25 mg/kg-day) or greater (Velsicol Chemical Corporation 1955). The no-observed-effect-level (NOEL) was 3 ppm (0.15 mg/kg-day). Applying an uncertainty factor of 300 to the NOEL, an oral RfD for heptachlor of 5.0×10^{-4} mg/kg-day was estimated (EPA 1990). In another study (Dow Chemical Company 1955), beagle dogs of both sexes fed heptachlor epoxide in their diet for 60 weeks developed increased liver-to-body weight ratios. No NOEL was determined from this study, but a lowest-observed-effect-level (LOEL) of 0.5 ppm (0.0125 mg/kg-day) was identified from the available data. An oral RfD of 1.3×10^{-5} mg/kg-day for heptachlor epoxide was estimated from these data by applying an uncertainty factor of 1,000 to the LOEL (EPA 1990).

REFERENCES

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1987. Toxicological Profile for Heptachlor/Heptachlor Epoxide. Draft for Public Comment. U.S. Public Health Service. October 1987

AHMED, F.E., R.W. HART, J.J LEWIS. 1977. Pesticide induced DNA damage and its repair in cultured human cells. Mutat. Res. 42:116-174.

DAVIS, K. 1965. Pathology Report on mice fed aldrin, dieldrin, heptachlor and heptachlor epoxide for two years. Internal FDA memorandum to Dr. A.J. Lehman, July 19

DOW CHEMICAL COMPANY. 1955. 60-Week Feeding Study with Dogs. MRID No. 00061912 (Unpublished) (As cited in EPA 1989)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Drinking Water Criteria Document for Heptachlor, Heptachlor epoxide and Chlordane. Final Draft. March 1985. PB86-117991 EPA 600/X.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Health Effects Assessment for Heptachlor. Final Draft. Environmental Criteria and Assessment Office, Cincinnati, Ohio. ECAO-CIN H085

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

EPSTEIN, S.S. 1976. Carcinogenicity of heptachlor and chlordane. Sci Total Environ 6:103.

MARKARJAN, D.S. 1966. Cytogenetic effect of some chlororganic insecticides on the nuclei of mouse bone-marrow cells. Genetika 1:132-137.

NATIONAL CANCER INSTITUTE (NCI). 1977. Bioassay of Heptachlor for possible carcinogenicity. NCI Carcinogenesis Tech. Rep Ser No. 9

VELSICOL CHEMICAL CORPORATION. 1955. 2-Year Feeding Study with Rats. MRID No. 00062599 (Unpublished) (As cited in EPA 1989)

VELSICOL CHEMICAL CORPORATION. 1973. Available from EPA. Write to F01, EPA, Washington, DC (as cited in EPA 1989)

HEXACHLOROCYCLOHEXANE (α-, β-, AND δ-ISOMERS)

The α-, β-, and δ-isomers are three of the eight isomers of hexachloro-cyclohexane (HCH) and are constituents of technical-grade HCH, which is approximately 40-45% gamma, 20-22% delta, 18-22% alpha, 4% beta, 1% epsilon and inerts, and 10% heptachlorocyclohexane by weight (Hooker Chemical Corporation 1969, IARC 1979). Human and animal data indicate that the α-, β-, and δ-isomers are absorbed following oral and inhalation exposure (Baumann et al. 1980, Czegledi-Janko and Avar 1970, Kashyap 1986, Nigam et al. 1986, Saxena et al. 1980, Saxena et al. 1981a, Saxena et al. 1981b, Albro and Thomas 1974). Absorption following ingestion has been reported to be greater than 90% for these isomers (Albro and Thomas 1974). In animals, acute oral exposure to β-HCH has caused renal effects (Srinivasan et al. 1984) while subchronic oral exposure to β-HCH has caused hematological, neurological, and reproductive effects and death (Van Velsen et al. 1986, Muller et al. 1981). Cardiovascular, immunological, and neurological effects have been observed in workers occupationally exposed to technical-grade HCH (Kashyap 1986). In animals, long-term oral exposure to α-, β-, and technical-grade HCH has resulted in hepatic and renal effects (Ito et al. 1973, Ito et al. 1975, Fitzhugh et al. 1950; Van Velsen et al. 1986). Oral exposure to technical-grade HCH and α-HCH has been reported to induce dominant lethal mutations in mice and to produce mitotic disturbances in rats, respectively (Lakkad et al. 1982, Hitachi et al. 1975). Hepatocellular carcinomas have been observed in mice and rats orally exposed to α- and β-HCH (Ito et al. 1973, Ito et al. 1975, Thorpe and Walker 1973).

EPA (1990) has classified α-HCH as a Group B2--Probable Human Carcinogen, β-HCH as a Group C--Possible Human Carcinogen, and δ-HCH as not classifiable as to human carcinogenicity. An oral cancer potency factor of 6.3 $(mg/kg\text{-}day)^{-1}$ was derived for α-HCH based on an increased incidence of liver tumors in mice exposed to α-HCH in the diet for 24 weeks (Ito et al. 1973, EPA 1990). An oral cancer potency factor of 1.8 $(mg/kg\text{-}day)^{-1}$ was derived for β-HCH based on increases in benign liver tumors in mice fed β-HCH in the diet (Thorpe and Walker 1973, EPA 1990). The inhalation cancer potency factors for these isomers have the same values as those for the oral route (6.3 and 1.8 for α- and β-HCH, respectively) and are based on the oral data (EPA 1990).

REFERENCES

ALBRO, P.W. and THOMAS, R. 1974. Intestinal absorption of hexachlorobenzene and hexa chlorocyclohexane isomers in rats. Bull Environ Contam Toxicol 12:378-384

BAUMANN, K., ANGERER, J., HEINRICH, R., LEHNERT, G. 1980. Occupational exposure to hexachlorocyclohexane. I. Body burden of HCH-isomers. Int Arch Occup Health 47:119-127

CZEGLEDI-JANKO, G. and AVAR, P. 1970. Occupational exposure to lindane: Clinical and laboratory findings. Br J Indust Med 27:283-286

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

FITZHUGH, O.G., NELSON, A.A., FRAWLEY, J.P. 1950. The chronic toxicities of technical benzene hexachloride and its alpha, beta and gamma isomers. J Pharmacol Exp Ther 100:59-66

HITACHI, M., YAMADA, K., TAKAYAMA, S. 1975. Brief communication: Cytologic changes induced in rat liver cells by short-term exposure to chemical substances. J Natl Cancer Inst 54:1245

HOOKER CHEMICAL CORPORATION. 1969. FBHC, Fortified Benzene Hexachloride, Bulletin No. 480, Industrial Chemicals Division, Niagara Falls, NY, pp. 1-4

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1979. IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans. Chemicals, industrial processes and industries associated wiht cancer. IARC Monogr Eval Carcinog Risk Chem Hum 24:133-135

ITO, N., NAGASAKI, H., ARAI, M., SUGIHARA, S., MAKIURA, S. 1973. Histologic and ultrastructural studies on the hepatocarcinogenicity of benzene hexachloride in mice. JNCI 51:817-826

ITO, N., NAGASAKI, H., AOE, H., SUGIHARA, S., MIYATA, Y., ARAI, M., SHIRAI, T. 1975. Development of hepatocellular carcinomas in rats treated with benzene hexachloride. JNCI 54:801-805

LAKKAD, B.C., NIGAM, S.K., KARNIK, A.B., THAKORE, K.N., ARAVINDA BABU, K., BHATT, D.K., KASHYAP, S.K. 1982. DominaNt-lethal study of technical-grade hexachlorocyclohexane in Swiss mice. Mutat Res 101:315-320

KASHYAP, S.K. 1986. Health surveillance and biological monitoring of pesticide formulators in India. Toxicol Lett 33:107-114

MULLER, D., KLEPEL, H., MACHOLZ, R.M., LEWERENZ, H.-J., ENGST, R. 1981. Electroneurophysiological studies on neurotoxic effects of hexachlorocyclohexane isomers and gamma-pentachlorocyclohexane. Bull Environ Contam Toxicol 27:704-706

NIGAM, S.K., KARNIK, A.B., MAJUMDER, S.K., VISWESWARIAH, K., SURYANARAYANA RAJU, G., MUKTH BAI, K., LAKKAD, B.C., THAKORE, K.N., CHATTERJEE, B.B. 1986. Serum hexachlorocyclohexane residues in workers engaged at a HCH manufacturing plant. Int Arch Occup Environ Health 57:315-320

SAXENA, M.C., SIDDIQUI, M.K.J., BHARGAVA, A.K., SETH, T.D., KRISHNAMURTI, C.R., KUTTY, D. 1980. Role of chlorinated hydrocarbon pesticides in abortions and premature labour. Toxicology 17:323-331

SAXENA, M.C., SIDDIQUI, M.K.J., SETH T.D., KRISHNA MURTI, C.R. 1981a. Organochlorine pesticides in specimens from women undergoing spontaneous abortion, premature or full-term delivery. J Anal Toxicol 5:6-9

SAXENA, M.C., SIDDIQUI, M.K.J., BHARGAVA, A.K., KRISHNA MURTI, C.R., KUTTY, D. 1981b. Placental transfer of pesticides in humans. Atch Toxicol 48:127-134

SRINIVASAN, K., RAMESH, H.P., RADHAKRINAMURTY, R. 1984. Renal tubular dysfunction caused dietary hexachlorocyclohexane (HCH) isomers. J Environ Sci Health 19:453-466

THORPE, E. and WALKER, A.I.T. 1973. The toxicology of dieldrin (HEOD). II. Comparative long-term oral toxicity studies in mice with dieldrin, DDT, phenobarbitone, beta-BHC, and gamma-BHC. Food Cosmet Toxicol 11:433-442

VAN VELSEN, F.L., DANSE, L.H.J., VAN LEEUWEN, F.X.R., DORMANS, J.A.M.A., VAN LOGTEN, M.J. 1986. The subchronic oral toxicity of the beta-isomer of hexachlorocyclohexane in rats. Fund Appl Toxicol 6:697-712

HEXANEDIOIC ACID, DIOCTYL ESTER

No toxicological information was available for hexanedioic acid, dioctyl ester, however, limited information was found for hexanedioic acid, a hydrolysis product under strong acidic conditions such as the stomach. Hexanedioc acid, also known as adipic acid, is generally recognized as safe for use as a sythetic flavoring agent (Guest et al. 1981). Hexanedioic acid is readily absorbed and metabolized following dietary exposure (Guest et al. 1981); absorption via the respiratory tract and skin can be inferred by the toxicity associated with these routes of exposure. Inhalation and dermal exposure to hexanedioic acid produces severe irritation to the eyes, nose, throat and skin (RTEC 1987, Sax and Lewis 1989, Weiss 1986). This compound is slightly toxic following acute exposure as indicated by the rat oral LD_{50} of 3.6 gm/kg (RTECS 1987). In humans, inhalation of vapor irritates mucous membranes of the nose and lungs inducing coughing, sneezing and difficult breathing. Contact with liquid hexanedioic acid irritates the eyes and has a pronounced drying effect on the skin which may progress to dermatitis (Weiss 1986). Hexanedioic acid does not appear to be teratogenic in mice following oral exposure (Guest et al. 1981) or to be carcinogenic in humans (IARC 1985). No health-based criteria have been established by EPA.

REFERENCES

GUEST D., G.V. Katz, B.D. Astill. 1981. Aliphatic Carboxylic Acid. In Clayton and Clayton eds. Patty's Industrial Hygiene and Toxicology, Volume IIc, p. 4945.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC), 1985. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans-Tobacco Smoking, Volume 38, Views and expert opinions of an IARC Working Group on the Evaluation of the Carcinogenic Risk of Chemicals to Humans.

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1987. Vol. 3A. National Institute for Occupational Safety and Health (NIOSH). U.S. Department of Health and Human Services.

SAX, N.I. and LEWIS, R.J. 1989. Dangerous Properties of Industrial Materials, Seventh Edition, Volume II, Van Nostrand Reinhold, New York.

WEISS, G., 1986. Hazardous Chemicals Data Book. Second Edition, Noyes Data Corporation, Park Ridge, New Jersey, p. 56.

HMX, or octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine, is poorly absorbed when administered orally and intravenously to rats and mice due to its low aqueous solubility (EPA 1988). No data were found in the available literature regarding pulmonary or dermal absorption. Although no adverse effects were reported in workers who had been potentially exposed to HMX at a munitions plant, acute oral doses of HMX administered to rats and mice have resulted in histologic liver changes, and CNS effects including ataxia and hyperkinesia. Animals receiving higher doses experienced convulsions (EPA 1988). Subchronic oral administration of HDX to rats caused transient weight loss and blood changes (e.g. reduced hemoglobin, hematocrit, and red blood cell counts) in all treated animals (DOD, 1985). At higher HDX levels, males exhibited liver necrosis and enlarged centrilobular cells while tubular kidney changes such as focal atrophy and dilation were seen in treated female rats (DOD 1985). These results suggest a sex difference in target organs of rats to HMX (DOD 1985). Microbial genetic toxicology assays suggest that HMX is not mutagenic, although only low concentrations of HMX were used in tests due to limited solubility (DOD 1977).

EPA (1990) has reported an oral reference dose (RfD) of $5x10^{-2}$ mg/kg-day based on a subchronic rat feeding study where administration of 150 mg/kg-day led to hepatic lesions (DOD 1985). The RfD was determined by applying an uncertainty factor of 1,000 to the no observed adverse effect level (NOAEL) of 50 mg/kg-day for males.

REFERENCES

DEPARTMENT OF DEFENSE (DOD). 1977. As cited in EPA 1990.

DEPARTMENT OF DEFENSE (DOD). 1985. AD-A171 601. Washington, D.C. As cited in EPA 1990.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Health Advisory on Octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine (HMX). Office of Drinking Water, Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, OH.

Gastrointestinal absorption of iron in humans ranges from 1% to 25% (EPA 1984). Absorption of iron following inhalation exposure has not been thoroughly studied. Iron is an essential element and is therefore nontoxic at doses necessary for maintaining normal health and nutrition (EPA 1984). However, overexposure to iron can cause adverse health effects. Gastrointestinal irritation is the primary health effect observed in humans following acute oral overexposure to iron. In humans, chronic oral overexposure to iron has been associated with gastrointestinal bleeding, metabolic acidosis, hepatic toxicity, hemosiderosis, and hemochromatosis (EPA 1984). Human fatalities have occurred following ingestion of iron at doses of 100 mg/kg-day (Venugopal and Luckey 1978). Chronic inhalation overexposure of humans to iron-containing dusts and fumes produces respiratory irritation and various pulmonary lesions (EPA 1984). There is limited evidence from studies with experimental animals that certain soluble iron salts are teratogenic. Certain iron compounds are also reported to be genotoxic (EPA 1984). Iron oxide enhances the carcinogenic action of various organic carcinogens (benzo[a]pyrene for example) and may act as a tumor promoter. Local sarcomas have been induced by subcutaneous injection of iron-dextran (EPA 1984).

The National Research Council of the National Academy of Sciences (NRC 1980) has suggested the recommended dietary allowances (RDAs) for iron of between 10 and 60 mg. Therefore, the maximum recommended daily intake of iron can be used as a conservative allowable intake for chronic exposure. No health based criteria have been derived by EPA.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Iron. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. ECAO-CIN-H054 (Final Draft)

NATIONAL RESEARCH COUNCIL (NRC). 1980. Drinking Water and Health. Safe Drinking Water Committee. National Academy Press, Washington, D.C. Vol. 3, 415 pp

VENUGOPAL, B. and LUCKEY, T.D. 1978. Metal Toxicity in Mammals. In Chemical Toxicity of Metals and Metalloids. Plenum Press, New York

ISOPROPYL METHYLPHOSPHONIC ACID (IMPA)

INTRODUCTION

Isopropyl Methylphosphonic acid (IMPA) is a breakdown product of diisopropyl methylphosphonate (DIMP) and sarin (agent GB) (Chemical Stockpile Disposal Program 1988). Hydrolysis products, such as IMPA are smaller, less complex molecules and are usually less potent than the parent agent (Chemical Stockpile Disposal Program 1988).

TOXICOKINETICS

The primary routes of potential exposure to the hydrolysis products, such as IMPA are oral and percutaneous absorption. Extensive dilutions of hydrolysis products usually occur under environmental conditions (Chemical Stockpile Disposal Program 1988). IMPA is not subject is enzymatic degradation in the rat. When IMPA was administered intraperitoneally, 40% and 85% were excreted in the rat urine after 48 and 72 hours, respectively (Hoskin 1956a,b). Mice exposed intravenously to sublethal doses of sarin had large concentrations of sarin and its metabolites (IMPA and MPA) in the brain, and important site for toxicity (Little et al. 1988). IMPA is the pharmacological inactive hydrolytic product of sarin. High concentrations of IMPA were also found in the kidneys indicating that this organ is the site of detoxification and excretion of sarin. Large quantities of free and bound IMPA were also found in the lung which suggested an important site for toxicity. Examination of the time course of sarin-induced motor hypoactivity and hypothermia revealed an immediate onset of action that lasted 24 hours, which suggested that only a small portion of phosphorylation in brain accounted for these pharmacological effects (Little et al. 1986).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

IMPA is not especially toxic since large doses are required to produce lethality in rodents. The oral LD_{50}'s are 7,650 and 6,070 mg/kg for male and female rats, respectively and 5,620 and 6,550 mg/kg for male and female mice (Mecler 1981). The eye irritation test revealed no signs of irritation in rabbit eyes. Application of IMPA to the intact and abraded skin of rabbits at doses of 2 mg/kg were mildly irritating but produced no systemic toxicity at a dose of 2 mg/kg. IMPA did not induce dermal sensitization in guinea pigs.

No evidence of toxicity followed administration of IMPA in the drinking water to rats at levels of 300, 1,000 and 3,000 ppm for 90 days. No changes in body weight, food intake, water intake, clinical chemistry or hematologic parameters were seen in treated rats compared to controls (Mecler 1981).

IMPA was not mutagenic to five strains of Salmonella indicator organisms in the Ames assay both with and without rat liver activation (Mecler 1981).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

No regulated standards or criteria have been promulgated for IMPA.

REFERENCES

CHEMICAL STOCKPILE DISPOSAL PROGRAM. 1988. Final Programmatic Environmental Impact Statement. Program Executive Officer--Program Manager for Chemical Demilitarization. Aberdeen Proving Ground MD.

HOSKIN, R.C.G., 1956a. Some observations concerning the biochemical inertness of methylphosphonic acid and isopropyl hydrogen methylphosphonate. Can J Biochem and Physiol. 34:743-745.

HOSKIN, R.C.G. 1956b. The enzymatic hydrolysis products of sarin. Can J Biochem and Physiol. 34:75-79.

LITTLE P.J., M.L. REYNOLDS, E.R. BOWMAN, B.R. MARTIN. 1986. Tissue disposition of [3H]sarin and its metabolites in mice. Toxicol Appl Pharmacol. 83(3):412-19.

LITTLE P.J., J.A. SCIMECA, B.R. MARTIN. 1988. Distribution of [3H]diisopropyl fluorophosphate, [3H]soman, [3H]sarin, and their metabolites in mouse brain. Drug Metab Dispos 16(4):515-520.

MECLER F.J. 1981. Mammalian Toxicological Evaluation of DIMP and DCPD (Phase 3--IMPA). Final Report. U.S. Army Medical Research and Development Command. Fort Detrick, MD. Contract Officer's Technical Representative: J.C. Dacre. Environmental Protection Research Division. U.S. Army Medical Bioengineering Research and Development Laboratory. Fort Detrick, MD.

Absorption of lead from the gastrointestinal tract of adult humans is estimated at 8%-45%. In children, absorption from non-paint sources ranges from 30% to 50% (Hammond and Beliles 1980, EPA 1986). There are other interpretations of the data (Duggan 1983) that suggest this may be as high as 70%. For adult humans, the overall absorption rate is 30%-50%, however essentially all of the particulate airborne lead deposited in the lower respiratory tract is absorbed. Lead is stored in the body in the kidney, liver, and bone (EPA 1984). The major adverse effects in humans caused by lead include alterations in the hematopoietic and nervous systems. The toxic effects are generally related to the concentration of this metal in blood. Blood concentration levels of over 80 μg/dl in children and over 100 μg/dl in sensitive adults can cause severe, irreversible brain damage, encephalopathy, and possible death. The Centers for Disease Control (CDC 1985) have used the value of 25 μg/dl as an acceptable level of blood lead. Recent information (EPA 1988), however, indicates that physiological and/or biochemical effects can occur even at lower levels. These include enzyme inhibition (16 μg/dl), elevated erythrocyte protoporphyrin (15 μg/dl), interference with Vitamin D metabolism, cognitive dysfunction in infants (10 to 15 μg/dl), electrophysiological dysfunction (6 μg/dl), and reduced childhood growth (4 μg/dl). Decreased fertility, fetotoxic effects, and skeletal malformations have been observed in experimental animals exposed to lead (EPA 1984). Chronic oral ingestion of certain lead salts (lead acetate, lead phosphate, lead subacetate) has been associated in experimental animals with increased renal tumors. Doses of lead that induced kidney tumors were high and were beyond the lethal dose in humans (EPA 1985).

EPA classified certain lead salts in Group B2 (Probable Human Carcinogen), although no cancer potency factor has been established (EPA 1990). This category applies to those agents for which there is sufficient evidence of carcinogenicity in animals and inadequate evidence of carcinogenicity in humans. EPA (1988) has recently proposed a maximum contaminant level goal (MCLG) of zero for lead. EPA (1990) has considered it inappropriate to develop a reference dose (RfD) for inorganic lead and lead compounds, since many of the health effects associated with lead intake (particularly changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development) occur essentially without a threshold. The new proposed MCLG is based on subtle effects of lead at low blood levels, the overall Agency goal of reducing lead exposures, and the probable carcinogenicity of lead at very high doses. Underlying this proposal was the assumption that blood lead levels in the range of 10-15 μg/dl are associated with serious effects. Additionally EPA noted that existing body burdens of lead were already in the range where adverse effects could result.

An alternative approach is also undergoing review by EPA to evaluate potential subchronic lead exposures to young children. This approach is based on a linear pharmacokinetic model used by EPA's Office of Air Quality Planning and Standards (OAQPS) for lead air quality standard setting (EPA 1989). The model, based on work by Harley and Kneip (1985), takes into account the uptake, retention and excretion of lead. It is referred to as the "Integrated Uptake/Biokinetic Model", and it estimates blood lead levels.

REFERENCES

CENTERS FOR DISEASE CONTROL (CDC). 1985. Preventing Lead Poisoning in Young Children. U.S. Department of Health and Human Services, Atlanta, Georgia

DUGGAN, M.J. 1983. The uptake and excretion of lead by young children. Arch. Env. Health 38147:246-247

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Lead. Environmental Criteria and Assessment Office, Cincinnati, Ohio. EPA 540/1-86-055

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. National primary drinking water regulations; synthetic organic chemicals, inorganic chemicals and microorganisms. Fed. Reg. 50:46937-47025 (November 13, 1985)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Air Quality Criteria for Lead. Office of Research and Development, Environmental Criteria and Assessment Office, Research Triangle Park, North Carolina. June 1986. EPA 600/8-83/023cF

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Drinking water regulations maximum contaminant levels goals and national primary drinking water regulations for lead and copper; proposed rule. Fed. Reg. 53:31516-31578 (August 18, 1988)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989. Review of the National Ambient Air Quality Standards for Lead: Exposure Analysis Methodology and Validation. Air Quality Management Division, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina

HAMMOND, P.B., and BELILES, R.P. 1980. Metals. In Doull, J., Klaassen, C.D., Amdur, M.O., eds. Casarett and Doull's Toxicology: The Basic Science of Poisons. 2nd ed. MacMillan Publishing Co., New York. Pp. 421-428

HARLEY, N.H., and KNEIP, T.H. 1985. An Integrated Metabolic Model for Lead in Humans of All Ages. Final report to U.S. Environmental Protection Agency. New York University School of Medicine, Department of Environmental Medicine. January 30, 1985. Contract No. B44899

LEWISITE

INTRODUCTION

Lewisite, also known as 2-chlorovinyl dichloroarsine, is a blistering agent. Lewisite blisters usually heal faster than blisters induced by mustard gas.

TOXICOKINETICS

Lewisite penetrates the skin readily because of its lipid solubility.

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

Lewisite is considered a lethal vesicant and a systemic poison when absorbed into the bloodstream (Chemical Stockpile Disposal Program 1988). Lethal exposures in humans can occur via inhalation, skin or eye contact or ingestion. The threshold for onset of severe systemic effects in humans is approximately 10 mg/kg to the skin (Chemical Stockpile Disposal Program 1988). Lewisite is lethal at about (LClo) 6 ppm for a 30 minute exposure and (LDlo) 38 μg/kg (ppb) for inhalation and dermal exposures, respectively (RTECS 1987). The medial lethal dosage is 1,200 to 1,500 mg-min/m^3 for humans while an exposure of 1,500 mg-min/m^3 produces severe and probable permanent corneal damage to the eyes (Army 1975).

Lewisite is capable of producing severe chemical burns upon direct contact with human tissues, especially the eyes, skin, and upper respiratory tract (Chemical Stockpile Disposal Program 1988, Goldman and Dacre 1989). However, when the humidity is high, lewisite hydrolyses so rapidly that it is difficult to maintain a concentration sufficient to blister bare skin. Lewisite interacts with cellular constituents to produce cell death in target tissues. In humans, concentrations of 0.05-0.10 mg/cm^2 induced erythema of the skin while larger concentrations such as 0.2 mg/cm^2 result in vesication or blistering (Ottinger et al. 1973). Gaseous lewisite at a concentration of 10 ppm for 15 minutes causes vesication, of 0.05 ppm for 15 minutes results in severe intoxication while inhalation of 0.5 ppm for 5 minutes is considered lethal. Lewisite vapor concentrations of 0.01 ppm can cause inflammation of the eyes and swelling of the eye lids after 15 minutes of exposure (Ottinger et al. 1973). At high doses it can cause permanent loss of eye sight if not removed immediately (Army 1975). A drop on the skin produces a immediate burning sensation which gradually intensifies. The pain associated with lewisite burns is initially an itching and stinging sensation that can persist and become intolerable if the contaminated area is extensive.

The liver, gall bladder, and bile duct are particularly vulnerable to lewisite exposure, although damage to the kidneys and urinary tract is also possible following dermal contact with large doses. As a systemic poison, lewisite also causes pulmonary edema, diarrhea, restlessness, weakness, and low blood pressure. Lewisite inhalation and ingestion severely damage the mucous membranes of the airways, mouth, stomach, and intestine (Chemical Stockpile Disposal Program 1988).

In a 13-week gavage study rats administered 2 mg/kg lewisite had decreased serum protein, creatinine, SGOT, and SGPT (males) and increased lymphocyte and platelet counts (females) (Sasser et al. 1989c). In addition, treatment-related lesions were detected in the forestomach of both sexes at 2 mg/kg and were characterized by necrosis of the stratified squamous epithelium accompanied by infiltration of neutrophils and macrophages, proliferation of neocapillaries, hemorrhage, edema and fibroblast proliferation. Mild acute inflammation of the stomach was also observed in some cases at 1 and 2 mg/kg. The estimated no-observable-effect level (NOEL) appears to be between 0.5 and 1 mg/kg.

Teratogenic properties and reprpductive toxicity of high-level lewisite exposure are suspected but have not been substantiated (Chemical Stockpile Disposal Program 1988). Maternal and fetal effects were investigated in pregnant rats and rabbits exposed to lewisite via gastric intubation (Hackett et al. 1987). In rats, the highest dose tested, 1.5 mg/kg did not induce toxic or teratogenic responses in maternal animals or their fetuses. In rabbits, maternal mortality ranged from 13% to 100% in the lewisite treatment groups and significant fetal effects, which were limited to signs of retarded development were observed only at dose levels that induced maternal mortality. Therefore, these results indicate that lewisite is not likely to be a mammalian teratogen.

In a two-generation reproductive study, solutions of lewisite were administered via intragastric intubation to rats prior to mating, during mating and after mating until the birth of their offspring (Sasser et al. 1989b). The dams continued to receive lewisite during lactation. At weaning, selected male and female offspring continued receiving lewisite during adolescence, mating and throughout gestation (Sasser et al. 1989b). Lewisite had no adverse effect on reproduction performance, fertility, or reproductive organ weights of male or female rats through two consecutive generations. No adverse effects were noted in offspring. In addition, lewisite did not cause gross or microscopic lesions in the male or female reproductive organs. The NOEL for reproductive effects in this study was greater than 0.6 mg/kg-day, the highest dose tested.

Recent in vitro mutagenicity studies in the Ames Salmonella/microsomal assay both with and without activation have obtained negative results for lewisite (Stewart et al. 1989). No mutagenic response was exhibited by any of the strains tested. All strains exhibited cytotoxicity at 1 µg/plate or higher; strain TA102 exhibited more cytotoxicity than the other strains. Another study, investigating the cytotoxic, clastogenic and mutagenic effects of lewisite on chinese hamster ovary cells found sister chromatid exchange (SCE) induction weakly positive, but not significantly greater than the control response (Jostes et al. 1989).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

No regulated standards or criteria have been promulgated for lewisite. However, for the purposes of this risk assessment Clement has derived an oral RfD using the subchronic oral data of Sasser et al. (1989c) in which histopathological alterations in the forestomach of rats were observed. In this study a no-observed-effect level (NOEL) was identified of between 0.5 and 1 mg/kg. Using the more conservative value of 0.5 mg/kg, adjusting for continuous exposure (5 days/7 days), and applying a safety factor of 1,000, (10 to account for animal to human extrapolation, 10 to account for subchronic to chronic duration, and 10 to protect sensitive members of the human population) an RfD of 3.6×10^{-4} mg/kg-day was calculated.

REFERENCES

ARMY. 1975. Military Chemistry and Chemical Compounds Field Manual. Department of the Army and Air Force, Washington, D.C. October 1975. FM 3-9, AFR 355-7.

CHEMICAL STOCKPILE DISPOSAL PROGRAM. 1988. FINAL PROGRAMMATIC ENVIRONMENTAL IMPACT STATEMENT. January. Program Executive Officer--Program Manager for Chemical Demilitarization. Aberdeen Proving Ground, MD

HACKETT, P.L., L.B. SASSER, R.L. ROMMEREIM, J.A. CUSHING, R.L. BUSCHBOM, D.R. KALKWARF. 1987. Teratology studies of lewisite and sulfur mustard agents: effects of lewisite in rats and rabbits. Pacific Northwest Laboratory. Richland WA. U.S. Army Medical Research and Development Command. Fort Detrick. MD.

GOLDMAN M. AND J.C. DACRE. 1989. Lewisite: Its chemistry, toxicology and biological effects. In Reviews of Environmental Contamination and Toxicology. Vol. 110:76-106.

ICF FEASIBILITY STUDY. Toxicity of contaminants suspected to be present at old "O" field. December 1987.

JOSTES, R.F. JR., L.B. SASSER, R.J. RAUSCH. 1989. Toxicology studies of lewisite and sulfur mustard agents: genetic toxicity of lewisite (L) in chinese hamster ovary cells. Pacific Northwest Laboratory. Richland WA. U.S. Army Medical Research and Development Command. Fort Detrick. MD.

OTTINGER R.S., J.L. BLUMENTHAL, D.F. DAL PORTO, G.I. GRUBER, M.J SANTY, C.C SHIH. 1973. Recommended methods of reduction, neutralization, recovery, or disposal of hazardous wastes. Volume VII. Propellants, explosives, chemical warfare. Report EPA-670/2-73-052-8. U.S. Environmental Protection Agency, Washington. DC (AD 419921).

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1987. Vol. 3A, National Institute for Occupational Safety and Health (NIOSH). U.S. Department of Health and Human Services.

SASSER L.B., J.A. CUSHING, D.R.KALKWARK, AND R.L. BUSCHBOM. 1989a. Toxicology studies on lewisite and sulfur mustard agents: modified dominant lethal study of sulfur mustard in rat. Pacific Northwest Laboratory. Richland WA. U.S. Army Medical Research and Development Command. Fort Detrick. MD.

SASSER L.B., J.A. CUSHING, D.R.KALKWARK, P.W. MELLICK, R.L. BUSCHBOM. 1989b. Toxicology studies on lewisite and sulfur mustard agents: two-generation reproduction study of lewisite in rats. Pacific Northwest Laboratory. Richland WA. U.S. Army Medical Research and Development Command. Fort Detrick. MD.

SASSER L.B., J.A. CUSHING, D.R.KALKWARK, P.W. MELLICK, R.L. BUSCHBOM. 1989c. Toxicology studies on lewisite and sulfur mustard agents: Subchronic toxicity study of lewisite in rats. Pacific Northwest Laboratory. Richland WA. U.S. Army Medical Research and Development Command. Fort Detrick. MD.

STERWART D.L., E.J. SASS, L.K. FRITZ, L.B. SASSER. 1989. Toxicology studies on lewisite and sulfur mustard agents. Mutageniticity of lewisite in the salmonella histidine reversion assay. Pacific Northwest Laboratory. Richland WA. U.S. Army Medical Research and Development Command. Fort Detrick. MD.

LEWISITE OXIDE (LO)

Lewisite oxide (LO) is also known as dichloro(2-chlorovinyl) arsine oxide (RTECS 1990). Lewisite oxide is a hydrolysis product of lewisite under conditions of environmental moisture (Rosenblatt et al. 1975). It has been suggested that the toxicity of lewisite and lewisite oxide are similar however, lewisite oxide appears to be more toxic as indicated by the rat oral LD_{50} of 5 mg/kg, compared to that for lewisite of 50 mg/kg (Rosenblatt et al. 1975). In addition, the oral LD_{50} for lewisite oxide in rabbits is 3 mg/kg, and in guinea pigs is 2 mg/kg (RTECS 1990). Little information is available on the toxicity of lewisite oxide in humans. Lewisite oxide is primarily a blistering agent causing severe blisters on the surface of the skin; however, studies indicate that exposure to this compound may also affect the respiratory tract and produce inflamation of the eyes (Rosenblatt et al 1975). In humans, lewisite oxide is a necrosant and it is assummed that the arsenical residue passes into the circulation, distributes to various organs, to elicit a general systemic poisoning, typical of arsenical compounds (Rosenblatt et al. 1975). Lewisite oxide interferes with the pyruvate oxidase system in the liver mitochondria, thus inhibiting the energy-generating capacity of the cell (Franke 1967, Peters et al. 1946). No health-based criteria have been derived for lewisite oxide by EPA. OSHA (1989) has recommended a 8-hour time-weighted-average (TWA) permissible exposure level (PEL) of 0.5 mg(AS)/m^3 for occupational exposure to lewisite oxide.

REFERENCES

FRANKE, S., 1967. Manual of Military Chemistry. Volume I-Chemistry of Chemical Warfare Agents. 114-133, 154-163. AD849866.

PETERS, R.A., H.M. SINCLAIR, AND R.H.S. THOMPSON. 1946. An analysis of the inhibition of pyruvate oxidation by arsenicals in relation to the enzyme theory of vesication. Biochem J 40:516-524.

OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION (OSHA). 1989. Federal Register vol. 54. No. 12. p. 2923.

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1990. National Institute for Occupational Safety and Health (NIOSH). U.S. Department of Health and Human Services.

ROSSENBLATT, D., T. MILLER, J.C. DACRE, I. MUUL, D.R. GOGLEY. 1975. Problem definitions on potential environmental pollutants II. physical, chemical, toxicological, and biological, properties of 16 substances. U.S. Army medical Bioengineering Research and Development Laboratory, Fort Detrick, Fredrick, MD.

MAGNESIUM

Magnesium is an essential nutrient which acts as a cofactor for many enzymes and plays an important role in neurological transmission and muscular excitability. Absorption of magnesium following oral exposures is dose-dependent in both humans and animals. At physiological doses of 3 to 10 milliequivalent (mEq), less than 10% appeared in the urine in 72 hours, while 59-88% was excreted in the feces in 120 hours (Stokinger 1981). An average adult in the United States ingests between 20 and 40 mEq of magnesium per day, one third of which is absorbed primarily in the small intestine. Normal magnesium plasma levels in humans range between 1.5 to 2.2 mEq per liter (Goodman and Gilman 1985). In humans, acute toxicity from inhalation of magnesium oxide fume results in metal fume fever (Sax 1984) and associated leukocytosis. Excessive magnesium can cause muscle weakness, hypotension, sedation, confusion, respiratory paralysis, coma and death in humans. Human exposure to particles of magnesium in subcutaneous tissue produce lesions that resist healing (Goyer 1986) and will cause skin and eye burns since it reacts with water to form caustic magnesium hydroxide (National Fire Protection Assoc. 1978). The oral lethal dose (LD_{50}) for dogs is 230 mg/kg. Dermal applications of magnesium powder to abraded surfaces of animals results in an inflammatory reaction (Stokinger 1981). Limited data are available on magnesium's carcinogenic potential. One study found intratracheal administration to hamsters produced olfactory tumors, and tumors of the lungs, thorax and respiratory system (RTECS 1987). No health-based criteria have been established by EPA.

REFERENCES

GOODMAN, L.S., AND GILMAN, A.G. EDS. 1985. The Pharmacological Basis of Therapeutics. 7th ed. MacMillan Publishing Co., New York. Pp. 874-878

GOYER, R.A. 1986. Toxic effects of Metals. In Doull, J., Klaassen, C.D., and Amdur, M.D., eds. Casarett and Doull, Toxicology. 3rd ed. MacMillan Publishing Co., New York. Pp. 624-625.

NATIONAL FIRE PROTECTION ASSOCIATION. 1978. Fire Protection Guide on Hazardous Materials. 7th ed. Boston, MA. Pp. 49-81

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1987. National Institute for Occupational Safety and Health (NIOSH). U.S. Government Printing Office, Washington, D.C.

SAX, N.I. 1984. Dangerous Properties of Industrial Materials. 6th ed. Van Nostrand Reinhold, New York. Pp 1720-1721

STOKINGER, H.E. 1981. The Metals. In Clayton, G., and Clayton, F., eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley Sons, New York. Vol. 2A. Pp. 1744-1749

MANGANESE

Manganese is absorbed at low levels following oral or inhalation exposure (EPA 1984a). The effects following acute exposure to manganese are unknown. Chronic oral and inhalation exposure of humans to high levels of manganese causes pneumonitis in exposed workers and has been associated with a condition known as manganism, a progressive neurological disease characterized by speech disturbances, tremors, and difficulties in walking (Kawamura et al. 1941). Altered hematologic parameters (hemoglobin concentrations, erythrocyte counts) have also been observed in individuals exposed chronically. Chronic oral exposure of rats to manganese chloride can result in central nervous system dysfunction (Leung et al. 1981, Lai et al. 1982). Manganese has not been reported to be teratogenic; however, this metal has been observed to cause depressed reproductive performance and reduced fertility in humans and experimental animals (EPA 1984a). Certain manganese compounds have been shown to be mutagenic in a variety of bacterial tests. Manganese chloride and potassium permanganate can cause chromosomal aberrations in mouse mammary carcinomal cells. Manganese was moderately effective in enhancing viral transformation of Syrian hamster embryo cells (EPA 1984a,b).

EPA (1990a) established an oral reference dose (RfD) of 1.0×10^{-1} mg/kg-day for manganese based on a no-observed-adverse-effects level (NOAEL) of 0.14 mg/kg-day in humans chronically exposed to manganese in food (WHO 1973, Schroeder et al. 1966, NRC 1989). An uncertainty factor of 1 was used to derive the reference dose. EPA (1990b) calculated an inhalation reference dose based upon an occupational study conducted by Saric et al. (1977) examining central nervous effects of manganese. Using a NOAEL of 2.1 mg/day and an uncertainty factor of 100, an inhalation RfD of 3.0×10^{-4} mg/kg-day was derived. Both the inhalation and oral values are based upon central nervous system effects (EPA 1990a,b).

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984a. Health Assessment Document for Manganese. Final Report. Environmental Criteria and Assessment Office, Environmental Protection Agency, Cincinnati, Ohio. August 1984. EPA 600/8-83-013F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984b. Health Effects Assessment for Manganese (and compounds). Environmental Criteria and Assessment Office, Washington, D.C. EPA 540/1-86-057

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Guidelines for carcinogen risk assessment. Fed. Reg. 51:33992-34003 (September 24, 1986)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washignton, D.C. July 1990

KAWAMURA, R., IKUTA, H., FUKUZUMI, S., et al. 1941. Intoxication by manganese in well water. Kitasato Arch. Exp. Med. 18:145-149

LAI, J.C.K., LEUNG, T.K.C., and LIM, L. 1982 Activities of the mitochondrial NAD-linked isocitric dehydrogenase in different regions of the rat brain. Changes in aging and the effect of chronic manganese chloride administration. Gerontology 28:81-85

LEUNG, T.K.C., LAI, J.C.K., and LIM, L. 1981. The regional distribution of monoamine oxidase activities towards different substrates: Effects in rat brain of chronic administration of manganese chloride and of aging. J. Neurochem. 36:2037-2043

NRC (NATIONAL RESEARCH COUNCIL). 1989. Recommended Dietary Allowances, 10th ed. Food and Nutrition Board, National Research Council, National Academy Press, Washington, D.C. 230-235.

SARIC, M., MARKICEVIC, S., and HRUSTIC, O. 1977. Occupational exposure to manganese. Br. J. Ind. Med. 34:114-118

SCHROEDER, H.A., BALASSA, D.D., AND TIPTON, I.H. 1966. Essential trace metals in man: Manganese, a study in homeostasis. J. Chron. Dis. 19:545-571.

WHO (WORLD HEALTH ORGANIZATION). 1973. Trace elements in human nutrition: Manganese. Report of a WHO Expert Committee. Technical Report Service, 532, WHO, Geneva, Switzerland. 34-36.

In humans, inorganic mercury is absorbed following inhalation and oral exposure, however only 7% to 15% of administered inorganic mercury is absorbed following oral exposure (EPA 1984, Rahola et al. 1971, Task Group on Metal Accumulation 1973). In humans, organic mercury is almost completely absorbed from the gastrointestinal tract and is assumed to be well absorbed via inhalation in humans (EPA 1984). A primary target organ for inorganic compounds is the kidney. Acute and chronic exposures of humans to inorganic mercury compounds have been associated with anuria, polyuria, proteinuria, and renal lesions (Hammond and Beliles 1980). Chronic occupational exposure of workers to elemental mercury vapors (0.1 to 0.2 mg/m^3) has been associated with mental disturbances, tremors, and gingivitis (EPA 1984). Animals exposed to inorganic mercury for 12 weeks have exhibited proteinuria, nephrotic syndrome and renal disease (Druet et al. 1978). Rats chronically administered inorganic mercury (as mercuric acetate) in their diet have exhibited decreased body weights and significantly increased kidney weights (Fitzhugh et al. 1950). The central nervous system is a major target for organic mercury compounds. Adverse effects in humans, resulting from subchronic and chronic oral exposures to organic mercury compounds, have included destruction of cortical cerebral neurons, damage to Purkinje cells, and lesions of the cerebellum. Clinical symptoms following exposure to organic mercury compounds have included paresthesia, loss of sensation in extremities, ataxia, and hearing and visual impairment (WHO 1976). Embryotoxic and teratogenic effects, including malformations of the skeletal and genitourinary systems, have been observed in animals exposed orally to organic mercury (EPA 1984). Both organic and inorganic compounds are reported to be genotoxic in eukaryotic systems (Leonard et al. 1984).

EPA (1990) has reported an oral reference dose for both chronic and subchronic exposures of $3x10^{-4}$ mg/kg-day for inorganic mercury based on several oral and parenteral studies conducted in the Brown Norway rat studies which observed kidney effects (Fitzhugh et al. 1950, Druet et al. 1978, Bernaudin et al. 1981). An uncertainty factor of 1,000 was used to derive the RfDs. EPA (1990) has also derived an inhalation RfD for inorganic mercury of $3x10^{-4}$ mg/kg-day for both chronic and subchronic exposures based on several human occupational studies in which neurotoxicity was observed (Fawer et al. 1987, Piikivi and Tolonen 1989, Piikivi and Hanninen 1989, Piikivi 1989). An uncertainty factor of 30 was used to derive both inhalation RfDs.

REFERENCES

BERNAUDIN JF, DRUET E, DRUET P, AND MASSE R. 1981. Inhalation or ingestion of organic or inorganic mercurials produces auto-immune disease in rats. Clin. Immunol. Immunopathol. 20: 488-494.

DRUET, P., DRUET, E., POTDEVIN, F., and SAPIN, C. 1978. Immune type glomerulonephritis induced by $HgCl_2$ in the brown Norway rat. Ann. Immunol. 129C:777-792

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria Document for Mercury. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, Ohio for the Office of Water Regulation and Standards, Washington, D.C. EPA 440/5-80-058. NTIS PB 81-117699

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Mercury. Environmental Criteria and Assessment Office, Cincinnati, Ohio. EPA 540/1-86-042

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment. Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Resposne, Office of Emergency and Remedial Response, Washington, D.C. July 1990

FAWER RF, DERIBAUPIERRE B, GUILLEMIN MP, BERODE M, LOBE M. 1983. Measurement of hand tremor induced by industrial exposure to metalic mercury. J. Ind Med. 40: 204-208.

FITZHUGH, O.G., NELSON, A.A., LAUG, E.P., and KUNZE, F.M. 1950. Chronic oral toxicities of mercury-phenyl and mercuric salts. Arch. Ind. Hyg. Occup. Med 2:433-441

HAMMOND, P.B., and BELILES, R.P. 1980. Metals. In Doull, J., Klaassen, C.D., and Amdur, M.O., eds. Casarett and Doull's Toxicology: The Basic Science of Poisons. 2nd ed. Macmillan Publishing Co., New York. Pp. 421-428

LEONARD, A., GERBER, G.B., JACQUET, P., and LAUWERYS, R.R. 1984. Mutagenicity, carcinogenicity, and teratogenicity of industrially used metals. In Kirsch-Volders, M., ed. Mutagenicity, Carcinogenicity and Teratogenicity of Industrial Pollutants. Plenum Press, New York. Pp. 59-126

PIIKIVI L. 1989. Cardiovascular reflexes and low long-term exposure to mercury vapor. Int Arch Occup Environ Health 61:391-395.

PIIKIVI L, HANNINEN H. 1989. Subjective symptoms and psychlogical performance of chlorine-alkali workers. Scand J Work Environ Health. 15:69-74.

PIIKIVI L, TOLONEN V. 1989. EEG findings in chlro-alkali workers subjected to low long-term exposure to mercury vapor. Br J. Ind MEd. 46:370-375.

RAHOLA, T., HATTULA, T., KORLAINEN, A., AND MIETTINEN, J.K. 1971. The biological halftime of inorganic mercury (Hg^{2+}) in man. Scand. J. Clin. Invest. 27(suppl. 116):77 (Abstract)

TASK GROUP ON METAL ACCUMULATION. 1973. Accumulation of toxic metals with special reference to their absorption, excretion and biological halftimes. Environ. Phys. Biochem. 3:65-67

WORLD HEALTH ORGANIZATION (WHO). 1976. Environmental Health Criteria, Mercury. Geneva

METHOXYCHLOR

Methoxychlor is an organochlorine pesticide that is primarily used in agriculture as a replacement for DDT. Feeding studies in animals indicate that methoxychlor is absorbed through the gastrointestinal tract and is not particularly persistent in the body (EPA 1985). Symptoms of methoxychlor poisoning in experimental animals include central nervous system depression, gastrointestinal distress, and local irritation of the nose and throat (Lehman 1951, Smith et al. 1946). Prolonged oral exposure to methoxychlor in animals primarily results in loss of body weight and growth retardation (Lehman 1951, NCI 1978). Methoxychlor induced maternal toxicity as evidenced by an excessive loss of litters (abortions) in rabbits treated orally with methoxychlor during days 7 through 19 of gestation (Kincaid Enterprises 1986). Methoxychlor was not found to cause cancer in a number of studies in rodents, dogs, and swine (NCI 1978, EPA 1985).

EPA (1990) developed an oral RfD of $5x10^{-3}$ mg/kg-day for methoxychlor based on maternal toxicity (excessive litter loss) in a rabbit teratology study (Kincaid Enterprises 1986). An uncertainty factor of 1,000 was used to develop the RfD.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Drinking Water Criteria Document for Methoxychlor. Environmental Criteria and Assessment Office, Cincinnati, Ohio. EPA 600/X-84-191

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office. Cincinnati Ohio.

KINCAID ENTERPRISES, INC. 1986. MRID No. 0015992. Available from EPA. (as cited in EPA 1990)

LEHMAN, A.J. 1951. Chemicals in foods: A report to the Association of Food and Drug Officials on current development. Part II. Pest. Assoc. Food Drug Off. 15:123-133

LEHMAN, A.J. 1965. Summaries of Pesticide Toxicity. FDA unpublished study.

NATIONAL CANCER INSTITUTE (NCI). 1978. Bioassay of Methoxychlor for Possible Carcinogenicity. NCI-CG-TR-35

SMITH, M.I., BAUER, H., STOHLMAN, E.F., and LILLIE, R.D. 1946. The pharmacologic action of certain analogues and derivatives of DDT. J. Pharmacol. Exper. Ther. 88:359-365

2-METHYL PHENOL (o-CRESOL)

Experimental evidence indicates that 2-methyl phenol is absorbed following ingestion, inhalation (EPA 1984), and dermal exposure (ACGIH 1986). Humans exposed to 2-methyl phenol for an unspecified time developed nasopharyngeal irritation (Uzhdavini et al. 1972). Effects following acute exposure to 2-methyl phenol include muscular weakness; gastroenteric disturbances; severe depression; edema of the lungs; injury to the eyes, skin, kidneys, liver, pancreas, spleen, and vascular system; collapse; and death (Deichmann and Keplinger 1981, NIOSH 1978). Effects in rats following subchronic exposure to 2-methyl phenol include increased mortality; reduction in body weight; increased kidney-to-body weight ratios; and CNS effects such as salivation, rapid respiration, lethargy, ataxia, coma, dyspnea, tremor, and convulsions (EPA 1987); hematopoietic effects; and sclerosis of the lungs (Uzhdavini et al. 1972). Lysol, a cresol-containing solution, produces extensive hemolysis, erosion of blood vessels, kidney tubular damage, liver necrosis, and death in humans following intravaginal application to induce abortion (Vance 1945, Presley and Brown 1956).

EPA (1990a) derived an oral reference dose of 0.05 mg/kg-day based on decreased body weights and neurotoxicity in rats administered 2-methyl phenol by gavage (EPA 1987). A safety factor of 1,000 was used to derive the RfD. EPA (1990b) derived a subchronic oral RfD of 0.5 mg/kg-day using a safety factor of 100, based on the same study and effect of concern.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. ACGIH, Cincinnati, Ohio

DEICHMANN, W.B., and KEPLINGER, M.L. 1981. Phenols and phenolic compounds. In Clayton, G., and Clayton, F., eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York. Vol. 2A, pp. 2597-2601

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Cresols. Environmental Criteria and Assessment Office, Cincinnati, Ohio, and Office of Emergency and Remedial Response, Washington, D.C. September 1984. EPA/540/1-86-050

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. o, m, p-Cresol. 90-Day Oral Subchronic Neurotoxicity Study in Rats. Office of Solid Waste, Washington, D.C. (As cited in EPA 1989)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1978. Criteria for a Recommended Standard--Occupational Exposure to Cresol. U.S. DHEW, PHS/CDC. U.S. Government Printing Office, Washington, D.C. February 1978

PRESLEY, J., and BROWN, W. 1956. Lysol-induced criminal abortion. Obstet. Gynecol. 8:368-370

UZHDAVINI, E., ASTAFYEVA, N., MAMYEVA, A., and BAKHTIZINA, G. 1972. [Inhalation toxicity of o-cresol.] Tr. Ufim Nauchno-Issled Inst. Gig. Profzabol. 7:115-119 (Russian)

VANCE, B. 1945. Intrauterine injection of Lysol as an abortifacient. Report of a fatal case complicated by oil embolism and Lysol poisoning. Arch. Pathol. 40:395-398

Experimental evidence indicates that 4-methyl phenol is absorbed following ingestion and inhalation (EPA 1984) and also after dermal exposure (NIOSH 1978). Effects following acute exposure to 4-methyl phenol include muscular weakness; gastroenteric disturbances; severe depression; edema of the lungs; injury to the eyes, skin, kidneys, liver, pancreas, spleen, and vascular system; collapse; and death (Deichmann and Keplinger 1981, NIOSH 1978). Effects in rats following subchronic exposure to 4-methyl phenol include increased mortality; reduction in body weight; increased kidney-to-body weight and liver-to-body weight ratios; and CNS effects such as salivation, rapid respiration, lethargy, ataxia, coma, dyspnea, tremors, diarrhea, and convulsions (EPA 1986, 1987). Lysol, a cresol-containing solution produces extensive hemolysis, erosion of blood vessels, kidney tubular damage, liver necrosis, and death in humans following intravaginal application to induce abortion (Vance 1945, Presley and Brown 1956).

EPA (1990) derived an oral reference dose of 0.05 mg/kg-day based on decreased body weights and neurotoxicity in rats administered 4-methyl phenol by gavage (EPA 1986, 1987). A safety factor of 1,000 was used to derive the RfD.

REFERENCES

DEICHMANN, W.B., and KEPLINGER, M.L. 1981. Phenols and phenolic compounds. In Clayton, G., and Clayton, F., eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York. Vol 2A, pp. 2597-2601

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Cresols. Environmental Criteria and Assessment Office, Cincinnati, Ohio, and Office of Emergency and Remedial Response, Washington, D.C. September 1984. EPA/540/1-86-050

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. o, m, p-Cresol. 90-Day Oral Subchronic Toxicity Studies in Rats. Office of Solid Waste, Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. o, m, p-Cresol. 90-Day Oral Subchronic Neurotoxicity Study in Rats. Office of Solid Waste, Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1978. Criteria for a Recommended Standard--Occupational Exposure to Cresol. U.S. DHEW, PHS/CDC. U.S.Government Printing Office, Washington, D.C. February 1978

PRESLEY, J., and BROWN, W. 1956. Lysol-induced criminal abortion. Obstet. Gynecol. 8:368-370

VANCE, B. 1945. Intrauterine injection of Lysol as an abortifacient. Report of a fatal case complicated by oil embolism and Lysol poisoning. Arch. Pathol. 40:395-398

METHYL PHOSPHONIC ACID

INTRODUCTION

Methylphosphonic acid (MPA) is a soluble, stable substance (Rosenblatt et al. 1975). Under environmental conditions MPA is a residue resulting from complete hydrolysis of the nerve agent sarin (GB) (Roach et al. 1987, Schott and Worthley 1974); however GB requires heat and strong acidic conditions to effect hydrolysis past the isopropyl methylphosphonate (IPMP) stage. MPA is also an hydrolysis product of VX (Chemical Stockpile Disposal Program 1988). MPA, an alkylphosphonic acid is an organophosphonate which has wide use as herbicides, insecticides, and anitbiotics (potent biocidal agents) (Roach et al. 1987).

TOXICOKINETICS

The primary routes of potential exposure to the hydrolysis products of chemical agents are oral and percutaneous absorption. Extensive dilutions of hydrolysis products has usually occurred (Chemical Stockpile Disposal Program 1988). Little is known about the absorption, distribution, metabolism and excretion of MPA. Ninety-two percent of the radiolabeled MPA intraperitoneally injected into rats was excreted in the urine within 48 hours (Hoskin 1956). Other toxicokinetic information is available from experiments with sarin (agent GB). MPA is a metabolite of sarin in mice exposed intraveneously to sublethal doses, and was distributed to areas of the brain in greater concentrations than sarin except the hypothalamus (Little et al. 1986, 1988). The higher concentrations of organophosphates (such as sarin) and their metabolites (IMPA and MPA) in the brain implicates that this area might be important with respect to the pharmacological effects or the toxicity of these compounds (Little et al. 1988). The major portion of radioactivity was free radiolabeled isopropyl methylphosphonic acid (IMPA), the pharmacological inactive hydrolytic product of sarin (Little et al. 1986). MPA is also presumably a metabolite of soman (Reynolds et al. 1985).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

MPA is reported to be nontoxic (presumably in reference to acute toxicity); no chronic toxicity data were found for MPA (Chemical Stockpile Disposal Program 1988). Fluoride is not a hazard except at relatively high doses; the beneficial effects of low levels on preventing tooth decay are well known. Chronic effects of isopropanol (rubbing alcohol) are relatively minor, and large amounts are required to produce acute toxic effects (Chemical Stockpile Disposal Program 1988).

There is a paucity of toxicological information on MPA. Preliminary pharmacological evaluation of newly synthesized derivatives of phosphonoamino acids in rats and mice demonstrated reduction of locomotor activity and anticonvulsant actions (Kleinrok et al. 1986).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

No regulated standards or criteria have been promulgated for MPA.

REFERENCES

CHEMICAL STOCKPILE DISPOSAL PROGRAM. 1988. Final Programmatic Environmental Impact Statement. Program Executive Officer–Program Manager for Chemical Demilitarization. Aberdeen Proving Ground MD.

HOSKIN, R.C.G. 1956. Some observations concerning the biochemical inertness of methyphosphonic and isopropyl hydrogen methylphosphonic acids. Can J Biochem and Physiol. 34:75-79.

KLEINROK Z., K. KOLASA, P. MASTALERZ, P. KAFARSKI. 1986. Preliminary pharmacological evaluation of newly synthesized derivatives of phosphonoamino acids. Pol J Pharmacol Pharm 38, Sept-Dec:435-442.

LITTLE P.J., M.L. REYNOLDS, E.R. BOWMAN, B.R. MARTIN. 1986. Tissue disposition of [3H]sarin and its metabolites in mice. Toxicol Appl Pharmacol. 83(3):412-19.

LITTLE P.J., J.A. SCIMECA, B.R. MARTIN. 1988. Distribution of [3H]diisopropyl fluorophosphate, [3H]soman, [3H]sarin, and their metabolites in mouse brain. Drug Metab Dispos 16(4):515-520.

ROACH M.C., L.W. UNGAR, R.N. ZARE, L.M. REIMER, D.L. POMPLIANO. 1987. Flourescence detection of alkylphosphonic acids using p-(9-anthroyloxy) phenacyl bromide. Army Research Office, Research Triangle Park, NC. Govt Reports Announcements and Index. Issue 18. NTIS/AD-A180 879/9.

REYNOLDS M.L., P.J. LITTLE, B.F. THOMAS, R.B. BAGLEY, B.R. MARTIN. 1985. Relationship between the biodisposition of [3H]soman and its pharmacological effects in mice. Toxicol Appl Pharmacol. 80(3):409-20.

ROSENBLATT, D.H, T.A. MILLER, J.A. DACRE, I. MUUL, D.T. COGLEY. 1975. Problem definition studies on potential environmental pollutants. II. physical, chemical, toxicological, and biological properties of 16 substances. U.S. Army Medical Bioengineering Research and Development Laboratory. Fort Detrick. Frederick, MD. Technical Report 7509.

SCOTT C.D., E.G., WORTHLEY. 1974. The toxicity of methylphosphonic acid to some aquatic species of plants. Edgewood arsenal technical memorandum. EB-TM-73011. Biomedical Laboratory. Edgewood Arsenal, Aberdeen Proving Grounds. MD. 21010.

METHYLENE CHLORIDE (Dichloromethane)

Methylene chloride is absorbed following oral and inhalation exposure. The amount of airborne methene chloride absorbed following inhalation exposure increases in direct proportion to its concentration in inspired air, the duration of exposure, and physical activity. Dermal absorption has not been accurately measured (EPA 1985a). Acute human exposure to methene chloride may result in irritation of eyes, skin, and respiratory tract; central nervous system depression; elevated carboxyhemoglobin levels; and circulatory disorders that may be fatal (EPA 1980). Chronic exposure of animals can produce renal and hepatic toxicity (NCA 1982). Methylene chloride is mutagenic for *Salmonella typhimurium* and produces mitotic recombination in yeast (EPA 1990a). Several inhalation studies conducted in animals provide clear evidence of methene chloride's carcinogenicity (NTP 1986). There is only suggestive evidence in experimental animals that hepatocellular carcinomas and neoplastic nodules arise from oral exposure (EPA 1985a,b).

EPA (1990a) classified methene chloride in Group B2--Probable Human Carcinogen. It has been concluded by EPA (1985b) that the induction of distant site tumors from inhalation exposure and the borderline significance for induction of tumors in a drinking water study are an adequate basis for concluding that methene chloride be considered a probable human carcinogen via ingestion as well as inhalation. EPA (1990a) derived an inhalation cancer potency factor of 1.4×10^{-2} $(mg/kg\text{-}day)^{-1}$ based on the results of a National Toxicology Program (NTP) inhalation bioassay conducted in rats and mice (NTP 1986). Mammary tumors were noted in rats, while lung and liver tumors were observed in mice. EPA (1990a) determined an oral cancer potency factor of 7.5×10^{-3} $(mg/kg\text{-}day)^{-1}$ based on the results of the NTP (1986) inhalation bioassay and on an ingestion bioassay conducted by the National Coffee Association (NCA 1983). In the NCA study, hepatocellular adenomas and/or carcinomas were observed in male mice. An oral reference dose (RfD) of 6×10^{-2} mg/kg-day has been developed for both chronic (EPA 1990a) and subchronic (1990b) exposures, based on a 2-year rat drinking water bioassay (NCA 1982) that identified no-observed-effect levels (NOELs) of 5.85 and 6.47 mg/kg-day for male and female rats, respectively. Liver toxicity was observed at doses of 52.58 and 58.32 mg/kg-day for males and females, respectively. An uncertainty factor of 100 was used to derive both the RfDs. EPA (1990b) has established an inhalation RfD of 3 mg/m^3 for both chronic and subchronic exposures based on a study by Nitschke et al. (1988) in which rats were exposed to 200 ppm (694.8 mg/m^3) for 2 years. This study identified a NOAEL for liver toxicity. A safety factor of 100 was used to derive both RfDs. The chronic inhalation RfD is currently undergoing verification by EPA (1990a).

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Halomethanes. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440.5-80-051

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Health Assessment Document for Dichloromethane. Office of Health and Environmental Assessment, Washington, D.C. February 1985. EPA/600/8-82004F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. Addendum to the Health Assessment Document for Dichloromethane. Office of Health and Environmental Assessment, Washington, D.C. September 1985. EPA/600/8-82-004F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment. Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

NATIONAL COFFEE ASSOCIATION (NCA). 1982. Twenty-four Month Chronic Toxicity and Oncogenicity Study of methylene Chloride in Rats. Final Report. Prepared by Hazelton Laboratories America, Inc., Vienna, Virginia. August 11, 1982

NATIONAL COFFEE ASSOCIATION (NCA). 1983. Twenty-fourth-Month Oncogenicity Study of methylene Chloride in Mice. Unpublished report prepared by Hazelton Laboratories, Inc., Vienna, Virginia

NATIONAL TOXICOLOGY PROGRAM (NTP). 1986. NTP Technical Report on the Toxicology and Carcinogenesis Studies of Dichloromethane in F344/N Rats and B6C3F1 Mice (Inhalation Studies). NTP TR306

NITSCHKE, K.D., BURED, J.D., BELL, T.J., et al. 1988. methylene Chloride: A 2 year inhalation toxicity and oncogenicity study in rats. Fund. Appl. Toxicology (in press) (as cited by EPA 1989b)

2-METHYLNAPHTHALENE

Little information is available on the health effects of 2-methylnaphthalene exposure. In humans, 2-methylnaphthalene is not a skin-irritant or photosensitizer (Gerarde 1960). In rats, oral administration of 5 ml/kg has been reported as lethal to all animals tested (Gerarde 1960, Sandmeyer 1981). 2-Methylnaphthalene is moderately toxic by ingestion and intraperitoneal routes; the oral rat LD_{50} is 1630 mg/kg (Sax and Lewis 1989). No health-based criteria have been derived by EPA.

REFERENCES

GERARDE, H.W. 1960. Toxicology and Biochemistry of Aromatic Hydrocarbons. Elsevier Publishing Co., London. (As cited in Sandmeyer 1981)

SANDMEYER, E.E. 1981. Aromatic hydrocarbons. In Clayton, G.D., and Clayton, F.E., eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York. Volume IIB. Pp. 3252-3431

SAX N.I., R.J. LEWIS. 1989. Dangerous Properties of Industrial Materials. Van Nostrand Reinhold, New York, 7th ed. Volume III. pgs. 2341-2.

MUSTARD (HD, H)

INTRODUCTION

Mustard is a blistering agent used extensively during World War I. It has a distinctive odor and a fairly long duration of effectiveness under normal weather conditions (Army 1975). Levinstein Mustard (H) contains about 30% impurities, which give it a pronounced odor; Distilled Mustard (HD), is a purified form with less odor and a slightly greater blistering power than H (Army 1975). The properties of H and HD are essentially the same, except sulfur impurities lessen its effectiveness (Army 1975). The toxicological effects are usually local at the point of agent contact with skin, eyes, or respiratory tract.

TOXICOKINETICS

Mustard penetrates the skin readily because of its lipid solubility. Wet skin is especially efficient in dermal absorption compared to dry skin. For this reason, HD exerts a casualty effect at lower concentrations in hot humid weather, since the body is then moist with perspiration (Army 1975). Mustard gas is also absorbed following inhalation exposure, as evidenced by its appearance in humans tissues (Drasch et al. 1987). Some of the vapors can be absorbed through the skin, with the majority being absorbed into the bloodstream (ATSDR 1990). The rate of mustard detoxification is very low, even small, repeated exposures are cumulative in their effects or more than cumulative due to sensitization (Army 1975). In rodents, mustard gas is hydrolyzed and binds with glutathione, since the major urinary metabolites are glutathione-conjugates (45%), thiodiglycol and conjugates (14%), and sulfone products (20%) (ATSDR 1990). Mustard gas is excreted primarily in the urine (ATSDR 1990).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

Mustard is not acutely lethal as the median lethal inhalation dosage (LCt_{50}) is 1,500 mg-min/m^3 for humans (estimated based on animal toxicity data), and the median incapacitating dosage (ICt_{50}) for dermal exposure is 2,000 mg-min/m^3 (Army 1975). The LCt_{50} dosage by inhalation is similar for a number of animal species (i.e., approximately 100 to 1,500 mg-min/m^3) (Chemical Stockpile Disposal Program 1988). Since the effects of HD are cumulative, the lethal dosage does not change significantly with exposure duration.

Mustard first acts as a cell irritant, and finally as a cell poison on tissue surfaces contacted (Army 1975). The physiological action of HD may be classified as local and systemic. The local action results in inflammation of the eyes and respiratory tract and erythema followed by blistering or ulceration. The eyes are very susceptible to low concentrations, and high mustard concentrations may cause permanent ocular damage (Army 1975). Mild exposures (20 to 70 mg-min/m^3) to HD vapor may produce lacrimation, and swelling, while more severe exposures (100 mg-min/m^3) can produce blepharospasm, blurring of vision, edema of the conjunctiva and eyelids, and mucous discharge (Chemical Stockpile Disposal Program 1988). The minimal effective dose for the development of mild to moderate erythema or rash is 50 mg-min/m^3. A latency period of several hours usually occurs before chemical burns and eye irritation become manifest (2-3 hours after exposure). In cases of severe exposure, the damage to the respiratory tract becomes evident; expectoration (phlegm discharge) is copious, rhinitis, laryngitis, tracheitis, and bronchitis are manifestations. Respiratory effects have been found in humans following both acute and chronic inhalation exposures to mustard (Somani and Babu 1989). Delayed effects after 2 years included chronic bronchitis and recurrent pneumonia, and deaths due to acute and chronic malignant respiratory disease, including influenza and pneumonia (Easton et al. 1988).

Mustard gas applied to the skin of rats produced local edema, and in rabbits, guinea pigs it produced vascular leakage, leukocyte infiltration (Vogt et al. 1984).

In the period of 4-16 hours following exposure, systemic effects of mustard become evident (Army 1975). Humans exposed dermally to mustard gas have experienced gastrointestinal effects manifested as nausea, vomiting, anorexia, abdominal pain, diarrhea, headache and lassitude (Sinclair 1948). Erythema, itching and blisters are the predominant dermal effects, and these reactions are usually delayed up to 48 hours (ATSDR 1990). Mustard at sufficiently high doses can also produce acute injury to the hematopoietic system (e.g. bone marrow, thymus, lymph nodes, and spleen) thereby causing a depression in circulating white cells, a consequent increased susceptibility to infection and possible subsequent bronchopneumonia which can be fatal (Army 1975, Chemical Stockpile Disposal Program 1988).

In a subchronic gavage study, rats were administered sulfur mustard (HD) at concentrations up to 0.3 mg/kg, 5 days/week for 13 weeks (Sasser et al. 1989c). A significant decrease ($p<0.05$) in body weight was observed in both sexes exposed to the highest dose tested (0.3 mg/kg). The only treatment-related histopathological lesion associated with gavage exposure was the appearance of epithelial hyperplasia of the forestomach of both sexes at 0.3 mg/kg (5/12 animals of each sex). The hyperplastic change was minimal and characterized by cellular disorganization of the basilar layer, an apparent increase in mitotic activity of the basilar epithelial cells, and thickening of the epithelial. Thus, a no-observed-effect level (NOEL) of 0.1 mg/kg was identified from this study.

There are epidemiological studies which suggest that humans exposed to mustard gas have a slight, but statistically significant ($p<0.01$) increase in the incidences of lung cancer deaths (Case and Lea 1955, Tokuoka et al. 1986, and Wada et al. 1968). In animal studies, inhalation exposure has also induced significantly higher incidences of pulmonary tumors in mice (Heston 1953).

No excess in fetal abnormalities were noted when rat dams were exposed during gestation (McNamara et al. 1975). However, this study was severely limited because it failed to report humidity, used whole-body exposures (combination inhalation and ingestion). Sulfur mustard (HD) was examined for teratogenic potential in rats and rabbits via intragastric intubation (Hackett et al. 1987). In rats, reductions in body weight were observed in maternal animals and their fetuses at the lowest administered dose (0.5 mg/kg), but the incidence of fetal malformations was not increased. In rabbits, the highest dose administered (0.8 mg/kg) induced maternal mortality and depressed body weight measures but did not affect fetal development. Thus, HD was not teratogenic in rats, and fetal effects were noted in rabbits only at doses which induced frank maternal toxicity.

Sasser et al. (1989a) examined the dominant lethal effect of HD in rats, and found significant male dominant lethal effects at 2 and 3 weeks post-exposure, which included fetal resorptions and implantation losses and decreases in total live embryo implants at 0.5 mg/kg. A significant increase in the percentage of abnormal sperm was also detected in males exposed to 0.5 mg/kg HD.

A variety of in vitro assays provide positive genotoxicity results (ATSDR 1990). Jostes et al. (1989) reported that micromolar amounts of HD are highly toxic, and induced chromosome aberration and SCE frequencies in a dose-dependent manner. Stewart et al. (1989) found that HD induced point mutations in Salmonella strain TA102 and frameshift mutations in TA97, but showed little or no mutagenicity against strains TA98 and TA100. HD induced cell killing with excision repair deficient strains (TA100, TA98, and TA97), but not with strain TA102. The in vitro data from both prokaryotic organisms (Salmonella typhimurium and Escherichia coli) and eukaryotic organisms (HeLa cells,

mouse lymphom, mouse L cells, rat lymphosarcoma) all support a mechanism of DNA alkylation leading to cross-link formation, inhibition of DNA synthesis and repair, point mutation, and chromosome and chromatid aberration formation. There are also data from Drosophila experiments in which sulfur mustard was injected into male flies, leading to sex-linked lethal mutations and point mutations at one of the loci affecting bristle formation (ATSDR 1990). All of these data are consistent with this agent being a powerful genotoxicant, which supports the recognized carcinogenicity of mustard gas.

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

No regulated standards or criteria have been promulgated for mustard. However, for the purposes of this risk assessment Clement has derived an oral RfD using the subchronic oral data of Sasser et al. (1989c) in which histopathological alterations in the forestomach of rat were observed. In this study a no-observed-effect level (NOEL) of 0.1 mg/kg was identified. Using this NOEL, adjusting for continuous exposure (5 days/7 days), and applying a safety factor of 1,000, (10 to account for animal to human extrapolation, 10 to account for subchronic to chronic duration, and 10 to protect sensitive members of the human population), an RfD of $7x10^{-5}$ mg/kg-day was calculated.

REFERENCES

ARMY. 1975. Military Chemistry and Chemical Compounds Field Manual. Department of the Army and Air Force, Washington, D.C. October 1975. FM 3-9, AFR 355-7.

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY. (ATSDR). 1990. Toxicological Profile for Mustard Gas. Prepared by Clement International. June 1990.

CASE R.A., A.J. LEA. 1955. Mustard gas poisoning, chronic bronchitis, and lung cancer: An investigation into the possibility that poisoning by mustard gas in the 1914-18 war might be a factor in the production of neoplasia. Br J Prev Soc Med. 9:62-72.

CHEMICAL STOCKPILE DISPOSAL PROGRAM. 1988. FINAL PROGRAMMATIC ENVIRONMENTAL IMPACT STATEMENT. January. Program Executive Officer–Program Manager for Chemical Demilitarization. Aberdeen Proving Ground, MD

DRASCH G., E. KRETSCHMER, M. PAHRM, ET AL. 1987. Concentrations of mustard gas bis-2-chloroethylsulfide in the tissue of a victim of a vesicant exposure. J Forensic Sci. 32:1788-1793.

EASTON D.F., J. PETO, R. DOLL. 1988. Cancers of the respiratory tract in mustard gas workers. Br J Ind Med. 45:652-659.

HACKETT, P.L., L.B. SASSER, R.L. ROMMEREIM, J.A. CUSHING, R.L. BUSCHBOM, D.R. KALKWARF. 1987. Teratology studies of lewisite and sulfur mustard agents: effects of lewisite in rats and rabbits. Pacific Northwest Laboratory. Richland WA. U.S. Army Medical Research and Development Command. Fort Detrick. MD.

HESTON W.E. 1953. Pulmonary tumors in Strain A mice exposed to mustard gas. Proceedings of the Society for Experimental Biology and Medicine. 82:457-460.

JOSTES, R.F. JR., L.B. SASSER, R.J. RAUSCH. 1989. Toxicology studies of lewisite and sulfur mustard agents: genetic toxicity of lewisite (L) in chinese hamster ovary cells. Pacific Northwest Laboratory. Richland WA. U.S. Army Medical Research and Development Command. Fort Detrick. MD.

MCNAMARA B.P., E.J. OWENS, M.K. CHRISTENSEN, ET AL. 1975. Toxicological basis for controlling levels of mustard in the environment. Edgewood Arsenal Special Publication. Aberdeen Proving Ground, Maryland: Department of the Army. EB-SP-74030.

SASSER L.B., J.A. CUSHING, D.R.KALKWARK, AND R.L. BUSCHBOM. 1989a. Toxicology studies on lewisite and sulfur mustard agents: modified dominant lethal study of sulfur mustard in rat. Pacific Northwest Laboratory. Richland WA. U.S. Army Medical Research and Development Command. Fort Detrick. MD.

SASSER L.B., J.A. CUSHING, D.R.KALKWARK, P.W. MELLICK, R.L. BUSCHBOM. 1989b. Toxicology studies on lewisite and sulfur mustard agents: two-generation reproduction study of lewisite in rats. Pacific Northwest Laboratory. Richland WA. U.S. Army Medical Research and Development Command. Fort Detrick. MD.

SASSER L.B., J.A. CUSHING, D.R.KALKWARK, P.W. MELLICK, R.L. BUSCHBOM. 1989c. Toxicology studies on lewisite and sulfur mustard agents: Subchronic toxicity study of lewisite in rats. Pacific Northwest Laboratory. Richland WA. U.S. Army Medical Research and Development Command. Fort Detrick. MD.

SINCLAIR D.C. 1948. The clinical features of mustard-gas poisoning in man. Br. Med. J. 290-294.

SOMANI S.M., S.R. BABU. 1989. Toxicodynamics of sulfur mustard. Int J Clin Pharmacol Ther Toxicol. 27:419-435.

STERWART D.L., E.J. SASS, L.K. FRITZ, L.B. SASSER. 1989. Toxicology studies on lewisite and sulfur mustard agents. Mutageniticity of lewisite in the salmonella histidine reversion assay. Pacific Northwest Laboratory. Richland WA. U.S. Army Medical Research and Development Command. Fort Detrick. MD.

TOKUOKA, S., Y. HAYASHI, K. INAI, ET AL. 1986. Early cancer and related lesions in the bronchial epithelium in former workers of a mustard gas factory. Acta Pathological Japonica. 36:533-542.

VOGT, R.F. JR., A.M. DANNENBERG JR., B.H. SCHOFIELD. 1984. Pathogenesis of skin lesions caused by sulfur mustard. Fund. Appl. Toxicol. 4:71-83.

WADA S., Y. NICHIMOTO, M. MIYANISH ET AL. 1962. Mustard gas as a cause of respiratory neoplasia in man. Lancet. 1:1161-1163.

NAPHTHALENE

Naphthalene is rapidly absorbed when inhaled but is more slowly absorbed by ingestion or through the skin (EPA 1982). Inhalation and oral exposure to naphthalene may cause nausea, headaches, vertigo, vomiting, abdominal pain, and liver and kidney damage in both humans and experimental animals (Linick 1983, Ojwang et al. 1985, Gidron and Leurer 1956, Gupta et al. 1979, Kurz 1987, Rao and Pandya 1981). Acute hemolytic anemia are reportedly caused by ingestion or inhalation of relatively large quantities of naphthalene (EPA 1982, Valaes et al. 1963). Optical neuritis, injuries to the cornea, and opacities of the lens also may result after inhalation exposure or ingestion (EPA 1982). Naphthalene is a mild eye irritant in rabbits, and cataracts can be induced after oral administration (EPA 1982). Application to the skin produces erythema and slight edema in rabbits (EPA 1982). Retarded cranial ossification and heart development are reported among offspring of rats injected intraperitoneally with naphthalene on gestational days 1 to 15 (EPA 1982). A significant reduction in the average number of live pups per litter was reported following a single oral dose of naphthalene (Plasterer et al. 1985). There are no epidemiologic or case studies available suggesting that naphthalene is carcinogenic in humans (EPA 1984). This compound is not generally considered to be carcinogenic in experimental animals (EPA 1984).

EPA (1990) developed an oral reference dose of $4x10^{-3}$ mg/kg-day for naphthalene based on the development of ocular and systemic lesions in rats (Schmahl 1955, EPA 1986) and occupational data on coke-oven workers. The oral RfD is currently under review. An uncertainty factor of 10,000 was applied to the animal data in the development of the reference dose.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1982. An Exposure and Risk Assessment for Benzo[a]pyrene and the Polycyclic Aromatic Hydrocarbons: Naphthalene. Final draft report. Office of Water Regulations and Standards. WH-553. Vol. II

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Naphthalene. Final draft. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. ECAO-CIN-HO14

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Health and Environmental Effects Profile for Naphthalene. Environmental Criteria and Assessment Office, Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington D.C. July 1990

GIDRON, E., and LEURER, J. 1956. Naphthalene poisoning. Lancet (February 4):228-230

GUPTA, R., SINGHAL, P.C., MUTHUSETHUPATHY, M.A., et al. 1979. Cerebral oedema and renal failure following naphthalene poisoning. J. Assoc. Physicians India 27:347-348

KURZ, J.M. 1987. Naphthalene poisoning: Critical care nursing techniques. Dimens. Crit. Care Nurs. 6:254-270

LINICK, M. 1983. Illness associated with exposure to naphthalene in mothballs—Indiana. MMWR 32:34-35

OJWANG P.J., AHMED-JUSHUF I.H., and ABDULLAH, M.S. 1985. Naphthalene poisoning following ingestion of moth balls: Case report. East Afr. Med. J. 62:72- 73

PLASTERER, M.R., BRADSHAW, W.S., BOOTH, G.M., CARTER, M.M., SCHULER, R.L., and HARDIN, B.D. 1985. Developmental toxicity of nine selected compounds following prenatal exposure in the mouse: Naphthalene, *p*-nitrophenol, sodium selenite, dimethyl phthalate, ethene thiourea, and four glycol derivatives. J. Tox. Environ. Health 15:25-38

RAO, G.S., and PANDYA, K.P. 1981. Biochemical changes induced by naphthalene after oral administration in albino rats. Toxicol. Lett. 8:311-315

SCHMAHL, D. 1955. Testing of naphthalene and anthracene as carcinogenic agents in the rat. Z. Krebsforsch. 60:697-710 (German with English translation)

VALAES, T., DOXIADIS S.A., and FESSAS, P. 1963. Acute hemolysis due to naphthalene inhalation. J. Pediatr. 63:904-915

Nickel compounds can be absorbed following inhalation, ingestion, or dermal exposure. The amount absorbed depends on the dose administered and the chemical and physical form of the particular nickel compound (EPA 1986). Dermal exposure of humans to nickel produces allergic contact dermatitis (EPA 1986). Adverse effects associated with acute exposure in animals have included depressed weight gain, altered hematological parameters, and increased iron deposition in blood, heart, liver, and testes (EPA 1987). Chronic or subchronic exposure of experimental animals to nickel has been associated with reduced weight gain, degenerative lesions of the male reproductive tract, asthma, nasal septal perforations, rhinitis, sinusitis, hyperglycemia, decreased prolactin levels, decreased iodine uptake, and vasoconstriction of the coronary vessels (EPA 1986). Teratogenic and fetotoxic effects have been observed in the offspring of exposed animals (EPA 1986). Inhalation exposure of experimental animals to nickel carbonyl or nickel subsulfide induces pulmonary tumors (EPA 1986). Several nickel salts cause localized tumors when administered by subcutaneous injection or implantation. Epidemiological evidence indicates that inhalation of nickel refinery dust and nickel subsulfide is associated with cancers of the nasal cavity, lung, larynx, kidney, and prostate (EPA 1986).

Nickel refinery dust and nickel subsulfide are both categorized as Group A carcinogens (Human Carcinogen) (EPA 1990a). These classifications are based on an increased incidence of lung and nasal tumors observed in workers occupationally exposed to nickel refinery dust (EPA 1986). These materials have inhalation cancer potency factors of 0.84 $(mg/kg\text{-}day)^{-1}$ and 1.7 $(mg/kg\text{-}day)^{-1}$, respectively (EPA 1990a). Nickel carbonyl is categorized in Group B2 (Probable Human Carcinogen); however, a potency factor has not been derived for nickel carbonyl (EPA 1990a). EPA derived an oral reference dose (RfD) for nickel of $2x10^{-2}$ mg/kg-day for both chronic (EPA 1990a) and subchronic (EPA 1990b) exposures based on a study by Ambrose et al. (1976) in which rats administered 5 mg/kg-day (NOAEL) nickel in the diet for 2 years did not experience decreased weight gain which was observed in animals administered 50 mg/kg-day (LOAEL). A safety factor of 300 was used to calculate the oral RfDs.

REFERENCES

AMBROSE, A.M., LARSON, P.S., BORZELLECA, J.R., and HENNIGAR, G.R. 1976. Long-term toxicologic assessment of nickel in dogs and rats. J. Food Sci. Technol. 13:181-187

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Health Assessment Document for Nickel and Nickel compounds. Office of Health and Environmental Assessment, Research Triangle Park, North Carolina. EPA 600/8-83-012FF

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Health Advisory for Nickel. Office for Drinking Water, Washington, D.C. March 31, 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment. Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Resposne, Office of Emergency and Remedial Response, Washington, D.C. July 1990

Both nitrate and nitrite are readily absorbed from the gastrointestinal tract after oral exposure (EPA 1987). The toxicity of nitrate in humans and experimental animals is due to its reduction to nitrite by bacteria in the stomach and subsequent reaction with stomach contents to form N-nitroso compounds (Parks et al. 1981). Nitrite reacts with hemoglobin in blood producing methemoglobin, thereby reducing the oxygen-carrying ability of red blood cells. Infants are particularly susceptible due to their high gut content of nitrate-reducing bacteria, and their lower enzymatic capacity to reduce methemoglobin to hemoglobin. Oral exposure to nitrite may result in acute toxic effects such as nausea, palpitations, numbness, and cyanosis due to methemoglobinemia in humans (EPA 1985, Walton 1951). Data concerning the chronic toxicity of nitrate in humans are limited, but there is some evidence that chronic use of drinking water high in nitrate may directly affect the central nervous system (CNS) (Petukhov and Ivanou 1970). Cardiovascular effects have also been observed in animals following acute oral exposure to high levels of nitrate (EPA 1987). In animals, toxicological effects following chronic ingestion of sodium nitrate include amyloidosis and hemosiderosis in the liver, kidney, spleen, and adrenals, and depletion of vitamins A and E (Chow et al. 1980, Fritsch et al. 1980, Inai et al. 1979). Chronic exposure to both nitrate and nitrite may lead to central nervous system toxicity in animals (EPA 1985). Nitrate and nitrite have not been associated with teratogenic effects in humans or laboratory animals and studies completed on livestock have not shown any relationship between nitrate and reproductive effects. However, developmental effects such as growth retardation and histological abnormalities of the liver, lungs, and spleens were observed in a three-generation study in rats following oral ingestion of nitrite (EPA 1985).

An oral reference dose (RfD) of 0.1 mg/kg-day for nitrite has been derived by EPA (1990). This value was based on an epidemiologic study in which the occurrence of methemoglobinemia was evaluated in infants that consumed formula prepared with nitrate-contaminated water (Walton 1951). The RfD was calculated for a 10-kg child drinking 1 liter of water/day, using a no-observed-effect-level (NOAEL) of 1.0 mg/kg-day and a modifying factor of 10 (EPA 1990).

REFERENCES

CHOW CK, CHEN CJ, GAIROLA C. 1980. Effect of nitrate and nitrite in drinking water on rats. Tox Lett. 6:199-206.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Final Draft for the Drinking Water Criteria Document on Nitrate/Nitrite. Prepared for the Criteria and Standard Division, Office of Drinking Water, Washington, D.C. PB86-117959

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Health Advisory: Nitrate/Nitrite. Office of Drinking WAter. Washington, D.C. (March 31, 1987)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

FRITSCH P, CANAL M, SAINT-BLANQUAT G, HOLLANDE E. 1980. Nutritional and toxicological impacts of nitrates and nitrites chronically administered (6 months) in rats. Ann Nutr Alim 34:1097-1114.

INAI K, AOKI Y, TOKUOKA S. 1979. Chronic toxicity of sodium nitrite in mice, with reference to ts tumorigenicity. Gann 70:203-208.

PARKS, N., KROHN, K., MATHIS, C., CHASKO, J., GEIGER, K., GREGOR, M., and PEEK, N. 1981. Nitrogen 13-labeled nitrite and nitrate: Distribution and metabolism after intratracheal administration. Science 212:58-61 (As cited in EPA 1987)

PETUKHOV NI, IVANOV AV. 1970. Investigation of certain psycho-physiological reactions in children suffering from methemoglobinemia due to nitrates in water. Hygiene and Sanitation. 35:29-31.

WALTON, G. 1951. Survey of literature relating to infant methemoglobinemia due to nitrate-contaminated water. Am. J. Public Health. 41:986-996

NITROBENZENE

Nitrobenzene is absorbed by all possible routes, but absorption primarily occurs through the respiratory tract and skin (EPA 1980); approximately 80% of inhaled nitrobenzene is absorbed (EPA 1980). In humans, long-term occupational exposure to nitrobenzene can result in cyanosis, methemoglobinemia, jaundice, anemia, sulfhemoglobinemia, and dark urine (EPA 1980). Short-term exposure to high levels of nitrobenzene can result in cyanosis, and if severe, the individual can go into a coma (Piotrowski 1967). Hematologic, adrenal, renal, and hepatic lesions have been reported in rats and mice exposed to nitrobenzene in air for 90 days (CIIT 1984). There is also limited evidence that exposure to nitrobenzene can result in changes in the tissues of the chorion and placenta in pregnant women (Dorigan and Hushon 1976); menstrual disturbances after chronic nitrobenzene exposure have also been reported (EPA 1980).

EPA (1990b) developed an inhalation RfD of $2x10^{-3}$ mg/m^3 for nitrobenzene based on a study in which hematological, adrenal, renal, and hepatic lesions were observed in mice following inhalation exposure to nitrobenzene (CIIT 1984); an uncertainty factor of 3,000 was used in the derivation. EPA (1989a) developed an oral RfD for nitrobenzene of $5x10^{-4}$ mg/kg-day based on the same CIIT (1984) study, using route-to-route extrapolation, the same endpoints of toxicity and an uncertainty factor of 10,000. EPA (1989a) is currently evaluating the carcinogenic potential of nitrobenzene.

REFEERENCES

CHEMICAL INDUSTRY INSTITUTE OF TOXICOLOGY (CIIT). 1984. Ninety-day Inhalation Study of Nitrobenzene in F344 Rats, CD Rats, and B6C3F1 Mice with cover letter dated 6/24/84 and EPA response dated 8/6/84. Unpublished study. FYI-OTS-0784-0333

DORIGAN, J., and HUSHON, J. 1976. Air Pollution Assessment of Nitrobenzene. U.S. Environmental Protection Agency, Washington, DC

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Nitrobenzene. Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-061

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

PIOTROWSKI, J. 1967. Further investigations on the evaluation of exposure to nitrobenzene. Br. J. Ind. Med. 24:60

NITROGEN AND COMPOUNDS

Nitrogen is an element that is commonly found as a nonflammable gas in the atmosphere (RTECS 1987). Because of its low boiling point, nitrogen has various commercial uses as a refrigerated liquid (RTECS 1987). However, contact with liquid nitrogen may cause frostbite (EPA 1990). Nitrogen has also been used as a miscellaneous additive in food (Hayes and Campbell 1986). Nitrogen can be absorbed following inhalation, or it can be obtained orally through one of many nitrogenous compounds. Toxicological information on nitrogen gas is limited due to its physiological inertness, however some nitrogenous compounds are highly injurious (ACGIH 1986, Amdur 1986). Nitrogen dioxide (NO_2) is an air pollutant which is oxidized from nitric oxide (NO) and is commonly found in cigarette smoke and in farmer's silos where ensilage is stored (Amdur 1986). Nitrogen dioxide is a deep lung irritant capable of producing pulmonary edema and emphysematous changes in the alveolar tissue of the lungs if inhaled in sufficient concentrations (Amdur 1986). Severe exposures to nitrogen dioxide can be fatal, causing death by asphyxiation (EPA 1990). Rats exposed for a lifetime to 15 ppm nitrogen dioxide had voluminous, dry lungs whose terminal bronchioles and alveolar ducts showed loss of cilia, epithelial hypertrophy, and narrowing (Amdur 1986). Limited evidence from animal studies suggest that exposure to nitrogen dioxide may also result in kidney, heart, and liver damage (EPA 1982). Inhalation exposure to nitrogen dioxide has not been found to be teratogenic, mutagenic, or oncogenic in experimental animals (EPA 1982). In water, nitrogen dioxide dissociates to form nitrates and nitrite, which results in the oxidation of hemoglobin to methemoglobin in humans (EPA 1990). ACGIH (1986) has recommended a time-weighted average threshold limit value of 3 ppm (6 mg/m^3) for nitrogen dioxide to protect against respiratory effects in individuals with bronchitis; however NIOSH recommends a ceiling value of 1 ppm nitrogen dioxide as a workplace environmental standard. No health-based criteria have been established by EPA.

REFERENCES

AMDUR, M.O. 1986. Air Pollutants. In Casarett and Doull's Toxicology. Klaassen C.D., Amdur M.O., and Doull J., eds. 3rd ed. Macmillan Publishing Co., New York. Pp 815-8.

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the threshold limit values and biological exposure indices. 5th ed. Cincinnati, OH, Pp. 435-436.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1982. Review of the National Ambient Air Quality Standards for Nitrogen Oxides: Assessment of Scientific and Technical Information. Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina. EPA 450/8-82-002.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Office of Health and Environmental Assessment, Cincinnati, Ohio.

HAYES, J.R. AND CAMPBELL, T.C. 1986. Food additives and contaminants. In Casarett and Doull's Toxicology. Klaassen C.D., Amdur M.O., and Doull J., eds. 3rd ed. Macmillan Publishing Co., New York. Pp 775-6.

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1987. Vol. 3A. National Institute for Occupational Safety and Health (NIOSH). U.S. Department of Health and Human Services.

4-NITROPHENOL

4-Nitrophenol, or para-nitrophenol is the most toxic mono-nitrophenol. Monophenols are readily absorbed by the gastrointestinal tract and lungs and are rapidly excreted, primarily in the urine (NRC 1981, Sittig 1985). In humans, ingestion may result in burning pain and white necrotic lesions in the mouth, esophagus and stomach. Other symptoms of oral exposure include: abdominal pain, vomiting, dizziness, tinnitus, shock, delirium, renal insufficiency, and cardiac effects such as heart blocks are arrhythmias. Convulsions are rare except in children. Although death from 4-nitrophenol is uncommon, it is usually due to cardiovascular collapse and renal dysfunction (Gosselin et al. 1984). Experimental studies in animals have resulted in methemoglobinemia, CNS depression, dyspnea, central and peripheral vagus stimulation (Beard and Noe 1981, NRC 1981), and hyperthermia (Sax 1984). This compound is reported to cause kidney and liver injury in animals (EPA 1980), and administration of 10 mg of 4-nitrophenol by gavage to anesthetized rats is reported to significantly increase respiratory volume. 4-Nitrophenol can inhibit chorine transport in red blood cells, suggesting a direct effect on cell membranes. Prolonged exposure in animals produced alterations of neurohumoral regulation and pathological changes manifested as colitis, enteritis, hepatitis, gastritis, hyperplasia of the spleen, and neuropathy (EPA 1980, NRC 1981). Dermal exposure results in pain followed by numbness, the skin becomes blanched, with formation of dry opaque eschar over the burn (Gosselin et al. 1984). 4-Nitrophenol is mutagenic in E. Coli (Kubinski 1981). Concentrations of 400 mg/kg administered to pregnant mice resulted in 18% maternal mortality; no toxic effects were observed in the offspring (Hardin 1987). No health-based criteria have been established by EPA.

REFERENCES

BEARD RR., NOE JT. 1981. Aromatic Nitro and Amino Compounds. Clayton and Clayton eds. in Patty's Industrial Hygiene and Toxicology. Vol 2C. Toxicology. 3rd ed. New York: John Wiley and Sons. p. 2473

ENVIRONMENTAL PROTECTION AGENCY. 1980. Ambient Water Quality Criteria for Nitrophenols. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, DC. October 1980. EPA 440/5-80-063.

GOSSELIN RE., SMITH RP., HODGE HC. 1984. Clinical Toxicology of Commercial Products. 5th ed. Baltimore: Williams and Wilkins, II-197.

HARDIN BD. et al. 1987. Teratog. Carcinog. Mutagen 7:29-48 (as cited in HSDB 1988).

HAZARDOUS SUBSTANCES DATA BANK (HSDB). 1988. National Library of Medicine, National Toxicology Informational Program. Bethesda MD.

NATIONAL RESEARCH COUNCIL. 1981. Drinking Water and Health, Washinghton DC. National Academy Press. Vol 4. p. 239-241.

SAX NI. 1984. Dangerous Properties of Industrial Materials. New York: Van Nostrand Reinhold. p. 972

SITTIG M. 1985. Handbook of Toxic and Hazardous Chemicals and Carcinogens. 2nd ed. Park Ridge, NJ. Noyes Data Corporation. p. 664.

1,4-OXATHIANE (1,4-THIOXANE)

INTRODUCTION

1,4-Thioxane, more commonly known as 1,4-oxathiane, is a sulfur vesicant-related hydrolysis product of mustard (D'Angostino et al. 1989). Oxathiane insecticides are monooxygen analogs of -dithianes, and showed the same level of insecticidal activity. Replacement of the sulfide link with oxygen however, reduced both mite and mammalian toxicity relative to the compounds with the sulfide link (Kurtz et al. 1987).

TOXICOKINETICS

The absorption, distribution, metabolism and excretion of 1,4-oxathiane are unknown at present.

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

There is a paucity of toxicological information on 1,4-oxathiane. 1,4-oxathiane is an ocular and dermal irritant in rabbits. It is of low toxicity as noted by the large oral LD_{50} for rats and guinea pigs of 2,830 and 3,960 mg/kg, respectively (RTECS 1990). The rat LC_{lo} for a 4-hour inhalation exposure was reported to be 4,000 ppm (RTECS 1990).

The mutagenic potential of 1,4-thioxane was assessed by using the Ames Salmonella/mammalian microsome mutagenicity assay (Sano and Korte 1985). Tester strains TA98, TA100, TA1535, TA1537, and TA1538 were exposed to doses 0.0016-5 muL/plate. The test compound was not mutagenic under conditions of this assay.

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

No regulated standards or criteria have been promulgated for 1,4-oxathiane.

REFERENCES

ANONYMOUS. 1986. Dermal, eye and oral toxicological evaluations. Phase II. Acute oral LD determinations of benzothiazole, dithiane, and oxathiane. Report; Iss Order No. AD-A172647/O/GAR, 71 pp.

D'AGOSTINO P.A., L.R. PROVOST, A.S. HANSEN, G.A. LUOMA. 1989. Identification of mustard related compounds in aqueous samples by gas chromatography/mass spectrometry. Biomed. Environ. Mass. Spectrum. 18(7):484-91.

KURTZ A.P., J.A. DURDEN JR., A.A. SOUSA, M.H. WEIDEN. 1987. Novel insecticidal oxathiolane and oxathiane oxime carbamates. J Agric. Food Chem. 35(1):106-14.

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1990. National Institute for Occupational Safety and Health (NIOSH). U.S. Department of Health and Human Services.

SANO S.K., D.W. KORT JR. 1985. Mutagenic potential of 1,4-thioxane. Report; ISS LAIR-206, Toxicololgy-Ser-94; Order No. AD-A165760/0/GAR. 26 pp.

PENTACHLOROPHENOL

Pentachlorophenol (PCP) is readily absorbed following oral and inhalation exposure; evidence from occupational studies indicates it is also absorbed following dermal exposure (EPA 1984). Acute exposure of humans to PCP can result in intoxication manifested as profuse sweating; fever; weight loss; gastrointestinal complaints; lung, eye, liver, and kidney damage; convulsions; heart failure; and even death (EPA 1987, EPA 1989). Acute intoxication in animals can result in coma and death (EPA 1987). Longer-term health effects observed in workers occupationally exposed to PCP include a higher incidence of low-grade infections or inflammations and depressed kidney function (EPA 1987). Subchronic health effects in laboratory animals administered PCP are an increase in liver and kidney weights (Johnson et al. 1973), a decrease in body weight (Deichmann et al. 1942, Goldstein et al. 1977), and the development of liver and splenic tumors (Kerkvliet et al. 1982a,b). Chronic health effects observed in rats include a reduced rate of body weight gain, increased SGPT enzyme activity, and pigmentation of the liver and kidneys (Schwetz et al. 1978). PCP produced fetotoxicity in rats (Larsen et al. 1975, Schwetz and Gehring 1973, Schwetz et al. 1974a,b, Schwetz et al. 1978) and hamsters (Hinkle 1973). Larsen et al. (1975) considered the fetotoxic effect of PCP exposure to be a reflection of maternal toxicity since PCP apparently does not cross the placental barrier (Larsen et al. 1975).

EPA (1990) reported an oral reference dose (RfD) for pentachlorophenol of 0.03 mg/kg-day based on liver and kidney pathology in rats administered PCP by gavage (Schwetz et al. 1978). A safety factor of 100 was used to derive to RfD.

REFERENCES

DEICHMANN, W., MACHLE, W., KITZMILLER, K., and THOMAS, G. 1942. Acute and chronic effects of pentachlorophenol and sodium pentachlorophenate upon experimental animals. J. Pharmacol. Exp. Therap. 76:104-117 (As cited in EPA 1984)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Pentachlorophenol. Environmental Criteria and Assessment Office, Cincinnati, OH and Office of Emergency and Remedial Response, Washington, D.C. September 1984. EPA/540/1-86-043

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Health Advisory for Pentachlorophenol. Office of Drinking Water. March 31, 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

GOLDSTEIN, J., FRIENSEN, M., LINDER, R., HICKMAN, P., HASS, J., and BERGMAN, H. 1977. Effects of pentachlorophenol on hepatic drug-metabolizing enzymes and porphyria related to contamination with chlorinated dibenzo-p-dioxins and dibenzofurans. Biochem. Pharmacol. 26:1549-155

HINKLE, D. 1973. Fetotoxic effects of pentachlorophenol on the Golden Syrian hamster. Toxicol. Appl. Pharmacol. 25:445

JOHNSON, R., GEHRING, P., KOCIBA, R., and SCHWETZ. B. 1973. Chlorinated dibenzodioxins and pentachlorophenol. Environ. Health Perspect., Exp. Issue No. 5, September, 1973. P. 171

KERKVLIET, N., BAECHER-STEPPAN, L., CLAYCOMB, A., CRAIG, A., and SHEGGEBY, G. 1982a. Immunotoxicity of technical pentachlorophenol (PCP-T): Depressed humoral immune responses to T-dependent and T-independent antigen stimulation in PCP-T-exposed mice. Fundam. Appl. Toxicol. 2(2):90-99. (CA 97: 176334d)

KERKVLIET, N., BAECHER-STEPPAN, L, and SCHMITZ, J. 1982b. Immunotoxicity of pentachlorophenol (PCP): Increased susceptibility to tumor growth in adult mice fed technical PCVP-contaminated diets. Toxicol. Appl. Pharmacol. 62(1):55-64

LARSEN, R., BORN, G., KESSLER, W., SHAW, S., and VANSICKLE, D. 1975. Placental transfer and teratology of pentachlorophenol in rats. Environ. Lett. 10:121-128

SCHWETZ, B. and GEHRING, P. 1973. The effect of tetrachlorophenol and pentachlorophenol on rat embryonal and fetal development. Toxicol. Appl. Pharmacol. 25:455

SCHWETZ, B., KEELER, P., and GEHRING, P. 1974a. The effect of purified and commercial grade pentachlorophenol on rat embryonal and fetal development. Toxicol. Appl. Pharmacol. 28:146-150

SCHWETZ, B., KEELER, P., and GEHRING, P. 1974b. Effect of purified and commercial grade pentachlorophenol on rat embryonal and fetal development. Toxicol. Appl. Pharmacol. 28:151-161

SCHWETZ, B., QUAST, J., KEELER, P., HUMISTON, C., and KOCIBA, R. 1978. Results of 2-years toxicity and reproduction studies on pentachlorophenol in rats. In: Pentachlorophenol: Chemistry, Pharmacology and Environmental Toxicology, K.R. Rao, Ed. Plenum Press, New York, New York. P. 301

Phenol is readily absorbed through the gut, by inhalation, and percutaneously (EPA 1980). Signs of acute phenol toxicity in humans and experimental animals are central nervous system depression, collapse, coma, cardiac arrest, and death. Acutely toxic doses can also cause extensive necrosis at the site of exposure (eyes, skin, oropharynx) (EPA 1980). In experimental animals subchronic oral and inhalation studies suggest that kidney, pulmonary, myocardial, and liver damage are associated with exposure, although many of these studies were poorly designed (EPA 1980, 1984). Oral administration of phenol to pregnant rats during gestational days 6 to 15 resulted in a significant reduction in fetal body weight (NTP 1983). Phenol exhibited tumor-promoting activity in the mouse skin painting system following initiation with 9,10-dimethyl-1,2-benzanthracene (DMBA) or benzo[a]pyrene (B[a]P), and it exhibited cutaneous carcinogenic activity in a sensitive mouse strain when applied at concentrations that produced repeated skin damage (EPA 1980).

EPA has established an oral chronic (EPA 1990a) and subchronic (EPA 1990b) reference dose (RfD) of 0.6 mg/kg-day for phenol based on reduced fetal body weight in rats (NTP 1983). A no-observed-adverse-effect level (NOAEL) of 60 mg/kg-day and a safety factor of 100 were used to derive both chronic and subchronic RfDs.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Phenol. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-066

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Phenol. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-007

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by the Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. October 1989

NATIONAL TOXICOLOGY PROGRAM (NTP). 1983. Teratologic evaluation of phenol in CD rats and mice. Report prepared by Research Triangle Institute, Research Triangle Park, NC. NTIS PB83-247726. Gov. Rep. Announce. Index 83(25):6247

PHOSGENE

INTRODUCTION

Phosgene (carbonyl chloride, carbon oxychloride, carbonic dichloride, chloroformyl chloride) is a highly toxic colorless gas at normal temperatures. It was used during World War I as a chemical warfare agent. Currently it is used industrially in the synthesis of isocyanates, carbonic acid esters, acid chlorides, dye intermediates, and pesticides and in the separation of ores (ACGIH 1986, Beard 1982).

TOXICOKINETICS

Phosgene is absorbed by inhalation and dermal exposure. In humans, phosgene, because its water solubility is only slight, is capable of reaching the alveolar region of the respiratory tract rather than being absorbed in the upper respiratory airways (Davies 1985). In contrast, the nasal airway structure of the rabbit allows absorption of phosgene in the nose (Davies 1985). Phosgene is rapidly hydrolyzed by water, producing carbon dioxide and hydrochloric acid, or can more rapidly acylate free amino, hydrazino, sulfhydryl, and hydroxy groups (Diller 1985). Acylation of tissue macromolecules (e.g., poly-L-lysine, human serum albumin) by phosgene has also been demonstrated (EPA 1986). It is thought that phosgene does not reach systemic circulation (EPA 1986). However, is has been reported that, in exposures to very high concentrations (200 ppm), the gas enters the lung capillaries; hemolysis occurs and capillary circulation is stopped, leading to death by acute overdistension of the right heart, "acute cor pulmonale" (Diller 1985).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

Most of the information on the health effects associated with exposure to phosgene are for the inhalation route. Phosgene, with its low water solubility, is capable of reaching the lower regions of the respiratory tract. The target of phosgene-induce toxicity is the lung. However, although it is known to be acutely toxic, producing respiratory effects (e.g., pulmonary edema) and sometimes, death, little is known about the health effects associated with chronic, low-level exposure to this gas (Cucinell 1974).

Based on the observations made during World War I, an LCT_{50} for humans of 3,200 mg/m^3 for a two-minute exposure has been estimated (Medical Division Status Summaries 1944). Concentration-effect relationships for exposure to phosgene have been reported as follows: perception of odor at >0.4 ppm, recognition of odor at >1.5 ppm, signs of irritation in eyes, nose, throat, and bronchi at >3 ppm, beginning lung damage at >30 ppm-min, clinical pulmonary edema at >150 ppm-min, LCT_1 of approximately 300 ppm-min, LCT_{50} of approximately 500 ppm-min, and LCT_{100} of approximately 1300 ppm-min (Diller 1985). The LCT_{50}s for mice, guinea pigs, monkeys and dogs have been reported to range from 1,000 mg/m^3 (monkeys) to 8,400 mg/m^3 (dogs) for one-minute exposure (Chasis 1944). In 30-minute exposures, the LCT_{50}s range from 1,000 mg/m^3 (monkeys) to 3,400 mg/m^3 (mice); the LCT_{50} for dogs for a 20-minute exposure is 4,200 mg/m^3.

Information on the human health effects of inhalation exposure to phosgene (e.g., pulmonary edema) comes primarily from case reports and epidemiological studies. The concentration and duration of exposure to phosgene are often not clearly quantified, as in cases involving exposure to phosgene as a thermal decomposition product of chlorinated hydrocarbons. In addition, the possibility of concurrent exposure to other toxic substances cannot be ruled out. The available human data indicate that the lung is the target of phosgene-induced toxicity. This is supported by the database of

animal studies, which are also primarily of acute and subchronic duration. Evidence of lung damage induced by phosgene in a number of species includes pulmonary edema, pneumonia, emphysema, bronchiolitis, bronchitis, and increased susceptibility to bacterial infection, leading to pneumonia and death.

A number of case reports indicate that respiratory effects are seen in humans following acute exposure to phosgene. Pulmonary edema, diffuse crepitant rales, and a depressed arterial oxygen tension were detected in a man who became ill after exposure to "excessive amounts" of phosgene (Cordasco and Stone 1973). Pulmonary edema was also detected in another man exposed to "concentrated gas" for 5 to 10 seconds (Bradley and Unger 1982). His symptoms included coughing, dyspnea, chest tightness, hypotension, tachycardia, tachypnea, cyanosis, and rales. This patient died when attempts to resuscitate him after after he developed ventricular fibrillation failed.

In addition to death, effects observed in animal studies in a number of species (e.g., rats, rabbits, dogs, cats, guinea pigs) include pulmonary edema, pneumonia, emphysema, bronchiolitis, and bronchitis (NIOSH 1976). Mongrel dogs repeatedly exposed to phosgene (24 to 40 ppm, 30 minutes per exposure, one to three times a week) developed acute bronchiolitis and peribronchiolitis involving terminal and respiratory bronchioles, chronic bronchitis, progressing to chronic obliterative bronchiolitis. The lungs of dogs exposed 30 or 40 times were most indicative of emphysema (Clay and Rossing 1964). Gross et al. (1965) exposed Wistar rats to 0.5 to 4 ppm phosgene for five minutes to eight hours and observed chronic pneumonitis. Exposure to a concentration of 3 ppm for five minutes was the lowest to produce a recognizable typical pulmonary lesion.

The information on chronic, low-level exposure is limited. Galdston et al. (1947) investigated the effects of repeated exposure in 5 workers. Although it is indicated that the workers were exposed repeatedly over a period of 1 1/2 and 3 1/2 years, no information was provided on the concentrations to which they had been exposed. In four out of five workers, the effects were concluded to be consistent with pulmonary emphysema; however, one of these four suffered from asthma from childhood and, therefore, the pulmonary effects can not be attributed to phosgene.

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

No regulated standards or criteria have been promulgated for phosgene. ACGIH (1986) has recommended at time-weighted-average threshold limit value (TWA-TLV) of 0.1 ppm (0.4 mg/m^3) for phosgene based in irritation of the respiratory tract.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the threshold limit values and biological exposure indices. 5th ed. Cincinnati, OH, Pp. 481.

BEARD R.R. 1982. Phosgene, $COCL_2$. In: Clayton, G.D., Clayton, F.E., eds. Patty's Industrial Hygiene and Toxicology: V. 2C, Toxicology. 3rd rev. ed. New York, NY: John Wiley and Sons; pp. 4126-4139.

BRADELY B.L AND UNGER K.M. 1982. Phosgene inhalation: a case report. Tex Med 78:51-53.

CHASIS H. 1944. Phosgene: Review of the literature on the effect of exposure in man and experimental animals. Contract W-49-036-CWS-1, unclassified report. (as cited in Cucinell 1974).

CLAY J.R., AND R.G. ROSSING. 1964. Histopathology of exposure to phosgene. Archives of Pathology. 78:544-551.

CORDASCO E.M. AND J.D. STONE. 1973. Pulmonary edema of environmental origin. Chest 64:182-185.

CUCINELL S.A. 1974. Review of the toxicity of long-term phosgene exposure. Arch Environ Health 28:272-275.

DAVIES C.N. 1985. Absorption of gases in the respiratory tract. Ann Occup Hyg. 29:13-25.

DILLER W.F. 1985. Pathogenesis of phosgene poisoning. Toxicol Ind Health. 1:7-15.

EPA. 1986. Health assessment document for phosgene. External review draft. Research Triangle Park, NC: US Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office. August 1986. EPA/600/8-86/022A.

GALDSTON M., J.A. LUETSCHER JR., W.T LONGCOPE, N.L. BALLICH. 1947. A study of the residual effects of phosgene poisoning in human subjects. II. After chronic exposure. Journal of Clinical Investigation. 26:169-181.

GROSS P, W.E, RINEHART, T, HATCH. 1965. Chronic pneumonitis caused by phosgene. Arch Environ Health 10:768-775.

MEDICAL DIVISION STATUS SUMMARIES. 1944. CWS Field Lab Memo 1-4-5, p. 305, unclassified report. (as cited in Cucinell 1974)

NIOSH. 1976. Occupational exposure to phosgene: Criteria for a recommended standard. National Institute for Occupational Safety and Health. DHEW (NIOSH) Publication No. 76-137.

PHOSPHORUS

INTRODUCTION

Phosphorus exists in several allotropic forms: white (or yellow), red, and black (Beliles 1981). White phosphorus is highly toxic while red and black phosphorus are essentially nontoxic. White phosphorus is a white to yellow soft, waxy solid with acrid fumes in air (NIOSH 1985). Red and black phosphorus are practically insoluble in water and stable except at high temperatures. Phosphorus is used in the manufacture of explosives, incendiaries, smoke bombs, chemicals, rodenticides, phosphor bronze and fertilizer (Beliles 1981).

TOXICOKINETICS

Phosphorus (white-yellow) can be absorbed through the skin, respiratory tract, and gastrointestinal tract (Beliles 1981). Experimental investigations in rats show the highest retention 5 days after oral administration in the liver, skeletal muscle, gastrointestinal tract, blood, and kidney. In the body, phosphorus is converted to phosphates. Urinary excretion, the chief mode of elimination, is largely as organic and inorganic phosphates (Beliles 1981).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

In humans, acute ingestion of white phosphorus affects the liver, kidney, hematopoietic system, brain, intestines, circulatory system, and the myocardium resulting in electrocardiographic changes (Davidson et al. 1987). Systemic and CNS effects have been noted in humans at oral doses of 16 mg/kg and 2,600 µg/kg, respectively and may be delayed for from a few hours to 3 days (Weiss 1986). Acute oral phosphorus intoxication generally has two stages. In the initial phase, gastrointestinal effects predominate and may include abdominal pain, nausea, vomiting, and belching. The second stage is characterized by the signs of hepatic, renal and cardiovascular dysfunction (jaundice, pitting edema, oliguria, high pulse rate, and low blood pressure). The most common pathological finding in deaths have been fatty degeneration of the liver and kidneys (Beliles 1981, NIOSH 1985). Deaths due to cardiovascular collapse usually occur within the first 24 hours after exposure. A minimum lethal dose of 1 mg/kg has been reported, and in a child, death has occurred after the consumption of as little as 3 mg while (Beliles 1981, Brewer and Haggerty 1958, Dacre and Rosenblatt 1974, Davidson et al. 1987).

White phosphorus vapors are highly irritating to the eyes, mucous membranes, respiratory tract and skin (NIOSH 1985). Dermal contact results in severe and painful burns; ocular damage may result from direct contact (Beliles 1981).

Red and black phosphorus are essentially harmless in single-dose exposures, but chronic ingestion of red phosphorus may induce systemic phosphorus poisoning (Gosselin et al. 1984). In general, red phosphorus is considered much less toxic than the white. Liver and kidney effects similar to those reported for white phosphorus are the primary observations in experimental animals subjected to red phosphorus. The cellular basis for the toxic effects of phosphorus is probably its reducing properties which may cause disturbance of intracellular oxidative processes (Beliles 1981).

In humans, the classical effect of chronic white phosphorus intoxication is on the bone, and most typically involves progressive necrotic disease of the jaw bones known as "phossy jaw" (Davidson et al. 1987, Beliles 1981). Cases of this disease have been observed among workers in the phosphorus match industry (white phosphorus is not longer used for this purpose), firecracker manufacture, and

white phosphorus production. The disease often takes years to develop and its pathogenesis currently is uncertain. The most widely held theory is that the phosphorus enters the jaw directly, reacts with the mouth flora, and subsequent infection develops followed by the disease. Other symptoms of chronic intoxication include gastrointestinal distress, low blood pressure, a phosphorus odor (garlic-like) of the breath, anemia, jaundice, delirium, coma and death (Weiss 1986).

White phosphorus is highly toxic in experimental animals; the oral LD_{50} in the rat is 3 mg/kg (RTECS 1987). Inhalation of 100 mg/m^3 phosphorus caused severe respiratory irritation, and high mortality due to bronchopneumonia or edema in rats. Dietary administration of white phosphorus to rabbits for 22 to 57 days resulted in decreased weight gain, and bone changes which included a narrowing of the epiphyseal cartilage plate of the long bones, reduction in number of cartilage cells/column, increased density in metaphyseal zone, and a greater number of trabeculae containing increased amounts of calcified cartilage matrix. Examination of the teeth revealed zones of abnormal dentin corresponding to periods of white phosphorus ingestion, the changes were considered nonspecific (Adams and Sarnat 1940).

Lifetime dietary exposure of rats to white phosphorus resulted in increased mortality incidences, retardation of weight gain, bone thickening of the epiphyseal line, and extension of the trabeculae into the shaft (Fleming et al. 1942).

No information on the carcinogenicity or mutagenicity of phosphorus was found. Female rats exposed to as little as 11 µg/kg of phosphorus on days 1 through 22 of gestation had decreased fertility, postimplantation mortality, and decreased litter size (RTECS 1987). Reproductive effects of white phosphors were investigated in rats administered white phosphorus by gavage 80 days prior to mating and continuing through weaning of two complete reproductive cycles (Condray 1985). High dose females (0.075 mg/kg-day) had a mortality rate of 53%, attributed to difficulty during parturition; hair loss was evident on the forelimbs of this group.

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

EPA (1990) has derived an oral RfD of $2x10^{-5}$ mg/kg-day for white phosphorus (elemental yellow phosphorus) based on parturition mortality and forelimb hair loss in a rat reproductive study (Condray 1985). A safety factor of 1,000 was applied to the no-observed-adverse-effect level of 0.015 mg/kg-day to calculate the RfD. ACGIH (1986) as recommended a 8-hour time-weighted-average threshold limit value (TWA-TLV) of 0.1 mg/m^3 (0.02 ppm) for phosphorus (white) to prevent acute poisoning. OSHA has set an air standard of 100 $\mu g/m^3$.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the threshold limit values and biological exposure indices. 5th ed. Cincinnati, OH.

ADAMS, C.O. AND B.G. SARNAT. 1940. Effects of yellow phosphorus and arsenic trioxide on growing bones and growing teeth. Arch Pathol. 30:1192-1201.

BELILES, R.P. 1981. Phosphorus, Selenium, and Tellurium. In Clayton and Clayton eds., Patty's Industrial Hygiene and Toxicology. pp. 2121-2124.

BREWER E., AND R.J. HAGGERTY. 1958. Toxic Hazards * Rat Poisons. II-Phosphorus. N Eng J Med. 258(3):147-148.

CONDRAY, J.R. 1985. Elemental yellow phosphorus one-generation reproduction study in rats. IR-82-215; IRD No. 401-189. Monsanto Company, St. Louis, MO.

DACRE J.C., AND D.H. ROSENBLATT. 1974. Mammalian toxicology and toxicity to aquatic organisms of four important types of waterborne munitions pollutants-- an extensive literature evaluation. Technical Report No. 7403. U.S. Army Medical Bioengineering Research and Development Laboratory, Aberdeen Proving Ground, Ft. Detrick, Frederick, MD. NTIS AS778-725.

DAVIDSON K.A., P.S. HOVATTER AND C.F. SIGMON. 1987. Water quality criteria for white phosphorus. Final Report ORNL--6336. Oak Ridge National Laboratory. AD-A186613.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

FLEMING R.B.L., J.W. MILLER, V.R. SWAYNE, JR. 1942. Some recent observations on phosphorus toxicology. J Ind Hyg Toxicol. 24(6):154-158.

GOSSELIN, R.E., SMITH, R.P., HODGE, H.C. 1984. Clinical Toxicology of Commercial Products. 5th ed. Williams and Wilkins, Baltimore.

NIOSH 1985. Pocket guide to Chemical Hazards. U.S. Dept. of Health and Human Services. September.

WEISS G. 1986. Hazardous Chemicals Data Book. 2nd ed. Noyes Data Corporation. Park Ridge, NJ.

PINACOLYL METHYLPHOSPHONIC ACID (PMPA)

INTRODUCTION

Pinacolyl methylphosphonic acid (PMPA) is a relatively stable hydrolysis product of soman (Johnsen and Blanch 1984). Hydrolysis products, such as PMPA are smaller, less complex molecules and therefore are usually less potent than the parent agent, soman (Chemical Stockpile Disposal Program 1988). PMPA is also a metabolite of soman in mice intravenously injected (Reynolds et al. 1985).

TOXICOKINETICS

The primary routes of potential exposure to the hydrolysis products, such as PMPA are oral and percutaneous absorption. Extensive dilutions of hydrolysis products usually occur under environmental conditions (Chemical Stockpile Disposal Program 1988). Mice exposed intravenously to sublethal doses of soman had large concentrations of soman and its metabolites (IMPA and PMPA) in the hypothalamus suggesting that this might be important with respect to the pharmacological effects or the toxicity of soman and PMPA (Little et al. 1988).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

There is virtually no toxicological information available for PMPA, however, since it is a hydrolysis product of soman, it can be expected to induce milder yet similar toxicity as its parent compound. In humans, the first signs of soman toxicity are general effects such as miosis, rhinorrhea, painful or impaired visual accommodation, lacrimation and salivation. Gastrointestinal effects include anorexia, nausea, vomiting, cramps, and diarrhea and respiratory effects include wheezing, coughing and a tightness in the chest, sweating, muscular fasiculations and frequent urination. These symptoms occur from the muscarinic stimulation of glands and smooth muscles innervated by the parasympathetic system. The high lipid solubility of soman may cause nicotinic effects in the central nervous system (CNS) during an episode of severe poisoning manifested as dreams, headaches, mood changes, dizziness, tremors and convulsions (LaBorde and Bates 1986).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

No regulated standards or criteria have been promulgated for PMPA.

REFERENCES

CHEMICAL STOCKPILE DISPOSAL PROGRAM. 1988. Final Programmatic Environmental Impact Statement. Program Executive Officer--Program Manager for Chemical Demilitarization. Aberdeen Proving Ground MD.

JOHNSEN B.A., AND J.H. BLANCH. 1984. Analysis of snow samples contaminated with chemical warfare agents. Arch Belg Med Soc, Hyg Med Trav Med Leg. Vol suppl, iss Proc-World Congr. "New Compd Biol Chem Warf". Toxicol Eval 1st. 20-30.

LABORDE J.B., H.K. BATES. 1986. Developmental toxicity study of agent GD-DCSM in NZW rabbits and CD rats. Division of Reproductive and Developmental Toxicology, National Center for Toxicological Research. Jefferson, Arkansas. Supported by the U.S. Army Medical Bioengineering Research and Development Laboratory, Fort Detrick, MD. Army Project No. 83-PP3812. FDA No. 224-83-0004.

LITTLE P.J., J.A. SCIMECA, B.R. MARTIN. 1988. Distribution of [3H]diisopropyl fluorophosphate, [3H]soman, [3H]sarin, and their metabolites in mouse brain. Drug Metab Dispos 16(4):515-520.

REYNOLDS M.S., P.J. LITTLE, B.F. THOMAS, R.B. BAGLEY, B.R. MARTIN. 1985. Relationship between the biodisposition of [3H]soman and its pharmacological effects in mice. Toxicol Appl Pharmacol. 80(3):409-20.

POLYCHLORINATED BIPHENYLS (PCBs)

PCBs are complex mixtures of chlorinated biphenyls. The commercial PCB mixtures that were manufactured in the United States were given the trade name of "Aroclor." Aroclors are distinguished by a four-digit number (for example, Aroclor 1260). The last two digits in the Aroclor 1200 series represent the average percentage by weight of chlorine in the product.

PCBs are readily absorbed through the gastrointestinal tract and somewhat less readily through the skin; PCBs are presumably readily absorbed from the lungs, but few data are available that experimentally define the extent of absorption after inhalation (EPA 1985). Dermatitis and chloracne (a disfiguring and long-term skin disease) have been the most prominent and consistent findings in studies of occupational exposure to PCBs. Several studies examining liver function in exposed humans have reported disturbances in blood levels of liver enzymes. Reduced birth weights, slow weight gain, reduced gestational ages, and behavioral deficits in infants were reported in a study of women who had consumed PCB-contaminated fish from Lake Michigan (EPA 1985). Reproductive, hepatic, immunotoxic, and immunosuppressive effects appear to be the most sensitive end points of PCB toxicity in nonrodent species, and the liver appears to be the most sensitive target organ for toxicity in rodents (EPA 1985). A number of studies have suggested that PCB mixtures are capable of increasing the frequency of tumors including liver tumors in animals exposed to the mixtures for long periods (Kimbrough et al. 1975, NCI 1978, Schaeffer et al. 1984, Norback and Weltman 1985). Studies have suggested that PCB mixtures can act to promote or inhibit the action of other carcinogens in rats and mice (EPA 1985).

EPA (1990) classified PCB as a Group B2 agent (Probable Human Carcinogen) based on sufficient evidence in animal bioassays and inadequate evidence from studies in humans. The EPA Carcinogen Assessment Group (EPA 1990) calculated an oral cancer potency factor of 7.7 $(mg/kg\text{-}day)^{-1}$ for PCBs based on the incidence of hepatocellular carcinomas and adenocarcinomas in female Sprague-Dawley rats exposed to a diet containing Aroclor 1260 as reported in a study by Norback and Weltman (1985). Clement Associates has derived an oral RfD of $1x10^{-4}$ mg/kg-day for Aroclor 1016 based on a chronic oral study conducted in monkeys (Barsotti and Van Miller 1984). A no-observed-adverse-effect level of 0.25 ppm (0.01 mg/kg-day) for fetotoxicity was identified from this study. A safety factor of 100 (10 to account for interspecies extrapolation and 10 to account for the variation among the members of the human population) was used to calculate the RfD.

REFERENCES

BARSOTTI, D.A., VAN MILLER, J.P. 1984. Accumulation of a commercial polychlorinated biphenyl mixture (aroclor 1016) in adult rhesus monkeys and their nursing infants. Toxicology 30:31-44

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Health Effects Criteria Document on Polychlorinated Biphenyls. Final Draft. Office of Drinking Water, Washington

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

KIMBROUGH, R.D., SQUIRE, R.A., LINDER, R.E., STRANDBERG, J.D., MONTALI, R.J. and BURSE, V.W. 1975. Induction of liver tumors in Sherman strain female rats by polychlorinated biphenyl Aroclor 1260. J. Natl. Cancer Inst. 55:1453

NATIONAL CANCER INSTITUTE (NCI). 1978. Bioassay of Aroclor 1254 for Possible Carcinogenicity. Cas. No. 27323-18-8. NCI Carcinogenesis Technical Report Series No. 38. DHEW (NIH) Publication No. 78-838

NORBACK, D.H., and WELTMAN, R.H. 1985. Polychlorinated biphenyl induction of hepatocellular carcinoma in the Sprague-Dawley rat. Environ. Health Perspect. 1:134-143

SCHAEFFER, E., GREIM, H., and GOESSNER, W. 1984. Pathology of chronic polychlorinated biphenyl (PCB) feeding in rats. Toxicol. Appl. Pharmacol. 75:278-288

POLYCYCLIC AROMATIC HYDROCARBONS (Carcinogenic)

PAHs occur in the environment as complex mixtures containing numerous PAHs of varying carcinogenic potencies. Only a few components of these mixtures have been adequately characterized, and only limited information is available on the relative potencies of different compounds.

PAH absorption following oral exposure is inferred from the demonstrated toxicity of PAHs following ingestion (EPA 1984a). PAH absorption following inhalation exposure is inferred from the demonstrated toxicity of PAHs following inhalation (EPA 1984a). PAHs are also absorbed following dermal exposure (Kao et al. 1985). It has been suggested that simultaneous exposure to carcinogenic PAHs such as benzo[a]pyrene and particulate matter can increase the effective dose of the compound (ATSDR 1987). Acute effects from direct contact with PAHs and related materials are limited primarily to phototoxicity; the primary effect is dermatitis (NIOSH 1977). PAHs have also been shown to cause cytotoxicity in rapidly proliferating cells throughout the body; the hematopoietic system, lymphoid system, and testes are frequent targets (Santodonato et al. 1981). Destruction of the sebaceous glands, hyperkeratosis, hyperplasia, and ulceration have been observed in mouse skin following dermal application of the carcinogenic PAHs (Santodonato et al. 1981). The carcinogenic PAHs have also been shown to have an immunosuppressive effect in animals (ATSDR 1987). Nonneoplastic lesions have been observed in animals exposed to the more potent carcinogenic PAHs but only after exposure to levels well above those required to elicit a carcinogenic response. Carcinogenic PAHs are believed to induce tumors both at the site of application and systemically. Neal and Rigdon (1967) reported that oral administration of 250 ppm benzo[a]pyrene for approximately 110 days led to forestomach tumors in mice. Thyssen et al. (1981) observed respiratory tract tumors in hamsters exposed to up to 9.5 mg/m^3 benzo[a]pyrene for up to 96 weeks.

Benzo[a]pyrene is representative of the carcinogenic PAHs and is classified by EPA in Group B2--Probable Human Carcinogen--based on sufficient evidence of carcinogenicity from animal studies and inadequate evidence from epidemiological studies (EPA 1984b, 1990). EPA (1984b) calculated an oral cancer potency factor of 11.5 $(mg/kg\text{-}day)^{-1}$ for carcinogenic PAHs (specifically benzo[a]pyrene) based on the study by Neal and Rigdon (1967).

REFERENCES

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1987. Draft Toxicological Profile for Benzo[a]pyrene. October 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984a. Health Effects Assessment for Polycyclic Aromatic Hydrocarbons (PAHs). Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-013

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984b. Health Effects Assessment for Benzo[a]pyrene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-022

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

KAO, J.K., PATTERSON, F.K., and HALL, J. 1985. Skin penetration and metabolism of topically applied chemicals in six mammalian species including man: An *in vitro* study with benzo[a]pyrene and testosterone. Toxicol. Appl. Pharmacol. 81:502-516 (As cited in ATSDR 1987)

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1977. Criteria for a Recommended Standard--Occupational Exposure to Coal Tar Products. DHEW (NIOSH) 78-107

NEAL, J., and RIGDON, R.H. 1967. Gastric tumors in mice fed benzo(a)pyrene: A quantitative study. Tex. Rep. Biol. Med. 25:553-557

SANTODONATO, J., HOWARD, P., and BASU, D. 1981. Health and ecological assessment of polynuclear aromatic hydrocarbons. J. Environ. Pathol. Toxicol. 5:1-364

THYSSEN, J., ALTHOFF, J., KIMMERLE, G., and MOHR, U. 1981. Inhalation studies with benzo(a)pyrene in Syrian golden hamsters. J. Natl. Cancer Int. 66:575-577

POLYCYCLIC AROMATIC HYDROCARBONS (Noncarcinogenic)

Polycyclic aromatic hydrocarbons (PAHs) occur in the environment as complex mixtures of which only a few components have been adequately characterized. Only limited information is available on the relative potencies of the "noncarcinogenic" PAHs. However, many have been shown to have some weak carcinogenic activity, or to act as promoters or cocarcinogens.

PAH absorption following oral and inhalation exposure is inferred from the demonstrated toxicity of PAHs following these routes of administration (EPA 1984). PAHs are also absorbed following dermal exposure (Kao et al. 1985). Acute effects from direct contact with PAHs and related materials are limited primarily to phototoxicity; the primary effect is dermatitis (NIOSH 1977). PAHs have also been shown to cause cytotoxicity in rapidly proliferating cells throughout the body; the hematopoietic system, lymphoid system, and testes are frequent targets (Santodonato et al. 1981). Some of the noncarcinogenic PAHs have been shown to cause systemic toxicity but these effects are generally seen at high doses (Santodonato et al. 1981). Slight morphological changes in the liver and kidney of rats have been reported following oral exposure to acenaphthene for 40 days (EPA 1984). Subchronic oral administration of naphthalene to rabbits and rats has resulted in cataract formation (Schmahl 1955).

EPA (1989) developed an oral reference dose of $4x10^{-3}$ mg/kg-day for naphthalene based on the development of ocular and systemic lesions in rats (Schmahl 1955, EPA 1986). An uncertainty factor of 10,000 was applied to the animal data in the development of the reference dose.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Polycyclic Aromatic Hydrocarbons (PAHs). Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-013

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Health and Environmental Effects Profile for Naphthalene. Environmental Criteria and Assessment Office, Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1989

KAO, J.K., PATTERSON, F.K., and HALL, J. 1985. Skin penetration and metabolism of topically applied chemicals in six mammalian species including man: An *in vitro* study with benzo[a]pyrene and testosterone. Toxicol. Appl. Pharmacol. 81:502-516 (As cited in ATSDR 1987)

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1977. Criteria for a Recommended Standard--Occupational Exposure to Coal Tar Products. DHEW (NIOSH) 78-107

SCHMAHL, D. 1955. Testing of naphthalene and anthracene as carcinogenic agents in the rat. Z. Krebsforsch. 60:697-710 (German with English translation)

SANTODONATO, J., HOWARD, P., and BASU, D. 1981. Health and ecological assessment of polynuclear aromatic hydrocarbons. J. Environ. Pathol. Toxicol. 5:1-364

POTASSIUM

Absorption of dietary potassium from the gastrointestinal tract is nearly complete (Goodman and Gilman 1985). Potassium is a reactive compound that is strongly caustic and corrosive upon contact with tissues (Wands 1981). Potassium is an essential element and more concern is generally associated with potassium deficiency (particularly in the elderly) than with toxicity. It functions in the maintenance of electrolyte balance, in the transmission of nerve impulses to muscle fibers, in the control of normal muscle contractility and cardiac rhythm, and it acts as an insulin antagonist in intermediary carbohydrate metabolism (NRC 1980). Ingestion of excess potassium results in hyperkalemia which alters the electrical activity of the heart. At potassium levels of 8 to 9 mEq per liter, there is profound depression in impulse generation and conduction in all cardiac tissues (Goodman and Gilman 1985). The National Research Council of the National Academy of Sciences (NRC 1980) has determined that the estimated adequate and safe intake level for potassium is between 1,875 and 5,600 mg/day for adults. NRC (1980) also noted that "it is not possible to induce hyperkalemia or potassium toxicity by dietary means in people with normal circulatory and renal function." However, acute poisoning with potassium chloride tablets has been observed in children at potassium levels as low as 2,000 mg/day (NRC 1980). No health-based criteria have been established for potassium by EPA.

REFERENCES

GOODMAN AND GILMAN. 1985. The Pharmacological Basis of Therapeutics. Gilman A., Goodman L., Rall, T., Murad F. eds. 7th. ed. MacMillan Publishing Co. New York. p. 866-874.

NATIONAL RESEARCH COUNCIL (NRC). 1980. Drinking Water and Health. Volume 3. Safe Drinking Committee, Washington, D.C. 415 pages.

WANDS, RC. 1981. Alkaline Materials. In Clayton G. and Clayton F., eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York. Vol 2B. pg. 3054-3055.

POTASSIUM 40

Potassium-40 is a natural radionuclide which spontaneously transforms itself into another nuclide by changing its nuclear configuration by the emission of beta particles (negative and positive). It has a half-life equal to the age of the earth and therefore, is ubiquitous in normal foods and human tissues. Cells which frequently undergo mitosis are the most radiosensitive to potassium-40 and include the hematopoietic stem cells, bone marrow cells, dividing cells in the intestinal glands, and germ cells (spermatocytes and oocytes). Clinical manifestations of high-level, acute exposure to potassium 40 include gastrointestinal effects (nausea, vomiting, diarrhea), bone marrow suppression (decreased circulating lymphocytes) and possibly central nervous system effects. Beta particles are ionizing, and therefore induce gene mutations and chromosome aberrations in both somatic and germ cells. Chronic, low-level exposure to ionizing radiation resulting from potassium 40, can result in the induction of cancer.

REFERENCES

HOBBS, C.H. AND R.O. MCCLELLAN. 1986. Toxic effects of Radiation and Radioactive Materials. In Casarett and Doull's Toxicology the Basic Science of Poisons. 3rd Ed. Klaassen, C.D, Amdur, M.O., and Doull J. eds. pp. 669-705.

RADIUM 226

Radium 226 (^{226}Ra) is an alkaline earth element, a decay product of Uranium 238 which further decays to Radon 222. It is an alpha-emitting radionuclide with a half-life of 1,602 years. Because radium is a metabolic analog of calcium it is deposited in the skeleton. The ^{226}Ra retained in the skeleton serves as a source of alpha radiation of bone and contiguous tissues at a dose rate that decreases slowly with time. The cells which frequently undergo mitosis are the most radiosensitive to ^{226}Ra and include hematopoietic stem cells, bone marrow cells, dividing cells in the intestinal glands, and germ cells (spermatocytes and oocytes). The early effects of exposure to ionizing radiation from ^{226}Ra result primarily from cell death. Depending on the size and distribution of the absorbed dose, the clinical manifestations of the acute radiation syndrome include hematopoietic depression, gastrointestinal denudation and central nervous system (CNS) disruption. Destruction of the gastrointestinal mucosa results in nausea, vomiting and diarrhea and may lead to ulceration and hemorrhage especially in the small intestine following severe exposures. Damage to the bone marrow cells is reflected by changes in the circulating blood (e.g., drastic fall in the number of lymphocytes). The severity of bone marrow depression and the latent period between exposure and appearance of the symptoms are related to the magnitude of dose. The germ cells of both males and females are radiosensitive. In males, acute doses of 10 to 100 rads (unit of radiation-absorbed dose) will cause a dose-related depression of the sperm count, which recovers slowly. With doses in the range of 500 rads, permanent sterility is likely. In females, destruction of oocytes results in permanent sterility due to a lack of stem cells in the adult ovary. Also, destruction of germinal epithelium of the ovary involves interruption of the production of the sex hormones. Tissues tolerate larger total doses of ionizing radiation when the dose is fractionated. Furthermore, ionizing radiation produces gene mutations and chromosome aberrations in both somatic and germ cells. It has clearly been established that radium induces osteosarcomas of the skeleton and carcinomas of the mastoid and paranasal air sinuses from investigations of radium dial painters and chemists involved in the luminous dial industry that flourished in the early 1900's. The radium dial painters were largely young women who used luminous paints containing ^{226}Ra, and as a consequence of an early industrial practice of "tipping" the brush on their lips, ingested significant quantities of ^{226}Ra.

REFERENCES

HOBBS, C.H. AND R.O. MCCLELLAN. 1986. Toxic effects of Radiation and Radioactive Materials. In Casarett and Doull's Toxicology the Basic Science of Poisons. 3rd Ed. Klaassen, C.D, Amdur, M.O., and Doull J. eds. pp. 669-705.

RDX (hexahydro-1,3,5-trinitro-1,3,5-triazine; cyclonite) is completely absorbed following oral exposure (EPA 1988). No data are available regarding dermal absorption. Workers exposed to RDX via inhalation and gastrointestinal routes suffered CNS effects, including headaches, nausea, vomiting, amnesia, clonic/tonic convulsions, and unconsciousness (Gosselin 1984; Kaplan 1965). These symptoms paralleled those previously reported in animal studies (Sunderman et al 1944; Von Oettingen et al 1949). However, a cross-sectional epidemiological study in a munitions plant did not identify any abnormalities in employees attributable to RDX exposure (Hathaway 1977). In acute toxicity studies, dogs exposed intravenously to RDX experienced decreased blood pressure and erratic electroencephalographic patterns at low doses, central nervous system hyperactivity and nonlethal convulsions at higher doses, and convulsions and death at the highest dose levels (EPA 1988). In subchronic feeding studies, mice experienced increased liver weights. Anemia was seen in male mice and rats, and female rats experienced increased liver weights (EPA 1988). Chronic oral exposure to RDX in rats and mice produced CNS effects, increased mortality, weight loss, anemia, hepatoxicity, renal toxicity, testicular degeneration, and inflammation of the prostate (Levine et al. 1983; EPA 1988). Decreased fertility, developmental effects, and embryotoxicity were observed in rats that were fed RDX. In rabbits, RDX caused maternal toxicity, and there was suggestive evidence for teratogenic effects (EPA 1988). No conclusive evidence of carcinogenicity has been shown for RDX. RDX was not found to be carcinogenic in Fisher 344 rats (Levine et al. 1983) or Sprague-Dawley rats (Hart 1977) exposed to RDX in the diet for 2 years. However, Lish et al. (1984) reported a statistically significant increase in the combined incidence of hepatocellular carcinomas and adenomas in female B6C3F1 mice fed RDX in the diet for two years.

EPA (1989) has classified RDX in Group C -- Possible Human Carcinogen -- and has developed an oral cancer potency factor of 0.11 $(mg/kg\text{-}day)^{-1}$. The potency factor is based on the increased incidence of combined hepatocellular carcinomas and adenomas in female mice receiving RDX in the diet for two years (Lish et al. 1984). EPA (1989) has derived a reference dose (RfD) of 0.003 mg/kg-day based on a chronic study in which rats receiving RDX in the diet for 24 months at varying dosages experienced inflammation of the prostate (Levine et al. 1983). A lowest observed adverse effect level (LOAEL) of 1.5 mg/kg-day was identified. An uncertainty factor of 100 was used to derive the RfD.

REFERENCES

American Conference of Governmental Industrial Hygienists Inc. (ACGIH). 1986. Documentation of the threshold limit values and biological exposure indices. 5th ed. Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Health Advisory for Hexahydro-1,3,5-Trinitro-1,3,5-Triazine (RDX). Criteria and Standards Division, Office of Drinking Water, Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989. Health Effects Assessment Summary Tables. October. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C.

GOSSELIN, R.E., SMITH, R.P, HODGE, H.C., AND BRADDOCK, J.E. 1984. Clinical Toxicology of Commercial Products. 5th ed. Williams and Wilkins, Baltimore.

HART, E.R. 1977. Two-year feeding study in rats. AD A040161. Litton Bionetics, Inc., Kensington, MD. Office of Naval Research, Contract No. N00014-73-C-0162.

HATHAWAY, J.A. ET AL. 1977. J. Occup. Med. 19:269-272. As cited in ACGIH.

KAPLAN, A.S., BERGHOUT, C.F., PECZENIK, A. 1965. Human intoxication from RDX. Arch. Environ. Health 10:877-883.

LEVINE, B.S., FUREDI, E.M. RAC, V.S., GORDON, D.E., LISH, P.M. 1983. Determination of the chronic mammalian toxicological effects of RDX: Twenty-four month chronic toxicity/carcinogenicity study of hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX) in the Fischer 344 rat. Phase V. Vol.1. AD A160774. Chicago, IL: IIT Research Institute. U.S. Army Medical Research and Development Command, Contract No. DAMD17-79-C-9161.

LISH, P.M., LEVINE, B.S., FUREDI, E.M., SAGARTZ, E.M., RAC, V.S. 1984. Determination of the chronic mammalian toxicological effects of RDX: Twenty-four month chronic toxicity/carcinogenicity study of hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX) in the B6C3F1 hybrid mouse. Phase VI. Vol.1. Chicago, IL: IIT Research Institute. U.S. Army Medical Research and Development Command, Contract No. DAMD17-C-9161.

SUNDERMAN, F. et al. 1944. National Defense Research Comm., Office of Scientific Research and Development. NDRC Contract OEM sr-96a.

VON OETTINGEN, W.F., DONAHUE, D.D., YAGODA, H., et al. 1949. J. Ind. Hyg. Tot. 31:21. As cited in ACGIH.

INTRODUCTION

Sarin, agent GB, or isopropyl methylfluorophosphate (IMPF), is a quick-acting nerve agent, and a marked cholinesterase inhibitor (ICF Feasibility Study 1987). Sarin has a high volatility and is very effective as a toxic inhalant but somewhat less effective as a skin penetrant, because it readily evaporates from the skin (Chemical Stockpile Disposal Program 1988). It is very soluble, and is therefore hydrolyzed rapidly, especially under conditions of high temperature and pH. Sarin is usually odorless, colorless, tasteless, nonirritating to the skin and is extremely toxic in both liquid and vapor forms (Chemical Stockpile Disposal Program 1988). Sarin is among the most potent of the chemical warfare agents; sarin is more acutely toxic to humans by inhalation than tabun (GA), but is less toxic than VX (Chemical Stockpile Disposal Program 1988).

TOXICOKINETICS

In vapor or aerosol form sarin can be inhaled or absorbed through the skin or the conjunctiva of the eye; as a liquid, it can be absorbed through the skin, conjunctiva, and upper digestive or gastrointestinal tract. The efficacy of percutaneous absorption varies among individuals (Chemical Stockpile Disposal Program 1988). Sarin is highly lipid soluble which can enhance absorption and cause systemic toxicity. Local effects of sarin are usually due to direct contact with gaseous or vaporized form. Sarin is slowly metabolized and essentially cumulative (Army 1975).

In rodents, sarin is unstable once it enters the body, and thus, less of the agent actually reaches the target tissues. Sarin is detoxified (hydrolyzed) by aliesterase (AE) (an enzyme in the plasma) which combines rapidly with sarin and prevents it from interacting with acetylcholinesterase (AChE). Human plasma does not contain AE. Metabolism studies in dogs demonstrated that the main detoxification product of sarin is isopropyl methylphosphonic acid (IMPA) and that this compound accounts for the majority of sarin activity found in brain tissue (Chemical Stockpile Disposal Program 1988). In mice exposed intravenously to sublethal doses of sarin, the metabolites of sarin (IMPA and MPA) were distributed to areas of the brain in greater concentrations than sarin (Little et al. 1986, 1988). The higher concentrations of sarin and its metabolites (IMPA and MPA) in the brain implicates that this area might be important with respect to the pharmacological effects or the toxicity of these compounds (Little et al. 1988). The high concentrations of sarin metabolites found in the kidneys implied that this organ played a major role in the detoxification and excretion of sarin. Large quantities of free and bound IMPA were also found in the lung which suggested an important site for toxicity (Little et al. 1986).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

Sarin is extremely toxic by inhalation and oral exposures or by dermal and ocular contact (ICF Feasibility Study 1987). Vapor and liquid forms do not injure skin due to rapid penetration; with more rapid penetration through the eye than the skin. In humans, the lowest vapor concentration at which toxic effects (miosis and alterations in cholinesterase levels) were noted (TC_{lo}) was 90 $\mu g/m^3$, while the lowest ingested dose (TC_{lo}) which induced muscle weakness, bronchiolar constriction, and nausea or vomiting has been reported to be 2 $\mu g/m^3$ (RTECS 1987). The median lethal concentration (LC_{50}) for humans is 70 mg/m^3 while the inhalation LC_{50}'s for rats, cats, dogs and guinea pigs are between 100-150 mg/m^3 for a 10 minute exposure (RTECS 1987). The dermal LD_{50}'s are 25 mg/kg, 925 $\mu g/kg$ and 1,080 $\mu g/kg$ for humans, rabbits and mice, respectively. The oral LD_{50} for rats is 550 $\mu g/kg$ (RTECS 1987).

The estimated LCt_{50} for percutaneous toxicity is 158 times higher than the estimated LCt_{50} for inhalation. Rapid evaporation from the skin is the primary factor in the relatively low percutaneous toxicity observed; with the prevention of evaporation the toxicity of sarin increases almost a hundredfold. Skin constituents also hinder dermal absorption, thereby attenuating the amount of sarin that reaches the target tissues (Chemical Stockpile Disposal Program 1988). The skin LCt_{50} for vapor is approximately 12,000-15,000 mg-min/m^3 while the median incapacitating dosage is approximately 8,000 mg-min/m^3 (Army 1975). The median incapacitating inhalation dosage (ICt50) is 35-75 mg-min/m^3 (Army 1975).

The toxic effects of sarin are due to its ability to irreversibly inhibit acetylcholinesterase (AChE), an enzyme responsible for the breakdown of the neurotransmitter acetylcholine (ACh). The mode of action involves the consequences of excessive ACh accumulation at nerve junction, and within portions of the nervous system that control smooth muscle, cardiac muscle, and endocrine-exocrine glandular function (Chemical Stockpile Disposal Program 1988). Manifestations include: drooling, increased bronchial secretions, bronchoconstriction, miosis, excessive sweating, vomiting, diarrhea, abdominal cramping, involuntary urination, and heartbeat irregularities (arrhythmias). In addition, ACh accumulation can affect the central nervous system, resulting in headache, anxiety, confusion, restlessness, giddiness, electroencephalographic (EEG) changes, or even convulsions and coma (Chemical Stockpile Disposal Program 1988). The third area affected by ACh accumulation is a portion of the nervous system controlling skeletal muscles. Thus, acute exposure to sarin can also result in a generalized weakness that increases with exertion, as well as muscle twitching and cramping. Respiratory failure, the immediate cause of death in lethal sarin exposure occurs due to neuromuscular block of the respiratory muscles and airway constriction (Army 1975). Serious and fatal cardiac complications can develop after apparent recovery from acute toxic effects, this is evident as electrocardiogram (EKG) abnormalities. Additionally cardiac lesions have been found in animals surviving high doses of nerve agents (Chemical Stockpile Disposal Program 1988).

The motor effects observed in rats, mice, or guinea pigs following exposure to sarin include tremors, hind-limb abduction (extension away from the body), rearing activity, chewing movements, decreases in spontaneous activity, and decreased coordination (Chemical Stockpile Disposal Program 1988). The motor effects are dose-dependent and range from mild (some salivation, fine tremors) to moderate (excessive salivation, and weeping, generalized tremors). A good correlation between brain AChE inhibition and the degree of toxicity is evident; rats injected with sarin exhibited a dose-dependent inhibition of brain AChE, with lethal doses producing >90% inhibition (Chemical Stockpile Disposal Program 1988).

Fischer 344 rats and Strain "A" mice (susceptible strains) exposed to airborne sarin for up to 52 weeks (maximum cumulative exposure of 10.5 mg-min/m^3) did not develop increased tumor incidences within the study duration of 1.5 years (Weimer et al. 1979). These results suggest that sarin is not carcinogenic, however, this study was not designed to be a definitive study of the carcinogenic activity of sarin, particularly with the limited dose employed (Chemical Stockpile Disposal Program 1988).

In a developmental study, sarin was administered orally to pregnant CD rats (0, 100, 240, and 380 µg/kg-day; gestational days 6-15) and pregnant NZW rabbits (0, 5, 10 and 15 µg/kg-day; gestational days 6-19) (Laborde and Bates 1986). At term, the maternal animals were sacrificed and the gravid uteri were weighed and examined for number and status of implants (alive, resorbed or dead). Individual fetal body weight and fetal malformations (external, internal and skeletal) were recorded. Both rat and rabbit dams in the high dose group exhibited statistically significant signs of maternal toxicity and increased maternal mortality. Examination of gravid uteri at sacrifice revealed no significant differences among treatment groups in the incidence of resorptions, dead or malformed

fetuses or in average body weight on live fetuses per litter. Thus, no evidence of developmental toxicity was found in either the CD rat or NZW rabbit, even at doses which produced significant maternal toxicity.

The mutagenic potential of sarin was studied in a series of in vitro and in vivo evaluations and it was concluded that sarin is not mutagenic (Goldman et al. 1987). Negative results were found in the Ames Salmonella bacterial gene mutation assay using 5 different strains exposed to up to 200 µg/100 µl concentrations. In mouse lymphoma cells exposed to sarin, no agent-related mutagenesis was found. Chromosomal damage as measured by sister chromatid exchanges (SCE) was not found in Chinese hamster ovary cells exposed in vitro to 200 µg/ml. Sister chromatid exchanges scored in lymphocytes from mice exposed in vivo to the maximally tolerated dose of 300 µg/kg sarin also showed no mutagenic effect. Rat hepatocytes, used to detect DNA damage in vitro by measuring unscheduled DNA synthesis, with and without metabolic activation were negative, indicating the sarin in not mutagenic.

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

No regulated standards or criteria have been promulgated for sarin (GB).

REFERENCES

ARMY. 1975. Military Chemistry and Chemical Compounds Field Manual. Department of the Army and Air Force, Washington, D.C. October 1975. FM 3-9, AFR 355-7.

CHEMICAL STOCKPILE DISPOSAL PROGRAM. 1988. FINAL PROGRAMMATIC ENVIRONMENTAL IMPACT STATEMENT. January. Program Executive Officer–Program Manager for Chemical Demilitarization. Aberdeen Proving Ground, MD

GOLDMAN M., A.K. KLEIN, T.G. KAWAKAMI, L.S. ROSENBLATT. 1987. Toxicity studies on agents GB and GD. Laboratory for Energy-Related Health Research, University of Calif. U.S. Army Medical Research and Development Command. Frederick, MD. APO 84PP4855, Study No. 4855.

ICF FEASIBILITY STUDY. 1987. Toxicity of contaminants suspected to be present at old "O" field. December 1987.

LABORDE, J.B., AND H.K. BATES. 1986. Developmental Toxicity Study of Agent GB-DSCM Types I and II in CD rats and NZW rabbits. National Center for Toxicological Research, FDA. U.S. Army Medical Research and Development Command, Fort Detrick, Maryland. Project Officer: J.c. Dacre. U.S. Army Medical Bioengineering R&D Laboratory.

LITTLE P.J., M.L. REYNOLDS, E.R. BOWMAN, B.R. MARTIN. 1986. Tissue disposition of [3H]sarin and its metabolites in mice. Toxicol Appl Pharmacol. 83(3):412-19.

LITTLE P.J., J.A. SCIMECA, B.R. MARTIN. 1988. Distribution of [3H]diisopropyl fluorophosphate, [3H]soman, [3H]sarin, and their metabolites in mouse brain. Drug Metab Dispos 16(4):515-520.

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1987. Vol. 3A. National Institute for Occupational Safety and Health (NIOSH). U.S. Department of Health and Human Services.

WEIMER ET AL. 1979. (as cited in CHEMICAL STOCKPILE DISPOSAL PROGRAM. 1988.)

SELENIUM

Results of studies with humans and experimental animals indicate that certain selenium compounds are readily absorbed from the gastrointestinal tract following oral exposure (EPA 1984). The pulmonary absorption of selenium following inhalation exposure has not been well studied, although there are reports suggesting that selenium is absorbed to some extent by this route (EPA 1984). Selenium is an essential element and therefore is nontoxic at doses necessary for normal health and nutrition. NAS (1980) reported that an adequate and safe selenium intake for an adult human ranges from 0.05 mg/day to 0.2 mg/day. However, exposure to selenium at levels that exceed these standards has been associated with adverse health effects. Subchronic or chronic oral exposure of experimental animals to various selenium compounds has produced anemia, reduced growth, increased mortality, and lesions of the liver, heart, kidney, and spleen (EPA 1984). In humans, chronic oral exposure to selenium has been associated with alopecia, dermatitis, discoloration of the skin, loss of fingernails, muscular dysfunction, convulsions, paralysis, and increased incidences of dental caries (EPA 1984, Yang et al. 1983). Headaches and respiratory irritation have been noted in humans following acute inhalation exposure (EPA 1984); dermatitis and gastrointestinal disturbances have resulted from occupational exposure (Glover 1967). Studies with a variety of animals have suggested that selenium may be teratogenic; however, these studies are limited in that exposure levels are not well characterized (EPA 1984).

An oral reference dose (RfD) of 3.0×10^{-3} mg/kg-day has been derived by EPA for selenious acid (1984, 1990). The oral RfD value was based on a study by Yang et al. (1983) in which humans who were exposed to seleniferous foodstuffs in the diet at doses of 3.2 mg/day developed loss of hair, loss of fingernails, dermatitis, and muscular dysfunction. An uncertainty factor of 15 was applied to the LOAEL of 3.2 mg/day, to determine the RfD.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Selenium (and Compounds). Office of Emergency and Remedial Response, Washington, D.C. EPA 540/1-86-058. September 1984

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

GLOVER, J.R. 1967. Selenium in human urine: A tentative maximum allowable concentration for industrial and rural populations. Ann. Occup. Hyg. 10:3-10

NATIONAL ACADEMY OF SCIENCES (NAS). 1980. Drinking Water and Health. National Academy Press, Washington, D.C. Vol. 3

YANG, G., WANG, S., ZHOU, R., and SUN, S. 1983. Endemic selenium intoxication of humans in China. Am. J. Clin. Nutr. 37:872-881

SILVER

Silver in various forms is absorbed to a limited extent following oral and inhalation exposures (EPA 1985). The acute toxic effects in humans following oral exposure to silver include corrosive damage to the GI tract leading to shock, convulsions, and death. In animals, acute exposure has been shown to affect the central nervous system and to cause respiratory paralysis (Hill and Pillsbury 1939). The primary effect of silver in humans following chronic exposures is argyria, a permanent bluish-metallic discoloration of the skin and mucous membranes, which can be either localized or generalized. Silver also accumulates in the blood vessels and connective tissue (EPA 1985).

EPA (1990a) derived an oral reference dose (RfD) for silver of 3.0×10^{-3} mg/kg-day for both chronic (1990a) and subchronic (EPA 1990b) exposures based on the human case reports of Gaul and Staud (1935), Blumberg and Carey (1934), and East et al. (1980). In these studies, argyria was observed at an average dose of silver of 0.0052 mg/kg-day, to which an uncertainty factor of 2 was applied.

REFERENCES

BLUMBERG, H. and CAREY, T.N. 1934. Argyremia: Detection of unsuspected and obscure argyria by the spectrographic demonstration of high blood silver. J. Am. Med. Assoc. 103:1521-1524

EAST, B.W., BODDY, K., WILLIAMS, E.D., MACINTYRE, D. and McLAY, A.L.C. 1980. Silver retention, total body silver and tissue silver concentrations in argyria associated with exposure to an anti-smoking remedy containing silver acetate. Clin. Exp. Dermatol. 5:305-311

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Drinking Water Criteria Document for Silver. Environmental Criteria and Assessment Office, Cincinnati, Ohio. PB 86-118288

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

GAUL, L.E. and STAUD, A.H. 1935. Clinical spectroscopy. Seventy cases of generalized argyrosis following organic and colloidal silver medication. J. Am. Med. Assoc. 104:1387-1390

HILL, W.R. and PILLSBURY, D.M. 1939. Argyria, the Pharmacology of Silver. Williams and Wilkins Co., Baltimore, Maryland.

SODIUM

Sodium is rapidly and almost completely absorbed from the gastrointestinal tract (NAS 1977). In humans, adverse effects of sodium occur as a result of ingestion of excess amounts of this element. Acute effects appear to occur only in neonates and young infants. Several studies suggest that permanent brain damage and sudden, unexpected deaths of infants between the ages of 2 weeks and 2 years may be due to hypernatremia (NAS 1977, NIOSH 1987, NRC 1980). Clinical and epidemiological studies suggest that prolonged ingestion of excess sodium may contribute to the development of hypertension in genetically susceptible people (NAS 1977, Wands 1981). In humans, sodium vapors and fumes are strongly alkaline and are extremely irritating and corrosive to the respiratory tract, eyes and skin (Wands 1981). Studies with experimental animals support the contention that excess sodium ingestion is related to the development of hypertension (NAS 1977). It is estimated that at least 40 percent of the population would benefit if consumption of sodium were limited to 2,000 mg/kg or less (EPA 1985, NAS 1977, NRC 1986). Sodium is reported to produce teratogenic and reproductive effects in experimental animals exposed to high doses by various routes. For example, mice exposed subcutaneously to over 2,000 mg/kg of sodium chloride on day 10 or 11 of gestation had an increased incidence of dead or resorbed young. Live young in this study had decreased body weights and an increased incidence of appendicular malformations, such as clubfoot and deviation of the digits (Nishimuri and Miyamoto 1969). No health-based criteria have been developed by EPA.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. National primary drinking water regulations; synthetic organic chemicals, inorganic chemicals and microorganisms; proposed rulemaking. Fed. Reg. 50:46936-47025.

NATIONAL ACADEMY OF SCIENCES (NAS). 1977. Drinking Water and Health. Safe Drinking Water Committee, Washington, D.C. p. 400-411

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1987. Registry of Toxic Effects of Chemical Substances. Data Base. Washington D.C.

NATIONAL RESEARCH COUNCIL (NRC). 1980. Drinking Water and Health. Volume 3. Safe Drinking Water Committee, Washington, D.C. 415 pages.

NISHIMOTO, H., AND MIYAMOTO, S. 1969. Teratogenic effects of sodium chloride in mice. Acta Anat. 74:121-124.

WANDS, RC. 1981. Alkaline Materials. In Clayton G. and Clayton F., eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York. Vol 2B. pg. 3056-3057.

INTRODUCTION

Soman, Agent GD, or pinacolyl methyl phosphonofluoridate, is a quick-acting lethal nerve agent first synthesized by the Germans between 1936 and 1938 (Army 1975, LaBorde and Bates 1986). It is a colorless liquid with a fruity odor; with impurities, it has an odor of camphor (Army 1975). The primary hydrolysis products of soman are hydrogen fluoride (HF) and pinacolyl methylphosphonic acid (MPMA). Soman is an organophosphate compound, that is a potent neurotoxic agent due to its effective inhibition of acetylcholinesterase (Goldman et al. 1987). It has a similar structure to that of sarin (GB) (LaBorde and Bates 1986).

TOXICOKINETICS

Soman is an organophosphate compound which is highly lipid soluble and can be absorbed via inhalation, topical contact or ingestion (LaBorde and Bates 1986). The rate of soman detoxification is low; it is slowly metabolized (Army 1975).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

The effects of soman may be local or systemic; the route of exposure and the chemical nature of the compound determine its action. Local effects from soman are usually due to direct contact with gaseous or vaporized form. Soman's high lipid solubility causes a systemic effects after exposure by any route.

Soman is highly irritating to the eyes following ocular exposure; exposure may cause cessation of breathing and death (ICF Feasibility Study 1987). Soman is also extremely toxic by skin absorption; the liquid does not injure the skin, but penetrates it rapidly. The lethal concentrations (LCIo) in humans following inhalation and dermal exposures are 70 mg/m^3 and 18 mg/kg, respectively (RTECS 1987). In the mouse the inhalation LC_{50} is 1 mg/m^3 for a 30 minute exposure, and the dermal LD_{50} is 7,800 $\mu g/kg$ (RTECS 1987). The median lethal inhalation dosage (LCt_{50}) for humans is 100 $mg\text{-}min/m^3$ (Army 1975); death usually occurs within 15 minutes after a fatal dosage is absorbed. The single oral dose LD_{50} for rabbits and rats are 350-470 $\mu g/kg$ and 400 $\mu g/kg$, respectively (LaBorde and Bates 1986).

Nerve agents such as soman interfere with acetylcholinesterase (a vital enzyme which prevents the accumulation of acetylcholine) to cause an increase in acetylcholine throughout the body. The subsequent accumulation of acetylcholine in the neuronal synapse results in constant stimulation of the post-synaptic acetylcholine receptors, causing an increase in parasympathetic tone (LaBorde and Bates 1986). Acetylcholine is vital in the innervation of skeletal muscles, autonomic ganglia, and many structures within the central nervous system. Anti-cholinesterase agents such as soman effect the loci that use acetylcholine for neuronal transmission like skeletal muscles, parasympathetic end organs, and the central nervous system (Army 1975).

In humans, the first signs of organophosphate toxicity are general effects such as miosis, rhinorrhea, painful or impaired visual accommodation, lacrimation and salivation. Gastrointestinal effects include anorexia, nausea, vomiting, cramps, and diarrhea. Other rapid symptoms of intoxication include respiratory impairment resulting in wheezing, coughing and a tightness in the chest, sweating, muscular fasiculations and frequent urination. These symptoms occur from the muscarinic stimulation of glands and smooth muscles innervated by the parasympathetic system. The high lipid solubility of

soman may cause nicotinic effects in the central nervous system (CNS) during an episode of severe poisoning manifested as dreams, headaches, mood changes, dizziness, tremors and convulsions. The most severe CNS symptom is the eventual paralysis of the central respiratory center, which is the most common cause of death in severe intoxication (Chemical Stockpile Disposal Program 1988).

Soman was administered via gavage to pregnant CD rats on days 6 through 15 of gestation at dose levels of 0, 37, 75, 150 or 165 µg/kg-day and to pregnant NZW rabbits on days 6 through 19 of gestation at dose levels of 0, 2.5, 5, 10 or 15 µg/kg-day (LaBorde and Bates 1986). Mean maternal weight changes, fetal implant status/litter (alive, dead, early and late resorbed), fetal weight and fetal malformations/litter (external, internal and skeletal) were compared among experimental groups by analysis of variance. Rats and rabbits in the high dose groups exhibited statistically significant signs of maternal toxicity and increased maternal mortality. In rabbits maternal mortality was 10% (2/21), 33% (7/21), and 42% (5/12) in the 5, 10 and 15 µg/kg-day dose groups, respectively. The dose of 2.5 µug/kg-day was a "no observable effect level (NOEL)" for mortality. Examination of gravid uteri at sacrifice revealed no significant dose related differences among treatment groups in the incidence of resorptions, dead or malformed fetuses or in average body weight of live fetuses per litter. These results show no evidence of developmental toxicity (embryo/fetotoxicity or teratogenic effect) in the CD rat or NZW rabbit following exposure to soman during embryonic differentiation and major organogenesis, even at a dose which produced overt maternal toxicity.

The mutagenic potential of soman was studied in a series of in vitro and in vivo evaluations and it was concluded that soman is not mutagenic (Goldman et al. 1987). Negative results were found in the Ames Salmonella bacterial gene mutation assay using 5 different strains exposed to concentrations up to 200 µg/100 µl concentrations. No agent-related mutagenesis was found in mouse lymphoma cells exposed to 50,100 or 200 µg/ml. Chromosomal damage as measured by sister chromatid exchanges (SCE) was not found in Chinese hamster ovary cells. Sister chromatid exchanges scored in lymphocytes from mice exposed in vivo to the maximally tolerated dose of 300 µg/kg soman also showed no mutagenic effect. Rat hepatocytes, used to detect DNA damage in vitro by measuring unscheduled DNA synthesis, with and without metabolic activation were negative.

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

No regulated standards or criteria have been promulgated for soman (GD).

REFERENCES

ARMY. 1975. Military Chemistry and Chemical Compounds Field Manual. Department of the Army and Air Force, Washington, D.C. October 1975. FM 3-9, AFR 355-7.

CHEMICAL STOCKPILE DISPOSAL PROGRAM. 1988. FINAL PROGRAMMATIC ENVIRONMENTAL IMPACT STATEMENT. January. Program Executive Officer--Program Manager for Chemical Demilitarization. Aberdeen Proving Ground, MD

GOLDMAN M., A.K. KLEIN, T.G. KAWAKAMI, L.S. ROSENBLATT. 1987. Toxicity studies on agents GB and GD. Laboratory for Energy-Related Health Research, University of Calif. U.S. Army Medical Research and Development Command. Frederick, MD. APO 84PP4855, Study No. 4855.

ICF FEASIBILITY STUDY. 1987. Toxicity of contaminants suspected to be present at old "O" field. December .

LABORDE J.B., H.K. BATES. 1986. Developmental toxicity study of agent GD-DCSM in NZW rabbits and CD rats. Division of Reproductive and Developmental Toxicology, National Center for Toxicological Research. Jefferson, Arkansas. Supported by the U.S. Army Medical Bioengineering Research and Development Laboratory, Fort Detrick, MD. Army Project No. 83-PP3812. FDA No. 224-83-0004.

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1987. Vol. 3A. National Institute for Occupational Safety and Health (NIOSH). U.S. Department of Health and Human Services.

SULFATE

Low doses of sulfate are absorbed at a high rate from the gastrointestinal tract, whereas larger doses may exceed absorptive capacity and are subsequently excreted in the urine (EPA 1985). Sulfate has few effects on humans and animals except at high doses. The primary toxic effects of excessive sulfate ingestion are attributable to the cathartic effects of the chemical, although some injury to the kidney and intestinal mucosa may occur. Human infants appear to be more sensitive to excess sulfate than adults. Chien et al. (1968) reported three case histories of infants who suffered from gastric distress due to consumption of formula containing 630 to 1,150 mg/liter sulfate. In chickens, very high amounts of sulfate consumed in drinking water have been shown to cause renal damage (Adams et al. 1975). There is no evidence that sulfate is mutagenic, carcinogenic or teratogenic. EPA (1985) has recommended a guidance level of 400 mg/liter sulfate in drinking water to protect infants from severe gastric distress. In addition, a secondary drinking water standard of 250 mg/liter, based upon aesthetic considerations has been recommended (EPA 1985). The U.S. Army has recommended limits for sulfate of 100 or 300 mg/liter for water consumption of 5 or 15 liters/day, respectively (Scofield and Hsieh ND).

REFERENCES

ADAMS, A.W., CUNNINGHAM, F.E., AND MUNGER, L.L. 1975. Some effects of layers of sodium sulfate and magnesium sulfate in their drinking water. Poult. Sci. 54:707-714 (As cited in EPA 1985)

CHIEN, L., ROBERTSON, H., AND GERRARD, J.W. 1968. Infantile gastroenteritis due to water with high sulfate content. Can. Med. Assoc. J. 99:102-104. (As cited in EPA 1985)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Drinking Water Criteria Water Regulations. Washington D.C. Federal Register vol 50, No. 219:46979-80.

SCOFIELD R, HSIEH D. ND. Criteria and Recommendations for standards for sulfate in military field water supplies. Univ of Calif. (as cited in EPA 1985)

SULFIDE SALTS

Sulfide salts are absorbed following oral and dermal exposure. Ingested sulfide salts are decomposed to hydrogen sulfide by gastric acid resulting in systemic poisoning (Gosselin et al. 1984). Alkaline sulfides are also strong local irritants to mucous membranes and skin (Gosselin et al. 1984). When sulfide salts come in contact with body fluids, they are completely hydrolyzed (Haggard 1921). Therefore, there is no toxicological distinction between sulfide salts and hydrogen sulfide. Sulfides affect the carotid body chemoreceptors to stimulate respiration (Anichkov and Belen'kii 1963); this invariably leads to central respiratory paralysis and ultimately death (Haggard et al. 1922). Sulfides can also cause a transient rise and sudden fall in blood pressure (Heymans et al. 1932). No health-based criteria are available for sulfide salts.

REFERENCES

ANICHKOV, S.V., and BELEN'KII, M.L. 1963. *Pharmacology of the Carotid Body Chemoreceptors.* The MacMillan Co., New York

GOSSELIN, R.E., SMITH, R.P., and HODGE, H.C. 1984. *Clinical Toxicology of Commercial* Products. 5th Edition. Williams and Wilkins, Baltimore

HAGGARD, H.W. 1921. The fate of sulfides in the blood. J. Biol. Chem 44:519-529

HAGGARD, H.W., HENDERSON, Y., CHARLTON, T. 1922. The influence of hydrogen sulfide upon respiration. Am. J. Physiol. 61:289-297

HEYMANS, C., BOUCKAERT, J.J., VON EULER, U.S., and DAUTREBANDE, L. 1932. Sinus carotidiens et reflexes vasomoteur. Arch. Int. Pharmacodyn. Ther. 43:86-110

TABUN (GA)

INTRODUCTION

Tabun, Agent GA, was the first nerve agent developed for chemical warfare; it contains cyanide instead of fluoride in its chemical structure (Chemical Stockpile Disposal Program 1988). Tabun is a lethal anticholinesterase and blister agent which is usually odorless, colorless, and tasteless. Tabun is highly toxic in both liquid and vapor forms, but is less toxic than the nerve agents sarin and VX (Chemical Stockpile Disposal Program 1988). Tabun is less volatile than sarin and would be expected to persist on the skin and in the environment somewhat longer. Under environmental conditions tabun is hydrolyzed to hydrogen cyanide (HCN) (Chemical Stockpile Disposal Program 1988).

TOXICOKINETICS

Tabun is an organophosphate compound which is highly lipid soluble and can be absorbed via inhalation, topical contact or ingestion. The rate of tabun detoxification proceeds at a slow rate by tabunase, which has been identified in several species, including humans (Chemical Stockpile Disposal Program 1988).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

The human toxicity by inhalation of tabun is approximately half that for sarin; this difference is well supported by animal data. Tabun appears to be more toxic to the ciliary muscles of the eyes than sarin because constriction of pupils occurs at a lower concentration of tabun [i.e., minimum effective doses (ECt_{50}) of 0.9 and 2 to 4 mg-min/m^3, respectively] (Chemical Stockpile Disposal Program 1988). For humans, the median lethal concentration (LCt_{50}) ranges from 135-150 mg-min/m^3 (mildly activity) to 200-400 mg-min/m^3 (resting) depending on physical exertion. The incapacitating dose (ICt_{50}) ranges from 100 mg-min/m^3 (mild activity) to 300 mg-min/m^3 (resting). The human LClo is 150 mg/m^3, and the rat LC_{50} is 304 mg/m^3 for a 10 minute exposure; the rat oral LD_{50} is 3.7 mg/kg (RTECS 1987). Respiratory doses and liquid tabun in the eye are lethal in 1 to 10 minutes while lethality varies for dermal absorption from 1-2 minutes to 1-2 hours (Army 1975).

Tabun differs from other G nerve agents in some of its biochemical effects on the brains of exposed animals and also in the rarity of tabun-induced convulsions, even at lethal doses (Chemical Stockpile Disposal Program 1988). Tabun has been found to cause acute motor and learning behavior effects in rats at doses of between 54 and 71% of the LD_{50}, with certain behavioral changes occurring the absence of obvious symptoms of tabun exposure (Chemical Stockpile Disposal Program 1988).

The clinical signs of tabun poisoning result from effects on smooth muscle and glands (drooling, excessive sweating, vomiting, diarrhea, involuntary urinations), effects on skeletal muscles (easy fatigue, weakness, muscle twitching) and effects on the CNS (headache, confusion, convulsions, and coma). Respiratory failure, the immediate cause of death appears to result primarily from depression of the brain's respiratory center (Chemical Stockpile Disposal Program 1988). Nerve agents such as tabun interfere with acetylcholinesterase (AChE) to cause an increase in acetylcholine throughout the body. The subsequent accumulation of acetylcholine in the neuronal synapse results in constant stimulation of the post-synaptic acetylcholine receptors, causing an increase in parasympathetic tone (Chemical Stockpile Disposal Program 1988). Acetylcholine is vital in the innervation of skeletal muscles, autonomic ganglia, and many structures within the central nervous system.

Information is limited on the possible chronic health effects from prolonged low-level exposure to tabun. It appears that tabun could induce delayed peripheral neuropathy. Despite the fact that tabun

contains a cyanide group instead of fluoride, tabun does not show in vitro inhibition of neurotoxic esterase (NTE) (Chemical Stockpile Disposal Program 1988). Intramuscular injections of the highest tolerated dose of tabun to adult laying hens for both acute and long-term (90 days) exposure did not induce behavioral or neurohistological evidence of organophosphate induce delayed neuropathy (OPIDN) (Henderson et al. 1989). It is postulated that even higher doses of tabun would be necessary to induce clinical signs of delayed peripheral neuropathy, and at these massive concentrations, death would more likely results prior to the onset of delayed neuropathy.

Tabun has been extensively evaluated for mutagenicity and it appears to be a weak mutagen. A linear dose-mutation response was observed in the point mutation assay at the thymidine kinase locus in mouse lymphoma cells exposed to tabun without rat liver S-9 activation (Kawakami et al. 1989a). Addition of rat liver S-9 did not enhance the dose-mutation response. Tabun was toxic to cultured chinese hamster ovary (CHO) cells at high levels (200 µg/ml) and behaved as a weak mutagen in the sister chromatid exchange (SCE) assay (Nasr et al. 1988). Chromatid exchanges increased linearly with tabun concentration but the number of exchanges never exceeded twice the number of the controls. Tabun was not genotoxic in the unscheduled DNA synthesis (UDS) assay as rat primary hepatocytes exposed to increasing concentrations of tabun had decreased UDS (Kawakami et al. 1988). In the bacterial (Ames) mutagenicity assay, tabun was not cytotoxic with metabolic activation but exhibited a slightly significant positive dose-response in 8/11 trials in tester strains TA 98, 100, 1535, and 1537 (Goldman et al. 1989). Tabun injected intraperitoneally in C57B1/6 mice at a sublethal dose of 700 µg/kg did not induce the SCE rate in the chromosomes of splenic lymphocytes (Kawakami et al. 1989b). In vitro, tabun was cytotoxic and inhibited oxygen consumption by the cell at millimolar levels, similar to free cyanide.

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

No regulated standards or criteria have been promulgated for tabun (GA).

REFERENCES

ARMY. 1975. Military Chemistry and Chemical Compounds Field Manual. Department of the Army and Air Force, Washington, D.C. October 1975. FM 3-9, AFR 355-7.

CHEMICAL STOCKPILE DISPOSAL PROGRAM. 1988. FINAL PROGRAMMATIC ENVIRONMENTAL IMPACT STATEMENT. January. Program Executive Officer--Program Manager for Chemical Demilitarization. Aberdeen Proving Ground, MD

GOLDMAN M., M. NASR, N. CONE, L.S. ROSENBLATT, B.W. WILSON. 1989. Toxicity studies on Agent GA. Mutagenicity of tabun (GA) in the ames mutagenicity assay. Laboratory for Energy-Related Health Research, Univ of Calif. U.S. Army Medical Research and Development Command. Fort Detrick. Frederick, MD.

HENDERSON J.D., R.J. HIGGINS, L. ROSENBLATT, B.W. WILSON. 1989. Toxicity studies on Agent GA. Delayed neurotoxicity- acute and repeated exposures of GA (tabun). Laboratory for Energy-Related Health Research, Univ of Calif. U.S. Army Medical Research and Development Command. Fort Detrick. Frederick, MD.

KAWAKAMI T.G., M. GOLDMAN, L. ROSENBLATT, B.W. WILSON. 1989a. Toxicity studies in agent GA. Mutagenicity of Agent GA (tabun) in the mouse lymphoma assay. Laboratory for Energy-Related Health Research, Univ of Calif. U.S. Army Medical Research and Development Command. Fort Detrick. Frederick, MD.

KAWAKAMI T.G., M. NASR, N. CONE, L. ROSENBLATT, M. GOLDMAN, B.W. WILSON. 1989b. Toxicity studies in agent GA. In vivo sister chromaide exchanges assay. Laboratory for Energy-Related Health Research, Univ of Calif. U.S. Army Medical Research and Development Command. Fort Detrick. Frederick, MD.

KAWAKAMI T.G., N. CONE, J.D. HENDERSON, M. GOLDMAN, L. ROSENBLATT, B.W. WILSON. 1988. Toxicity studies in agent GA. Unscheduled DNA synthesis in rat hepatocytes after exposure to tabun (GA). Laboratory for Energy-Related Health Research, Univ of Calif. U.S. Army Medical Research and Development Command. Fort Detrick. Frederick, MD.

NASR M., N. CONE, T.G. KAWAKAMI, M. GOLDMAN, L. ROSENBLATT, B.W. WILSON. 1988. Toxicity studies on Agent GA (Phase I). Mutagenicity of Agent GA (Tabun) in the in vitro cytogenetic sister chromatid exchange test. Laboratory for Energy-Related Health Research, Univ of Calif. U.S. Army Medical Research and Development Command. Fort Detrick. Frederick, MD.

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1987. Vol. 3A. National Institute for Occupational Safety and Health (NIOSH). U.S. Department of Health and Human Services.

TELVAR/MONURON

INTRODUCTION

Telvar, (3-(4-chlorophenyl)-1,1-dimethylurea), more commonly known as monuron, is a broad-spectrum herbicide that was widely used for soil application and for control of many grasses and herbaceous weeds on non-cropland areas, such as industrial sites and drainage ditch banks (Gosselin et al. 1984, IARC 1976). In addition, telvar was registered for selective control of weeds in several crops such as sugar cane, citrus, pineapple, cotton, and asparagus (Hayes 1982). Resides of 1-7 mg/kg were tolerated on a variety of fruits and vegetable (IARC 1976). In July 1973, the use of telvar was curtailed in the U.S. due to its high oncogenic potential (Gosselin et al. 1984, IARC 1976). Telvar is available as a technical grade product, and as a granular formulation containing both telvar and trichloroacetic acid (IARC 1976).

TOXICOKINETICS

Telvar is absorbed from the gastrointestinal and respiratory systems. Dermal absorption is unknown (Hayes 1982). In rats given 875 mg/kg orally, peak blood concentrations occurred 2 hours after dosing; thereafter, the compound was distributed evenly throughout the body. Telvar-related material was excreted in the urine and secreted into milk of lactating animals. After administration of 175 mg/kg-day for 60 days or of 0.1-20 mg/kg-day for 6 months, tissue retention of telvar metabolites occurred in the lungs > heart > liver, brain and kidneys > mild, bone-marrow and thyroid galnd (Fridman 1968). In mammals, telvar is metabolized (i) by oxidative N-demethylation, (ii) by hydroxylation of the aromatic nucleus and (iii) by fission of the urea residue to give choroaniline derivatives (Ernst 1969, Ernst and Bohme 1965). Hydroxylation of the 2-position is favored rather than the 3-position. Phenolic metabolites are excreted in the urine as conjugates (Ernst 1969, Ernst and Bohme 1965). The majority of metabolites excreted in the urine retain the urea configuration and result from hydroxylation and dealkylation of the parent compound (Hayes 1982).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

Acute oral toxicity was manifested as dose-related ataxia, drowsiness, tachypnea, dyspnea, irritability, hyperrefexes, diarrhea, diuresis, hypothermia, glycosuria, proteinuria and aciduria; death was attributed to cardiac or respiratory failure (Hayes 1982, Gosselin et al. 1984). Autopsies revealed gastroenteritis, fatty necrosis of the liver, renal and splenic pallor and degenerative changes in kidneys, muscle, salivary glands and testes (Gosselin et al. 1984). The toxicity of telvar has been shown in rats to be exacerabated by protein-deficient diets. Repeated doses in rats produce anemia, liver damage and methemoglobinemia, the latter is due to the formation of p-chloroaniline by metabolic hydrolysis (Gosselin et al. 1984).

In a chronic dietary bioassay, the presence of an abnormal pigment, thought to be sulfhemoglobin, correlated with the extent of hemolysis and hemosiderin deposits (Gosselin et al. 1984). In chronic feeding studies concentrations of 2,500 ppm for 2 years to rats and dogs showed liver enlargement, but no histological changes, and no evidence of cancinogenicity (Hayes 1982). Diuron causes dose-related microsomal enzyme induction.

The available data suggest that telvar is carcinogenic. Telvar has been tested in mice and rats by oral administration and in mice by single subcutaneous injection. Male mice orally exposed to commercial grade telvar had an increased ($p<0.05$) incidence of lung tumors (6/16 compared with 9/30 for controls) (Innes et al. 1969). In a subsequent study in mice, an increased incidence of liver tumors

was observed, but survival rates in controls were not reported. Oral administration of telvar to male rats resluted in tumors at various sites; none were observed in concurrent controls (IARC 1976).

In a 13-month study, mice of two respective strains were adminstered telvar via milk (Rubenchik et al. 1970). Tumors occurred in 13/13 mice of one bred, and 7/25 mice of the other strain while controls had 0 and 1 tumors, respectively; however survival rates among control rats were not reported. The same investigators examined 50 randon-bred male rats adiministered dietary concentrations of 450 mg/kg-day for 18 months. Tumors occurred in 14/50 rats; stomach tumors, intestinal tumor, liver-cell carcinomas, alveolar carcinomas, and other lung carcinomas were observed compared to none in controls.

There is evidence to suggest that telvar induces effects on fertility, and on the embyro or fetus (RTECS 1987). Pre-implantation mortality, fetotoxicity, extra embyronic structures, and ear, eye and craniofacial abnormalities were among those reported (RTECS 1987). There were no untoward findings in a three-generation study in rats maintained at a dietary level of 125 ppm (6 mg/kg-day) (Hayes 1982).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

No regulated standards or criteria have been promulgated for telvar (monuron).

REFERENCES

ERNST, W. 1969. Metabolism of substituted dinitrophenols and ureas in mammals and methods for the isolation and identification of metabolites. J. Sud-Afrik Chemi Instit 22:S79-S88 (as cited in IARC 1976).

ERNST, W., C. BOHME. 1965. Uber den Stoffwechsel von Harnstoff-Herbiciden in der Ratte. I. Monuron und Aresin. Fd Cosmet Toxicol 3:789-796. (as cited in IARC 1976).

FRIDMAN, E.B. 1968. Distribution and accumulation of monuron in animals, and methods of its excretion its excretion. Zdravookhr. Turkm. 12:25-27 (as cited in IARC 1976).

HAYES W.J. 1982. Pesticides Studied in Man. Williams and Wilkins, Baltimore/London. pp. 540-541.

INNES, J.R.M., B.M. ULLAND, M.G. VALERIO, I. PETRUCELLI, L. FISHBEIN, E.R. HART, A.J. PALLOTTA, R.R. BATES, H.L. FALK, J.J. GART, M. KLEIN, I. MITCHELL, J. PETERS. 1969. Bioassay of pesticides and industrial chemicals for tumorigenicity in mice: a preliminary note. J Natl Cancer Instit. 42:1101-1114.

INTERNATIONAL AGENCY FOR RESEARCH ON CARCINOGENS (IARC). 1976. IARC monographs on the evaluation of the carcinogenic risk of chemicals to man: Some Carbamates, Thiocarbamates and Carbazides. Vol 12, pgs. 167-174.

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1987.

RUBENCHIK, B.L., N.E. BOTSMAN, G.P. GORBANJ. 1970. On carcinogenic effect of herbicide monuron. Vop. Onkol. 16:51-53 (as cited in IARC 1976).

In humans, absorption of a single inhalation dose of 1,1,2,2,-tetrachloroethane vapor was reported to be 97%; absorption of this chemical from the gastrointestinal tract is inferred from studies in which an increased incidence of liver tumors was reported in mice exposed in the diet (EPA 1984). The effects associated with occupational exposure to 1,1,2,2-tetrachloroethane by inhalation or dermal routes are primarily neurological and include, tremors, headache, numbness, excessive perspiration, and anorexia (EPA 1984). In experimental animals, subchronic inhalation exposure to 1,1,2,2-tetrachloroethane is associated with liver effects, decreased hemoglobin content of red blood cells, decreased hematocrit, and fluctuations in white blood cell count (Schmidt et al. 1972, Navrotskiy et al. 1971, Horiuchi et al. 1962). 1,1,2,2-Tetrachloroethane is a liver carcinogen when administered orally to mice (NCI 1978).

EPA (1990) classified 1,1,2,2-tetrachloroethane in Group C--Possible Human Carcinogen based on increased incidence of hepatocellular carcinoma in mice. EPA (1990) developed an oral cancer potency factor of 0.2 $(mg/kg\text{-}day)^{-1}$ based on the study conducted by NCI (1978) in which a highly significant dose-related increase in the incidence of hepatocellular carcinomas was observed in both male and female mice. An inhalation cancer potency factor of 0.2 $(mg/kg\text{-}day)^{-1}$ was also calculated from these data (EPA 1990). EPA (1987) has also derived an interim oral reference dose (RfD) of 4.6×10^{-4} mg/kg-day for 1,1,2,2-tetrachloroethane based on an inhalation study by Schmidt et al. (1972) in which rats were exposed to 1,1,2,2-tetrachloroethane vapor for 5 hours/day for 265 days. In this study decreased body weight, increased white blood cell count and increased hepatic fat content were observed. Using a LOAEL of 0.456 mg/kg-day and applying a safety factor of 1,000 the interim RfD was derived.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for 1,1,2,2-Tetrachloroethane. Environmental Criteria Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-032

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

HORIUCHI, K., HORIGUCHI, K., KADOWAKI, K., and ARATAKE, K. 1962. Studies on the industrial tetrachloroethane poisoning. Osaka City Med. J. 8:29-38

NAVROTSKIY, V.K., KASHIN, L.M., KULINSKAYA, I.L., ET AL. 1971. Comparative assessment of the toxicity of a number of industrial poisons when inhaled in low concentrations for prolonged periods. Trudy S'ezda Gigenistov Ukranisoi 8:224-226 (Russian)

NATIONAL CANCER INSTITUTE (NCI). 1978. Biassay of 1,1,2,2-Tetrachloroethane for Possible Carcinogenicity. NCI Tech. Rep. Ser. No. 27

SCHMIDT, P., BINNEWIES, S., GOHLKE, R., and ROTHE, R. 1972. Subacute action of low concentrations of chlorinated ethanes on rats with and without additional ethanol treatment. I. Biochemical and toxicometric aspects, especially results in subacute and chronic toxicity studies with 1,1,2,2-tetrachloroethane. Int. Arch. Arbeitsmed. 30:283-298 (German)

TETRACHLOROETHENE

Tetrachloroethene is absorbed following inhalation (IARC 1979) and oral (EPA 1985a,b) exposure. Tetrachloroethene vapors and liquid also can be absorbed through the skin (EPA 1985a,b). The principal toxic effects of tetrachloroethene in humans and animals following acute and longer-term exposures include central nervous system (CNS) depression and fatty infiltration of the liver and kidney with concomitant changes in serum enzyme activity levels indicative of tissue damage (EPA 1985a,b, Buben and O'Flaherty 1985). Humans exposed to doses of between 136 and 1,018 mg/m^3 for 5 weeks develop central nervous system effects, such as lassitude and signs of inebriation (Stewart et al. 1974). The offspring of female rats and mice exposed to high concentrations of tetrachloroethene for 7 hours daily on days 6-15 of gestation developed toxic effects, including a decrease in fetal body weight in mice and a small but significant increase in fetal resorption in rats (Schwetz et al. 1975). Mice also exhibited developmental effects, including subcutaneous edema and delayed ossification of skull bones and sternebrae (Schwetz et al. 1975). In a National Cancer Institute bioassay (NCI 1977), increased incidences of hepatocellular carcinoma were observed in both sexes of B6C3F1 mice administered tetrachloroethene in corn oil by gavage for 78 weeks. Increased incidences of mononuclear cell leukemia and renal adenomas and carcinomas (combined) have also been observed in long term bioassays in which rats were exposed to tetrachloroethene by inhalation (NTP 1986).

EPA (1990b) classifies tetrachloroethene as a Group B2 carcinogen (Probable Human Carcinogen). EPA (1990b) has derived an oral slope factor of $5x10^{-2}$ $(mg/kg\text{-}day)^{-1}$ based on liver tumors observed in the NCI (1977) gavage bioassay for mice. The inhalation cancer potency factor for tetrachloroethene of $3.3x10^{-3}$ $(mg/kg\text{-}day)^{-1}$ is based on an NTP (1986) bioassay in rats and mice in which leukemia and liver tumors were observed (EPA 1990b). Both cancer potency factors are currently under review by EPA (1990a). EPA (1990a,b) has also derived an oral reference dose (RfD) of $1x10^{-2}$ mg/kg-day for tetrachloroethene based on a 6-week gavage study by Buben and O'Flaherty (1985). In this study, mice and rats suffered significantly increased liver weight/body weight ratios and hepatotoxicity when treated with 71 mg/kg-day tetrachloroethene. Effects were not seen in animals treated with 14 mg/kg-day. The RfD was derived using a NOAEL of 14 mg/kg-day and applying an uncertainty factor of 1,000. EPA (1990b) established a subchronic oral RfD of $1x10^{-1}$ mg/kg-day, using an uncertainty factor of 100 and based on the same study and effect of concern.

REFERENCES

BUBEN, J.A., and O'FLAHERTY, E.J. 1985. Delineation of the role of metabolism in the hepatotoxicity of trichloroethene and perchloroethene: A dose-effect study. Toxicol. Appl. Pharmacol. 78:105-122

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Health Assessment Document for Tetrachloroethene (Perchloroethene). Office of Health and Environmental Assessment, Washington, D.C. July 1985. EPA 600/8-82-005F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. Drinking Water Criteria Document for Tetrachloroethene. Office of Drinking Water, Criteria and Standards Division, Washington, D.C. June 1985

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Remedial Response, Washington, D.C. July 1990

INTERNATIONAL AGENCY FOR RESAERCH ON CANCER (IARC). 1979. IARC Monographs on the evaluation of the carcinogenic risks of chemicals to humans. Vol. 20: Some Halogenated Hydrocarbons. World Health Organization, Lyon France

NATIONAL CANCER INSTITUTE (NCI). 1977. Bioassay of Tetrachloroethene for Possible Carcinogenicity. CAS No. 127-18-4. NCI Carcinogenesis Technical Report Series No. 13, Washington, D.C. DHEW (NIH) Publication No. 77-813

NATIONAL TOXICOLOGY PROGRAM (NTP). 1986. Toxicology and Carcinogenesis Studies of Tetrachloroethene (Perchloroethene) (CAS No. 127-18-4) in F344/N Rats and B6C3F1 Mice (Inhalation Studies). NTP Technical Report Series No. 311, Research Triangle Park, North Carolina. DHEW (NIH) Publication No. 86-2567

SCHWETZ, B.A., LEONG, B.K.J., and GEHRING, P.J. 1975. The effect of maternally inhaled trichloroethene, perchloroethene, methyl chloroform, and methene chloride on embryonal and fetal development in mice and rats. Toxicol. Appl. Pharmacol. 55:207-219

STEWART, R.D., HAKE, C.L., FORSTER, H.V., LEBRUN, A.J., PETERSON, J.F., and WU, A. 1974. Tetrachloroethene: Development of a biologic standard for the industrial worker by breath analysis. Medical College of Wisconsin, Milwaukee, Wisconsin. NIOSH-MCOW-ENUM-PCE-74-6

Tetryl is absorbed through oral, inhalation, and dermal routes. It is a skin and mucous membrane irritant, with the most common reaction being skin sensitization and dermatitis. Industrial exposure to tetryl has caused severe upper respiratory tract irritation with coughing and epistaxis, edema, headache, irritability, malaise, lassitude, sleeplessness, and conjunctivitis (Witkowski et al. 1942). Dermal exposure may stain the skin and hair yellow (Hamilton and Hardy 1974). Additionally, heavy airborne exposure to tetryl may cause liver damage (Hardy and Maloof 1950; Schwartz 1942). Acute exposure to tetryl in rabbits (via gavage) and dogs (subcutaneously) led to severe acute inflammation at the injection site, varying degrees of edema and hemorrhaging, degeneration of muscle tissue, and toxic degeneration of the kidneys. Dog livers showed varying degrees of necrosis in the centers of the lobules and severe fatty degeneration of the liver cells. Rabbit livers showed almost no changes (Wells 1920). Tetryl appears to be a potent, direct-acting mutagen in three microbial mutagenicity test systems (*Neurospora crassa, Salmonella typhimurium, and Saccharomyces cerevisiae* D_4)even at low concentrations (Whong et al. 1980). EPA has not derived health-based criteria for tetryl.

REFERENCES

HAMILTON, A., HARDY, H. 1974. Industrial Toxicology, 3rd. ed., p. 308. Pub. Sciences Group, Inc., Acton, MA.

HARDY, W.L., MALOOF, C.C. 1950. Evidence of systemic effect of tetryl. AMA Arch. Ind. Hyg. Occup. Med. 1:545.

SCHWARTZ, L. 1942. Industrial dermatitis in our war industries. Ind. Med. 11(10):457.

WELLS, H.G., LEWIS, J.H., SANSUM, W.D. ET AL. 1920. Observations on the toxicity of teranitromethyl aniline (Tetryl), tetranitroxylene (T.N.X.), tetranitroaniline (T.N.A.), dinitrodichlorobenzene, parazol, and metanitroaniline. J. Ind. Hyg. 2:247.

WHONG, W.-Z., SPECINER, N.D., AND G.S. EDWARDS. 1980. Mutagenic activity of tetryl, a nitroaromatic explosive, in three microbial test systems. Toxicology Letters 5:11-17.

WITKOWSKI, L.J. FISHER, C.N., MURDOCK, H.D. 1942. Industrial illness due to tetryl. JAMA 119(17):1406.

THALLIUM

Thallium and its salts are readily and rapidly absorbed through the skin, lungs, and mucous membranes of the mouth and gastrointestinal tract. Percutaneous absorption has also been reported to occur through rubber gloves (Rumack 1986). Thallium is acutely toxic to humans regardless of the chemical form of the compound or route of administration. Hundreds of cases of thallotoxicosis due to ingestion of thallium-based pesticides have been reported (ACGIH 1986). Children poisoned by thallium ingestion have exhibited neurological abnormalities including mental retardation and psychoses (ACGIH 1986). The effects of thallium toxicity are similar in humans and animals. The most commonly noted response to thallium exposure is alopecia, but neurological and gastrointestinal findings are frequently found. Such effects include ataxia, lethargy, painful extremities, peripheral neuropathies, convulsions, endocrine disorders, psychoses, nausea, vomiting, and abdominal pains (Bank 1980). It has been noted that the degree and duration of exposure to thallium and its salts can influence the clinical picture of thallium intoxication. Subchronic feeding studies conducted with rats observed marked growth depression and a nearly complete loss of hair (EPA 1986, Clayton and Clayton 1981). Exposure to thallium salts during critical developmental stages in chicks and rats has been reported to be associated with the induction of adverse developmental outcomes (Karnofsky et al. 1950). Pre- and postnatally exposed rat pups have exhibited hydronephrosis, fetal weight reduction and growth retardation (Clayton and Clayton 1981, Gibson and Becker 1970). Thallium has also been shown to cross the placenta and, presumably, enter the fetal blood system (Clayton and Clayton 1981). Thallium has not been demonstrated to be carcinogenic in humans or experimental animals and may have some antitumor activity (Clayton and Clayton 1981).

EPA (1990b) developed an oral reference dose (RfD) of 7×10^{-5} mg/kg-day for thallium in soluble salts based on a subchronic feeding study in which rats received 0.20 mg thallium/kg-day administered as thallium sulfate (MRI 1986, EPA 1986). Increased blood chemistry parameters (SGOT and serum LDH) and alopecia were observed. An uncertainty factor of 3,000 was used to calculate the RfD. EPA (1990a) also derived oral RfDs for certain thallium salts (i.e., thallium acetate, thallium carbonate, thallium chloride, thallium nitrate, thallium selenite and thallium sulfate) of between $8\text{-}9 \times 10^{-5}$ mg/kg-day based on the same EPA (1986) 90-day subchronic rat study. The same endpoints of toxicity were observed and an uncertainty factor of 3,000 was used to derive the RfDs.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. ACGIH, Cincinnati, Ohio

BANK, W.J. 1980. Thallium. In Spencer, P.S., and Schaumberg, H.H., eds. Experimental and Clinical Neurotoxicology. Williams and Wilkins, Baltimore. P. 571

CLAYTON, G.D., and CLAYTON, F.E., eds. 1981. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York. P. 1916

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Subchronic (90-day) toxicity of thallium (I) sulfate in Sprague-Dawley rats. Final Report. Prepared for the Office of Solid Waste, U.S. EPA, Washington, D.C. Project No. 8702-1(18)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Washington, D.C. July 1990

GIBSON, J.E., and BECKER, B.A. 1970. Placental transfer, embryo toxicity, and teratogenicity of thallium sulfate in normal and potassium-deficient rats. Toxicol. Appl. Pharmacol. 16:120-132

KARNOFSKY, D.A., RIDGWAY, L.P., and PATTERSON, P.A. 1950. Production of achondroplasia in the chick embryo with thallium. Proc. Soc. Exp. Biol. Med. 73:255-259

MIDWEST RESEARCH INSTITUTE (MRI). 1986. Subchronic (90-day) toxicity study of thallium sulfate in Sprague-Dawley rats. Office of Solid Waste. U.S. EPA Washington, D.C

RUMACK, B.H., ed. 1986. Poisindex. Microfiche ed. Micromedix, Inc., Denver, Colorado, in association with the National Center for Poison Information, with updates, 1975-present

THIODIGLYCOL

Thiodiglycol, a hydrolysis product of mustard gas, is reported to have low toxicity (Hawley 1981). It can be inferred from the available animal toxicity studies that thiodiglycol is absorbed following acute exposure. In humans, thiodiglycol is a urinary metabolite of mustard gas. Very little toxicological information is available on thiodiglycol. It has been shown to be a mild skin and eye irritant in animal studies (ICF Feasibility Study, 1987). The acute oral LD_{50} in rats is 6,610 mg/kg (Smyth et al. 1941). Thiodiglycol, unlike mustard gas, has no effect on the cardiovascular system in rabbits or dogs (Anslow et al. 1946). Because of its low toxicity thiodiglycol is not a contaminant of concern; concentrations resulting from the hydrolysis of mustard gas in the environment are unlikely to be toxicologically significant (RCRA, ND). No health-based criteria have been derived by EPA.

REFERENCES

ANSLOW, W.P. and HOUCK, C.R. 1946. Systemic pharmocology and pathology of sulfer and nitrogen mustards. In: Chemical Warfare Agents, and Related Chemical Problems, Chapter 22, Summary of Technical Report of Division 9, NDRC, 440-478 and 731- 737. (as cited in Rossenblatt et al., 1975)

HAWLEY. 1981. (as cited in RCRA Facility Assessment, ND)

ICF Feasibility Study. 1987.

RCRA Facility Assessment. ND. Edgewood Area, AP6, MD.

ROSSENBLATT, D., MILLER, T., DACRE, J., MAJ, I., COGLEY, D. 1975. Problem definitions on potential environmental pollutants II. physical, chemical, toxicological, and biological, properties of 16 substances. Fredrick, MD. United States Medical Research and Development Laboratory.

SMYTH, H., SEATON, J., FISCHER, L. 1941. The single dose toxicity of some glycols and derivatives. J. Industr. Hyg. Toxicol. 23:259-268. (as cited in Rosenblatt et al., 1975)

TITANIUM AND COMPOUNDS

Titanium (Ti) is a reactive metallic element which occurs most often in the +4 oxidation state (titanic), but the +3 and +2 oxidation states do occur in several organometallic compounds (Goyer 1986). Following an oral exposure to titanium, approximately 3 percent is absorbed into the bloodstream, the majority of which is excreted in the urine (Goyer 1986). Although occupational exposure to titanium may be heavy, with concentrations in air up to 50 mg/m^3, the metal and its inorganic salts are relatively nontoxic (Goyer 1986). Virtually no toxicological information is available on titanium metal due to its physiological inertness, however some of its compounds are highly injurious (Stokinger 1981). The most common titanium compound, titanium dioxide (TiO_2), has been classified as a nuisance particulate (Goyer 1986). Slight fibrosis of lung tissue has been reported following inhalation exposure to TiO_2; otherwise TiO_2 is considered physiologically inert by all routes (ingestion, inhalation, dermal, and subcutaneous) (Goyer 1986). Titanium tetrachloride ($TiCl_4$), however, is extremely toxic in humans and animals; acute exposure to $TiCl_4$ vapor can cause severe irritation and damage to eyes, coughing, headache, dizziness, lung damage, and bronchial pneumonia (Weiss 1986). Dermal exposure to liquid $TiCl_4$ causes thermal and acid burns of exposed areas, while ingestion causes nausea, vomiting, cramps, diarrhea, and possible tissue ulceration (Weiss 1986). In acute inhalation studies involving dogs, $TiCl_4$ caused respiratory distress, vomiting, intense bronchitis and some edema following 3 days of exposure (Stokinger 1981). Dogs exposed for longer periods of time (9 weeks) to an average concentration of 6.8 ppm $TiCl_4$ displayed numerous monocytes, necrotic cells, and brown Ti granules grouped around the bronchi of the lung (Stokinger 1981). These lesions resulted in reduced pulmonary capacity and increased susceptiblilty to infection (Stokinger 1981). Titanium carbide (TiC) was found to be associated with pulmonary fibrosis in workers occupationally exposed in the manufacture of cutting edges for tools (Menzel and Amdur 1986). There is equivocal evidence that titanium is tumorigenic in rats, causing blood lymphomas (including Hodgkin's disease) and tumors at the site of application (RTECS 1987). Titanocene, an organo-titanium compound, caused a variety of tumors, hepatomas, fibrosarcoma, lymphoblastoma when injected intramuscularly into tumor-resistant mice; titanium dioxide, however, showed no evidence of carcinogenicity in rats and mice at dietary levels of 25,000 and 50,000 ppm for 103 weeks (Stokinger 1981). No health-based criteria have been derived by EPA for titanium and its compounds.

REFERENCES

GOYER R.A. 1986. The toxic effects of metals. In Casarett and Doull's Toxicology. 3rd ed. Macmillan Publishing Co., New York. Pp 627-8.

MENZEL D.B. AND AMDUR M.O. 1986. Toxic responses of the respiratory system. In Klaassen C.D., Amdur M.O., and Doull J., eds. Casarett and Doull's Toxicology. 3rd ed. Macmillan Publishing Co., New York. Pp 348.

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1987. Vol. 3A. National Institute for Occupational Safety and Health (NIOSH). U.S. Department of Health and Human Services.

STOKINGER, H.E. 1982. The metals. In Patty's Industrial Hygiene and Toxicology. Clayton, G.D. and Clayton, P.B., eds. 3rd ed. John Wiley and Sons, New York. Vol. 2C. Pp 1968-1980.

WEISS G. 1986. Hazardous chemicals data book. 2nd ed. Noyes Data Corporation, Park Ridge, New Jersey. Pp 966.

WILLIAMS G.M. AND WEISBURGER J.H. 1986. Chemical carcinogens. In Casarett and Doull's Toxicology. 3rd ed. Macmillan Publishing Co., New York. Pp 348.

TOLUENE

Toluene is absorbed in humans following both inhalation and dermal exposure (EPA 1985). In humans, the primary acute effects of toluene vapor are central nervous system (CNS) depression and narcosis. These effects occur at concentrations of 200 ppm (754 mg/m^3) (von Oettingen et al. 1942a,b). In experimental animals, acute oral and inhalation exposures to toluene can result in central nervous system (CNS) depression and lesions of the lungs, liver, and kidneys (EPA 1987). The earliest observable sign of acute oral toxicity in animals is depression of the CNS, which becomes evident at approximately 2,000 mg/kg (Kimura et al. 1971). In humans, chronic exposure to toluene vapors at concentrations of approximately 200 and 800 ppm has been associated with CNS and peripheral nervous system effects, hepatomegaly, and hepatic and renal function changes (EPA 1987, Anderson et al. 1983). Toxic effects following prolonged exposure of experimental animals to toluene are similar to those seen following acute exposure (Hanninen et al. 1976, von Oettingen et al. 1942a). In rats, chronic exposure to toluene via inhalation results in CNS toxicity and a dose-related reduction in hematocrit values (CIIT 1980). The liver and kidney (NTP 1989a) are the target organs in rats following prolonged gavage administration (NTP 1989a). There is some evidence in mice that oral exposure to greater than 0.3 ml/kg toluene during gestation results in embryotoxicity (Nawrot and Staples 1979). Inhalation exposure of up to 1,000 mg/m^3 by pregnant rats during gestation has been associated with significant increases in skeletal retardation (Hudak and Ungvary 1978).

EPA (1990a) has derived an oral risk reference dose (RfD) of 0.2 mg/kg-day for toluene based on a gavage study in which rats were given doses as high as 5,000 mg/kg 5 days/week for 13 weeks and changes in liver and kidney weights were observed (NTP 1989). No adverse effects were observed in any of the treated animals at 223 mg/kg-day (NOAEL). An uncertainty factor of 1,000 was used to calculate the oral RfD. EPA (1990b) derived an oral subchronic RfD of $4x10^{-1}$ based on a gavage study in which rats experienced CNS effects (Wolf et al. 1956). An uncertainty factor of 100 was used to calculate the RfD. EPA (1990b) reported chronic and subchronic inhalation RfDs for toluene of 2 mg/m^3 based on the development of adverse CNS effects in humans (Anderson et al. 1983). An uncertainty factor of 100 was used for both exposures.

REFERENCES

ANDERSON et al. 1983. (Not provided in EPA 1989b)

CHEMICAL INDUSTRY INSTITUTE OF TOXICOLOGY (CIIT). 1980. A Twenty-Four Month Inhalation Toxicology Study in Fischer 344 Rats Exposed to Atmospheric Toluene. Executive Summary and Data Tables. October 15, 1980

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Drinking Water Criteria Document for Toluene. Final Draft. Office of Drinking Water, Washington, D.C. March 1985

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Health Advisory for Toluene. Office of Drinking Water, Washington, D.C. March 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

HANNINEN, H., ESKELININ, L., HUSMAN, K., and NURMINEEN, M. 1976. Behavioral effects of long-term exposure to a mixture of organic solvents. Scand. J. Work Environ. Health 2:240-255 (As cited in EPA 1987)

HUDAK, A., and UNGVARY, G. 1978. Embryotoxic effects of benzene and its methyl derivatives: Toluene, xylene. Toxicology 11:55-63

KIMURA, E.T., EBERT, D.M., and DODGE, P.W. 1971. Acute toxicity and limits of solvent residue for sixteen organic solvents. Toxicol. Appl. Pharmacol. 19:699-704

NAWROT, P.S., and STAPLES, R.E. 1979. Embryo-fetal toxicity and teratogenicity of benzene and toluene in the mouse. Teratology 19:41A

NTP (National Toxicology Program). 1989. Toxicology and carcinogenesis studies of toluene in F344/N rats and B6C3F$_1$ mice. Technical Report Series No. 371. Research Triangle Park, NC.

VON OETTINGEN, W.F., NEAL, P.A., DONAHUE, D.D., et al. 1942a. The Toxicity and Potential Dangers of Toluene, with Special Reference to its Maximal Permissible Concentration. PHS Publication No. 279. P. 50 (As cited in EPA 1987)

VON OETTINGEN, W.F., NEAL, P.A., DONAHUE, D.D., et al. 1942b. The toxicity and potential dangers of toluene—preliminary report. J. Am. Med. Assoc. 118:579-584 (As cited in EPA 1987)

WOLF, M.A., ROWE, V.K., MCCOLLISTER, D.D., et al. 1956. Toxicological studies of certain alkylated benzenes and benzene. Arch. Ind. Health 14:387

1,2,3-TRICHLOROBENZENE

1,2,3-trichlorobenzene (1,2,3-TCB) is used to combat termites. 1,2,3-TCB is one of three isomers of trichlorobenzenes and is usually found as an isomeric mixture (Cameron et al 1937). It can be inferred from the available literature that this compound has a relatively low toxicity. Little information is available on the toxicological properties of 1,2,3-TCB; however, studies indicate that this compound may be an eye and respiratory irritant (Cameron et al. 1937). Exposure may also produce adverse effects on the liver in humans and animals (Cameron et al 1937). Because of its low toxicity, this compound is not a contaminant of concern. No health-based criteria have been derived by EPA.

REFERENCES

CAMERON, G.R., THOMAS, J.C., ASHMORE,S.A. ET AL., 1984. The Toxicity of certain chlorine derivatives of benzene with special reference to o-dichlorobenzene. J. Pathol. Bacteriol. 44:281-196, 1937 In: GOSSELIN, R.E., SMITH, R.P., HODGE, H.C., Clinical Toxicology of Commerical Products, Fifth Edition, Williams and Wilkins, Baltimore, MD.p.173.

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1987. Vol. 3A. National Institute for Occupational Safety and Health (NIOSH). U.S. Department of Health and Human Services.

VERSHUEREN, K., Handbook of Environmental Data on Organic Chemicals. 2nd Edition, Van Nostrand Reinhold Co., New York, 1983. pp. 1121-2.

Information inferred from data describing the toxicity or excretion of trichlorobenzenes suggests that they are absorbed following oral, dermal, and inhalation exposure (EPA 1985). Human exposure to 1,2,4-TCB in air can result in eye and respiratory irritation. The effects in laboratory animals of acute exposure to trichlorobenzenes include local irritations, convulsions, and death. Liver, kidneys, adrenals, mucous membranes, and brain ganglion cells appear to be target organs, with effects including edema, necrosis, fatty infiltration of the liver, increased organ weights, porphyrin induction, and microsomal enzyme induction (EPA 1985). Studies on the toxic effects of trichlorobenzenes following subchronic exposure indicate that, in general, the liver and kidneys are target organs (Kociba et al. 1978, Coate et al. 1977, Watanabe et al. 1978). Subchronic oral studies have found that 1,2,4-TCB induces hepatic enzymes and liver porphyrins, increases liver weight, and causes fatty infiltration of the liver (Carlson and Tardiff 1976, Carlson 1977, Smith et al. 1978). Topical doses of 1,2,4-TCB have been reported to result in extensor convulsions, necrotic foci in the liver, and death in guinea pigs (Powers et al. 1975, Brown et al. 1969). Teratogenicity studies after administration by the oral route in rats showed mild osteogenic changes in pups and significantly retarded embryonic development as measured by growth parameters (Black et al. 1983, Kitchin and Ebron 1983). Maternal toxicity was observed at doses causing effects in the pups. Increased incidences of non-neoplastic lesions were seen in multiple organs in both male and female mice exposed to 1,2,4-TCB painted on the skin for 2 years (Yamamoto et al. 1957).

EPA (1990) developed an oral reference dose (RfD) for 1,2,4-trichlorobenzene of 0.02 mg/kg-day based on a study by Carlson and Tardiff (1976) that identified increased liver-to-body weight ratios in male rats exposed at 40 mg/kg-day but not at 20 mg/kg-day; an uncertainty factor of 1,000 was used to develop the RfD. EPA (1990) developed an inhalation RfD of 3×10^{-3} mg/kg-day based on increased uroporphyrin levels in rats exposed for 3 months (Watanabe et al. 1978); an uncertainty factor of 1,000 was used to develop the RfD.

REFERENCES

BLACK, W.D., VALLIE, V.E.O., RUDDICK, J.A., and VILLENEUVE, D.C. 1983. The toxicity of three trichlorbenzene isomers in pregnant rats. The Toxicologist 3:30

BROWN, V.K.H., MUIR, C., and THORPE, E. 1969. The acute toxicity and skin irritant properties of 1,2,4-trichlorobenzene. Ann. Occup. Hyg. 12:209-212

CARLSON, G.P. 1977. Chlorinated benzene induction of hepatic porphyria. Experientia 33:1627-1629

CARLSON, G.P., and TARDIFF, R.G. 1976. Effect of the chlorinated benzenes on the metabolism of foreign organic compounds. Toxicol. Appl. Pharmacol. 36:383-394

COATE, W.B., SCHOEFISCH, W.H., LEWIS, T.R., and BUSEY, W.M. 1977. Chronic inhalation exposure of rats, rabbits, and monkeys to 1,2,4-trichloro- benzene. Arch. Environ. Health 32:249-255

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Health Assessment Document for Chlorinated Benzenes. Office of Health and Environmental Assessment. Washington, D.C. January 1985. EPA/600/8-84/015F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

KITCHIN, K.T., and EBRON, M.T. 1983. Maternal hepatic and embryonic effects of 1,2,4-trichlorobenzene in the rat. Environ. Res. 31:362-373

KOCIBA, R.J., LEONG, B.K., and HEFNER, R.E., Jr. 1981. Subchronic toxicity study of 1,2,4-trichlorobenzene in the rat, rabbit, and beagle dog. Drug Chem. Toxicol. 4:229-249

POWERS, M.B., COATE, W.B., and LEWIS, T.R. 1975. Repeated topical applications of 1,2,4-trichlorobenzene: Effects on rabbit ears. Arch. Environ. Health 30:165-167

SMITH, C.C., CRAGG,S.T., and WOLFE, G.F. 1978. Subacute toxicity of 1,2,4-trichlorobenzene (TCB) in subhuman primates. Fed. Proc. 37:248

WATANABE, P.G., KOCIBA, R.J., HEFNER, R.E., Jr., YAKEL, H.O., and LEONG, B.K.J. 1978. Subchronic toxicity studies of 1,2,4-trichlorobenzene in experimental animals. Toxicol. Appl. Pharmacol. 45:322-333

YAMAMOTO, H., OHNO, Y., NAKAMORI, K., OKUYAMA, T.,IMAI, S., and ISUBURA, Y. 1957. [Chronic toxicity and carcinogenicity test of 1,2,4-trichlorobenzene on mice by dermal painting.] J. Nara. Med. Assoc. 33:132-145 (Japanese)

1,1,1-TRICHLOROETHANE

Like other chlorinated aliphatic hydrocarbons, 1,1,1-trichloroethane (1,1,1-TCA, methyl chloroform) is rapidly and completely absorbed following both oral and inhalation exposure. Pulmonary absorption is initially large and gradually decreases to a steady-state condition. Absorption through the skin is slow. 1,1,1-TCA is distributed throughout the body and readily crosses the blood-brain barrier (EPA 1984). The most notable toxic effects of 1,1,1-TCA inhalation exposure in humans and animals are central nervous system depression, including anesthesia at very high concentrations, and impairment of coordination, equilibrium, and judgment at lower concentrations (350 ppm and above). In both humans and animals, cardiovascular effects, including premature ventricular contractions, decreased blood pressure, and sensitization to epinephrine-induced arrhythmia can result from acute exposure to high concentrations of 1,1,1-TCA vapor (EPA 1985). Fatty liver changes have been reported in guinea pigs following subchronic inhalation exposure (Torkelson et al. 1958). NTP (1984) reported preliminary results of bioassays in rats and mice indicating that oral administration of 1,1,1-TCA increases the incidence of hepatocellular carcinomas in female mice but not for male rats. This study was inadequate to evaluate the carcinogenicity of 1,1,1-TCA in female rats and male mice.

EPA (1990a) calculated an oral reference dose (RfD) for 1,1,1-trichloroethane based on an inhalation study by Torkelson et al. (1958) in which rats, rabbits, guinea pigs and monkeys were exposed to 1,1,1-TCA vapor. A no-observed-adverse-effect (NOAEL) of 500 ppm (2,730 mg/m^3, or 90 mg/kg-day) was identified for hepatotoxicity from this study. Using the NOAEL of 90 mg/kg-day and an uncertainty factor of 1,000, a RfD of $9x10^{-2}$ mg/kg-day was derived. An inhalation RfD of 0.3 mg/kg-day for 1,1,1-TCA also has been determined by EPA (1990b) based on this same study, in which hepatotoxicity was observed in guinea pigs. An uncertainty factor of 1,000 was used in calculating the RfD. EPA (1990b) established subchronic oral and inhalation RfDs of $9x10^{-1}$ mg/kg-day and 3 mg/kg-day, respectively, based on the same study and same effect of concern. An uncertainty factor of 100 was used in calculating the subchronic RfDs.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for 1,1,1-Trichloroethane. Final Draft. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. ECAO-CIN-H005

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Drinking Water Criteria for 1,1,1-Trichloroethane (Draft). Office of Drinking Water, Criteria and Standards Division, Washington, D.C. January 1985

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by the Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio for the Office of Solid Waste and Remedial Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

NATIONAL TOXICOLOGY PROGRAM (NTP). 1984. Annual Plan for Fiscal Year 1984. DHHS, Public Health Service, Research Triangle Park, North Carolina. NTP-84-023

TORKELSON, T.R., OYEN, F., McCOLLISTER, D.D., and ROWE, V.K. 1958. Toxicity of 1,1,1-trichloroethane as determined on laboratory animals and human subjects. Am. Ind. Hyg. Assoc. J. 19:353-362

1,1,2-TRICHLOROETHANE

1,1,2-Trichloroethane (1,1,2-TCA) is rapidly absorbed from oral, inhalation and dermal exposures (Torkelson and Rowe 1981, Arena 1979). In humans, acute oral and inhalation exposures to 1,1,2-TCA result in central nervous system (CNS) depression, equilibrium disturbances, vertigo, headaches, lassitude, hypotension, anesthesia and coma (Arena 1979). Acute oral and inhalation administration to animals produces liver and kidney damage, irritation to the eyes and nose, CNS depression, and death due to respiratory arrest (ACGIH 1986, Torkelson and Rowe 1981). In dogs the hepatotoxic effects include hepatocyte vacuolation, enzyme induction, fatty degeneration and necrosis (NRC 1977, Torkelson and Rowe 1981). The hepatoxicity and nephrotoxicity of 1,1,2-TCA has been found to be potentiated by pretreatment with certain halogenated organic compounds and solvents. Subchronic oral administration to mice produced alterations in clinical serum levels indicative of adverse liver effects (White et al. 1985, Sanders et al. 1985). Dermal exposures result in irritation and injury to the skin from defatation (Torkelson and Rowe 1981). Evidence suggests that 1,1,2-TCA is embryo toxic to chicken eggs (Elovaara 1979). 1,1,2-TCA was found to be weakly mutagenic in S. Cerevisiae (Torkelson and Rowe 1981). Oral administration of 1,1,2-TCA has been associated with the induction of hepatocellular carcinomas and pheochromocytomas in mice but not in rats (NCI 1978, Weisburger 1977)

EPA has classified 1,1,2-TCA in group C (Possible Human Carcinogen). This category applies to agents for which there is limited evidence of carcinogenicty in animals. EPA (1990a) has derived a cancer potency factor of 5.7×10^{-2} $(mg/kg\text{-}day)^{-1}$ for both oral and inhalation exposures based on an increased incidence of liver tumors in mice (NCI 1978). EPA (1990a) has also established an oral reference dose (RfD) of 4.0×10^{-3} mg/kg-day for 1,1,2-TCA based upon clinical chemistry alterations in mice given 3.9 mg/kg-day in drinking water (White et al 1985, Sanders et al 1985). An uncertainty factor of 1,000 was used to calculate the RfD. EPA (1990b) has derived a subchronic oral RfD of 4.0×10^{-2} mg/kg-day, using an uncertainty factor of 100, and based on the same study and effect of concern.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. AGCIH, Inc., Cincinnati, Ohio

ARENA, J.M. 1979. Poisoning: Toxicology, Symptoms, Treatments. 4th ed., Charles C. Thomas, Publisher, Springfield, Illinois. P. 207

ELOVAARA, E., et al. 1979. Effects of methene chloride, trichloroethane, trichloroethene, tetrachloroethene, and toluene on the development of chick embryos. Toxicology. 12:111

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Health Criteria and Assessment Officer, Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

NATIONAL CANCER INSTITUTE (NCI). 1978. Bioassay of 1,1,2-trichloroethane for possible carcinogenicity. U.S. Dept. Health, Educ, Welfare. Pub. No. NCI-CG-TR-74

NATIONAL RESEARCH COUNCIL (NRC). 1977. Drinking Water and Health, Volume 1, National Academy Press, Washington, D.C. P. 775-776

SANDERS, V.M., WHITE Jr., K.L., SHOPP Jr., G.M., and MUNSON,A.E. 1985. Humoral and cell mediated immune status of mice exposed to 1,1,2-trichloroethane. Drug Chem. Toxicol 8:357-372

TORKELSON, T.R., AND ROWE, U.K. 1981. Halogenated alphatic hydrocarbons. In Clayton, G.D., and Clayton, P.B., eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York. Vol. 2B., p. 3510-3512

WHITE Jr., K.L., SANDERS, V.M., BARNES, V.W., SHOPP Jr., G.M., and MUNSON,A.E. 1985. Toxicology of 1,1,2-trichloroehtane in the mouse. Drug Chem. Toxicol 8: 333-355

WEISBURBER, E.K. 1977. Carcinogenicity studies on halogenated hydrocarbons. Environ. Health Perspect. 21:7-16

TRICHLOROETHENE

Absorption of trichloroethene (TCE) from the gastrointestinal tract is virtually complete. Absorption following inhalation exposure is proportional to concentration and duration of exposure (EPA 1985). TCE is a central nervous system depressant following acute and chronic exposures. In humans, single oral doses of 15 to 25 ml (21 to 35 grams) of TCE have resulted in vomiting and abdominal pain, followed by transient unconsciousness (Stephens 1945). High-level exposure can result in death due to respiratory and cardiac failure (EPA 1985). Hepatotoxicity has been reported in human and animal studies following acute exposure to TCE (EPA 1985). Nephrotoxicity has been observed in animals following acute exposure to TCE vapors (ACGIH 1986, Torkelson and Rowe 1981). Subacute inhalation exposures of mice have resulted in transient increased liver weights (Kjellstrand et al. 1983). Industrial use of TCE is often associated with adverse dermatological effects including reddening and skin burns on contact with the liquid form, and dermatitis resulting from vapors. These effects are usually the result of contact with concentrated solvent, however, and no effects have been reported following exposure to TCE in dilute, aqueous solutions (EPA 1985). Trichloroethene has caused significant increases in the incidence of hepatocellular carcinomas in mice (NCI 1976) and renal tubular-cell neoplasms in rats exposed by gavage (NTP 1983), and pulmonary adenocarcinomas in mice following inhalation exposure (Fukuda et al. 1983, Maltoni et al. 1986). Trichloroethene was mutagenic in *Salmonella typhimurium* and in *E. coli* (strain K-12), utilizing liver microsomes for activation (Greim et al. 1977).

EPA (1990) classified trichloroethene in Group B2--Probable Human Carcinogen based on inadequate evidence in humans and sufficient evidence of carcinogenicity from animal studies. An oral cancer potency factor of 1.1×10^{-2} $(mg/kg\text{-}day)^{-1}$ has been derived by EPA (1990) based on two gavage studies conducted in mice in which an increased incidence of liver tumors were observed (Maltoni et al. 1986, Fukuda et al. 1983). An inhalation cancer potency factor of 1.7×10^{-2} $(mg/kg\text{-}day)^{-1}$ has been derived for trichloroethene (EPA 1990) based on an increased incidence of lung tumors in mice (NCI 1976). EPA (1987) developed a chronic oral reference dose (RfD) of 7.35×10^{-3} mg/kg-day based on a subchronic inhalation study in rats in which elevated liver weights were observed following exposure to 55 ppm, 5 days/week for 14 weeks (Kimmerle and Eben 1973). A safety factor of 1,000 was used to calculate the RfD. However, this RfD is currently under review by EPA.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. ACGIH, Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Trichloroethene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. EPA 540/1-86-046

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Health Assessment Document for Trichloroethene. Environmental Criteria and Assessment Office. Research Triangle Park, North Carolina. EPA/600/8-82/006F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Health Advisory for Trichloroethene. Office of Drinking Water, Washington, D.C. March 31, 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by the Office of Health and Environmental Assessment. Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

FUKUDA, K., TAKEMOTO, K., and TSURUTA, H. 1983. Inhalation carcinogenicity of trichloroethene in mice and rats. Ind. Health 21:243-254

GREIM, H., BIMBOES, D., EGERT, G., GIGGELMANN, W., and KRAMER, M. 1977. Mutagenicity and chromosomal aberrations as an analytical tool for *in vitro* detection of mammalian enzyme-mediated formation of reactive metabolites. Arch. Toxicol. 39:159

KIMMERLE, G., and EBEN, A. 1973. Metabolism, excretion and toxicology of trichloroethene after inhalation. 1. Experimental exposure on rat. Arch. Toxicol. 30:115

KJELLSTRAND, P., HOLQUIST, B., ALM, P., KANJE, M., ROMARE, S., JONSSON, I., MANNSON, L., and BJERKEMO, M. 1983. Trichloroethene: Further studies of the effects on body and organ weights and plasma butyl cholinesterase activity in mice. Acta. Pharmacol. Toxicol. 53:375-384 (As cited in EPA 1985)

MALTONI, C., LEFEMINE, G., COTTI, G. 1986. Experimental Research on Trichloroethene. Carcinogenesis Arch. Res. Industrial Carcinogenesis Series. Maltoni, C., Mehlman, M, A., Eds. Vol. V. Princeton Scientific Publishing Co., Inc., Princeton, NJ. p. 393

NATIONAL CANCER INSTITUTE (NCI). 1976. Carcinogenesis Bioassay of Trichloroethene. CAS No. 79-01-6. Carcinogenesis Technical Report Series No. 2. PB-264 122

NATIONAL TOXICOLOGY PROGRAM (NTP). 1983. Carcinogenesis Studies of Trichloroethene (Without Epichlorohydrin), CAS No. 79-01-6, in F344/N rats and B6C3F$_1$ mice (Gavage Studies). Draft. August 1983. NTP 81-84, NTP TR 243.

STEPHENS, C. 1945. Poisoning by accidental drinking of trichloroethene. Br. Med. J. 2:218

TORKELSON, T.R., and ROWE, V.K. 1981. Halogenated aliphatic hydrocarbons. In Clayton, G.D., and Clayton, P.B., eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York. Vol. 2B, pp. 3553-3559

TRICHLOROFLUOROMETHANE

Trichlorofluoromethane is rapidly absorbed in humans and animals following oral and inhalation exposures (ACGIH 1986, Mergner et al. 1975, Adir et al. 1975). Oral administration of 2.5 ml/kg in rats resulted in neither death or liver toxicity (Slater 1965) and application to the eyes or skin caused only mild reversible irritation (Scholz 1962, Quevauviller et al. 1964, Hood 1964). The LC_{50} of trichlorofluoromethane in rats after a 4 hour exposure has been determined to be 26,200 ppm (ACGIH 1986). Metabolic changes (e.g., increased blood glucose and lactic acid, decreased oxygen uptake) were reported in rats and rabbits exposed to 50,000 ppm trichlorofluoromethane for one hour daily for 15 days (Paulet 1975). Dogs exposed to the same concentration for 20 minutes showed increases in blood glucose and lactic acid. A subchronic study using dogs exposed to trichlorofluoromethane in the air for 90 days showed elevated BUN levels and lung lesions (Jenkins et al. 1970). Evidence of cardiotoxicity (cardiac arrhythmias) in dogs exposed to 5000 ppm trichloromethane and intravenous epinephrine was provided by Reinhardt et al. (1971). Subsequent experiments have shown the effects to be brief with the animals recovering shortly after the end of exposure (Clark et al. 1972). Other animals exposed to trichlorofluoromethane concentrations between 5,000 to 15,00 ppm have also been shown to exhibit cardiovascular and circulatory abnormalities (Flowers et al. 1975, Taylor and Drew 1975, Taylor 1975, Aviado 1975). The available evidence suggests that trichlorofluoromethane is not carcinogenic (NCI 1978). This study did show an association between dose and increased incidence of mortality in rats.

EPA (1990) has reported chronic and subchronic inhalation RfDs for trichlorofluoromethane of 0.2 mg/kg-day and 2 mg/kg-day, respectively, based on studies investigating elevated BUN levels and lung lesions in dogs (Jenkins et al. 1970); uncertainty factors of 10,000 and 1,000 were used to develop the RfDs for chronic and subchronic exposures, respectively. A chronic (EPA 1990a) and subchronic (EPA 1990b) oral reference doses of 0.3 mg/kg-day and 0.7 mg/kg-day have been reported, respectively, for trichlorofluoromethane based on a chronic rat study in which increased mortality was observed (NCI 1978). An uncertainty factor of 1,000 was used to derive both the chronic and subchronic RfDs.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th Edition. Cincinnati, Ohio

ADIR, J., BLAKE, D.A., and MERGNER, G.M. 1975. J. Clin. Pharmacol. 15:760

AVIADO, D.M. 1975. Toxicology 3:311

BARRAS, C.E. 1974. Unpublished data. Haskell Laboratory, DuPont Co., Wilmington, DE. October 1974

CLARKE, D.G., and TINSTON, D.J. 1972. Proc. Eur. Soc. Drug Toxicity 13:212

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office. Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

FLOWERS, N.C., HAND, R.C., and HORAN, L.G. 1975. Arch. Env. Health. 30:353

HOOD, D.B. 1964. Unpublished data. Haskell Laboratory, DuPont Co., Wilmington, DE. September 1964

JENKINS, L.J., JONES, R.A., COON, R.A., and SIEGEL, J. 1970. Tox. Appl. Pharm. 16:133

LESTER, D., and GREENBERG, L.A. 1950. Arch. Ind. Hyg. Occup. Med. 2:335

MERGNER, G.W., BLAKE, D.A., and HELRICH, M. 1975. Anesthesiology 42:345

NATIONAL CANCER INSTITUTE (NCI). 1978. Bioassay of Trichlorofluoromethane for Possible Carcinogenicity. NCI Carcinogenesis Tech. Rep. Ser. No. 106. (Also publ as DHEW (NIH) 78-1356)

PAULET, G., RONCIN, G., and VIDAL, E., et al. 1975. Tox. Apl. Pharm. 34:197

QUEVAUVILLER, A., SCHRENZEL, M., and HUYEN, V.N. 1964. Therapie 19:247

REINHARDT, C.F., AZAR, A., and MAXFIELD, ME., et al. 1971. Arch. Env. Health. 22:265

SCHOLZ, J. 1962. Fortschr. Biol. Aerosolforsch. Jahren 1957-1961. Ber. Aerosol-Kongr. 4:420

SLATER, T.F. 1965. Biochem. Pharmacol. 14:178

TAYLOR, G.J. 1975. Arch. Env. Health. 30:349

TAYLOR, G.J., and DREW, R.T. 1975. Tox. Appl. Pharm. 32:177

N,N'-BIS-(2,4,6-TRICHLOROPHENYL)UREA
(246TCPU)

INTRODUCTION

N,N'-Bis-(2,3,6-trichlorophenyl)urea (TCPU) has an extremely low solubility, is stable, immobile and persistent in the aqueous environment (Army 1987, Harvey et al. 1990).

TOXICOKINETICS

The absorption, distribution, metabolism and excretion of TCPU are unknown at present.

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

The lowest lethal dose (LDlo) for TCPU in rats has been reported to be 50 mg/kg (RTECS 1990). The guinea pig was the most sensitive species for evaluating compounds structurally similar to TCPU (Army 1987). In a study conducted by the army, the approximate lethal dose was greater than 1,000 mg/kg for both guinea pigs and rats given TCPU in a suspension of water, and greater than 1,500 mg/kg in animals given TCPU in a corn oil vehicle (Army 1987). No toxic signs were exhibited by any of the test animals during the 14-day observation period and there were no changes in weight gain, organ weight, gross necropsy, or histopathologic findings associated with TCPU administration. In rats receiving TCPU in oil vehicle, the only significant difference observed was increased thymus weight in male rats, but no abnormal histopathology was evident (Army 1987).

TCPU was evaluated for mutagenic activity in microbial assays with and without the addition of mammalian metabolic activation preparations. Five strains of Salmonella typhimurium (TA-1535, TA-1537, TA-1538, TA-98 and TA-100) were used in evaluating mutagenic potential. TCPU did not exhibit mutagenic activity to the Salmonella typhimurium indicator organisms. In addition, no significant increased frequencies were found for chromosomal abberations in chinese hamster ovary cells, for mutation frequencies in the mouse lymphoma assay with and without metabolic activation, or for unscheduled DNA synthesis (UDS) in rat hepatocytes (Army 1987).

The results of the Army (1987) studies indicate that the test material when ingested in single oral doses is relatively nontoxic. In vitro studies indicate that TCPU poses no hazard as a mutagen. Due the the extremely low solubility of TCPU, it appears that massive doses would be required to produce toxic manifestations.

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

No regulated standards or criteria have been promulgated for TCPU.

REFERENCES

ARMY. 1987. Determination of the acute toxicity of N,N'-Bis-(2,4,6-Trichlorophenyl) urea in male and female rats and guinea pigs. Study No. 75-51-0523-87. March 1987. United States Army Environmental Hygiene Agency. Aberdeen Proving Ground, MD.

HARVEY, S.D., D.D DAUBLE, B.D. MCVEETY, R.J. FELLOWS, R.M. BEAN, D.A. CATALDO, J.A. CAMPBELL. 1990. Fate of N,N'-bis(2,4,6-Trichlorophenyl)-urea in a freshwater sediment. Pacific Northwest Laboratory, Richland WA. Chemical Research Development and Engineering Center. U.S. Army Armament Munitions Chemical Commans. Aberdeen Proving Ground, MD

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1990. National Institute for Occupational Safety and Health (NIOSH). U.S. Department of Health and Human Services.

1,1,2-TRICHLORO-1,2,2-TRIFLUOROETHANE (CHLOROFLUROCARBON-113)

Chlorofluorocarbons (CFC) are absorbed following inhalation and dermal exposure; gastrointestinal absorption has not been quantitatively studied, but it is known to occur. Inhalation exposure results in an initial rapid increase in blood levels followed by a slower increase to maximum concentrations (EPA 1983). In humans, acute exposure to 2500 ppm was found to slightly impair psychomotor performance and higher doses resulted in a loss of concentration, drowsiness, "heaviness" in the head and dizziness upon lateral shaking of the head (Stopps and McLaughlin 1967). Longer-term occupational exposure to CFC-113 has not been shown to result in adverse health effects at average dose levels of approximately 500 ppm (EPA 1983). Acute or subchronic inhalation exposure of laboratory animals to CFC-113 at doses above 2000 ppm has been reported to result in detrimental cardiovascular effects including arrhythmias, tachycardia, hypotension, diminished myocardial contractility and cardiac sensitization to the effects of exogenous epinephrine. Higher doses have been shown to result in nervous system effects ranging from slight narcosis to convulsions and death. CFC-113 has also been reported to cause liver and kidney congestion and kidney and thyroid enlargement in certain experiments (EPA 1983).

EPA (1990) has derived an oral reference dose (RfD) for CFC-113 of 30 mg/kg-day based on an epidemiologic study in which psychomotor impairment was observed (Imbus and Adkins 1972). A no-observed-adverse-effect level (NOAEL) of 272 mg/kg-day and a safety factor of 10 were used to calculte the RfD.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA) 1983. Health Assessment Document for 1,1,2-Trichloro-1,2,2-trifluoroethane (Chlorofluorocarbon CFC-113). Final Report. Office of Health and Environmental Assessment, Research Triangle Park. N.C. EPA 600/8-82-002F.

ENVIRONMENTAL PROTECTION AGENCY (EPA) 1990. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office. Cincinnati, Ohio.

IMBUS, H.R., ADKINS, C. 1972. Physical examination of workers exposed to trichlorotrifluoroethane. Arch. Environ. Health. 24(4): 257-261.

STOPPS, G.J., AND McLAUGHLIN, M. 1967. Psychophysiological Testing of Human Subjects Exposed to Solvent Vapors. Amer. Ind. Hyg. Assoc. J. 23:43-50

1,3,5-TRINITROBENZENE

Little information is available for 1,3,5-trinitrobenzene (TNB). Therefore, toxicity information is based on the structurally similar 1,3-dinitrobenzene (RTECS 1983). 1,3-dinitrobenzene is absorbed orally. In subchronic drinking water studies, rats experienced decreased hemoglobin concentrations, splenic weight gain and enlargement with hemosiderin deposits, decreased body weight gain in females, and testicular atrophy in males in the two highest dose levels (Cody et al. 1981). 1,3,5-TNB has been shown to be mutagenic in DNA repair assays (McGregor et al. 1980).

EPA (1989) has derived an oral reference dose (RfD) for 1,3,5-TNB of 5.00×10^{-5} mg/kg-day based on a subchronic study whereby rats experienced increased spleen weights when given 1,3-dinitrobenzene in drinking water (Cody et al. 1981). The RfD for 1,3,5-TNB was derived by analogy from 1,3-dinitrobenzene. A NOAEL of 0.4 mg/kg-day for 1,3-DNB (equivalent to 0.51 mg/kg-day 1,3,5-TNB) was identified from this study. An uncertainty factor of 10,000 was used to calculate the RfD for 1,3,5-TNB.

REFERENCES

CODY, T.E., WITHERUP, S., HASTINGS, L., STEMMER, K., AND CHRISTIAN, R.T. 1981. 1,3-Dinitrobenzene: Toxic effect in vivo and in vitro. J. Toxicol. Environ. Health 7(5): 829-847.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989. Health Effects Assessment Summary Tables. October. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C.

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1983. U.S. Department of Health and Human Services, National Institute for Occupational Safety and Health.

2,4,6-TRINITROTOLUENE (TNT)

HUMAN HEALTH EFFECTS

2,4,6-Trinitrotoluene (TNT) is absorbed by humans and laboratory animals following oral, inhalation and dermal exposure (EPA 1989). Once absorbed, TNT is distributed throughout the body and is metabolized extensively (EPA 1980). The hematopoietic system and the liver are the principle target organs of TNT toxicity in humans and experimental animals (Levine et al. 1983; NRC 1982). Increased incidences of cataracts (Harkonen et al. 1983), neurological manifestations, and nephrotoxicity have been observed in humans occupationally exposed to TNT (Kaganov et al. 1970, as cited in Zakhari and Villaume 1978). Acute exposure in rats is associated with respiratory paralysis, cyanosis, ataxia, and sometimes death (Lee et al. 1975). Testicular atrophy and degeneration have been observed in rats subchronically exposed to TNT in the diet (Dilley et al 1982). TNT has been found to cause urinary bladder papillomas and carcinomas in female rats (U.S. DOD 1984). Mutagenic activity was observed in strains TA98, TA1537, TA1538, and TA100 of *Salmonella typhimurium* with and without metabolic activation (Spanggord et al. 1982).

EPA (1990) has assigned TNT a weight-of-evidence classification of C -- Possible Human Carcinogen -- based on urinary bladder papillomas and carcinomas at 50 mg/kg-day in female Fischer 344 rats (U.S. DOD 1984). An oral cancer potency factor of 3.0×10^{-2} $(mg/kg\text{-}day)^{-1}$ has been assigned to TNT. EPA (1990) has developed a reference dose (RfD) for TNT based on a study in beagle dogs in which an increased incidence of liver effects was observed (Levine et al. 1983). Animals received TNT in gelatin at daily oral doses of 0, 0.5, 2.0, 8.0 or 32 mg/kg/bw for 26 weeks. Gross and histologic examination revealed examination revealed toxic liver effects and/or lesions in all dose groups with lesions in the low dose group (0.5 mg/kg-day) described as trace to mild. No lesions were observed in the control animals. The 0.5 mg/kg-day dose is the lowest-observed-adverse-effect level (LOAEL) for this study. An RfD of 5.00×10^{-4} mg/kg-day was derived based on this LOAEL and an uncertainty factor of 1,000.

ENVIRONMENTAL EFFECTS

Information on the toxic effects of TNT on terrestrial plants and wildlife was not found in the available literature. In laboratory mammal species (potential surrogates for wildlife species), oral LD_{50} values have been reported to range between 660 mg/kg (mice) to 3,190 mg/kg (rats), with most values range between 800 to 1,000 mg/kg (Ryon 1987). Acute effects following dosing in rats and mice included central nervous system disturbances with inactivity, tremors, and seizures followed by death (Ryon 1987).

In aquatic systems, TNT can result in a general decrease in water quality, including increases in the COD, and in the levels of nitrogen species and sulfates in the receiving waters (Ryon et al. 1984). TNT is not highly acutely toxic to aquatic invertebrates with LC_{50} values in the range of 6.5 mg/liter for isopods to 27 mg/liter for insects (midges) (Liu et al. 1983). Acute values in fish range from 1.34 mg/liter for rainbow trout to 2.81 mg/liter for channel catfish (Ryon 1987). Chronic toxicity tests with invertebrates in a microcosm resulted in no significant effects on survival or reproduction at the maximum concentration tested of 1.03 mg/liter (Bailey et al. 1985). In early life-stage tests with rainbow trout, TNT concentrations greater than or equal to 0.49 mg/liter resulted in decreased fry survival. TNT concentrations of 0.04 mg/liter resulted in decreased fathead minnow fry survival the first 30 days of exposure and reduced fry length and width following 60 days of exposure.

Little information is available on the bioconcentration of TNT, but based on the available data and analogy to dinitrotoluene, TNT is not expected to accumulate in aquatic organisms. A whole-body BCF of 25 was reported by Ryon et al. (1984) for 2,4 DNT.

Ryon (1987) derived a maximum (acute) concentration criterion of 557 µg/L TNT and a tentative continuous (chronic) concentration criterion of 40 µg/L TNT to protect aquatic life. Using an Acceptable Daily Intake (ADI) of 0.28 mg/day and a no-observed-effect level of 0.4 mg/kg-day, a human health criterion of 135 µg/L was derived (Ryon 1987).

REFERENCES

BAILEY, H.W., SPANGGORD, R.J., JAVITZ, H.S. AND LIU, D.H.W. 1985. Toxicity of TNT Wastewaters to Aquatic Organisms. Volume III. Chronic Toxicity of LAP Wastewater and 2,4,6-Trinitrotoluene. Final Report. SRI International, Menlo Park, CA. DAMD17-75-C-5056. 80pp.

DILLEY, J.V., TYSON, C.A., SPANGGORD, R.J., SASMORE, D.P., NEWELL, G.W., and DACRE, J.C. 1982. Short-term oral toxicity of 2,4,6-trinitrotoluene in mice, rats, and dogs. J. Tox. Environ. Health 9:565-585.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Dinitrotoluene. Criteria and Standards Division, Washington, D.C. EPA 440/5-80-045. NTIS PB 81-117566.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1989. Trinitrotoluene. Health Advisory. Office of Drinking Water, U.S. EPA. Washington, D.C. 20460.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Integrated Risk Information System. EPA, Washington, D.C.

HARKONEN, H., KARKI, M., LAHTI, A., and SAVOLAINEN, H. 1983. Early equational cataracts in workers exposed to trinitrotoluene. Am. J. Ophth. 95:807-810.

KAGANOV A.L., FEDYUSHINA, N.A., BOCHENKOVA, Y.K. 1970. [A study of the nervous system under prolonged exposure to trinitrotoluene.] Gig. Tr. Prof. Zabol. 14: 47-48 (Russian), as cited in Zakhari and Villaume, 1978.

LEE, C-C, DILLEY, J.V., HODGSON, J.R. ET AL. 1975. Mammalian toxicity of munition compounds: Phase I. Acute oral toxicity, primary skin and eye irritation, dermal sensitization, and disposition and metabolism. Report No. 1, Midwest Research Institute, Project No. 3900-B, Kansas City, MO. DAMD 17-74C-4073. AD-B011 150.

LEVINE, RUSH, BURNS, AND LISH. 1983. Determination of chronic mammalian effects of TNT: 26-Week subchronic oral toxicity of TNT in beagle dogs. Department of Defense. Cameron Station, Alexandria, Virginia. AD-A157-002.

LIU, D.H.W., SPANGGORD, R.J., BAILEY, H.C., JAVITZ, H.S. AND JONES, D.C.L. 1983. Toxicity of TNT Wastewaters to Aquatic Organisms. Volume I. Acute Toxicity of LAP Wastewater and 2,4,6-Trinitrotoluene. Final Report. AD A142 144. SRI International, Menlo Park, CA. DAMD17-75-C-5056. 85pp.

NATIONAL RESEARCH COUNCIL. 1982. National Research Council. Evaluation of the health risks of ordinance disposal waste in drinking water. Committee on Toxicology. National Academy Press. Washington, D.C. N00014-80-C-0161. PB83 153635.

RYON, M.G. 1987. Water Quality Criteria for 2,4,6-Trinitrotoluene (TNT). Final Report. Oak Ridge National Laboratory, Oak Ridge, Tennessee.

RYON, M.G., PAL, B.C., TALMAGE, S.S., AND ROSS, R.H. 1984. Database Assessment of the Health and Environmental Effects of Munitions Production Waste Products. Oak Ridge National Laboratory, Oak Ridge, Tennessee.

SPANGGORD, R.J., MORTELMANS, K.E., GRIFFIN, A.F., and SIMMON, V.F. 1982. Mutagenicity in *Salmonella typhimurium* and structure-activity relationships of wastewater compounds emanating from the manufacture of trinitrotoluene. Environ. Mutagen. 4: 163-179.

U.S. DOD (DEPARTMENT OF DEFENSE). 1984. Cameron Station, Alexandria, Virginia. AD-A168637.

ZAKHARI A., VILLAUME, J.E. 1978. A Literature Review - Problem definition studies on selected toxic chemicals. Volume 3 of 8. Occupational health and safety aspects of 2,4,6-trinitrotoluene (TNT). Final Report, Science Information Services Organization, Philadelphia, PA. DAMD 17-77-C-7020, AD-055 683.

VINYL CHLORIDE

Vinyl chloride is rapidly absorbed in rats following oral and inhalation exposure, while dermal absorption of vinyl chloride is minor (EPA 1985). At high inhalation exposure levels, workers have experienced dizziness, headaches, euphoria, and narcosis. In experimental animals, inhalation exposure to high levels of vinyl chloride can induce narcosis and death. Lower doses result in ataxia, narcosis, congestion and edema of the lungs, and hyperemia in the liver (EPA 1985). Chronic inhalation exposure of workers to vinyl chloride is associated with hepatotoxicity, central nervous system disturbances, pulmonary insufficiency, cardiovascular toxicity, gastrointestinal toxicity, and acro-osteolysis (EPA 1985). Experimental animals chronically exposed via inhalation or ingestion have exhibited effects involving the liver, spleen, kidneys, hematopoietic system, and skeletal system (EPA 1984). Feron et al. (1975) found that administration of vinyl chloride to rats by gavage resulted in hematologic, biochemical, and organ-weight effects at doses above 30 mg/kg-day. Evidence for an association between human exposure to vinyl chloride and birth defects or fetal loss is conflicting (EPA 1987). Human exposure to vinyl chloride has been associated with an increased incidence of hepatic angiosarcoma and brain, lung, and hemolymphopoietic cancers. In animal studies, chronic inhalation and ingestion of vinyl chloride at levels as low as 1.7 and 5 mg/kg-day have induced cancer in the liver and in other tissues of rats and mice (IARC 1979; Feron et al. 1981; Maltoni et al. 1980, 1981).

EPA (1990) has classified vinyl chloride in Group A (Human Carcinogen) based on adequate evidence of carcinogenicity from epidemiological studies. EPA (1990) reported an oral slope factor of 2.3 $(mg/kg\text{-}day)^{-1}$ for vinyl chloride based on the long-term ingestion study in rats in which lung tumors were observed (Feron et al. 1981). The inhalation slope factor for vinyl chloride is 2.95×10^{-1} $(mg/kg\text{-}day)^{-1}$ (EPA 1990) and is based on chronic inhalation studies conducted by Maltoni et al. (1980, 1981) in which liver tumors were observed in rats.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Vinyl Chloride. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-036

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Drinking Water Criteria Document on Vinyl Chloride. Final Draft. Office of Drinking Water, Washington, D.C. January 1985

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Memo from Sandra Lee (Program Manager, Regional Support Team, Toxics Integration Branch) to Addresses, Subject: Updated Reference Dose and Cancer Potency Numbers for Use in Risk Assessments. Office of Solid Waste and Emergency Response, Washington, D.C. October 1984. 19 pp.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by the Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Remedial Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

FERON, V.J., SPEEK, A.J., WILLIAMS, M.I., VAN BATTUM, D., and DE GROOT, A.F. 1975. Observations on the oral administration and toxicity of vinyl chloride in rats. Food. Cosmet. Toxicol. 13:633-638

FERON, V.J., HENDRIKSEN, C.F.M., SPEEK, A.J., TIL, H.P., and SPIT, B.J. 1981. Lifespan oral toxicity study of vinyl chloride in rats. Food Cosmet. Toxicol. 19:317-333

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1979. IARC Monographs on the Evaluation of the Carcinogenic Risks of Chemicals to Humans. Some Halogenated Hydrocarbons. World Health Organization, Lyon, France. Vol. 20

MALTONI, C., G. LEFEMINE, A. CILIBERTI, et al. 1980. Vinyl chloride carcinogenicity bioassays (BT project) as an experimental model for risk identification and assessment in environmental and occupational carcinogenesis. Epidemiol. Anim. Epidemiol. Hum: Cas Chlorure Vinyle Monomere. Reun Club Cancerog. Chim. 20th, Meeting Date 1979. Publ Essent, Paris, France. Pp. 11-112

MALTONI, C., G. LEFEMINE, A. CILIBERTI, et al. 1981. Carcinogenicity bioassays of vinyl chloride monomer: A model of risk assessment on an experimental basis. Environ. Health Perspect. 41:329

INTRODUCTION

VX (where "V" stands for venom) is the most acutely lethal nerve agent which acts as a cholinesterase inhibitor (Chemical Stockpile Disposal Program 1988). It is odorless, colorless, tasteless and extremely toxic in both liquid and vapor forms, especially following skin and eye absorption. The V agents are more stable than the G agents and are more resistent to detoxification, less volatile, and more efficient at skin penetration (Chemical Stockpile Disposal Program 1988).

TOXICOKINETICS

VX is a highly lipid soluble organophosphate compound which can be absorbed via inhalation, topical contact or ingestion (Chemical Stockpile Disposal Program 1988). Liquid VX also rapidly penetrates the eye (Army 1975). The evaporation of VX from the skin is almost negligible; VX suffers virtually no degradation as it slowly penetrates the skin enabling more VX to reach the bloodstream. In rabbits, essentially 100% of dermally applied VX reached the circulatory system within 4.5 hours (Chemical Stockpile Disposal Program 1988). It has been speculated that its larger molecular size and different solubility characteristics may reduce the diffusion of VX through tissues and cell membranes to the target tissues (Chemical Stockpile Disposal Program 1988). The rate of detoxification is slow; VX is essentially cumulative (Army 1975).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

VX is most effective as a skin penetrant and lethal contact agent. The rate of action is rapid; death usually occurs within 15 minutes after a fatal dosage (Army 1975). The estimated median lethal respiratory dosages (LCt_{50}) for VX in humans and rats are 20-50 mg-min/m^3, and 17 mg-min/m^3, respectively while the human incapacitating dose (ICt_{50}) is 5-15 mg-min/m^3 (Chemical Stockpile Disposal Program 1988). In humans, VX is estimated to be approximately twice as toxic as sarin by inhalation, 10 times as toxic by oral administration and approximately 170 times as toxic following skin exposure (Chemical Stockpile Disposal Program 1988). The animal oral LD_{50} value is 0.1 mg/kg for rats, 10 times the oral toxicity observed for sarin. VX apparently has a slower toxic action as compared with the G agents, both in terms of onset of symptoms and in recovery (Chemical Stockpile Disposal Program 1988).

Percutaneous absorption is a more likely route of VX exposure than inhalation; moreover, percutaneous toxicity is more likely to occur from the absorption of VX aerosol or liquid than from the vapor (Chemical Stockpile Disposal Program 1988). Following absorption, VX not only irreversibily inhibits acetylcholinesterase (AChE), an enzyme responsible for the breakdown of the neurotransmitter acetylcholine (ACh), but also reacts directly with the ACh receptors and other neurotransmitter receptors. The high toxicity of VX is attributed to its very specific reaction with, and subsequent inhibition of AChE (Chemical Stockpile Disposal Program 1988). The mode of action involves the consequences of excessive ACh accumulation at nerve junction, and within portions of the nervous system that control smooth muscle cardiac muscle, and endocrine-exocrine glandular function (Chemical Stockpile Disposal Program 1988). Manifestations include: drooling, increased bronchial secretions, bronchoconstriction, miosis, excessive sweating, vomiting, diarrhea, abdominal cramping, involuntary urination, and heartbeat irregularities (arrhythmias). In addition, ACh accumulation can affect the central nervous system (CNS; the brain and spinal cord), resulting in headache, anxiety, confusion, restlessness, giddiness, electroencephalographic (EEG) changes, or even convulsions and coma (Chemical Stockpile Disposal Program 1988). The third area affected by ACh accumulation is a

portion of the nervous system controlling skeletal muscles. Thus, acute exposure to VX can also result in a generalized weakness that increases with exertion, as well as muscle twitching and cramping. Respiratory failure, the immediate cause of death in VX exposure, is an example of an effect that occurs as the result of ACh accumulation at several sites in the nervous system, in addition to neuromuscular block of the respiratory muscles, airway constriction, and increased agent-induced respiratory failure (Army 1975).

In a comprehensive evaluation of VX recently conducted by the Army, it was concluded that VX did not induce adverse effects related to genotoxic, teratogenic, reproductive, subchronic, or delayed neuropathy at concentrations up to 4 µg/kg. Sprague Dawley rats subcutaneously injected with up to 4 µg/kg VX for 90 days did not exhibit consistent effects for organ weights, clinical chemistry parameters, or histopathology (Goldman et al. 1988). In addition, no evidence for organophosphate-induced delayed neuropathy from VX was observed following a single subcutaneous exposure or chronic subcutaneous exposures up to 90 days in the chicken.

In a three-generation reproduction study, Sprague Dawley rats were subcutaneously injected with VX at concentration up to 4 µg/kg (Goldman et al. 1988). Reproductive toxicity indices included decreased fertility, premature delivery, litter sizes, pup survival, and size of offspring. Neither these nor any changes in reproductive behavior, clinical observations, or gross necropsy or histopathology indicated any untoward VX effects. Mutagenic effects of VX on germinal cells were tested using the dominant lethal study, but there were no effects on preimplantation losses.

The teratogenic potential of VX was evaluated in Sprague Dawley rats and New Zealand rabbits exposed to VX at concentrations of up to 4 µg/kg subcutaneously during gestation (Goldman et al. 1988). No teratogenic activity was observed in either rats or rabbits, and no effects were noted for fetal weight, dead fetuses, resorptions, sex ratios, external visceral or skeletal abnormalities.

VX did not appear to be mutagenic using the Ames Salmonella assay, with and without metabolic activation for five different revertant bacterial strains. In addition, VX did not increase the recombinant activity in yeast Saccharomyces cerevisiae, and failed to induce forward mutations in the mouse lymphoma cells (Goldman et al. 1988).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

No regulated standards or criteria have been promulgated for VX.

REFERENCES

ARMY. 1975. Military Chemistry and Chemical Compounds Field Manual. Department of the Army and Air Force, Washington, D.C. October 1975. FM 3-9, AFR 355-7.

CHEMICAL STOCKPILE DISPOSAL PROGRAM. 1988. FINAL PROGRAMMATIC ENVIRONMENTAL IMPACT STATEMENT. January. Program Executive Officer--Program Manager for Chemical Demilitarization. Aberdeen Proving Ground, MD

GOLDMAN M., L.S. ROSENBLATT, B.W. WILSON, T.G. KAWAKAMI, M.R. CULBERTSON, J.P. SCHREIDER, J.F. REMSEN, M. SHIFRINE. 1988. Toxicity studies on agent VX. Laboratory for Energy-Related Health Research, Univ of Calif. U.S. Army Medical Research and Development Command. Fort Detrick. MD.

ICF FEASIBILITY STUDY. Toxicity of contaminants suspected to be present at old "O" field. December 1987.

RCRA FACILITY ASSESSMENT, n.d. Edgewood Area, APG, MD.

XYLENES

The three xylene isomers, compounds that have the same chemical constituents in a different configuration, have similar toxicological properties and are discussed together. Data from animals and humans suggest that approximately 60% of an inhaled dose is absorbed. Inference from metabolism and excretion studies suggests that absorption of orally administered xylene is nearly complete. Dermal absorption is reported to be minor following exposure to xylene vapor but may be significant following contact with the liquid (EPA 1985). In humans, acute inhalation exposures to relatively high concentrations of xylene adversely affect the central nervous system and lungs and can irritate mucous membranes (EPA 1987, Hake et al. 1981). Savolainen et al. (1980) observed that body balance and manual coordination were impaired in eight male students following inhalation exposure to m-xylene. However, tolerance against the observed effects developed during one work week. In experimental rats, long-term inhalation exposure to o-xylene resulted in hepatomegaly (Tatrai et al. 1981). Oral exposure to 200 mg/kg xylene in the diet for up to 6 months was also associated with liver toxicity, specifically the development of intracellular vesicles (Bowers et al. 1982). Prolonged oral exposures in mice resulted in hyperactivity, a manifestation of CNS toxicity (NTP 1986). Xylene appears to be fetotoxic and may increase the incidence of visceral and skeletal malformations in offspring of exposed experimental animals (Mirkova et al. 1983). There is suggestive evidence that xylene is carcinogenic in experimental animals when exposed by oral gavage (Maltoni et al. 1985).

EPA (1990a) calculated a chronic oral reference dose (RfD) for mixed xylenes of 2 mg/kg-day based on an NTP (1986) study in which male rats given a gavage dose of 179 mg/kg-day for 103 weeks did not exhibit hyperactivity, decreased body weight or significant increased mortality. The oral RfD was derived using the no-observed-adverse-effect level (NOAEL) of 179 mg/kg-day and an uncertainty factor of 100. EPA (1990b) calculated a subchronic oral RfD of 4 mg/kg-day for mixed xylenes based on a 13 week gavage study where no adverse effects were seen in rats. An uncertainty factor of 100 was used to calculate the RfD. EPA (1990b) reported a chronic and subchronic inhalation RfD for mixed xylenes of 0.3 mg/m^3 based on a study in which CNS effects, and nose and throat irritation were observed in humans exposed to 20 ppm (27 mg/m^3) for 5 days (Hake et al. 1981, Carpenter et al. 1975); an uncertainty factor of 100 was used to develop the RfD for both exposure levels.

REFERENCES

BOWERS, D.E., Jr., CANNON, M.S., and JONES, D.H. 1982. Ultrastructural changes in livers of young and aging rats exposed to methylated benzenes. Am. J. Vet. Res. 43:679-683

CARPENTER, C.P., KINKEAD, E.R., GEARY, D.L., Jr., SULLIVAN, L.J., and KING, J.M. 1975. Petroleum hydrocarbon toxicity studies: V. Animal and human response to vapors and mixed xylenes. Toxicol. Appl. Pharmacol. 33:543-558

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Drinking Water Criteria Document for Xylenes (Final Draft). Environmental Criteria and Assessment Office, Cincinnati, Ohio. March 1985. ECAO-CIN-416. EPA 600/X-84-185-1

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Health Advisory for Xylenes. Office of Drinking Water, Washington, D.C. March 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

HAKE, ET AL. 1981 (as cited in EPA 1989b)

MALTONI, C., CONTI, B., COTTI, G., and BELPOGGI, F. 1985. Experimental studies on benzene carcinogenicity at the Bologna Institute of Oncology: Current results and ongoing research. Am. J. Ind. Med. 7:415-416

MIRKOVA, E., ANTON, G., MIKHAILOVA, A., KHINKOVA, L., and BENCHEV, I.V. 1983. Prenatal toxicity to xylene. J. Hyg. Epidemiol. Microbiol. Immunol. 27:337-343

NATIONAL TOXICOLOGY PROGRAM (NTP). 1986. Toxicology and Carcinogenesis Studies of Xylenes (Mixed) [60% *m*-Xylene; 14% *p*-Xylene; 9% *o*-Xylene; 17% Ethylbenzene] in F344/N Rats and B6C3F$_1$ Mice (Gavage Studies). December 1986. NTP TR 327

SAVOLAINEN, K., RIIHIMAKI, V., SEPPALAINEN, A.M., and LINNOILA, M. 1980. Effects of short-term *m*-xylene exposure and physical exercise on the central nervous system. Int. Arch. Occup. Environ. Health 37:205-217

TATRAI, E., UNGVARY, G., and CSEH, I.R. 1981. The effect of long-term inhalation of *o*-xylene on the liver. Ind. Environ. Xenobiotics Proc. Int. Conf. Pp. 293-300

Zinc is absorbed in humans following oral exposure; however, insufficient data are available to evaluate absorption following inhalation exposure (EPA 1984). Zinc is an essential trace element that is necessary for normal health and metabolism and therefore is nontoxic in trace quantities (Hammond and Beliles 1980). Exposure to zinc at concentrations that exceed recommended levels has, however, been associated with a variety of adverse effects. In humans, chronic and subchronic inhalation exposure to zinc has been associated with gastrointestinal disturbances, dermatitis, and metal fume fever, a condition characterized by fever, chills, coughing, dyspnea, and muscle pain (EPA 1984). Chronic oral exposure of humans to zinc may cause anemia and altered hematological parameters (Pories et al. 1967, Prasad et al. 1975). Reduced body weights have been observed in studies in which rats were administered zinc in the diet. There is no evidence that zinc is teratogenic or carcinogenic (EPA 1984).

EPA (1990) has derived an oral reference dose (RfD) of $2x10^{-1}$ mg/kg-day for both chronic and subchronic exposures based on studies in which anemia and reduced blood copper were observed in humans exposed to oral zinc doses of 2.14 mg/kg-day (Pories et al. 1967, Prasad et al. 1975). A safety factor of 10 was used to develop the RfD.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Zinc (and Compounds). Office of Emergency and Remedial Response, Washington, D.C. EPA 540/1-86-048. September 1984

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1990. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. July 1990

HAMMOND, P.B., and BELILES, R.P. 1980. Metals. In Doull, J., Klaassen, C.D., and Amdur, M.O., eds. Casarett and Doull's Toxicology: The Basic Science of Poisons. 2nd ed. Macmillan Publishing Co., New York. Pp. 409-467

PORIES, W.J., HENZEL, J.H., ROB, C.G., and STRAIN, W.H. 1967. Acceleration of wound healing in man with zinc sulfate given by mouth. Lancet. 1:121-124

PRASAD, A.S., SCHOOMAKER, E.B., ORTEGA, J., et al. 1975. Zinc deficiency in sickle cell disease. Clin. Chem. 21:582-587

APPENDIX C

ECOLOGICAL TOXICITY PROFILES

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ALUMINUM

AQUATIC TOXICITY

The mechanism of aluminum toxicity in aquatic organisms is not known. It is possible that aluminum ions (Al^{3+}) compete with Mg^{2+} and Ca^{2+} in biological membrane functions (Wood 1985). Havas and Hutchinson (1983 in Malley and Chang 1985) found that aluminum may disrupt ionic balance. The principal site of damage following aluminum exposure appears to be the gill (Malley and Chang 1985).

Aluminum is amphoteric, with solubility lowest at a pH of 5.5 and increasing as pH deviates from 5.5 in either direction (EPA 1988). Attempts to relate toxicity with pH have produced conflicting results. Some researchers have reported increased aluminum toxicity with increased pH (Freeman and Everhardt 1971, Hunter et al. 1980 in EPA 1988) while others have reported decreased aluminum toxicity (Call 1984, Boyd 1979, and Kimball manuscript in EPA 1988). Due to aluminum's complex physico-chemical properties, it has not been possible to determine which form or species of aluminum is the most toxic. Inorganic monomers are believed to be the most toxic dissolved forms of aluminum (Palmer et al. 1988). Aluminum is known to complex with naturally occurring humic acids and form aggregates referred to as "floc" (EPA 1988). Floc has been demonstrated to affect smaller aquatic organisms such as the water flea (Daphnia magna) by physically impeding movement (Lamb and Bailey 1981).

Toxicity of aluminum to aquatic invertebrates, vertebrates, and plants is summarized below.

Invertebrates

Acute toxic effects in freshwater invertebrates have been reported at aluminum concentrations of 1,900 ug/L for Ceriodaphnia dubia (McCauley et al. 1986 in EPA 1988), 55,500 ug/L for the snail Physa sp. (Call 1984 in EPA 1988), and >79,900 ug/L for the midge Tanytarsus disimilis (Lamb and Bailey 1981). In chronic toxicity tests using D. magna, a 29% reduction in survival was noted at a concentration of 1,020 ug/L (Kimball manuscript in EPA 1988). In surviving organisms at this concentration, no effect on reproduction was observed. Chronic tests indicate that the invertebrates may be more sensitive to aluminum than vertebrates (EPA 1988).

Malley and Chang (1985) studied the effects of aluminum and pH on calcium uptake by post-molt crayfish (Orconectes virilis). At a neutral pH, an aluminum concentration of 200 ug/L had no effect on Ca^{2+} uptake. At pH 5.5 with no aluminum, a 70 % reduction in Ca^{2+} uptake from that in the control was reported. Aluminum concentrations as low as 200 ug/L at pH 5.5 reduced Ca^{2+} uptake by 80% from that in the control. However, the degree of inhibition did not increase directly with increased aluminum concentrations and therefore no dose-response relationship was identified.

Fish

A 48-hour LC_{50} of 4,000 ug/L was reported by Muramoto (1981 in EPA 1988) for carp (Cyprinus carpio) and a 96-hour LC_{50} of 3,680 ug/L was reported for brook trout (Salvelinus fontinalis) (Decker and Menendez 1974 in EPA 1988). In a test using rainbow trout (Salmo gairdneri) exposed for 72 hours, an LC_{50} of 5,200 ug/L was reported (Freeman and Everhardt 1971 in EPA 1988). In early-life stage tests using fathead minnows (Pimephales promelas) at concentrations of 2,300 and 4,700 ug/L, survival was equal to or better than survival in the control group (Kimball manuscript in EPA 1988).

Palmer et al. (1988) investigated the comparative sensitivities of bluegill (Lepomis macrochirus), channel catfish (Ictalurus punctatus), and fathead minnow to aluminum at different pHs in 96-hour tests. They found that fathead minnow were the most sensitive, followed by channel catfish and bluegill. They reported that at pH 6.5 to 7.5, aluminum was not toxic to these species. At pH 5.5, however, aluminum concentrations of 100 and 200 ug/L caused complete mortality in fathead minnow and channel catfish, respectively. An aluminum concentration of 400 ug/L caused only 73% mortality in juvenile bluegills at pH 5.5.

Toxicity values are available for two fish species that occur at or near Aberdeen Proving Ground. Species mean acute values reported by EPA (1988) for juvenile channel catfish and yellow perch (Perca flavescens) are >47,900 and >50,000 ug/L, respectively. Out of seven fish species tested, channel catfish and yellow perch are 5th and 6th with regard to species sensitivity.

Amphibians

Birge (1978 in EPA 1988) and Birge et al. (1978 and 1979 in EPA 1988) conducted embryo-larval tests using two amphibian species. They report a 7-day EC_{50} for death and deformity of 50 ug/L aluminum (pH=7.4) for the narrow-mouthed toad (Gastrophryne carolinensis), and an 8-day EC_{50} of 2,280 ug/L aluminum for the marbled salamander (Ambystoma opacum).

Plants

Tests for aquatic phytotoxicity of aluminum report effects at concentrations as low as 460 ug/L for the green algae Selenastrum capricornutum (Call 1984 in EPA 1988). Decreased root weights were reported in the Eurasian watermilfoil at 2,500 ug/L (Stanley 1974 in EPA 1988).

Sediment Toxicity

Data regarding toxicity of aluminum-containing sediments were not available in the literature.

Bioaccumulation

Data regarding bioaccumulation in invertebrate species were not found in the literature. Cleveland et al. (1986 in EPA 1988) reported whole-body bioconcentration factors (BCFs) for "eyed-embryo" (post-hatch) and 37 day-old brook trout. Following 15 days of exposure, BCFs of 147 and 231 were reported for the hatchlings and juveniles, respectively. Following 30 days of exposure, BCFs for these life stages had decreased to 50 and 136, respectively.

Criteria

EPA has established 1-hour and 4-day ambient water quality criteria of 750 and 87 ug/L, respectively, for protection of freshwater aquatic life (EPA 1988). Too few data regarding aluminum toxicity to saltwater species are available to derive criteria.

TERRESTRIAL TOXICITY

Toxicity information for terrestrial wildlife is limited. Aluminum has no known biological function (Wood 1985). Although the mechanism of aluminum toxicity is not known, the most probable mechanism is alteration of membrane function (Wood 1985) similar to that in aquatic organisms. The toxicity of aluminum to higher organisms depends upon the inorganic speciation of its salts. Aluminum uptake

has been correlated with diseases of the central nervous system (Wills and Savory 1985, Perl 1985 in Wood 1985). NAS (1980) reports that aluminum toxicity in domestic birds and mammals is generally expressed as a secondary phosphorus deficiency. Aluminum is believed to bind with phosphorus in the intestine, making it unavailable for absorption.

Birds

Shafer et al. report a LD_{50} of greater than 111 mg/kg body weight in red-winged blackbirds (Agelaius phoeniceus) (Shafer et al. 1983). Cakir et al. (1978 in NAS 1980) report no effect concentrations of 486 ppm aluminum in the diet for both turkeys and chicks.

Mammals

No toxicity studies using wild mammalian species were located in the literature. No adverse effects were observed in calves and sheep fed 1,200 and 1,000 ppm respectively aluminum in the diet (Valdivia et al. 1978 and Bailey 1977 in NAS 1980). Yokel (1983) reported learning deficiencies in adult rabbits dosed with 0.5 and 1.1 mg aluminum/kg body weight. Rabbits dosed with 1.6 mg/kg required 50% more food to maintain a body weight comparable to that of the control animals. The author hypothesized that this may be due to alterations in phosphorus metabolism, reduced glucose absorption, or disturbances in carbohydrate metabolism induced by aluminum.

Plants

No data were located regarding toxicity of aluminum to wild plants. Wallace and Romney (1977 in Gough et al. 1979) found that concentrations of 8,000 ug/L in culture solutions resulted in toxic threshold concentrations in rice shoots and soybean leaves of 20 mg/kg and 30 mg/kg, respectively. They found that most aluminum remained in the roots. Soil pH influences the availability of aluminum in the soil to plants.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Ambient Water Quality Criteria for Aluminum-1988. Office of Water Regulations and Standards, Criteria and Standards Division. Washington, D.C. EPA 440/5-86-008.

GOUGH, L.P., SHACKLETTE, H.T., and CASE, A.A. 1979. Element concentrations toxic to plants, animals, and man. U.S. Government Printing Office Washington, D.C. Geological Survey Bulletin 1466.

LAMB, D.S. and BAILEY, G.C. 1981. Acute and chronic effects of alum to midge larva (Diptera: Chironomidae). Bull. Environ. Contam. Toxicol. 27:59-67.

MALLEY, D.F. and CHANG, P.S. 1985. Effects of aluminum and acid on calcium uptake by the crayfish Orconectes virilis. Arch. Environ. Contam. Toxicol. 14:739-747.

NATIONAL ACADEMY OF SCIENCES (NAS). 1980. Mineral Tolerance of Domestic Animals. Subcommittee on mineral toxicity in animals. National Research Council. Washington, D.C.

PALMER, R.E., KLAUDA, R.J., and LEWIS, T.E. 1988. Comparative sensitivities of bluegill, channel catfish and fathead minnow to pH and aluminum. Environ. Toxicol. and Chem. 7:505-516.

SCHAFER, E.W., JR., BOWLES, W.A., JR. and HURLBUT, J. 1983. The acute oral toxicity, repellency, and hazard potential of 998 chemicals to one or more species of wild and domestic birds. Arch. Environ. Contam. Toxicol. 12:355-382.

WOOD, J. 1985. Effects of acidification on the mobility of metals and metalloids: an overview. Environ. Health Perspec. 63:115-119.

YOKEL, R.A. 1983. Repeated systemic aluminum exposure effects on classical conditioning of the rabbit. Neurobehav. Toxicol. and Teratol. 5:41-46.

AMMONIA

AQUATIC TOXICITY

In aqueous solutions, unionized ammonia (NH_3 H_20) exists in equilibrium with the ammonium ion (NH_4^+) and hydroxide. The toxic effect of ammonia in aquatic species is due principally to the unionized form which penetrates cell membranes more readily. Ammonia is acutely and chronically toxic to both freshwater and saltwater aquatic life (EPA 1986).

Invertebrates

Acute toxicity to freshwater invertebrates is reported at concentrations of 530 to 22,800 ug/L, and chronic toxicity is reported at concentrations as low as 304 ug/L (EPA 1986). Acute toxicity to saltwater invertebrates is reported at concentrations as low as 189 ug/L (EPA 1987). Only one chronic study was available regarding saltwater invertebrates (EPA 1987). This study reported a chronic value of 122 ug/L for mysid shrimp, Mysidopsis bahia. Invertebrates appear to be more tolerant than fish to elevated ammonia (EPA 1987).

Fish

Acute toxicity to freshwater fish is reported at NH_3 concentrations ranging from 83 to 4,600 ug/L, and chronic toxicity in fish is reported at concentrations as low as 17 ug/L (EPA 1986). Concentrations of ammonia acutely toxic to freshwater fish can cause loss of equilibrium, hyperexcitability, increased breathing, oxygen uptake, and in severe cases, convulsions and death (WHO 1986). At lower concentrations, ammonia can affect reproduction and induce pathological changes in gill, liver and kidney tissue (WHO 1986). Acute and chronic toxicity increase with decreasing pH; acute toxicity increases with decreasing temperature.

Acute toxicity to saltwater fish species occurs at concentrations ranging from 140 ug/L for juvenile Atlantic silverside (Menidia menidia) to 5600 for the three-spined stickleback (Gasterostaus aculeatus). Acute data are available for 8 species found at APG. The most sensitive species other than Atlantic silverside (reported above) was striped bass (Morone saxatilis), with an LC_{50} of 330 reported for larval-stage individuals. Values for other species ranged from 390 ug/L for embryo-larval stage of red drum (Sciaenops ocellatus) to 2,130 for larval stages of white perch (Morone americana) (EPA 1987).

Amphibians

Data regarding toxicity of ammonia to amphibian species were not located in the literature.

Plants

Ammonia low concentrations functions as a nitrogen source, but excess ammonia can be toxic to plants. Based on limited data, freshwater plant species appear to be considerably more tolerant to NH3 than invertebrates or fish (EPA 1986). Saltwater plants appear to be much more sensitive to the presence of ammonia. A concentration of 39 ug/L reduced reproduction in the red macroalgae Champia parvula, while a concentration of 5 ug/L had no effect on the species (Thursby 1986 in AQUIRE 1990).

Sediment Toxicity

No sediment toxicity data were available in the literature.

Bioaccumulation

Bioaccumulation data were not found in the literature.

Criteria

EPA (1986) presents ambient water quality criteria for unionized ammonia for the protection of freshwater aquatic life based upon ambient water temperature and pH and for situations where salmonids and other sensitive coldwater species were present and absent . Ambient water criteria can be determined by assuming an average pH of 7 for APG surface water, an average temperature of 20°C, and that salmonids and other sensitive coldwater species are absent. The corresponding 4-day and 1-hour criteria are 8.3 and 93 ug/L, respectively.

EPA (1987) has proposed criteria for unionized ammonia for the protection of saltwater aquatic life. These criteria are not dependent on pH or temperature. Based on the proposed criteria, saltwater species should not be adversely affected if the 4-day average concentration of unionized ammonia does not exceed 13 ug/L and the 1-hour average concentration does not exceed 108 ug/L more than once every 3 years.

TERRESTRIAL TOXICITY

Wildlife

Data regarding toxicity to terrestrial wildlife were not located in the literature. Although ammonia is toxic to terrestrial animals, it is found in the urinary tract, where it regulates the pH of the urine by neutralizing excess acids (Schmidt-Nielsen 1983).

Plants

Nitrogen is one of the six major elements essential to plants (N,P,K,Ca,Mg,S). Plants obtain nitrogen from the atmosphere via nitrogen fixation (Galston et al. 1980). Ammonium ion (NH^{4+}) can be absorbed by plant roots, but it is usually oxidated by soil microorganisms to nitrate (NO_3), which is then readily absorbed by plants (Schmidt-Nielsen 1983).

REFERENCES

AQUATIC INFORMATION RETRIEVAL (AQUIRE). 1990. Computerized database. Chemical Information Systems, Inc. Baltimore, Maryland.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Quality Criteria for Water. Office of Water Regulations and Standards. EPA 440/5-86-001.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Water quality criteria: availability of document. Fed. Reg. 52(40):6213-6216 (March 2, 1987).

GALSTON, A.W., DAVIES, R.L., and SATTER, R.L. 1980. The Life of the Green Plant. Prentice-Hall, Inc. Englewood Cliffs, New Jersey.

SCHMIDT-NIELSEN, K. 1983. Animal physiology: Adaptation and Environment. Cambridge University Press. New York, New York.

WORLD HEALTH ORGANIZATION (WHO). 1986. Environmental Health Criteria. Ammonia. Geneva.

ANTIMONY

AQUATIC TOXICITY

The aquatic toxicity of antimony (Sb) has been reviewed by EPA (1988). There are primarily two oxidation states of antimony in natural waters: Sb(III), and Sb(V) (EPA 1988). The trivalent state predominates under moderately oxidizing conditions, whereas the pentavalent form occurs in aerobic alkaline environments (EPA 1988); however, toxicity to antimony is attributed to the trivalent form (EPA 1988). Precipitation of antimony, primarily as antimony trioxide of antimony oxychloride, can be an important factor limiting soluble antimony in natural waters (EPA 1988). However, the precipitation of antimony can be influenced by the aqueous chlorine concentration (EPA 1988).

Invertebrates

Hydra are the most sensitive freshwater animal species to antimony (III) based on the nine freshwater genera tested. The mean acute value for the Hydra oligactic is 500 ug/L, which is at least 51 times more sensitive than the most resistant genus (EPA 1988). Cladocerans are the second most sensitive freshwater animals species. Mean acute value for these species are: Ceriodaphnia dubia 3,470 ug/L and Daphnia magna 18,140 ug/L (EPA 1988). Acute toxicity values reported for the caddisfly (Pycnopshye sp.), amphipod (Gammarus psedolimnaeus), and annelid (Lumbriculus variegatus) exceed the highest soluble antimony (III) levels attained, 25,700 ug/L (EPA 1988). The only freshwater invertebrate chronic toxicity value is 3,218 ug/L for Daphnia magna (EPA 1988).

Acute toxicity values (species mean acute values) for antimony (III) for seven species of saltwater invertebrates range from 3,780 ug/L for the adult sea urchin Lytechinus pictus to greater than 53,400 ug/L for the adult isopod Idothea resecata (EPA 1988). No chronic toxicity values were found for saltwater invertebrates.

Fish

For four species of freshwater fish, the mean acute values for antimony (III) range from 21,800 ug/L to greater than 25,800 ug/L (which exceeded the highest soluble antimony (III) levels attained) (EPA 1988). The 8-day dietary LC_{50} for juvenile fathead minnows (Pimephales promelas) of 20,200 ug/L (EPA 1988); whereas, an 8-day EC_{50} (based on death and deformity) of 11,300 ug/L for embryo-larval goldfish (Carassius auratus) exposed to antimony (III) was reported by Birge (1978 in EPA 1988). A 27-day EC_{50} for embryo-larval rainbow trout (Salmo gairdneri) is 660 ug/L (Birge et al. 1980 in EPA 1988). The chronic value for fathead minnow for trivalent antimony is 1,616 ug/L (EPA 1988).

Species mean values for saltwater fish species range from 4,800 ug/L to greater than 1,000,000 ug/L (based on four species). The chronic values reported from two early life-stage tests with the inland silverside (Menidia beryllina) are 2,874 and 3,016 ug/L (Hughes and Boothman 1987 in EPA 1988). There was no significant reduction in survival or growth observed at 1,130 ug/L (EPA 1988).

Toxicity values are available for only a limited number of fish species that occur at or near the Aberdeen Proving Ground (APG). The only species mean acute value for antimony (III) for freshwater fish species associated with APG is bluegill (Lepomis machrochirus) >25,800 ug/L (EPA 1988). An 8-day EC_{50}, based on death and deformity, of 11,300 ug/L was determined for embryo-larval stages of the goldfish exposed to antimony (III) (EPA 1988). The species mean values for saltwater fish species are: mummichog (Fundulus heterclitus) >1,000,000 ug/L and inland silverside 7,830 ug/L. The inland silverside chronic values are 2,874 and 3,016 ug/L (EPA 1988).

Amphibians

Birge et al. (1979 in EPA 1988) determined a 7-day EC_{50} of 300 ug/L for embryo larva of the narrow-mouthed toad Gastrophryne carolinensis based on death and deformity within 4 days of post-hatching.

Plants

Data on the effects of antimony (III) on freshwater plants is limited to two 4-day studies. An EC_{50} (reduction in chlorophyll a) of 610 ug/L for the green alga (Selenastrum capricornutum) was reported by the EPA (1978 in EPA 1988); whereas, no effects were observed in the duchweed Lemna minor at the highest soluble concentration of antimony trichloride, 25,200 ug/L (EPA 1988).

Toxicity values for saltwater plants is limited to one 96-hr EC_{50} (reduction in chlorophyll a) of 4,200 ug/L for the diatom Skeletonema costatum reported by Brooke et al. (1986 in EPA 1988).

Sediment Toxicity

Barrick and Beller (1989) reported sediment toxicity values in terms of apparent effect thresholds (AETs). For antimony, they report an amphipod AET of 200 mg/kg, and a benthic AET of 150 mg/kg (expressed as dry weight).

Bioaccumulation

Limited information is available on the bioaccumulation of antimony in freshwater aquatic organisms. Barrows et al. (1980 in EPA 1988) observed no significant accumulation in whole body residues of antimony (III) in bluegill after 28 days of exposure.

No data are available on the bioaccumulation of antimony (III) in salt water organisms, but antimony is known to occur in the tissues of saltwater species (EPA 1988).

Criteria

The EPA (1988) has proposed ambient water quality criteria for antimony (III). For freshwater aquatic organisms, the 4-day average concentration criterion (chronic criterion) is 30 ug/L (not to be exceeded more than once every three years on the average) and the 1-hour average concentration (acute criterion) of 88 ug/L for antimony (III). For saltwater organisms, the acute and chronic criteria are 1,500 ug/L and 500 ug/L, respectively. EPA considers the acid-soluble measurement of trivalent antimony to provide the most appropriate measurement for aquatic life criteria; however, because there is no EPA-approved method for this measurement, EPA suggests that measurement of acid-soluble antimony (III), acid-soluble antimony, and total recoverable antimony be evaluated (EPA 1988).

TERRESTRIAL TOXICITY

Information on the toxicity of antimony to terrestrial wildlife is quite limited. Available information is discussed below.

Birds

Shepard (1980 in HSDB 1990) reported no adverse effects in chicks given 0.1 mg antimony potassium tartrate on the 4th day. No other avian toxicity information was found.

Mammals

Symptoms of acute antimony poisoning in animals include vomiting, diarrhea, and gatrointestinal tract irritation, marked weight loss, hair loss, increase in the number of eosinophilic cells, and heart failure (HSDB 1990).

Information on the toxicity of antimony to mammalian laboratory and wildlife species is limited. No fetal changes were observed in four sheep given 2 mg/kg of antimony potassium tartrate during the major portion of gestation (Shepard 1980 in HSDB 1990). Chronic incorporation of 5 mg antimony/L in drinking water increased serum cholesterol levels and reduced serum glucose levels in rats (Schroeder et al. 1970 in HSDB 1990).

Plants

No information was found on the phytotoxicity of antimony.

REFERENCES

BARRICK, R.C. and BELLER, H.R. 1989. Reliability of sediment quality assessment in Puget Sound. In: Oceans '89 Proceedings. Inst. of Electrical and Electronic Engineers. Piscataway, New Jersey. Publication No. 89CH2780-5.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Ambient Water Quality Criteria for Antimony (III) - Draft. Office of Water Regulations and Standards. Washington, D.C.

HAZARDOUS SUBSTANCES DATABANK (HSDB). 1990. Computerized Database.

AQUATIC TOXICITY

The aquatic toxicity of arsenic has been reviewed by EPA (1985), Eisler (1988), and Sorensen (1987). Arsenic is among the most toxic of the metalloids to freshwater fish (Sorensen 1987). Acute toxicity in fish is most likely a result of suffocation as a result of mucus build-up over the gills (Sorensen 1987). The liver, because of its detoxication role, is considered the primary target organ in chronic arsenic exposures (Sorensen 1987). Some fish species have shown some indication of acclimation to arsenic exposure, that is, toxicity has decreased in organisms previously exposed to arsenic (Rand and Petrocelli 1985). The trivalent form (arsenite or arsenic [III]) is significantly more toxic to fish than the pentavalent form of arsenic (arsenate or arsenic [V]) (Sorensen 1987, Burton et al. 1987). In addition, the inorganic forms, generally, are more toxic than the organoarsenicals (Eisler 1988). The acute toxicity of arsenic can be affected by a number of factors including water temperature, pH, phosphate concentration, and suspended solids (Eisler 1988).

Invertebrates

Freshwater amphipods and cladocerans are more sensitive than freshwater fish to arsenic. Mean acute values for these species are: Gammarus pseudolimnaeus 874 ug/L, Simocephalus spp. 1,175 ug/L, Ceriodaphnia reticulata 1,800 ug/L, and Daphnia spp. 2,444 ug/L (EPA 1985). Stoneflies are relatively tolerant of arsenic; the acute value for Pteronarcys californica is 22,040 ug/L (EPA 1985). No acute toxicity values were reported by EPA (1985) for mayflies or caddisflies, and toxicity information for these groups is limited. The chronic value for the cladoceran Daphnia magna exposed to arsenic (III) is 914.1 ug/L (EPA 1985).

Saltwater invertebrates are more sensitive than saltwater fish to arsenic (III). Acute toxicity values (species mean acute values) for arsenic (III) for nine species of saltwater invertebrates range from 252 ug/L for dungeness crab to 10,120 ug/L for polychaete worms (EPA 1985). The acute values for arsenic (V) are 2,319 ug/L for mysid and 4,611-5,110 ug/L for the amphipod Ampellsca abdita (EPA 1985). Only one chronic toxicity value for arsenic is available for saltwater invertebrates. The chronic value for arsenic (III) for the mysid is 895.2 ug/L (EPA 1985). This value is approximately two times less than the acute value for mysid of 1,740 ug/L.

Fish

Species mean acute values for arsenic (III) for seven species of freshwater fish range from 13,340 ug/L to 41,760 ug/L (EPA 1985). Trout are among the most sensitive freshwater fish species tested with arsenic and the most sensitive fish are rainbow trout (Oncorhynchus mykiss) with acute effects occurring at 10,800 ug/L (arsenic[V]) in fish two months old under static conditions (Hale 1977). A 96-hour LC_{50} for adult rainbow trout of 23,000 ug/L was reported for static conditions by Johnson and Finley (1980). An acute LC_{50} for brook trout (Salvelinus fontinalis) of 14,960 ug/L for flow-through conditions was reported by Cardwell et al. (1976 in EPA 1985). The 28-day EC_{50} (based on death and deformity) for embryo-larval rainbow trout exposed to arsenic (III) is 550 ug/L (EPA 1985; Pickering et al. 1983). The EC_{10} for this study was 134 ug/L (Birge et al. 1980, 1981 in EPA 1985). The chronic value for fathead minnow (Pimephales promelas) for trivalent arsenic is 3,026 ug/L and for pentavalent arsenic is 891.6 ug/L (EPA 1985). The flagfish (Jordanella floridae) chronic value for trivalent arsenic of 2,962 ug/L is similar to the chronic value for fathead minnow (EPA 1985).

Species mean acute values for saltwater fish exposed to trivalent arsenic : nge from 12,700 ug/L to 16,033 ug/L (based on three species) (EPA 1985). No chronic values for saltwater fish are available in EPA (1985).

Toxicity information is available for some fish species that occur at or near the Aberdeen Proving Ground (APG). The available "species mean acute values" for arsenic (III) for freshwater fish associated with APG are: bluegill (Lepomis macrochirus) 41,760 ug/L, goldfish (Carassius auratus) 26,040 ug/L, and channel catfish (Ictalarus puntatus) 18,100 ug/L. The species mean acute value for Atlantic silverside (Menidia menidia), a saltwater species, is 16,033 ug/L (EPA 1985). An 8-day EC_{50}, based on death and deformity, of 42,100 ug/L was determined for largemouth bass (Micropterus salmoides) embryo and larvae exposed to trivalent arsenic (EPA 1985).

Amphibians

Two studies reporting effects on amphibians were found. For the narrow-mouthed toad (Gastrophryne carolinenesis) in an early life-stage test, an EC_{50} of 40 ug/L (As [III]) was reported for a 7-day exposure (Birge 1978). Death or malformations of embryos were observed at this concentration (Eisler 1988). An EC_{50} of 4,450 ug/L (As[III]), based on fatalities and deformities, was reported for the marbled salamander (Ambystoma opacum) after 8 days of exposure (Birge et al. 1978 as cited in EPA 1985).

Plants

Based on limited information, arsenic (V) may be more toxic to aquatic plants than arsenic (III). The freshwater algae, Selenastrum capricornutum showed 50 percent growth inhibition in 4 days at 690 ug/L arsenic (V). The lowest concentration of arsenic (III) resulting in 50 percent growth inhibition in the same species of alga was 31,200 ug/L (EPA 1985). Three species of freshwater algae (Cladophora sp., Spirogyra sp., and Zygnema sp.) showed 100 percent kill within two weeks when exposed to 2,320 ug/L arsenic (III) (EPA 1985). Potamogeton sp. (a freshwater submerged plant species) had 95 percent kill within one month at 2,320 ug/L arsenic (III) (EPA 1985). The most sensitive freshwater plant species to arsenic (V) was the alga Scenedesmus obliquus which had a 14-day EC_{50} of 48 ug/L (EPA 1985). Two other algae species showed decrease growth at 75 ug/L (EPA 1985). The most sensitive saltwater plant tested is the alga Skeletonema costatum which showed growth inhibition at 19 ug/L arsenic (III) and 13 ug/L arsenic (V) (EPA 1985). A wide range of sensitivities occurs among saltwater plant species. The highest acute concentrations reported to have no effect on growth were for the alga Hymenomonas carterae, 10,000 ug/L for trivalent arsenic, and 150,000 ug/L for pentavalent arsenic (EPA 1985).

Sediment Toxicity

Information on the toxicity of arsenic-contaminated sediments is limited. Pavlou and Weston (1983 in Pavlou 1987) reviewed proposed limits for arsenic in sediments and found values ranging from 3-8 mg/kg. Using the equilibrium partitioning approach (assuming 2 percent organic carbon in sediments), Pavlou and Weston estimated a safe level of arsenic in marine sediments of 16 mg/kg (dry weight). Barrick and Beller (1989) reported sediment toxicity values in terms of apparent effect thresholds (AETs). They report an AET values for arsenic of 57, 93, and 700 mg/kg (dry weight) for amphipods, oysters, and benthic organisms, respectively. Burton et al. (1987) showed that the toxicity of arsenic to Daphnia magna was reduced by the addition of lake sediments to the test chamber. They concluded that arsenic became less available to the test organism via adsorption to the sediments.

Bioaccumulation

EPA (1985) reported that arsenic may bioaccumulate in lower forms of aquatic life more readily than in fish, and that bioaccumulation potential is similar for As(III) and As(V). Bottom feeding fish tend to accumulate more arsenic than pelagic fish (Sorensen 1987). Whole body bioconcentration factors (BCF) of 3 and 17 have been reported for arsenic (III) for the snails Stagnicola emarginata and Helisoma campanulatum, respectively (EPA 1985). BCF values for the same species exposed to arsenic (V) are 3 and 6, respectively. Spehar et al. (1980) reported a BCF of 219 for the cladoceran Daphnia magna after 21-days exposure to arsenic (III) (96 ug/L - static concentration). A BCF for arsenic (V) of 131 was reported for the stonefly Pteronarcys dorsata, based on a 28-day flow through study (Spehar et al. 1980).

The only BCF reported for a saltwater species is a 112-day value of 350, based on arsenic (III) in soft parts of the eastern oyster (Crassostrea virginica) (EPA 1985).

EPA (1985) found no accumulation of arsenic (both inorganic and organic forms) in rainbow trout (Salmo gairdneri) (whole body) after 28-day exposures. A BCF of 4 and a biologic half-life of one day was derived for bluegills (Lepomis macrochirus) for arsenic (III) (EPA 1978). A BCF of 3 was reported for fathead minnow when exposed to arsenic (V) for 30 days (DeFoe 1982 as cited in EPA 1985). Eisler (1988) also indicated that bioconcentration of arsenic by aquatic organisms is generally low (BCF less than 17). Oladimeji et al. (1982 in EPA 1985) found that preexposure of rainbow trout to arsenic (III) enhanced the elimination of subsequent dosing. These studies suggest that some elimination pathway is induced in freshwater fish through exposure to low (non-lethal) doses of arsenic (III). Reduced growth and survival has been reported in immature bluegills when arsenic concentrations in their muscle tissue exceed 1.3 mg/kg fresh weight (NRCC 1978 in Eisler 1988).

No BCFs were found for saltwater fish.

Anderson et al. (1980 in EPA 1985) reported BCFs of 2 to 5 for four species of freshwater aquatic plants, after 42 days of exposure. The plants evaluated were Hydrophila lacustris, water hyacinth (Eichhornia crassipes), alligator weed (Alternanthera philoxeroides), and duckweed (Lemna minor).

No BCFs were found for saltwater plants.

Criteria

EPA (1985) has established, for freshwater aquatic species, a 4-day average concentration criterion (chronic criterion) of 190 ug/L (not to be exceeded more than once every three years) and a 1-hour average concentration criterion (acute criterion) of 360 ug/L for trivalent (III) arsenic. EPA (1985) considers the acid-soluble form of arsenic to be the most appropriate measurement for toxicity, however, because there is currently no EPA-approved method for this measurement, EPA recommends that total recoverable arsenic be evaluated. Thus, these criteria may be overly protective when based on total recoverable arsenic. Insufficient data are available to derive criteria for pentavalent (V) arsenic according to EPA (1985). The acute and chronic freshwater LOELs for pentavalent arsenic are 850 ug/L and 48 ug/L, respectively (EPA 1986) based on toxic effects on freshwater plants. The acute and chronic marine criteria for trivalent arsenic are 69 ug/L and 36 ug/L, respectively (EPA 1986). There is insufficient information to develop marine criteria for pentavalent arsenic; the acute and chronic marine LOELs are 2,319 ug/L and 13 ug/L, respectively (EPA 1986).

TERRESTRIAL TOXICITY

The toxicity of arsenic (As) to terrestrial wildlife has been recently reviewed by Eisler (1988). Trivalent arsenic toxicity is primarily due to reaction of arsenic with sulfhydryl groups of proteins followed by enzyme inhibition (Eisler 1988). Pentavalent arsenic probably acts by uncoupling oxidative phosphorylation; it does not react with sulfhydryl groups as readily (Eisler 1988). Chronic poisonings in wild animals have not been clearly documented. In laboratory species, arsenic has been found to be carcinogenic, teratogenic, embryotoxic, and fetotoxic. Information on the essentiality of arsenic in animals is limited (NAS 1980, Eisler 1988). Signs of arsenic deficiency have been reported in rats. Deficiency has also been reported in pigs and goats at dietary levels of less than 0.05 mg/kg (Eisler 1988).

Birds

A median lethal dietary concentration of 480 ppm (approximately 58 mg/kg-body weight) has been reported for the bobwhite quail (Colinus virginianus) (Heath et al. 1972, Hill et al. 1975). Mallards (Anas platyrhynchos) may be less sensitive to arsenic than are bobwhite quail based on the results of an acute oral study that reported an LD_{50} for mallards of 323 mg/kg-body weight (Eisler 1988). No chronic toxicity studies with mallards were located in the literature reviewed for this assessment. The NOEL for chickens (22 weeks old) is 10 ppm (in diet) after 56 days of exposure; at 100 ppm, decreased body weight, feed intake, and egg production occurred (Hermayer et al. 1977 in NAS 1980). In this study there were four birds per treatment. The level of 10 ppm in feed is equivalent to a dose of approximately 1.25 mg/kg-body weight, based on the dietary conversion factor for young chickens in Lehman (1954) of 0.125 mg/kg-body weight per 1 ppm in feed.

Mammals

For mammals, median lethal doses (LD_{50}) have been reported in the range of 10.5 to 40.4 mg/kg body weight (NRC 1977). Lethal oral doses for most domestic mammals appear to be between 1 to 25 mg/kg, as sodium arsenite, and 3 to 10 times that range as arsenic trioxide.

NAS (1980) has proposed the following maximum tolerable levels for dietary arsenic for domestic animals: 50 ppm for inorganic arsenic and 100 ppm for organic forms of arsenic. NAS (1974) recommends a safe upper limit for the concentration of arsenic in drinking water for livestock and poultry of 0.5 mg/L. Puls (1988) recommends that arsenic in drinking water for livestock does not exceed 0.05 mg/L, and for poultry 0.2 mg/L.

Plants

Arsenic is toxic to terrestrial plants. It can inhibit mitosis, photosynthesis, and respiration, and interfere with nucleic acid and protein synthesis. Soil arsenic concentrations of 500 mg/kg have been reported to completely inhibit growth in six vegetable crops (NRC 1977). Concentrations of arsenic in soil (dry weight) from 15 to 50 mg/kg (mean of five studies = 28 mg/kg) have been reported to be phytotoxic (Kabata-Pendias and Pendias 1984).

REFERENCES

BARRICK, R.C. and BELLER, H.R. 1989. Reliability of sediment quality assessment in Puget Sound. In: Oceans '89 Proceedings. Inst. of Electrical and Electronic Engineers. Piscataway, New Jersey. Publication No. 89CH2780-5.

BIRGE, W.J. 1978. Aquatic toxicology of trace elements of coal and fly ash. In: Thorp, J.H., and Gibbons, J.W. (eds.). Energy and Environmental Stress in Aquatic Systems. National Technical Information Service, Springfield, Virginia. CONF-771114.

BURTON, G.A. Jr., LAZORCHAK, J.M., WALLER, W.T. and LANZA, G.R. 1987. Arsenic toxicity changes in the presence of sediment. Bull. Env. Cont. Tox. 38:491-499.

EISLER, R. 1988. Arsenic Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Department of the Interior, Fish and Wildlife Service. Laurel, Maryland. Contaminant Hazard Reviews Report No. 12, Biological Report 85(1.12).

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1978. In-depth Studies on Health and Environmental Impacts of Selected Water Pollutants. U.S. Environmental Protection Agency. Contract No. 68-01-4646.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Ambient Water Quality Criteria for Arsenic-1984. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-84-003. PB85-227445.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Quality Criteria for Water 1986. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-86-001.

HALE, J.G. 1977. Toxicity of metal mining wastes. Bull. Environ. Contam. Toxicol. 17:66.

HEATH, R.J., SPANN, J.W., HILL, E.F. and KREITZER, J.F. 1972. Comparative Dietary Toxicities of Pesticides to Birds. U.S. Department of Interior. Washington, D.C. Fish and Wildlife Service Special Scientific Report--Wildlife. No. 152.

HILL, E.F., HEATH, R.G., SPANN, J.W. and WILLIAMS, J.D. 1975. Lethal Dietary Toxicities of Environmental Pollutants to Birds. U.S. Department of the Interior. Washington, D.C. Fish and Wildlife Service, Special Scientific Report--Wildlife No. 191.

JOHNSON, W.W., and FINLEY, M.T. 1980. Handbook of Acute Toxicity of Chemicals to Fish and Aquatic Invertebrates. U.S. Fish and Wildlife Service. Washington, D.C. Resource Publication 137.

KABATA-PENDIAS, A. and PENDIAS, H. 1984. Trace Elements in Soil and Plants. CRC Press. Boca Raton, Florida.

LEHMAN, A.J. 1954. Appraisal of the study of chemicals in food, drugs, and cosmetics. U.S. Association of Food and Drug Officials. 18:66.

NATIONAL ACADEMY OF SCIENCES (NAS). 1974. Nutrients and Toxic Substances in Water for Livestock and Poultry. National Research Council. Washington, D.C.

NATIONAL ACADEMY OF SCIENCES (NAS). 1980. Mineral Tolerance of Domestic Animals. National Research Council, Subcommittee on Mineral Toxicity in Animals. Washington, D.C.

NATIONAL RESEARCH COUNCIL (NRC). 1977. Drinking Water and Health. National Academy of Sciences. Washington, D.C.

PAVLOU, S.P. 1987. The use of the equilibrium partitioning approach in determining safe levels of contaminants in marine sediments. In: Dickson, K.L., Maki, A.W., and Brungs, W.A. (eds.). Fate and Effects of Sediment-Bound Chemicals in Aquatic Systems. Proceedings of the Sixth Pellston Workshop, Florissant, Colorado, August 12-17, 1984. SETAC Special Publication Series, Pergamon Press. New York, New York. Pp. 388-412.

PICKERING, Q.H., HUNT, E.P., PHIPPS, G.L., ROUSH, T.H., SMITH, W.E., SPEHAR, D.L., STEPHENS, C.E., and TANNER, D.K. 1983. Effects of pollution on freshwater fish and amphibians. JWPCF. 55(6):840-862.

PULS, R. 1988. Mineral Levels in Animal Health. Sherpa International. Clearbrook, British Columbia.

RAND, G.M., and PETROCELLI, S.R. 1985. Fundamentals of Aquatic Toxicology. Hemisphere Publishing Corp. Washington, D.C.

SORENSEN, E.M.B. 1987. The effects of arsenic on freshwater teleosts. Reviews in Env. Tox. 3:1-53.

SPEHAR, R.L., FIANDT, J.T., ANDERSON, R.L and DeFOE, D.L. 1980. Comparative toxicity of arsenic compounds and their accumulation in invertebrates and fish. Arch. Env. Cont. & Toxicol. 9:53-63.

AQUATIC TOXICITY

Barium is readily oxidized in water to form barite ($BaSO_4$) and witherite ($BaCO_3$), both of which are highly insoluble in water. Other barium salts are reportedly toxic (NAS 1974); however, these forms are not expected to occur in natural aquatic environments. Barium ions in general are rapidly precipitated or removed from solution by adsorption and sedimentation (NAS 1974). In most natural waters, there is sufficient sulfate or carbonate to precipitate the barium present in the water, rendering it an insoluble nontoxic compound.

Invertebrates

Bijan and Deschiens (1956 in NAS 1972) reported that barium concentrations of 10,000 to 15,000 ug/L were lethal to two species of snails.

Fish

A 28-day LC_{50} of 42,700 ug/L is reported for rainbow trout (Salmo gairdneri) (Pickering et al. 1983). A concentration of 50,000 affected the nervous system in coho salmon (Oncorhynchus kisutch) exposed for 72 hours and 158,000 ug/L killed 90 % of the test species (ORSANCO 1960 in NAS 1972). An LC50 of 200,000 ug/L is reported by Bijan and Deschiens 1956 in NAS 1972).

Amphibians

Data regarding toxicity in amphibians were not located in the literature.

Plants

Bijan and Deschiens (1956 in NAS 1972) reported that barium concentrations of 10,000 to 15,000 ug/L were lethal to an aquatic plant species.

Sediment toxicity

No sediment toxicity data were available in the literature.

Bioaccumulation

Barium can be concentrated in the goldfish (Carassius auratus) by a factor of 150 (Templeton 1958 in NAS 1972). Radioactivity studies showed accumulation of barium in organs, bones, scales, and gills of marine fish from the northeast Pacific (Moiseev and Kardashev 1964 in NAS 1972). Lowman et al. (1971 in NAS 1972) listed bioconcentration factors (BCFs) of 17,000, 900, and 8 in phytoplankton, zooplankton, and fish muscle, respectively.

Criteria

As the physical and chemical properties of barium will generally preclude the existence of the toxic soluble form under usual freshwater conditions, EPA (1986) has not established criteria for the protection of aquatic life.

TERRESTRIAL TOXICITY

Data regarding toxicity to terrestrial wildlife were not located in the literature. Toxicity in laboratory mammals is discussed below in absence of more applicable data.

Barium has not been shown to be an essential element in vertebrates (NAS 1980). Ingestion of barium has shown such effects as vomiting and diarrhea, and central nervous system effects such as violent tonic and clonic spasms followed in some cases by paralysis (NAS 1980). Animal studies indicate that barium exhibits a steep toxicity curve and that the acute lethal dose of barium varies with the compound, route of administration, species, strain, and age (EPA 1985). Rats given $BaSO_4$ intragastrically died, only after the dosage reached 25% to 50% of body weight, from stomach rupture (Boyd and Abel 1966). Clearance of barium is dependent on its solubility as well as the particle size and surface area.

Plants

Chaudhry et al. (1977) found that 2,000 ug barium per gram of soil as $Ba(NO_3)_2$ decreased yields considerably and showed an uptake of barium by the plants (2% barium in bush bean leaves and 1% in barley leaves).

Criteria

NAS (1980) recommends a maximum tolerable level of 20 ppm in the diets of domestic animals including cattle, sheep, swine, horses, rabbits, and poultry. Based on conversion factors by Lehman (1954) these levels are equal to doses of 2.5 mg/kg-body weight for birds and 0.6 mg/kg-body weight for rabbits. Puls (1988) has recommended a maximum concentration in drinking water for livestock of 1.0 mg/L.

REFERENCES

BOYD, E.M. and ABEL, M. 1966. The acute toxicity of barium sulfate administered intragastrically. Can. Med. Assoc. J. 94:849-853.

CHAUDRY, F.M., WALLACE, A., and MUELLER, R.T. 1977. Barium toxicity in plants. Commun. in Soil Sci. and Plant Analysis 8(9): 795-797.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Drinking Water Criteria Document for Barium. Criteria and Standards Division. Office of Drinking Water. PB86-117835. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Quality Criteria for Water 1986. Office of Water Regulations and Standards. May 1, 1986. EPA 440/5-86-001. Washington, D.C.

LEHMAN, A.J. 1954. Appraisal of the study of chemicals in food, drugs, and cosmetics. Association of Food and Drug Officials U.S. Vol 18:66.

NATIONAL ACADEMY OF SCIENCES (NAS). 1972. Water Quality Criteria 1972. EPA R3/73/033 Washington, D.C. March

NATIONAL ACADEMY OF SCIENCES (NAS). 1974. Nutrients and Toxic Substances in Water for Livestock and Poultry. Washington, D.C.

NATIONAL ACADEMY OF SCIENCES (NAS). 1980. Mineral Tolerance of Domestic Animals. Subcommittee on mineral toxicity in animals. National Research Council. Washington, D.C.

PICKERING, Q.H., HUNT, E.P., PHIPPS, G.L., ROUSH, T.H., SMITH, W.E., SPEHAR, D.L., STEPHENS, C.E., and TANNER, D.K. 1983. Effects of pollution on freshwater fish and amphibians. JWPCF. 55(6):840-862

PULS, R. 1988. Mineral levels in animal health. Sherpa International. Clearbrook, British Columbia, Canada. 240 pp.

BENZENE HEXACHLORIDE ISOMERS
(BHCs)

AQUATIC TOXICITY

Invertebrates

Johnson and Finley (1980) report acute toxicity values for 7 invertebrate species exposed to gamma-BHC, (lindane). 48-hour EC50 values range from 3.2 ug/L for the ostracod Cypridopsis sp. to 520 ug/L for the cladoceran Simocephalus.

Fish

Sublethal exposures of fish to lindane produced focal necrotic lesions in the liver and damage to the convoluted tubules in kidney glomeruli (Johnson and Finley (1980).

Johnson and Finley (1980) report acute toxicity values for 13 fish species. The most sensitive fish species is brown trout (Salmo trutta), with a 96-hour LC50 of 1.7 ug/L and the most tolerant species is the goldfish (Carassius auratus), with a 96-hour LC50 of 131 ug/L. LC50 values for species at APG are: 90 ug/L for carp (Cyprinus carpio), 64 ug/L for black bullhead (Ictalurus melas), 44 ug/L for channel catfish (Ictalurus punctatus), 68 ug/L for bluegill (Lepomis macrochirus), 32 ug/L for largemouth bass (Micropterus salmoides), and 68 ug/L for yellow perch (Perca flavescens).

Amphibians

No data regarding BHC toxicity to amphibians were found in the literature.

Plants

No data regarding BHC toxicity to aquatic plants were found in the literature.

Bioaccumulation

Matsumura (1977) calculated a bioconcentration factor (BCF) of 363 for mosquito larvae (Aedes aegypti) exposed for 48-hours. BCFs ranging from 32 to 143 were calculated for pink shrimp (Pinaeus duorarum) exposed to water concentrations of 0.61 to 0.13 ug/L, and BCFs ranging from 25 to 80 were calculated for grass shrimp (Paleomonetes pugio) exposed to concentrations of 1.0 to 5.5 ug/L (Menzie 1980). BCFs decreased with increasing water concentrations. BCFs ranging from 167 to 287 are reported for pinfish (Lagodon rhomboides) exposed in water concentrations of 18.4 to 31.3; and BCFs ranging from 337 to 727 are reported for sheepshead minnow (Cyprinodon variegatus); again, BCFs decreased with increasing water concentrations (Menzie 1980).

Sediment Toxicity

No sediment toxicity data were available in the literature.

Criteria

For gamma-BHC (Lindane), EPA (1986) has established an acute (24-hour average) freshwater ambient water quality criterion (AWQC) of 0.080 ug/L, and a chronic AWQC of 2.0 ug/L not to be exceeded at any time. Although data are inadequate for deriving an AWQC for other BHC isomers,

available data indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 100 ug/L and would occur at lower concentrations in species more sensitive than those tested.

For saltwater aquatic life, an AWQC of 0.16 ug/L has been established not to be exceeded at any time. Available data for a mixture of BHC isomers indicates that acute toxicity to saltwater aquatic life occurs at concentrations as low as 0.34 ug/L and would occur at lower concentrations in species more sensitive than those tested.

TERRESTRIAL TOXICITY

Mammals

Newell et al. (1987) report a no-observed-effect level (NOEL) of 0.3 mg/kg bw based on neurotoxicity in dogs exposed for 4 months.

Birds

Newell et al (1987) report a no-observed-effect level (NOEL) of 0.02 mg/kg bw based on reduced hatchability in chickens exposed to BHCs for 3 months. Hill (1975) reported 5-day LD_{50} concentrations of 425, 561, 882, and > 5,000 mg/kg diet for bobwhite (Colinus virginianus), Japanese quail (Coturnix c. japonica), ring-necked pheasant (Phasianus colchicus), and mallard (Anas platyrhynchos), respectively.

Plants

No data regarding toxicity to terrestrial plant species were found in the literature searched.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Quality Criteria for Water 1986. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-86-001.

HILL, E.F., HEATH, R.G., SPANN, J.W., and WILLIAMS, J.D. 1975. Lethal Dietary Toxicities of Environmental Pollutants to Birds. U.S. Fish and Wildlife Service. Special Scientific Report–Wildlife No. 191. Washington, D.C.

JOHNSON, W.W. and FINLEY, M.T. 1980. Handbook of Acute Toxicity of Chemicals to Fish and Aquatic Invertebrates: Summaries of Toxicity Tests Conducted at Columbia National Fisheries Research Laboratory, 1965-1978. U.S. Fish and Wildlife Service. Resource Publication 137. Washington, D.C.

MATSAMURA, F. 1977. Absorption, accumulation, and elimination of pesticides by aquatic organisms. Env. Sci. Res. 10:77-105.

MENZIE, C.M. 1980. Metabolism of Pesticides - Update III. U.S. Fish and Wildlife Service, Washington, DC. Special Scientific Report - Wildlife No. 232.

NEWELL, A.J., JOHNSON, D.W., and ALLEN, L.K. 1987. Niagara River Biota Contamination Porject: Fish Flesh Criteria for Piscivorous Wildlife. New York State Department of Environmental Conservation, Division of Fish and Wildlife, Bureau of Environmental Protection. Albany, New York.

BERYLLIUM

AQUATIC TOXICITY

The toxicity of beryllium to aquatic wildlife has been reviewed by EPA (1980). Available data suggests that beryllium concentrations as low as 130 and 5.3 µg/L induce acute and chronic toxicity to freshwater species, respectively, and more sensitive species may experience toxic effects at lower concentrations (EPA 1980). The data on freshwater fishes indicate that hardness affects toxicity; beryllium is more toxic in soft water than in hard water. The correlation between hardness and invertebrate species was unavailable; however, it was determined that a significant difference exists between acute and chronic exposures of beryllium in invertebrates (EPA 1980). Chronic toxicity data of beryllium for saltwater species was not found in the available literature.

Invertebrates

When exposed to beryllium, the cladoceran Daphnia magna had acute toxicity values from 2,500 to 7,900 µg/L (EPA 1980). A chronic study indicated that effects on reproduction were observed in Daphnia magna at 7.3 µg/L; however, no effects on reproduction were observed at 3.8 µg/L (EPA 1980). From this study a chronic toxicity value for Daphnia magna was determined to be 5.3 µg/L. This data indicate that there is a significant difference in toxicity between acute and chronic exposures.

When exposed for one hour to a beryllium concentration of 9,010 µg/L, abnormal embryonic development in the saltwater sea urchin (Paracontrotus lividus) was observed (EPA 1980).

Fish

It has been determined that when exposed in soft water, compared to hard water, fish are more sensitive to toxic effects of beryllium. Acute toxicity values for the guppy (Poecilia reticulata) ranged from 130 µg/L to 32,000 µ/L for soft and hard water, respectively (EPA 1980). Acute toxicity values for the bluegill (Lepomis macrochirus), a fish species that occurs at or near the Aberdeen Proving Ground (APG), were 1,300 µg/L in soft water and 12,000 µg/L in hard water (EPA 1980). Another species associated with APG, the channel catfish (Ictalurus punctatus), had a 96-hour LC_{50} value of greater than 5,090 µg/L in hard water; a soft water value was not reported (EPA 1980).

The saltwater mummichog (Fundulus hereoclitus), a species associated with APG, experienced reduced alkaline phosphatase activity when exposed to beryllium concentrations of 9 µg/L (EPA 1980).

No information was available concerning toxicity to fish chronically exposed to beryllium.

Amphibians

One study concerning acute toxicity of beryllium to salamanders (Ambystoma maculatum) demonstrated water hardness differences that were similar to effects seen in freshwater fish. Acute 96-hour LC_{50} values were 3,150 µg/L for soft water and 18,200 µg/L for hard water (EPA 1980).

Plants

For aquatic plants information was limited to a study performed with green alga, Chlorella vannieli, where growth was inhibited at a concentration of 100,000 µg/L (EPA 1980).

Sediment Toxicity

No information was found on the sediment toxicity of beryllium.

Bioaccumulation

A bioconcentration factor (BCF) of 19 was determined for bluegill that were exposed to beryllium for 28 days (EPA 1980). A whole body half-life of one day also was found for bluegill (EPA 1980).

Criteria

EPA (1980) has proposed for freshwater aquatic species an acute criterion of 130 µg/L and a chronic criterion of 5.3 µg/L for beryllium; however, species more sensitive than those tested may have lower values. Acute or chronic toxicity criteria for saltwater species are not available due to the limited data base (EPA 1980).

TERRESTRIAL TOXICITY

Available information is limited concerning the ingestion of beryllium in drinking water or diet by animals. A chronic toxicity value for beryllium sulfate administered to rats in their diet was determined to be 2,750 mg/kg bw/day (NRC 1977). Beryllium is of greatest concern when exposure occurs by inhalation; however, exposure of beryllium to wildlife by this route would not be expected.

Birds

No avian information was found in the available literature concerning beryllium exposure or toxicity.

Mammals

According to NRC (1977) the dietary LD_{50} for laboratory rats (genus Rattus) exposed to beryllium sulfate for 172 days (approximately 5.7 months) was 2,750 mg/kg bw/day. In EPA (1980) it was reported that the oral LD_{50} of beryllium chloride in rats was 9.7 mg/kg as Be; however, no indication of exposure duration was given. Laboratory rats fed 6.0 mg/day of beryllium in their diet for 2 years, equivalent to 1.0 mg/kg bw/day, exhibited no observable effects related to beryllium consumption (NRC 1977). Female mice fed 5.0 mg/L in drinking water during their lifetime experienced slight effects on body weight; however, this was not true for the male rats in the experiment, and no effects on the life span of either sex were observed (NRC 1977). No adverse effects were observed in four dogs that ingested 10 mg/kg bw for 19 months. After 9 months one of the dogs was sacrificed but no indication of tissue damage was found (NRC 1977).

Plants

No information relating to exposure or toxicity of beryllium and plants was found in available literature.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Beryllium-1980. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-80-024. PB81-117350.

NATIONAL RESEARCH COUNCIL (NRC). 1977. Drinking Water and Health. National Academy of Sciences. Washington, D.C.

AQUATIC TOXICITY

The aquatic toxicity of boron has been reviewed by Eisler (1990). Borax and boric acid are the most ecologically important boron compounds that enter or degrade in the environment. From available data, no-observable-effect levels (NOELs) for boron were reported to be 13,600 and 37,000 ug/L in freshwater and marine biota, respectively. Aquatic plants are highly species-specific with regard to boron toxicity and several diatoms require boron as a micronutrient. A toxicity value of 12,000 ug/L (16-day LC_{50}) indicates that the coho salmon (Oncorhynchus kisutch) is the fish species most sensitive to boron.

Invertebrates

Freshwater invertebrate acute toxicity values for boric acid ranged from 53,000 to 420,000 ug/L in the cladoceran (Daphnia magna). A Maximum Acceptable Toxicant Concentration (MATC) of 6,400 ug/L and a Lowest Observed Effect Level (LOEL) of 13,600 ug/L is reported for this species.

An acute toxicity value for boric acid in the sea urchin (Anthocidaris crassispina) was 75,000 ug/L. Normal development (NOEL) was observed when sea urchins were exposed to 37,000 ug/L (Eisler 1990). No other information concerning acute or chronic toxicity values for saltwater invertebrates was found in the available literature.

Fish

For the adult mosquito fish (Gambusia affinis), 96-hour LC_{50} values for boric acid and sodium borate were 5,600,000 mg/L (979,000 ug/L boron) and 3,600,000 ug/L, respectively (Eisler 1990). The bluegill (Lepomis macrochirus), when exposed to boron trifluoride, had an acute toxicity value of 15,000,000 ug/L. The adult rainbow trout had an acute toxicity value of 338,000 ug/L (Eisler 1990).

Toxicity information is available for the goldfish (Carassius auratus) and channel catfish (Ictalurus punctatus), fish species that occur at or near the Aberdeen Proving Ground (APG). Embryo-larvae of freshwater fish exposed to boric acid in soft water through day 4 post-hatch had 28-day LC_{50}'s which ranged from 46,000 ug/L in the goldfish to 155,000 ug/L in the channel catfish (Eisler 1990). The 28 LC_{50}'s ranged from 22,000 ug/L for the channel catfish to 79,000 ug/L for the rainbow trout (Salmo gairdneri) when embryo-larval stages were exposed to boric acid in hard water (Eisler 1990). For embryo larvae exposed to borax in soft and hard water, the acute toxicity values ranged from 27,000 to 54,000 ug/L for rainbow trout and 71 ug/L to 155,000 ug/L for channel catfish, respectively. Therefore, the species most sensitive to boric acid in soft water is the embryo-larval stage of the goldfish; in hard water, the channel catfish. The rainbow trout embryo is the most sensitive to borax in soft or hard water.

Available data indicates that the coho salmon (Oncorhychus kisutch) is the saltwater fish most sensitive in acute exposures to boron with a LC_{50} value of 12,000 ug/L. For the dab (Limanda limanda), a 96 hour LC_{50} of 74,000 ug/L is reported (Eisler 1990).

Plants

Boron has highly species-specific effects on aquatic plants and there is some evidence that pH may affect the toxicity of boron in freshwater plants. A reduction in growth and chlorophyll in the alga

(Anacystis nidulans) was observed after 72 hours at 75,000 to 100,000 ug/L as boric acid, while no adverse effects were observed at 50,000 ug/L. Exposure to concentrations of 50,000 to 100,000 ug/L produced abnormal cell division in Chlorella pyrenoidosa (Eisler 1990).

Borate is a necessary trace nutrient for some marine plants (i.e., diatoms). The diatoms Bellarochea polymorpha and Skeletonema costatum experienced positive effects, such as prolonged survival, when exposed to 10,000 ug/L and 50,000 ug/L of boric acid, respectively (Eisler 1990).

Amphibians

An acute exposure of 874,000 ug/L of boric acid has been shown to cause malformations of embryos in the toad (Bufo vulgaris). Limited data indicate that water hardness does not affect the chronic toxicity of boron to amphibians. Chronic toxicity values for amphibian embryos exposed to boric acid ranged from 130,000 to 135,000 ug/L for the leopard frog (Rana pipiens) to 123 to 145 ug/L for the Fowler's toad (Bufo fowleri) in soft and hard water, respectively (Eisler 1990).

Sediment Toxicity

No information was found on the sediment toxicity of boron.

Bioaccumulation

Bioaccumulation factors (BCFs) of 4 to 5 are reported for the green algae Chlorella pyrenoidosa exposed to boric acid for 7 days (Eisler 1990). NO BCFs were available for fish.

Criteria

Ambient water criteria have not been established for boron.

TERRESTRIAL TOXICITY

The toxicity of boron to terrestrial animals has been recently reviewed by Eisler (1990). Requirement for boron in birds and mammals is not known with certainty.

Birds

Recent studies indicate that adverse effects on growth, behavior, and brain biochemistry have resulted when mallards (Anas platyrhynchos) were fed concentrations of 300 to 400 ppm in their diet (Eisler 1990). No adverse effects were observed when mallards consumed diets contain 13 ppm boron. When adult mallards received dietary levels of 30 ppm boron for 3 weeks and the offspring continued on the same diets for an additional 3 weeks, the growth rate of the ducklings was adversely affected (Eisler 1990). Another study indicated that female mallard ducklings experienced reduced growth when fed 100 ppm in their diet (Eisler 1990). Adult chickens fed 875 ppm boron in their diet over a 6 day period stopped producing eggs (Eisler 1990).

Mammals

Boron is toxic to mammals. Acute oral doses administered to rats resulted in LD_{50}'s of 550 to 710 mg/kg body weight (bw) for boric acid and 510 to 690 mg/kg bw for borax. An acute toxic dose of 100 to 300 gm (equivalent dose of 200 to 600 mg/kg bw) in cattle was reported in Eisler (1990).

Several studies indicate that boron may retard growth: in cattle exposed to approximately 15 mg/kg/day of boron in drinking water; in dogs which consumed dietary levels of 1,750 mg/kg; in rabbits that consumed greater than 140 mg/kg/day in their diet; and in rats that ingested of 150 mg/L in drinking water or 1,060 mg/kg in their diet. However, no adverse effects were observed in cattle that consumed 2 to 2.5 g/day for 40 days; or in dogs chronically exposed to dietary levels of 174 to 524 mg/kg of borax and boric acid, respectively (Eisler 1990).

A 60 day chronic study indicated that adverse changes in testes occurred in rats fed 0.015 to 0.3 mg/kg bw/day (boric acid). Rats that ingested an equivalent dose of 0.05 mg/kg bw/day (boric acid) during a 6 month drinking water study experienced decreased spermatozoid count and reduction in spermatozoid activity.

Plants

Boron is an essential element for the growth of plants and some species of fungi, bacteria, and algae; however, excess boron may be phytotoxic. Phytotoxicity has been reported in several plant species including trees, grasses, fruits, vegetables, and grains. Generally, concentrations of boron in soil water at 5,000 to 12,000 ug/L induce toxic effects in plants. Some plant species more sensitive to boron include citrus, stone fruits, and nut trees (Eisler 1990).

Criteria

For the protection of sensitive species, certain criteria recommended by the EPA include: in livestock diet, maximum tolerable level (as borax) of 150 mg B/kg dry weight; and in livestock drinking waters, maximum allowable level of 5 mg B/L (Eisler 1990).

REFERENCES

EISLER, R. 1990. Boron Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Department of the Interior, Fish and Wildlife Service. Laurel, Maryland. Contaminant Hazard Reviews Report No. 20. Biological Report 85(1.20).

AQUATIC TOXICITY

The toxicity of cadmium to aquatic life has been reviewed by EPA (1985) and Eisler (1985). For aquatic organisms, the toxicity of cadmium generally decreases as hardness increases. Chronic toxicity values for fathead minnows (Pimephales promelas) and Daphnia magna tested over a range of hardness values found a significant correlation between hardness and toxicity (EPA 1985). Thus, ambient water quality criteria (AWQCs) have been developed to reflect the relationship of toxicity and hardness. It is important to note that the EPA AWQCs are derived assuming that water hardness serves as a surrogate indicator of two other water quality parameters that also influence the toxicity of some metals; these parameters are pH and alkalinity. These three parameters can affect toxicity in a number of ways, for example, increased calcium and magnesium (i.e., increased hardness) can compete with divalent cations (such as Cd^{2+}) for activity sites at the fish gill; and alkalinity and pH affect the complexation and speciation of metals. Generally, less toxic forms of cadmium result from increasing alkalinity and pH. Fish may develop some level of tolerance to cadmium, possibly through induction of metallothionein (Hodson 1988). White suckers (Catostomus commersoni) that were pre-exposed to sublethal levels of cadmium had subsequent LC_{50}s that were approximately 2.5 times greater than fish that were not pre-exposed to cadmium.

Invertebrates

Daphnia (including D. magna and D. pulex) are the most sensitive freshwater invertebrates to cadmium according to EPA (1985). This genus ranked third in sensitivity out of 44 genera of freshwater invertebrates and fish tested (EPA 1985). The genus mean acute toxicity value for Daphnia spp. (at a hardness of 50 mg/L as $CaCO_3$) is 26.06 ug/L (EPA 1985). Seventeen species of invertebrates (in 14 genera) have mean acute values of less than 1,000 ug/L (EPA 1985). Aquatic insects are relatively tolerant of cadmium. Mean acute values for mayflies are 322.8 ug/L (Paraleptophlebia praepedita) and 2,310 ug/L (Ephemerella grandis) (EPA 1985). The mean acute value for a caddisfly species is 3,400 ug/L. The most tolerant of 32 invertebrate species reported in EPA (1985) is a damselfly species with a mean acute value of 8,100 ug/L. Williams et al. (1985) determined that the amphipod Gammarus pulex was the most sensitive to cadmium of ten species of macroinvertebrates tested (96-hr LC_{50} = 20 ug/L) and that the caddisfly Hydropsyche angustipennis was the most tolerant (96-hr LC_{50} = 520,000 ug/L). The mean water hardness in the Williams et al. toxicity tests was 152 mg/L as $CaCO_3$.

Chronic values for cladocerans range from 0.12 to 3.932 ug/L (adjusted for a hardness of 50 mg/L) (EPA 1985). For the snail Aplexa hypnorum, the chronic value is 3.739-6.269 ug/L (adjusted for a hardness of 50 mg/L) (EPA 1985).

Marine invertebrates are relatively more tolerant of cadmium than freshwater invertebrates (Eisler 1985). For saltwater invertebrates, the species mean acute values range from 41.29 ug/L for the mysid Mysidopsis bahia to 135,000 ug/L for the oligochaete Monopylephorus cuticalatus (based on 29 genera of invertebrates) (EPA 1985). Twelve species, in nine genera, have mean acute values of less than 1,000 ug/L. Chronic values for mysids are 7.141 and 8.237 ug/L (EPA 1985).

Fish

Mean acute values for 20 species of freshwater fish range from 1.638 ug/L for brown trout (Salmo trutta) to 8,325 ug/L for goldfish (Carassius auratus) (EPA 1985). Salmonids comprise the four most

sensitive species. The species mean acute value for rainbow trout (Salmo gairdneri) (for hardness = 50 mg/L) is approximately 3.6 ug/L (EPA 1985). The acute LC_{50} for rainbow trout, based on flow-through tests was 1.75 ug/L at a hardness of 31 mg/L (Davies 1976). A 28-day EC_{50} (based on death and deformity) of 140 ug/L at a hardness of 104 mg/L has been reported for rainbow trout (Birge 1978, Birge et al. 1980 in EPA 1985). The LC_{50} for brown trout, based on static tests is 1.4 ug/L at a hardness of 39-48 mg/L (Spehar and Carlson 1984a,b in EPA 1985). Reported acute values for brook trout (Salvelinus fontinalis), are quite divergent, thus their sensitivity to cadmium is not clear based on available information. Holcombe et al. (1983 in EPA 1985) reported an LC_{50} of 5,080 ug/L for brook trout at a hardness of 47.4 mg/L (Holcombe et al. 1983 in EPA 1985). However, Carroll et al. (1979 in EPA 1985) reported an LC_{50} of less than 1.5 ug/L at a hardness of 42 mg/L. At a hardness of 330-350 mg/L the 96-hour LC_{50} was only slightly higher at 3.8-4.4 ug/L (Carroll et al. 1979 in Eisler 1985). Seven-day studies with brook trout resulted in 4.4 percent mortalities at 3.6 ug/L and 30.6 percent mortalities at 60 ug/L (Hamilton and Mehrle 1987 in Lehnertz 1989).

Brook trout are the most sensitive freshwater fish with respect to chronic toxicity of cadmium. Chronic values for brook trout of 2.045 ug/L and 1.732 ug/L were reported for hardness values of 44 and 37 mg/L, respectively (Eaton et al. 1978, Sauter et al. 1976 both in EPA 1985). Testicular damage was reported for brook trout exposed to 10 ug/L cadmium for 21 days at a hardness of 10 mg/L (Sangalang and O'Halloran 1972, 1973 in EPA 1985). A chronic toxicity value (based on early life-stage effects) of approximately 6.7 ug/L was reported for brown trout (Salmo trutta) at a hardness of 44 mg/L (as $CaCO_3$) (Eaton et al. 1978 in EPA 1985). A number of long-term exposure studies have been conducted with rainbow trout but chronic toxicity values have not been determined. Birge et al. (1981 in EPA 1985) observed reduced survival in rainbow trout exposed for 18 months to 0.2 ug/L at a hardness of 112 mg/L. Hughes et al. (1979 in EPA 1985) reported increased gill diffusion in rainbow trout exposed for 234 days to 2 ug/L cadmium at a hardness of 240 mg/L. Physiological effects were reported by Arillo et al. (1982 in EPA 1985) in rainbow trout exposed to 10 ug/L cadmium for 4 months at a hardness of 320 mg/L. Reduced growth and survival were observed by Woodworth and Pascoe (1982 in EPA 1985) in rainbow trout exposed to 100 ug/L cadmium for 47 days at a hardness of 98.6 mg/L. Physiological effects were also reported by Majewski and Giles (1984 in EPA 1985) for rainbow trout exposed for 178 days to 3.6-6.4 ug/L cadmium, at a hardness of 82 mg/L. Reduced survival of embryo-larval rainbow trout was reported at a concentration of less than 5 ug/L and a hardness of 100 mg/L after 62 days of exposure (Dave et al. 1981 in EPA 1985).

The lowest mean acute value for a saltwater fish species in EPA (1985) is 779.8 ug/L for Atlantic silverside (Menidia menidia). The other acute mean acute values reported in EPA (1985) are: winter flounder 14,297 ug/L, striped killifish 21,000 ug/L, mummichog (Fundulus heteroclitus) 50,570 ug/L, and sheepshead minnow (Cyprinodon variegatus) 50,000 ug/L. No chronic studies were reported in EPA (1985) for saltwater fish.

Species mean acute toxicity values for freshwater fish species associated with Aberdeen Proving Ground are as follows: banded killifish (Fundulus diaphanus) 98.79 ug/L, carp (Cyprinus carpio) 215.5 ug/L, American eel (Anguila rostrata) 736.4 ug/L, white sucker (Catostomus commersoni) 3,514 ug/L, bluegill (Lepomis macrochirus) 6,961 ug/L, pumpkinseed (Lepomis gibbosus) 1,347 ug/L, threespine stickleback (Gasterosteus aculeatus) 4,977 ug/L, channel catfish (Ictalurus punctatus) 5,708 ug/L, white perch (Morone americana) 7,544 ug/L, mosquitofish (Gambusia affinis) 7,685 ug/L, and goldfish (Carassius auratus) 8,325 ug/L (EPA 1985). Mean acute toxicity values for saltwater species associated with APG are: Atlantic silverside (Menidia menidia) 779.8 ug/L, winter flounder (Pseudopleuronecthes americanus) 14,297 ug/L, and mummichog (Fundulus heteroclitus) 50,570 ug/L (EPA 1985).

Amphibians

Limited toxicity information is available for amphibians. The 7-day EC_{50} (based on death and deformity) for embryo-larva of the narrow-mouthed toad (Gastrophyryne carolinensis) is 40 ug/L at a hardness of 195 mg/L (Birge 1978 in EPA 1985). Birge et al. (1978 in EPA 1985) reported an 8-day EC_{50}, based on death and deformity, of 150 ug/L (hardness = 99 mg/L) for embryo-larva of the marbled salamander (Ambystoma opacum). The African clawed frog (Xenopus laevis) appears to be less sensitive than the two amphibian species discussed above; acute (48-hour) LC_{50}s of 3,200 and 11,700 ug/L were reported for hardness values of 170 and 209 mg/L, respectively (EPA 1985). Inhibited development was reported for this species after 100 days exposure to 650 ug/L (hardness = 170 mg/L) (Canton and Slooff 1982 in EPA 1985).

Plants

Water quality criteria which are sufficient to protect freshwater animals would also be protective of freshwater plants according to EPA (1985). The lowest toxicity value reported in EPA (1985) for a freshwater plant is 2 ug/L based on a ten-fold decrease in growth rate in the diatom Asterionella formosa. A significant population decline was reported for algae (a mixture of species) at 5 ug/L (Giesy et al. 1979 in EPA 1985). Duckweed (Lemna valdiviana) showed a decrease in the number of fronds at 10 ug/L (Hutchinson and Czyrska 1972 in EPA 1985). All acute EC_{50} values reported in EPA (1985) are at least 10 times higher than the toxicity values given above. Rachlin et al. (1982 in EPA 1985) determined 96-hour EC_{50} values for a number of freshwater plant species, including: 105 ug/L for the green alga Chlorella saccarophila, 120 ug/L for the alga Anabaena flos-aquae, 310 ug/L for the diatom Navicula incerta, 480 ug/L for the diatom Nitzschia costerium, and 3,700 ug/L for the green alga Chlorella vulgaris.

Red alga are the most sensitive saltwater species reported; reduced female growth and cessation of sexual reproduction have been reported in Champla parvula at 22.8 ug/L (Steele and Thursby 1983 in EPA 1985). Toxicity values for a number of diatom species are as follows: Ditylum brightweillii 5-day EC_{50} = 60 ug/L, Thalassiosira psuedonana 96-hr EC_{50} = 160 ug/L, Skeletonema costatum 96-hr EC_{50} = 175 ug/L, and Asterionella japonica 72-hr EC_{50} = 224.8 ug/L (EPA 1985). The 8-day EC_{50} for the kelp Laminana saccharina is 860 ug/L (Markham et al. 1980 in EPA 1985).

Sediment Toxicity

Limited information is available on the toxicity of sediments contaminated with cadmium. Pavlou and Weston (1983 in Pavlou 1987) reviewed literature for proposed limits for cadmium in sediments and found values ranging from 1-6 mg/kg (dry weight). Francis et al. (1984) conducted static toxicity tests with embryo-larval goldfish (Carassius auratus), largemouth bass (Micropterus salmoides) and leopard frog (Rana pipiens) exposed to cadmium-enriched freshwater sediments (2.26 percent organic matter). Cadmium concentrations in sediments ranged from 1 to 1,000 mg/kg, and the concentrations in the overlying water ranged from 1.1 to 76.5 ug/L, respectively. Only largemouth bass exposed to the highest concentration (averaged measured concentrations were 1,079 mg/kg in sediments and 43.9 ug/L in water) resulted in statistically significant mortality (75 percent survived to 4-days post-hatch). Birge et al. (1977 in Birge et al. 1987) estimated a threshold concentration of 31 mg/kg for cadmium based on early life stage tests in rainbow trout.

Midge larvae have shown lower survival rates, reduced size, and decreased rates of emergence after exposure to sediments contaminated with a mixture of cadmium, chromium, and zinc (Wentsel et al. 1977b, 1978 in Francis et al. 1984). Midge larvae have also shown avoidance behavior to sediments

contaminated with more than 422 mg/kg cadmium and more than 8,330 mg/kg zinc (Wentsel et al. 1977a in Francis et al. 1984).

Barrick and Beller (1989) reported sediment toxicity values in terms of apparent effect thresholds (AETs). They report AET values of 5.1, 6.7, and 9.6 mg/kg for benthic invertebrates, amphipods, and oysters, respectively.

Bioaccumulation

In general, cadmium does not readily accumulate in edible fish tissues (Phillips and Russo 1978 in Heiskary and Helwig 1983). Accumulated cadmium is slowly excreted by freshwater organisms (Benoit et al. 1976 in EPA 1985, Kumada et al. 1980 in EPA 1985). Kumada et al. (1980 in EPA 1985) found faster elimination of cadmium that was consumed in the diet than that taken up from the water column. BCFs for cadmium in freshwater fish range from 3 for muscle of brook trout (based on 490 days of exposure) (Benoit et al. 1976 in EPA 1985) to 7,440 for whole body mosquitofish (Gambusia affinis) (based on 26 weeks of exposure) (Giesy et al. 1977 in Eisler 1985). BCFs for brook trout muscle were somewhat higher for shorter exposure durations. A BCF of 151 was determined for 84 days of exposure (Benoit et al. 1976 in EPA 1985), and a BCF of 22 was reported for 93 days of exposure for brook trout (Sangalang and Freeman 1979 in EPA 1985). Whole body BCFs of 33 and 540 were reported for rainbow trout based on 70 and 140 days of exposure, respectively (Kumada et al. 1973, 1980 in EPA 1985). Cadmium is preferentially accumulated in the liver, thus reducing concentrations in the muscle. Rainbow trout exposed to 10 ug/L cadmium for 3 months had BCFs of 4,900 for the liver, 1,740 for gill tissue, 740 for kidney, 160 for spleen, and 100 for heart tissues (Roberts et al. 1979 in Eisler 1985).

For the saltwater mummichog (Fundulus heteroclitus) a whole-body BCF of 15 was reported based on three weeks of exposure (Eisler et al. 1972 in Eisler 1985).
BCFs are reported in EPA (1985) for a wide variety of freshwater invertebrates. Whole-body BCFs for snails (Physa integra) and clams (Corbicula fluminea) range from 1,750 to 3,770. BCFs for insects (including mayfly, dragonfly, damselfly, stonefly, beetle, caddisfly, and midge) range from 164 to 4,190. The highest value of 4,190 is based on a 28-day study with the caddisfly Hydropsyche betteni (Spehar et al 1978 in EPA 1985). Whole-body values for crustaceans are 320-484 for cladocerans and 184 for crayfish.

A BCF of 3,500 was determined from a 66-week study with the marine crustacean Pontoporeia affinis (Sundelin 1983 in Eisler 1985).

BCFs for freshwater plants include 603 for duckweed (Lemna valdiviana) and 960 for fern (Salvinia natans) (EPA 1985). A BCF of 2,550 has been reported for the algae, Chlorella vulgaris, based on 1.4 weeks of exposure (Ferard et al. 1983 in Eisler 1985).

A BCF of 1,680 has been determined for the marine phytoplankton species Phaeodactylum tricornutum (Hardy et al. 1984).

Criteria

Ambient water quality criteria (AWQC) proposed by EPA (1985) state that freshwater organisms will not be unacceptably affected if the one-hour average concentration does not exceed the value given by $e^{(1.128[\ln(\text{hardness})] - 3.828)}$ in ug/l, and if the 4-day average concentration is not in excess of $e^{(0.7852[\ln(\text{hardness})] - 3.490)}$ in ug/L. At a hardness of 100 mg/l $CaCO_3$, the acute criterion for cadmium is

3.9 ug/L and the chronic criterion is 1.1 ug/L. For protection of saltwater organisms, EPA (1986) has established acute and chronic AWQC of 43 and 9.3 ug/L. The criteria and the values reported in the criteria document (EPA 1985) are considered equivalent to the acid soluble form. EPA considers the acid soluble measurement of metals to provide a more scientifically appropriate basis for establishing criteria for metals. However, no EPA-approved methods are available for acid-soluble measurements, and until methods are available, EPA recommends applying the criteria using the total recoverable method (EPA 1985). The concentration of the acid-soluble form is expected to be less than the value for total recoverable cadmium, thus, concentration expressed as total cadmium may somewhat overestimate the bioavailable (and potentially toxic) concentration.

TERRESTRIAL TOXICITY

The toxicity of cadmium to terrestrial organisms has been reviewed by Eisler (1985). Cadmium has not been shown to be biologically essential or beneficial (Eisler 1985).

Birds

Sublethal effects of cadmium toxicity in birds include growth retardation, anemia, and testicular damage (Hammons et al. 1978 in Eisler 1985). Increased dietary calcium, iron, selenium, zinc, or ascorbic acid, decrease the toxicity of dietary cadmium. Dietary lead or mercury increase the toxicity of cadmium (Hammons et al. 1978 in Eisler 1985).

In a study by DiGiulio and Scanlon (1984), mallard ducks (Anas platyrhynchos) were chronically exposed to cadmium-spiked food. They found significant effects on energy metabolism at 450 ppm, but not at 150 ppm. The 450 ppm group also had reduced body and liver weights. White and Finley (1978 in Eisler 1985) reported no loss in body weight in adult mallards fed 200 ppm cadmium for 90 days. Mallard ducklings showed mild to severe kidney lesions when fed 20 ppm dietary cadmium for 12 weeks (Cain et al. 1983 in Eisler 1985). After 8 weeks the birds showed reduced hemoglobin concentrations and packed cell volume, and increased serum glutamic pyruvic transaminase. Japanese quail (Coturnix japonica) given 75 ppm dietary Cd for six weeks had anemia, bone marrow hypoplasia, and heart damage (Eisler 1985). Reduced body weight occurs in poultry at 400 ppm and egg fertility and hatchability are adversely affected at 100 ppm (Puls 1988). This dietary concentration corresponds to a dosage of 12.5 mg/kg bw assuming a dietary conversion factor of 0.125 mg/kg bw per 1 ppm in the diet (Lehman 1954). Puls (1988) reported that poultry exhibited nephritis at 3 ppm dietary cadmium, but egg production was increased. At 8-60 ppm dietary cadmium reduced food consumption, reduced egg production, and eggshell-thinning have been reported in poultry (Puls 1988). Adverse effects (intestinal disease) have been reported in Japanese quail at dietary levels as low as 1 ppm. At 0.5 ppm, Japanese quail showed increased tissue concentrations of cadmium, but no other adverse effects were reported (NAS 1980). In this assessment, the 0.5 ppm level is used as a NOEL for birds and is equivalent to a dose of 0.06 mg/kg-body weight, based on the dietary conversion factor for young chickens in Lehman (1954) of 0.125 mg/kg-body weight per 1 ppm in the diet.

Mammals

Mammals have no effective mechanism for the elimination of ingested cadmium; therefore, with time, the cadmium tends to accumulate in the liver and kidney. It tends to be very persistent in the kidney and can cause renal tubular damage (NAS 1980). Toxic effects include decreased growth rates, anemia, infertility, fetus abnormalities, abortions, kidney disease, intestinal disease, and hypertension.

Cadmium is a teratogen, carcinogen, and a probable mutagen according to Eisler (1985). The lowest lethal oral doses in rats and guinea pigs range from 150 to 250 mg/kg (Eisler 1985). Doyle et al. (1972) found that 30 to 60 ppm cadmium in the diet of sheep for 191 days reduced growth and feed intake. In a 30-month study with rats elevated blood pressure occurred at the lowest level tested of 1 ppm (Perry et al. 1977 in NAS 1980).

The maximum tolerable dietary cadmium level recommended by NAS (1980) for domestic mammals and poultry is 0.5 ppm. This level is equivalent to a dose of 0.015 mg/kg for rabbits and 0.0625 mg/kg for chickens (based on dietary conversion factors in Lehman 1954). NAS (1974) recommends a safe upper limit for drinking water for livestock and poultry of 0.05 mg/L. Puls (1988) recommends that the cadmium concentration in drinking water not exceed 0.01 mg/L for livestock.

Plants

Cadmium in soil is absorbed passively by plants and translocated freely within the plant. Its phytotoxicity is related to alteration of cell membrane permeability and at least some toxic effects are linked specifically to interference of zinc-dependent uptake and translocation processes (Foy et al. 1978). Chlorosis is one of the general symptoms of cadmium toxicity in plants and appears to be caused by direct or indirect interaction of cadmium with foliar iron (Foy et al. 1978). Allaway (1968) noted that 3 mg/kg cadmium in the tissues of plants depressed growth. Traynor and Knezek (1973) reported that corn grown on cadmium-enriched soils readily absorbed and translocated the element. They also found growth reduction in corn at its maximum when 281 mg/kg cadmium was added to the soil resulting in a plant concentration of 131 mg/kg (ash weight basis). Cadmium has been found to concentrate in plants as high as ten times the soil concentration (Chaney and Hornick 1977 in Kabata-Pendias and Pendias 1984). Cadmium toxicity to soybeans was described as a 10 percent reduction in yield and discoloration of the plants at soil concentrations as low as 2.5 mg/kg. This soil concentration was also attributed to a 21 percent and 40 percent yield reductions in wheat and lettuce, respectively (Haghiri 1973). Levels of 3 to 8 mg/kg (mean of four studies = 5.25 mg/kg) were reported as phytotoxic by Kabata-Pendias and Pendias (1984).

REFERENCES

ALLAWAY, W.H. 1968. Agronomic controls over the environmental cycling of trace elements. In: Norman, A.G. (ed.). Advances in Agronomy. Academic Press, New York, New York. 20:235-274.

BARRICK, R.C. AND BELLER, H.R. 1989. Reliability of sediment quality assessment in Puget Sound. In: Oceans '89 Proceedings. Inst. of Electrical and Electronic Engineers. Piscataway, New Jersey. Publication No. 89CH2780-5.

BIRGE, W.J., BLACK, J.A., WESTERMAN, A.G., and FRANCIS, P.C. 1987. Toxicity of sediment-associated metals to freshwater organisms: biomonitoring procedures. In: Dickson, K.L., Maki, A.W., and Brungs, W.A. (eds.). Fate and Effects of Sediment-Bound Chemicals in Aquatic Systems. Proceedings of the Sixth Pellston Workshop, Florissant, Colorado, August 12-17, 1984. SETAC Special Publication Series, Pergamon Press. New York, New York. Pp. 199-218.

DAVIES, P.H. 1976. The need to establish heavy metal standards on the basis of dissolved metals. In: Andrew, R.W., Hodson, P.V. and Konasewich, D.E. (eds.). Toxicity to Biota of Metal Forms in Natural Water. International Joint Commission, Windsor, Ontario. 5:93-126.

DIGIULIO, R.T. and SCANLON, R.F. 1984. Sublethal effects of cadmium ingestion on mallard ducks. Arch. Environ. Contam. Toxicol. 13:765.

DOYLE, J.J., PFANDER, W.H., GREBING, S.E. and PIERCE, J.O., II. 1972. Effects of dietary cadmium on growth and tissue levels in sheep. In: Hemphill, D.D. (ed.). Trace Substances in Environmental Health - VI. Univ. of Missouri Ann. Conf., 6th Proc. Pp. 181-186.

EISLER, R. 1985. Cadmium Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Department of the Interior, Fish and Wildlife Service. Patuxent Wildlife Research Center. Laurel, Maryland. Contaminant Hazard Reviews Report No. 2.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Ambient Water Quality Criteria for Cadmium-1984. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-85-032. PB85-227031.

FOY, C.D., CHANEY, R.L. and WHITE, M.C. 1978. The physiology of metal toxicity in plants. Ann. Rev. Physiol. 29:511-566.

FRANCIS, P.C., BIRGE, W.J., and BLACK, J.A. 1984. Effects of cadmium-enriched sediment on fish and amphibian embryo-larval stages. Ecotoxicology and Environmental Safety. 8:378-387.

HAGHIRI, F. 1973. Cadmium uptake by plants. J. Environ. Qual. 2(1):93-96.

HARDY, J.T., SULLIVAN, M.F., CRECELIUS, E.A. and APTS, C.W. 1984. Transfer of cadmium in a phytoplankton-oyster-mouse food chain. Archives of Env. Contam. & Tox. 13:419-425.

HEISKARY, S.A., and HELWIG, D.D. 1983. Acid Rain Intensive Study Lakes Program, Status Report for the 1981 Study Lakes. Minnesota Pollution Control Agency, Division of Water Quality, Monitoring and Analysis Section.

HODSON, P.V. 1988. The effect of metal metabolism on uptake, disposition and toxicity in fish. Aquatic Toxicology. 11:3-18.

KABATA-PENDIAS, A. and PENDIAS, H. 1984. Trace Elements in Soil and Plants. CRC Press. Boca Raton, Florida.

LEHMAN, A.J. 1954. Appraisal of the study of chemicals in food, drugs, and cosmetics. U.S. Association of Food and Drug Officials. 18:66.

LEHNERTZ, C. 1989. Clear Creek Basin 1989: The Effects of Mining on Water Quality and the Aquatic Ecosystem-Draft. Prepared for the Colorado Division of Wildlife. Denver, Colorado.

NATIONAL ACADEMY OF SCIENCES (NAS). 1974. Nutrients and toxic substances in water for livestock and poultry. National Research Council. Washington, D.C.

NATIONAL ACADEMY OF SCIENCES (NAS). 1980. Mineral Tolerance of Domestic Animals. National Research Council, Subcommittee on Mineral Toxicity in Animals. Washington, D.C.

PAVLOU, S.P. 1987. The use of the equilibrium partitioning approach in determining safe levels of contaminants in marine sediments. In: Dickson, K.L., Maki, A.W., and Brungs, W.A. (eds.). Fate and Effects of Sediment-Bound Chemicals in Aquatic Systems. Proceedings of the Sixth Pellston Workshop, Florissant, Colorado, August 12-17, 1984. SETAC Special Publication Series, Pergamon Press. New York, New York. Pp. 388-412.

PULS, R. 1988. Mineral Levels in Animal Health. Sherpa International. Clearbrook, British Columbia.

TRAYNOR, M.F. and KNEZEK, B.D. 1973. Effects of nickel and cadmium contaminated soils on nutrient composition of corn plants. In: Hemphill, D.D. (ed.). Trace Substances in Environmental Health - VII. Univ. of Missouri Ann. Conf., 7th Proc. Pp. 82-87.

WILLIAMS, K.A., GREEN, D.W.J. and PASCOE, D. 1985. Studies on the acute toxicity of pollutants to freshwater macroinvertebrates. 1. Cadmium. Arch. Hydrobiol. 102:461-471.

CHROMIUM

The toxicity of chromium (Cr) to fish and wildlife has been reviewed by Eisler (1986). Both Cr(III) and Cr(VI) occur in natural waters, and they may be converted to one another under naturally occurring conditions (EPA 1985). The chemical and toxicological properties of these two forms are different (EPA 1985).

AQUATIC TOXICITY

The toxicity of chromium (VI) to aquatic species appears to increase as pH and/or hardness decreases (USEPA 1985).

Invertebrates

Cladocerans are the most acutely sensitive species tested with Chromium (VI). Genus mean acute values (hardness = 50 mg/L) are 28.94 ug/L for Daphnia sp., 36.35 ug/L for Simocephalus sp., and 45.10 ug/L for Ceriodaphnia reticulata (EPA 1985). Amphipods are also quite sensitive to chromium (VI); the acute mean value for Gammarus pseudolimnaeus is 67.1 ug/L. The lowest mean acute value reported by EPA (1985) for an aquatic insect for Cr (VI) is 57,300 ug/L for the midge Tanytarsus dissimilis. The lowest values for damselfly (Enallagma aspersum) and stonefly (Neophasganophora capitata) are 140,000 ug/L and 1,870,000 ug/L, respectively (EPA 1985).

Chromium (III) is less acutely toxic than Chromium (VI). The most acutely sensitive species to Cr (III) is the mayfly (Ephemerella subvaria) with a mean acute value of 2,221 ug/L. Amphipods (Gammarus sp.) were the second most sensitive with a mean acute value of 3,200 ug/L (EPA 1985). The lowest reported toxicity values for damselflies and caddisflies are 43,100 ug/L and 50,000 ug/L, respectively. No acute toxicity values for Cr(III) were reported for Ceriodaphnia sp.; the lowest value for a cladoceran is 16,010 ug/L (for Daphnia magna).

Chronic toxicity values for invertebrates are limited. The chronic value for Ceriodaphnia reticulata for Cr(VI) is 40 ug/L at a hardness of 45 mg/L. The chronic value for Cr(III) at a hardness of 52 mg/L is 66.11 ug/L (EPA 1985). No other chronic values were reported for Ceriodaphnia, amphipods, or aquatic insects.

Fish

In general, fish are not as sensitive to chromium as invertebrates. The lowest acute mean value for a fish species for Cr(VI) is 30,000 ug/L (for the guppy, Poecilia reticulata) (EPA 1985). The mean acute value for the fathead minnow is 41,050 ug/L (Cr[VI]). For trout species the mean acute values are 59,000 ug/L for brook trout and 69,000 for rainbow trout (for Cr[VI]). The guppy is also the most sensitive fish species to Cr(III), with a mean acute value of 7,053 ug/L. For fathead minnows the Cr(III) mean acute value is 10,320 ug/L. The mean acute values for rainbow trout is 9,669 ug/L. No other mean acute toxicity values for trout were reported by EPA (1985).

Chronic toxicity values of 264.6 ug/L have been reported for Cr(VI) (Benoit 1976 in EPA 1985) for brook (life-cycle) and rainbow trout (early life-stage) at a hardness of 45 mg/L. Mortality was the most sensitive indicator for both of these species. At a hardness of 34 mg/L a chronic value of 73.18 ug/L (Cr[VI]) has been reported for rainbow trout (ELS) (Sauter et al. 1976 in EPA 1985). This value was based on a reduction in growth at 60 days post-hatch. For Chromium (III), the chronic value (based on survival) for rainbow trout is 68.63 ug/L at a hardness of 26 mg/L (EPA 1985). Brown trout showed

immune system effects after 38 weeks of exposure to 1,010 ug/L Cr(VI) at a hardness of 207 mg/L (O'Neill 1981 in EPA 1985).

Amphibians

Chromium is apparently quite toxic to some amphibians. The 7-day EC50, based on death and deformity, for embryo-larval narrow-mouthed toads (Gastrophryne carolinensis), is 30 ug/L Cr[VI] at a hardness of 195 mg/L (Birge 1978 in EPA 1985). Embryo-larval marbled salamanders (Ambystoma opacum) are not as sensitive; the 8-day EC50 at a hardness of 99 mg/L is 2,130 ug/L Cr[VI] (Birge et al. 1978 in EPA 1985).

Plants

Toxicity values of 2 to 7,800 ug/L (Cr[VI]) have been reported for freshwater aquatic plants (EPA 1985). For the diatom, Navicula seminulum, concentrations of 245 ug/L to 335 ug/L (Cr[VI]) caused 50 percent reduction in growth in soft and hard water, respectively (EPA 1985). Aquatic phytotoxicity information for Cr(III) is quite limited, but based on available information it is apparently less toxic than Cr(VI) (EPA 1985).

Sediment Toxicity

Barrick and Beller (1989) report sediment values in terms of apparent effect thresholds (AETs). They report AETs of 260 and 270 mg/kg (dry weight) for benthic invertebrates and amphipods, respectively.

Bioaccumulation

Bioaccumulation has been found to vary among species. Tissue concentrations are normally highest at lower trophic levels and lowest with the top predators, indicating that biomagnification does not occur. A BCF of 3.4 was found by Calamari et al. (1982) for hexavalent chromium in rainbow trout. All three rainbow trout BCFs reported for CR(VI) by EPA (1985) were less than 3 (for both whole body and muscle). No information is available for Cr(III).

Criteria

Water quality criteria have been developed to reflect the effect of hardness on toxicity, as well as the differences in toxicity of the two oxidation states. The Federal ambient water quality criteria for chromium (VI) are 11 ug/L for a 4-day average concentration, and 16 ug/L for the 1-hour average concentration (USEPA 1986). According to EPA (1985), these concentrations should be treated as the acid-soluble form. For chromium (III) the 4-day average concentration is defined by the value given by $e^{(0.8190[\ln(\text{hardness})]+1.561)}$ and the 1-hour average is defined by $e^{(0.8190[\ln(\text{hardness})]+3.688)}$. At a hardness of 100 mg/L the chronic criteria is 210 ug/L and the acute criteria is 1,700 ug/L.

TERRESTRIAL TOXICITY

Birds

Data on hexavalent chromium effects on birds are few. Male domestic chickens fed a diet containing up to 100 ppm hexavalent chromium for 32 days showed no adverse effects in survival, growth, or food utilization efficiency (Rosomer et al. 1961 in Eisler 1986).

Mammals

It appears that the primary source of uptake of chromium by small mammals is through ingestion of contaminated soil while grooming (Taylor 1980 in Eisler 1986). Short biologic half-life and fractional assimilation indicates a reduced toxic effect, especially under chronic exposures. Feeding studies done on cotton rats demonstrated low assimilation (0.8%) and rapid loss (99% in one day) of hexavalent chromium. The LD_{50} is 19.8 mg/kg chromium (VI) and 600-2,600 mg/kg for chromium III (USEPA 1987). Hexavalent chromium was fatal to dogs in three months when fed 100 ppm in their diet, but was not lethal at 11.2 ppm over four years when administered in their water (Steven et al. 1976 in Eisler 1986). They also found the toxic threshold in rats to be 1,000 ppm chromium (VI) in the diet and 100% survival when exposed to 134 ppm in their drinking water for three months.

Plants

The chromium content of plants is controlled mainly by the amount of soluble chromium in the soils. Chromium (VI) is the most soluble and available to plants, but it is also the most unstable form under normal soil conditions. Usually chromium distribution in plants results in the highest concentrations in the roots, then the leaves and stems and the lowest concentrations in the grain (Kabata-Pendias and Pendias 1984). Typical symptoms of chromium phytotoxicity are wilting of plant tops, root injury, chlorosis in young leaves, brownish-red leaves and chlorotic bands on cereals (Kabata-Pendias and Pendias 1984). Kabata-Pendias and Pendias (1984) reported levels of 75 to 100 mg/kg (dry weight) as phytotoxic; the mean value of four studies was approximately 94 mg/kg.

Criteria

NAS (1980) recommends that the maximum level of chromium in the diet is 1,000 ppm for livestock and poultry. Concentrations in drinking water should not exceed 1.0 mg/L for livestock and poultry according to recommendations by NAS (1974). Puls (1988) recommends that the maximum concentration in drinking water should be less than 0.05 mg/L for livestock and poultry.

REFERENCES

BARRICK, R.C. and BELLER, H.R. 1989. Reliability of sediment quality assessment in Puget Sound. In: Oceans '89 Proceedings. Inst. of Electrical and Electronic ENgineers, Publication No. 89CH2780-5, Piscataway, NJ

CALAMARI, D., GAGGINS, G.F. and PACCHETTI, G. 1982. Toxicokinetics of low levels of Cd, Cr, Ni, and their mixtures in long-term treatment on Salmo gairdneri. Chemosphere 11:59-70.

EISLER, R. 1986. Chromium hazards to fish, wildlife, and invertebrates: a synoptic review. U.S. Department of the Interior, Fish and Wildlife Service. Biological Report 85(1.6). January, 1986. Contaminant Hazard Reviews Report No. 6. 60 pp.

KABATA-PENDIAS, A. and H. PENDIAS. 1984. Trace Elements in Soil and Plants. CRC Press. Boca Raton, Florida.

NATIONAL ACADEMY OF SCIENCES (NAS). 1980. Mineral Tolerance of Domestic Animals. Subcommittee on mineral toxicity in animals. National Research Council. Washington, D.C.

NATIONAL ACADEMY OF SCIENCES (NAS). 1974. Nutrients and toxic substances in water for livestock and poultry. National Research Council. Washington, D.C.

PULS, R. 1988. Mineral levels in animal health. Sherpa International. Clearbrook, British Columbia, Canada. 240 pp.

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1985. Ambient Water Quality Criteria for Chromium-1984. Office of Water Regulations and Standards. EPA 440/5-84-029. PB85-227478. January, 1985. Washington, D.C.

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1986. Quality Criteria for Water 1986. Office of Water Regulations and Standards, Washington D.C. May 1, 1986. EPA 440/5-86-001

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1987. Drinking Water Health Advisories. Final. March 31, 1987. Washington, D.C.

COPPER

AQUATIC TOXICITY

Copper is a minor nutrient for aquatic life at low levels (1 to 10 ppb), but at concentrations only slightly higher is toxic to aquatic life. Copper toxicity has been shown to be related primarily to the activity of the cupric (Cu^{2+}) ion, and possibly to some of the hydroxy complexes (EPA 1985).

In fresh water, toxicity decreases with increasing water hardness. Data suggest that acclimation increases tolerance of some aquatic organisms to copper (EPA 1985). In fish, acute toxicity may result from disrupted osmoregulatory function. Continued ingestion of copper in excess of nutritional requirements leads to accumulation, especially in the liver (Rand and Petrocelli 1985). Acute and chronic exposures in invertebrates can result in decreased survival and reproduction. In algae, copper can inhibit growth, photosynthesis, and sexual reproduction. Information on copper toxicity in higher plants is not available.

Toxicity of copper to fresh- and saltwater invertebrates, vertebrates, and plants is summarized below.

Invertebrates

Sensitivities of fresh and saltwater invertebrate species appear to be similar. In acute assays summarized by EPA (1985), the most sensitive freshwater species is Daphnia magna, with an EC_{50} of 6.5 ug/L, and the most tolerant species is the stonefly Acroneuria lycorias, with an EC_{50} of 8,300 ug/L. Saltwater invertebrate EC_{50} values range from 5.3 ug/L for the Pacific oyster (embryo) Crassostrea gigas to 8,000 ug/L for the common rangia Rangia cuneata.

Nelson et al. (1988) reported 96-hour LC_{50} values of 29, 51, and 122 ug/L for juvenile bay scallops (Argopeten irradians), surf clams (Spisula solidissima), and blue mussels (Mytilus edulis), respectively. Devi and Rao (1989) studied the effects of heavy metals on two species of fiddler crabs (Uca annulipes and U. triangularis) obtained from polluted and unpolluted sources. They reported 96-hour LC_{50} values of 12.82 and 14.81 ug/L for U. annulipes and U. triangularis from polluted water, and 96-hour LC_{50} values of 9.42 and 8.28 ug/L for U. annulipes and U. triangularis, respectively, obtained from unpolluted water. This indicates that organisms from polluted waters may have a higher tolerance to toxic effects of heavy metals including copper.

Shaner and Knight (1985) reported Daphnia magna 24-hour LC_{50}s of 1,332 and 1,578 ppm copper in sediment at alkalinities of 600 and 1000 mg/liter, respectively. They found that increasing alkalinity resulted in decreased mortality at a given sediment copper concentration.

Chronic values for freshwater invertebrates range from 6.066 ug/L for the amphipod Gammarus pseudolimnaeus to 29.33 ug/L for D. magna (EPA 1985). It is interesting to note that D. magna was the most sensitive freshwater species in the acute tests but the least sensitive in chronic tests. In a life cycle test, chronic toxicity to the saltwater mysid shrip Mysidopsis bahia is reported at 54.09 ug/L (EPA 1985).

Fish

Acute toxicity is noted in freshwater fish species at concentrations ranging from 10 ug/L for the chinook salmon Oncorhyncus tshawytscha to 10,200 ug/L for the bluegill Lepomis macrochirus, a species found at APG (EPA 1985). Acute toxicity in saltwater fish is reported at concentrations

ranging from 66.6 ug/L for the Atlantic silverside (Menidia menidia) to 510 ug/L for the Florida pompanao Trachinotus carolinus (EPA 1985).

Palawski et al. (1985) investigated the sensitivity of young striped bass (Morone saxatilis, an APG species) to copper in fresh and saline water. They report 96-hour LC_{50} values of 7, 10, and 21 ug/L in hard water, 1% saline water, and 5% saline water. Species mean acute values for 14 other species occurring at APG range from 69.81 ug/L for the brown bullhead Ictalurus nebulosis to 5,860 ug/L for white perch Morone americana (EPA 1985).

Pedder and Maly (1985) report that high copper concentrations in water attract some fish species. They calculated a 96-hour EC_{50} for rainbow trout Salmo gairdneri of 500 to 750 ug/L copper based on avoidance. This EC_{50} value range is higher than the species mean acute value for rainbow trout of 42.50 ug/L copper calculated by EPA (1985), indicating that fish may be attracted to areas where copper concentrations are acutely toxic to them.

Henry and Atchison (1986) studied the effects of copper on the behavior of social groups of bluegill sunfish (Lepomis macrochirus). They noted that aggressiveness of the dominant fish increased following exposure to copper concentrations as low as 34 ug/L.

Chronic toxicity values for freshwater species range from 3.873 ug/L for the brook trout Salvelinus fontinalis to 60.36 ug/L for northern pike Esox lucius (EPA 1985). No chronic toxicity for saltwater vertebrate species were available in the literature.

Amphibians

EPA (1985) reported the toxic effects of copper on embryo-larval stages of 3 toad, 2 frog, and 1 salamander species. Seven-minute EC_{50} values based on death and deformity were 40 and 26,960 ug/L for the southern gray tree frog (Hyla sp.) and Fowler's toad (Bufo woodhouse fowleri), respectively. An EC_{50} of 100 ug/L based on avoidance was reported for the American toad (Bufo americanus) following 80-minutes of exposure. EC_{50} values of 40, 50, and 770 ug/L were reported for the narrow-mouthed toad (Gastrophryne sp.), the leopard frog (Rana sp.), and the marbled salamander (Ambystoma opacum), respectively, following 7 to 8 days of exposure.

Plants

Steeman-Nielsen and Wium-Anderson (1970 in EPA 1985) reported an EC_{50} for growth reduction of 1 ug/L for the green algae Chlorella sp.

Copper acts as a micronutrient for plant growth at very low concentrations, but inhibits plant growth, photosynthesis, and nitrogen fixation at higher concentrations (Jain et al. 1989, Azzez and Banerjee 1986). Jain et al (1989) state that aquatic plants may be used to remove heavy metals from polluted water. They conducted a study of uptake of copper (1,000, 2,000, 4,000, and 8,000 ug/L) in solution by two aquatic plants, duckweed (Lemna minor) and water velvet (Azolla pinnata). They calculated a bioconcentration factor for copper of 51.20 for duckweed; however, when iron was added to the test solution at equal concentrations, the bioconcentration factor was 26.53. They conclude that the presence of iron inhibits biouptake of copper by aquatic plants.

Bioaccumulation

Wright et al. (1985) reported copper concentrations ranging from 11.8 to 103.2 ug/g wet weight in oysters collected from the Central Chesapeake Bay Region in June 1979, with a mean over all sites of 46.68 ug/g. Eastern oysters (Crassostrea virginica) bioaccumulated copper up to 28,200 times in a 14-day study by Shuster and Pringle (1969 in EPA 1985), and became bluish-green, without significant mortality.

Kay and Haller (1986) researched the bioaccumulation of copper by waterhyacinth weevils (Neochetina eichhorniae) feeding on water hyacinth. Water hyacinth grown in solutions containing 0, 1,000, and 2,500 ug/L copper, had copper concentrations in the leaves of 15.35, 21.62, and 44.77 mg/kg, respectively. Weevils feeding on these leaves contained copper at concentrations of 30.42, 38.37, and 32.77 mg/kg, and experienced no adverse effects.

A muscle bioconcentration factor of 1.0 was reported for bluegill exposed for 660 days (Benoit 1975 in EPA 1985).

Sediment Toxicity

Barrick and Beller (1989) reported sediment toxicity values in terms of apparent effect thresholds (AETs). They report AETs of 390, 530, and 1,300 mg/kg (dry weight) for oyster, benthic invertebrates, and amphipods, respectively.

Criteria

EPA (1985) has developed ambient water quality criteria for the protection of fresh- and saltwater aquatic life. Because copper toxicity decreases with increasing water hardness in freshwater, these criteria depend upon water hardness in a particular water body. EPA (1985) recommended that the 4-day average concentration of copper (in ug/L) should not exceed the value given by $e^{(0.8545[\ln(\text{hardness})]-1.465)}$, and the 1-hour average concentration should not exceed the value given by $e^{(0.942[\ln(\text{hardness})]-1.464)}$. At a water hardness of 100 mg/liter as $CaCO_3$, the corresponding values are 12 and 18 ug/L respectively.

EPA (1985) developed ambient water quality criteria for the protection of saltwater aquatic life from acutely toxic concentrations of copper. The recommended 1-hour criterion is 2.9 ug/L, not to be exceeded more than once every three years. No criterion was developed for chronic exposures.

TERRESTRIAL TOXICITY

Mammals

Data on copper toxicity in wild mammalian species could not be located in the available literature. However, an acute oral LD_{50} of 60 mg Cu/kg-body weight has been reported for horses (NAS 1980). The LD_{50} for $CuSO_4$ in rats is approximately 300 mg/kg bw, or 120 mg Cu/kg bw (NAS 1980). Copper tolerance in mammals varies widely partly due to differences in sulfur metabolism and in dietary levels of other trace elements such as iron, molybdenum, selenium, sulfur, and zinc (NAS 1980). NAS (1980) suggested the following maximum tolerable levels for dietary copper: 25 ppm for sheep, 100 ppm for cattle, rabbits 200 ppm, 250 ppm for swine, and 800 ppm for horses.

Birds

Data are available on the toxicity of copper in wild birds. Canada geese (Branta canadensis) ingesting pond water containing 100 ppm copper as copper sulfate developed acute copper toxicosis (NAS 1980). Copper toxicity is associated with hemolytic crisis, hepatic necrosis, and death. Maximum tolerable dietary levels for turkey and chickens are 300 ppm (NAS 1980).

Plants

Kabata-Pendias and Pendias (1984) reported soil copper levels as low as 60 mg/kg as phytotoxic to some plants. Other data regarding toxicity of copper to terrestrial plants were not located in the literature.

REFERENCES

AZEEZ, P.A. and BANERJEE, D.K. 1986. Effect of copper and cadmium on carbon assimilation and uptake of metals by algae. Toxicol. and Environ. Chem. 12:77-86.

BARRICK, R.C. AND BELLER, H.R. 1989. Reliability of sediment quality assessment in Puget Sound. In: Oceans '89 Proceedings. Inst. of Electrical and Electronic Engineers. Piscataway, New Jersey. Publication No. 89CH2780-5.

DEVI, V.U. and RAO, Y.P. 1989. Heavy metal toxicity to fiddler crabs, Uca annulipes and Uca triangularis: respiration on exposure to copper, mercury, cadmium, and zinc. Bull. Environ. Contam. Toxicol. 43:165-172

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Ambient Water Quality Criteria for Copper-1984. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-84-031.

HENRY, M.G. and ATCHISON, G.J. 1986. Behavioral changes in social groups of bluegills exposed to copper. Trans. Am. Fish. Soc. 115:590-595.

JAIN, S.K., VASUDEVAN, P., and JHA, N.K. 1989. Removal of some heavy metals from polluted water by aquatic plants; studies on duckweed and water velvet. Biol. Wastes. 28:115-126.

KABATA-PENDIAS, A. and PENDIAS, H. 1984. Trace Elements in Soil and Plants. CRC Press. Boca Raton, Florida.

KAY, S.H. AND HALLER, W.T. 1986. Heavy metal bioaccumulation and effect on water hyacinth weevils, Nexchetina eichhorniae, feeding on water hyacinth, Eichhornia crassipes. Bull Environ. Contam. Toxicol. 37:239-245.

NATIONAL ACADEMY OF SCIENCES (NAS). 1980. Mineral Tolerance of Domestic Animals. National Research Council, Subcommittee on Mineral Toxicity in Animals. Washington, D.C.

NELSON, D.A., MILLER, J.E, and CALABRESE, A. 1988. Effect of heavy metals on bay scallops, surf clams and blue mussels in acute and long-term exposures. Arch. Environ. Contam. Toxicol. 17:595-600.

PALAWSKI, D., HUNN, J.B., and DWYER, F.J. 1985. Sensitivity of young striped bass to organic and inorganic contaminants in fresh and saline waters. Trans. Am. Fish. Soc. 114:748-753.

PEDDER, S.C.J. and MALY, E.J. 1985. The effect of lethal copper solutions on the behavior of rainbow trout, Salmo gairdneri. Arch. Environ. Contam. Toxicol. 14:501-507.

RAND, G.M. and PETROCELLI, S.R. 1985. Fundamentals of Aquatic Toxicology. Hemisphere Publishing Corp. Washington, D.C.

SHANER, S.W. and KNIGHT, A.W. 1985. The role of alkalinity in the mortality of Daphnia magna in bioassays of sediment-bound copper. Comp. Biochem. Physiol. 82C(2):273-277.

WRIGHT, D.A., MIHURSKY, J.A., and PHELPS, H.L. 1985. Trace metals in Chesapeake Bay oysters: intra-sample variability and its implications for biomonitoring. Marine Environ. Res. 16:181-197.

AQUATIC TOXICITY

The toxicity of cyanide to aquatic organisms is similar to terrestrial animal species and is mainly due to the HCN species. Following acute exposures to high concentrations, cyanides exert their toxic effects by inhibiting cellular respiration. Chronic exposures can decrease reproduction, impair swimming ability, increase respiration, disrupt osmo- and iono-regulation, and induce histopathological effects in fish (EPA 1985). Toxicity of cyanide in aquatic organisms has been shown to increase with a decrease in temperature and dissolved oxygen below saturation (EPA 1985). Studies have demonstrated that embryos, sac fry, and warm water species of fish tend to be more resistent to the acute toxicity of cyanide (EPA 1985).

Invertebrates

In general, freshwater invertebrates are more resistent than fish to the acute toxicity of cyanide (EPA 1985). Cladocerans and amphipods (Gammarus pseudolimnaeus) exhibit comparable sensitivity to cyanide as fish. The genus Daphnia was the most sensitive of 7 invertebrate species reported and ranked 7th among the 15 freshwater organisms tested; the mean acute value is 123.6 ug/L. The genus Gammarus was the tenth most sensitive of the species tested; the mean acute value is 167 ug/L. The midge (Tanytarsus dissimilis) and the isopod (Asellus communis) were the most tolerant species tested, with acute values of 2,490 ug/L and 2,326 ug/L respectively (EPA 1985). In chronic studies, the lowest Maximum Acceptable Tolerant Concentration (MATC) reported for a freshwater invertebrate was 18.3 ug/L for Gammarus pseudolimneaus (EPA 1985).

Species mean acute values for 7 species of marine invertebrates range from 4.9 ug/L for embryos of the rock crab (Cancer irroratus) to over 10,000 ug/L for larvae of the Atlantic slippershell (Crepidula fornicata) (EPA 1985). These two species were the most sensitive and resistent species, respectively, to cyanide of the 9 marine species tested. The rock crab was six times more sensitive to cyanide than the next most sensitive species tested, the copepod (Acartia tonsa) (EPA 1985). The mean species acute value for this species is 30 ug/L. The only chronic value reported in EPA (1987) for a saltwater invertebrate is a value of 69.7 ug/L for the mysid (Mysidopsis bahia).

Fish

Species mean acute values for cyanide for 17 species of freshwater fish range from 44.7 ug/L to 318 ug/L (EPA 1985). Trout are among the most sensitive freshwater fish species tested with cyanide and the most sensitive fish are rainbow trout (Salmo gairdneri); a 96-hour LC_{50} of 27 ug/L was reported in juveniles exposed to cyanide under flow-through conditions (Kovacs and Leduc 1982 in EPA 1985). Acute LC_{50}s of 52 ug/L and 90 ug/L were reported for juvenile brook trout (Salvelinus fontinalis) and juvenile Atlantic salmon (Salmo salar), respectively, tested under flow-through conditions (EPA 1985). An acute LC_{50} of 61.5 ug/L for the trout Oncorhynchcus mykiss was reported by Meyer (1981 in EPA 1985). Leduc (1978 in EPA 1985) reported abnormal embryo and larval development in the Atlantic salmon following 58 days of exposure to cyanide at 9.6 ug/L. A concentration of 10 ug/L reduced fecundity in rainbow trout (Pickering et al. 1983). The chronic values for cyanide of 7.9 ug/L, 13.6 ug/L, and 16.4 ug/L are reported for the brook trout Salvelinus fontinalis), the bluegill (Lepomis macrochirus), and the fathead minnow (Pimephales promelas), respectively (EPA 1985).

Toxicity data for cyanide in saltwater fish is limited. Mean acute values for the three species reported by EPA (1985) are: Atlantic silverside (Menidia menidia), 59 ug/L; sheepshead minnow (Cyprinodon

variegatus), 300 ug/L; and winter flounder (Pseudopleuronectes americanus), 372 ug/L. Cheng and Ruby (1981 in EPA 1985) reported reduced fecundity and hatching in the flagfish (Jordanella floridae) when exposed to 63 ug/L cyanide for 10 days. A chronic value of 36 ug/L was determined from an early life-stage study with the sheepshead minnow (EPA 1985).

Toxicity information is available for a several of the fish species that occur at or near the Aberdeen Proving Grounds. The species mean acute values for freshwater fish are: bluegill (Lepomis macrochirus), 99.3 ug/L; black crappie (Pomoxis nigromaculatus), 102 ug/L; largemouth bass (Micropterus salmoides), 102 ug/L; and goldfish (Carassius auratus), 318 ug/L (EPA 1985). A chronic values of 13.6 ug/L was determined from an early life-stage study for the bluegill (Lepomis macrochirus) (EPA 1985). Species mean acute values were reported for two saltwater fish: the Atlantic silverside (Menidia menidia), 59 ug/L; and the winter flounder (Pseudopleuronectes americanus), 372 ug/L.

Amphibians

No information was found on the toxicity of cyanide to amphibians.

Plants

Both freshwater and saltwater plant species demonstrate a wide range of susceptibilities to cyanide (EPA 1985). The saltwater red macroalga (Champia parvula) is extremely sensitive to cyanide toxicity with growth and reproductive effects evident at 11 to 25 ug/L (EPA 1985). EPA concluded that adverse effects of cyanide on plants are unlikely at concentrations which have not been shown to cause chronic effects in most freshwater and marine animal species (EPA 1985). Therefore, plants will be protected if the most sensitive animals are protected.

Sediment Toxicity

No information was found on the toxicity of cyanide-contaminated sediments to aquatic organisms.

Bioaccumulation

While long-term survival and growth of various freshwater fish species are known to be substantially reduced under conditions of 20 to 50 ug/L free cyanide, no accumulation or biomagnification in the food chain has been demonstrated (Towill et al. 1978 in EPA 1985). Also, field studies have demonstrated that despite cyanide-induced mortality among invertebrate fauna, populations of these organisms can rapidly recover in lakes treated with cyanide (Leduc et al. 1973 in EPA 1985).

Criteria

EPA (1985) has established for freshwater aquatic organisms and their uses a 4-day average concentration criterion (chronic criterion) for cyanide of 5.2 ug/L (not to be exceeded more than once every three years on the average) and a 1-hour average concentration criterion (acute criterion) of 22 ug/L for cyanide. For marine waters, the criterion is 1 ug/L both as a 4-day and 1-hour average concentration. EPA regards the measurement of free cyanide to provide the more scientifically appropriate measurement for aquatic life criteria; however, because there is no EPA-approved method for this measurement, EPA recommends that measurement of total cyanide be applied (EPA 1985).

TERRESTRIAL TOXICITY

The mechanism of toxicity of cyanide, inhibition of cellular respiration, appears to be similar regardless of phylogentic order. The acute toxicity and, to a lesser extent, the chronic toxicity of cyanide have been studied primarily in laboratory animals (NAS 1980). Little information is available concerning the adverse effects of cyanide in terrestrial wildlife.

Birds

Acute oral toxicity values ranged from 4 mg/kg to 21 mg/kg in the American kestrel and domestic chicken, respectively (Wiemeyer et al. 1986). More sensitive to the acute effects of cyanide were carnivorous birds. In the Wiemeyer et al. (1986) study, the carnivores included the American kestrel, black vulture, and Eastern screeching owl. The herbivores studied were the domestic chicken, Japanese quail, and the European starling.

Mammals

Cyanide is acutely toxic to mammals. Median lethal doses (LD_{50}'s) between 3 and 4 mg/kg body weight have been reported for mice and rats (EPA 1985). Cyanide has caused death and toxicosis in cows and goats acutely exposed to large amounts of cyanide in forage, but dose-response data in these species are limited (Gurnsey et al. 1977, Shaw 1986). Data are limited on the toxic effects of cyanide following chronic exposures. Cyanide has induced physiological changes in rats following long-term dietary exposure to concentrations of 73 ppm (approximately 3.7 mg/kg, Lehman 1954), and has caused toxic effects in piglets exposed during gestation to cyanide in the diet of the mother at concentrations of approximately 277 ppm (approximately 13.9 mg/kg, Lehman 1954) (EPA 1985).

Plants

No information is available on the toxicity of cyanide to plants. However, some plants, themselves, may be a toxicologically significant source of cyanide.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Ambient Water Quality Criteria for Cyanide-1984. Office of Water Regulations and Standards. EPA 440/5-84-028. PB85-227460.

GURNSEY, M.P., JONES, W.T., MERRALL, M. and REID, C.S.W. 1977. Cyanide poisoning in cattle: two unusual cases. New Zealand Veterinary Journal. 25:124-125.

LEHMAN, A.J. 1954. Appraisal of the study of chemicals in food, drugs, and cosmetics. U.S. Association of Food and Drug Officials. 18:66.

NATIONAL ACADEMY OF SCIENCES (NAS). 1980. Mineral Tolerance of Domestic Animals. National Research Council, Subcommittee on Mineral Toxicity in Animals. Washington, D.C.

PICKERING, Q.H., HUNT, E.P., PHIPPS, G.L., ROUSH, T.H., SMITH, W.E., SPEHAR, D.L, STEPHENS C.E. and TANNER, D.K. 1983. Effects of pollution on freshwater fish and amphibians. Journal of the Water Pollution Control Federation. 55(6):840-862.

SHAW, J.M. 1986. Suspected cyanide poisoning in two goats caused by ingestion of crab apple leaves and fruits. Veterinary Record. 119:242-243.

WIEMEYER, S.N., HILL, E.F., CARPENTER, J.W. and KRYNLTSKY, A.D. 1986. Acute oral toxicity of sodium cyanide in birds. Journal of Wildlife Diseases. 22:538-546.

AQUATIC TOXICITY

Invertebrates

Data on the aquatic toxicity of DDT and its metabolites are limited primarily to acute toxicity data and information on bioaccumulation and biomagnification in the food chain. LC_{50} values between 0.2 and 1,230 ug/L have been reported for aquatic invertebrates exposed to DDT, DDD, or DDE (EPA 1980). Mayer and Ellersieck (1986) and Connell and Miller (1984) present acute toxicity data for a variety of taxa. The most sensitive freshwater invertebrate is the water flea Daphnia pulex, with a reported 48-hour EC_{50} of 0.36 ug/L based on immobilization (Mayer and Ellersieck 1986). 96-Hour LC_{50}s of 1 and 4 ug/L are reported for the freshwater amphipod Gammarus lacustris and the isopod Asellus brevicaudus, respectively. LC_{50}s for mosquito larvae (Culex fatigans and Anophetes albimanus) and stonefly naiad (Pteronarcys california) are 70, 10, and 7 ug/L, respectively. Immature crayfish (Orconectes nais) up to 5 weeks of age were markedly more susceptible to DDT than were older crayfish. LC_{50}s for crayfish less than 5 weeks old ranged from 0.18 to 0.9 ug/L, while those for 8 week old and mature crayfish were 28 and 100 ug/L, respectively (Mayer and Ellersieck 1986). LC_{50} values for marine invertebrates range from 0.6 ug/L for the sand shrimp (Crangon septemispinosa) to 6 ug/L for the hermit crab (Pagurus longicarpus) and 9 ug/L for the eastern oyster Crassostrea virginica (Connell and Miller 1984; Mayer and Ellersieck 1986).

Fish

DDT in water is absorbed by fish directly through the skin, and is also accumulated by invertebrates which are ingested by fish. A range of LC_{50} values from 2 to 21 ug/L is reported in Connell and Miller (1984) for freshwater fish. LC_{50} values for freshwater fish species are presented in Mayer and Ellersieck (1986); data are available for several species occurring at APG. The most sensitive species is the largemouth bass (Micropterus salmoides), with a 96-hour LC_{50} of 1.5 ug/L. Other LC_{50}s are 4.9, 5.0, and 15 ug/L for bluegill sunfish Lepomis macrochirus, black bullhead Ictalurus melas, and channel catfish Ictalurus punctatus, respectively. Values for estuarine fish range from 0.4 to 89 ug/L (Connell and Miller 1984).

Chronic life-cycle test with fathead minnows (Pimephales promelas) resulted in toxic effects at DDT concentrations of 0.74 ug/L (EPA 1980). No chronic tests using saltwater species were available in the literature.

Amphibians

Amphibian species are reportedly less susceptible to DDT poisoning than fish (Brown 1978 in Connell and Miller 1984). LC_{50} values for 1 week, 5 week, 6 week, and 7 week old Fowler's toad tadpoles are 750, 1,000, 100, and 30 ug/L,respectively (Mayer and Ellersieck 1986). A 96-hour LC_{50} of 1,000,800 ug/L is reported for the tadpole Pseudacris trisciata (Connell and Miller 1984).

Aquatic Plants

DDT is reportedly toxic to most or all algae at concentrations of 5,000 ug/L (Connell and Miller 1984).

Sediment Toxicity

EPA (1988) proposed an interim sediment criteria value for DDT of 0.008 mg/kg based on the chronic AWQC for DDT and assuming a 10% organic carbon content. The AWQC was derived to be protective of birds feeding on animals from DDT-contaminated areas, but also is protective of aquatic life.

Bioaccumulation

Bioconcentration factors for pinfish (Lagodon rhomboides) and Atlantic croaker (Micropogon undulatus) were 10,000 and 25,000, respectively (EPA 1980).

Criteria

For freshwater organisms, EPA (1986) has established an acute ambient water quality criterion (AWQC) of 1.1 ug/L and a chronic AWQC of 0.001 ug/L. For saltwater organisms, the acute and chronic AWQC are 0.001 and 0.13 ug/L, respectively.

TERRESTRIAL TOXICITY

At sufficiently high doses, DDT can induce death in organisms by disrupting central nervous system transmission. DDT is believed to interfere with ionic movement into and out of axons by binding with the axonal membrane lipoproteins. This changes the structure of the membrane and allows for continued passage of sodium ions (Connell and Miller 1984). DDT is metabolized to the primary metabolites DDE and DDD, and some other minor metabolites. The combination of DDT and its metabolites is often expressed as total DDT residue or DDTR. Acute and chronic DDT toxicity in animals has been studied. The primary acute effect studied has been death. Chronic effects include changes in behavior (e.g., aggression, responsiveness and eating rates) and reproduction (e.g., mating, eggshell thinning, egg hatchability, and hatchling survival). Additionally, DDT's high lipophilicity results in its storage in animal fat at concentrations that, when later mobilized under stress, can cause adverse effects. If the animal is consumed as prey, the fat-stored pesticide serves as an effective dosing mechanism for higher trophic levels.

Birds

Acute median lethal dosage (LD_{50}s) for birds include an LD_{50} of >2,240 mg/kg for mallard ducks and 841 mg/kg for Japanese quail (Hudson et al. 1984). Following chronic exposures to 100 ppm DDT in the diet, 50% of exposed adult mallards died in about 1 year. DDT concentrations of 30 ppm in the diet were not lethal to either mallards Anas platyrhynchos or bobwhite quail Colinus virginianus exposed for 90 days (Hudson et al. 1984).

Many of the sublethal effects of DDT in birds have been attributed to the metabolite DDE. DDE has been found to cause eggshell thinning in birds consuming a diet containing DDTR. Lincer (1975 in Weimeyer et al. 1986) found 14 to 15% shell thinning in American kestrels Falco sparverius daily dosed with 3 ppm DDE for less than 7 months. Stendell et al. (1989) fed 3 captive American kestrels pine voles (Microtus pinetorum) from pesticide-contaminated apple orchards. The pine voles contained 48 mg/kg bw DDE, 3.5 mg/kg bw DDD, and 14.1 mg/kg bw DDT. One kestrel which died at 31 days contained 147 ppm DDE in the carcass (wet weight) and 63 ppm in the brain, while the other two which were sacrificed at 60 days contained 232 ppm DDE in the carcass and only 20.4 ppm in the brain. Wiemeyer et al. (1986) found a 10% thinning in American kestrels dosed with 10 ppm DDE

for 1 year. Field studies have shown poisoning in robins (Turdus migratorius) associated with 53-204 ppm DDTR in earthworms (Lumbricus sp.); in blackbirds Turdus merula and thrushes (Turdidae) it was associated with 13-29 ppm DDTR (Collett and Harrison 1968 in Blus 1978). Laboratory populations of white-throated sparrows (Zonotrichia leucophrys) demonstrated delayed migratory conditions at doses of 5 ppm. DDTR doses at 8 ppm reduced fledgling success of Bengalese finches (Jefferies 1971 in Weimeyer et al. 1986).

The lowest dietary LOEL of 3 ppm, reported for American kestrels, is used to derive a critical toxicity value for the assessment of DDTR impacts in birds. The toxicity data for robins is not used here to assess impacts in robins (a selected indicator species) because it was derived from a field study with undefined exposures and because the toxic endpoint was death (a maximally severe response). Eggshell thinning (the endpoint in the kestrel study) is a more sensitive endpoint and believed to be more reflective of potential subtle toxic effects in wild bird populations. The dietary level of 3 ppm can be converted to a dosage of 1.3 mg/kg body weight (bw) by assuming a kestrel weighs 0.125 kg and ingests 0.052 kg of food each day (APHIS 1987). Applying an uncertainty factor of 100 (10 for interspecies differences and 10 for extrapolation of a NOEL from a LOEL) results in a toxicity criterion of 0.013.

Mammals

Median lethal dietary concentrations in the range of 651 to 1160 ppm have been reported for short-tailed shrews exposed for up to 17 days via the diet to DDT in corn oil (Blus 1978). The lower value of 651 ppm will be conservatively used in this assessment to evaluate the potential for acute impacts to shrews at the site. The dietary concentration of 651 ppm is converted to a dosage of 1,953 mg/kg/day by assuming a shrew weighs 0.016 kg and ingests daily an amount of food equal to 3 times its body weight (0.048 kg food) (Burt and Grossenheider 1976). A critical acute toxicity criterion of 39 mg/kg day is derived by applying an uncertainty factor of 50 (5 to estimate safe acute doses and 10 to account for differences in the two genera of shrews potentially occurring at this site) to the estimated median lethal dosage. This approach does not consider chronic toxicity.

Plants

Residues of DDT are taken up in plants by the roots system. Residues in the vapor phase penetrate plant leaves (Finlayson and MacCarthy 1973 in Connell and Miller 1984). Eno and Everett (1958) found a 35% reduction in root weight and an 11% reduction in the top weight of Black Valentine beans when grown in soil containing 12.5 mg/kg DDTR.

REFERENCES

ANIMAL AND PLANT HEALTH INSPECTION SERVICE (APHIS). 1987. Rangeland Grasshopper Cooperative Management Program. Final Environment Impact Statement-1987. U.S. Department of Agriculture. APHIS FEIS 87-1.

BLUS, L.J. 1978. Short-tailed shrews: toxicity and residue relationships of DDT, dieldrin, and endrin. Arch. Environ. Contam. Toxicol. 7:83-98.

BURT, W.H. and GROSSENHEIDER, R.P. 1976. A Field Guide to the Mammals. The Peterson Field Guide Series, Third Edition. Houghton Mifflin Company. Boston, Massachusetts.

CONNELL, D.W. AND MILLER, G.J. 1984. Chemistry and Ecotoxicology of Pollution. John Wiley and Sons. New York, New York. Pp. 162-227.

ENO, C.F. and EVERETT, P.H. 1958. Effects of soil applications of 10 chlorinated hydrocarbon insecticides on soil microorganisms and the growth of stringless black valentine beans. Proc. Soil Sci. Soc. Am. 22:235-238.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for DDT. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-80-038.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Quality Criteria for Water. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-86-001.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Application of Interim Sediment Criteria Values at Sullivan's Ledge Superfund Site. Memo from Christopher Zarba to Jane Downing. Office of Water. Washington, D.C. April 11, 1988.

HUDSON, R.H., TUCKER, R.K. and HAEGLE, M.A. 1984. Handbook of Toxicity of Pesticides to Wildlife. U.S. Department of the Interior, Fish and Wildlife Service. Washington, D.C. Resource Publication 153.

MAYER, F.L. and ELLERSIECK, M.R. 1986. Manual of Acute Toxicity: Interpretation and Data Base for 410 Chemical and 66 Species of Freshwater Animals. U.S. Department of the Interior, Fish and Wildlife Service. Washington, D.C. Resource Publication 160.

STENDELL, R.C., BEYER, W.N., AND STEHN, R.A. 1989. Accumulation of lead and organochlorine residues in captive American kestrels fed pine voles from apple orchards. J. Wildl. Diseases. 25(3):388-391.

WIEMEYER, S.N., PORTER, R.D., HENSER, G.L. and MAESTRELLI, J.R. 1986. DDE, DDT, and Dieldrin: Residues in American Kestrals and Relations to Reproduction. U.S. Fish and Wildlife Service. Fish and Wildlife Technical Report 6.

1,3-DICHLOROPROPENE

AQUATIC TOXICITY

Aquatic toxicity data for 1,3-dichloropropene are limited. Aquatic toxicity data were obtained from the CIS-AQUIRE database (AQUIRE 1991). Toxicity data for saltwater organisms were not located in the literature. Toxicity to freshwater organisms is discussed below.

Invertebrates

Invertebrate toxicity data are limited to one species, the water flea Daphnia magna. LeBlanc (1981 in AQUIRE 1991) reports 24- and 48-hour LC_{50} values of 7,200 and 6,200 ug/L, and a 48-hour no-observed-adverse effect concentration (NOEC) of 410 ug/L. Johnson and Finley (1980 in AQUIRE 1991) report a 48-hour EC_{50} based on immobilization of 90 ug/L.

Fish

96-hour LC_{50} values ov 1,080, 3,650, 4,100, and 6,100 ug/L are reported for walleye (Stizostedian vitreum), largemouth bass (Micropterus salmoides), fathead minnow (Pimephales promelas) (Johnson and Finley 1980 in AQUIRE 1991), and bluegill sunfish (Lepomis machrochirus) (Buccafusco et al. 1981 in AQUIRE 1991), respectively.

Data regarding toxicity of 1,3-dichloropropene to amphibian and plant species were not located in the literature. No sediment toxicity or bioaccumulation data regarding 1,3-dichloropropene were located.

Criteria

Due to lack of toxicity data, criteria have not been established for 1,3-dichloropropene.

TERRESTRIAL TOXICITY

No data regarding toxicity to terrestrial wildlife species were located in the literature. Data regarding toxicity to laboratory mammals are reported below in absence of more applicable data.

1,3-Dichloropropene is reported to be irritating to skin, eyes, and mucous membranes. Liver and kidney injury have been produced in experimental animals. Oral LD_{50}s of 713 and 470 mg/kg are reported in male and female rats, respectively (Budavari et al. 1989). An LD_{50} of 504 mg/kg is reported in rabbits exposed dermally (Budvari et al. 1989).

REFERENCES

AQUATIC INFORMATION RETRIEVAL (AQUIRE). 1991. Computerized database. Chemical Information Systems, Inc. Balitmore, Maryland.

BUDVARI, S., O'NEIL, M.J., SMITH, A., and HECKELMAN, P.E. (Eds). 1981. The Merck Index - An Encyclopedia of Chemicals, Drugs, and Biologicals. Eleventh Edition. Merck and Co., Inc. Rahway, NJ.

DIELDRIN

AQUATIC TOXICITY

Invertebrates

Toxicity data for several freshwater invertebrate species are reported in Johnson and Finley (1980). LC50 values vary from 0.5 ug/L in the stoneflies Pteronarcys sp. and Pteronarcella sp. to 740 ug/L for the crayfish Orconectes sp. A concentration of 560 ug/L was not acutely toxic to midge larvae (Chironomus sp.), but at 180 ug/L these organisms were unable to survive over 14 days, and they were unable to complete metamorphasis at 5.6 ug/L.

Dieldrin toxicity values for several estuarine invertebrates are reported by Mayer (1987). The most sensitive estuarine species is the pink shrimp Penaeus duorarum, with a reported 48 hour EC50 of 0.30 ug/L. 96-hour LC50s of 3.7 and 4.5 are reported for the mysid Mysidopsis bahia, and a 96-hour LC50 of 8.6 ug/L is reported for the grass shrimp Palaemonetes pugio. Other invertebrate species appear to be slightly less sensitive. A 48-hour EC50 of 240 is reported for the blue crab Callinectes sapidus, and 96-hour EC50s of 15 and 31 ug/L are reported for the eastern oyster Crassostrea virginica.

Fish

In fish, exposure to dieldrin in the diet has been reported to result in significantly altered serum amino acid composition, adrenal and thyroid function, ammonia detoxification, and phenyl keto acid metabolism (Johnson and Finley 1980).

96-Hour LC50 for freshwater species range from 1.2 ug/L for rainbow trout Salmo gairdneri to 19 ug/L for channel catfish Ictalurus punctatus. LC50s for estuarine species are 3.2 ug/L for spot Leiostomus xanthurus and striped mullet Mugil cephalus) and 10 ug/L for sheepshead minnow Cyprinodon variegatus (Mayer 1987).

Bioaccumulation

Dieldrin has an estimated BCF of 269 based on Veith et al. (1979).

Criteria

Ambient water quality criteria established by EPA (1986) state that freshwater organisms will not be unacceptably affected if the one-hour average concentration does not exceed 2.5 ug/L, and if the 24-hour average does not exceed 0.0019 ug/L.

Based in the Equilibrium Partition Approach, EPA (1988) has proposed a sediment quality criteria of 0.199 mg/kg.

TERRESTRIAL TOXICITY

Birds

Hudson et al. (1984) reported the following LD_{50}s in mg/kg body weight for birds: mallard, 381, fulvous whistling duck, 100-200, Canada goose, <141, pheasant, 79.0, Japanese quail, 69.7, house sparrow, 47.6, rock dove, 26.6, chukar, 25.3, gray partridge, 8.84, and California quail, 8.78.

Mammals

Acute LC_{50}s are 75-150 mg/kg for mule deer and 100-200 mg/kg for domestic goat (Hudson et al. 1984). An acute oral LD_{50} for rats of 37-87 mg/kg is reported in Meister's Farm Chemicals Handbook (1988).

Plants

Information regarding toxicity to plants were not located in the literature.

REFERENCES

HUDSON, R.H., TUCKER, R.K. and HAEGELE, M.A. 1984. Handbook of toxicity of pesticides to wildlife. U.S. Department of the Interior, Fish and Wildlife Service. Resource Publication 153. Second Edition. Washington, D.C.

MAYER, F.L., Jr. 1987. Acute toxicity handbook of chemicals to estuarine organisms. Environmental Research Laboratory, Gulf Breeze, FL. EPA 600/8-87-017.

MEISTER, R.T. 1988. Farm Chemicals Handbook. Meister Publishing Company. Willoughby, Ohio.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Quality Criteria for Water 1986. Office of Water Regulations and Standards, Washington D.C. May 1, 1986. EPA 440/5-86-001

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Interim Sediment Criteria Values for Nonpolar Hydrophobic Organic Compounds. Office of Water. Washington, DC

VEITH, G.D., DeFOE, D.L., and BERGSTEDT, B.V. 1979. Measuring and Estimating the Bioconcentration Factor of Chemicals in Fish. J. Fish Res. Board Can. 36:1040-1048

1,3-DINITROBENZENE

AQUATIC TOXICITY

Aquatic toxicity data for 1,3-dinitrobenzene are limited. Aquatic toxicity data obtained from the AQUIRE (1990) database. Toxicity to saltwater organisms were not located in the literature. Toxicity to freshwater organisms is discussed below.

Invertebrates

Invertebrate toxicity data are limited to one species, the water flea Daphnia magna. Pearson et al. (1979) report a 48-hour EC_{50} of 53,000 ug/L for D. magna. In a 16-day study, an EC_{50} based on reproduction of 3,200 ug/L was reported (Hermens et al. 1984 in AQUIRE 1990). The lowest concentration reported to produce toxic effects is 1,200 ug/L, which resulted in a 10% reduction in growth of D. magna (Deneer et al. 1988 in AQUIRE 1990).

Fish

96-Hour LC_{50} values range from 5,000 to 15,900 ug/L for fathead minnow Pimephales promelas (AQUIRE 1990). Acute toxicity was reported in the golden orfe Leuciscus idus at a concentration of 10,000 ug/L (Juhnke and Luedemann 1978 in AQUIRE 1990). A no effect concentration of 159 mg/kg diet was reported in carp Cyprinus carpio exposed for 21 hours (Loeb and Kelly 1963 in AQUIRE 1990).

Amphibians

Data regarding toxicity of 1,3-dinitrobenzene to amphibian species were not located in the literature.

Plants

Data regarding toxicity to aquatic plants are limited to algae species. The most sensitive species reported in the literature is the blue-green algae Anacystis aeruginosa, with a lowest observed effect concentration (LOEC) of 170 ug/L based on population growth following 8 days of exposure (Bringmann and Kuhn 1978 in AQUIRE 1990). In the green algae Scenedesmus quadricauda, an 8-day LOEC of 700 ug/L is reported (Bringmann and Kuhn 1978 in AQUIRE 1990).

Sediment Toxicity

No sediment toxicity data regarding 1,3-dinitrobenzene were located in the literature.

Bioaccumulation

Data regarding bioaccumulation of 1,3-dinitrobenzene are not available.

Criteria

Due to a lack of toxicity data, criteria have not been established for 1,3-dinitrobenzene.

TERRESTRIAL TOXICITY

No data regarding toxicity to terrestrial wildlife species were located in the literature. Studies reporting toxicity in laboratory rats were found, and are reported below in absence of more applicable data.

1,3-Dinitrobenzene has been reported to induce methemoglobinemia and anemia, splenic enlargement, and cyanosis in mammals following prolonged exposure (Blackburn et al. 1988). It is also believed that 1,3-DNB interferes with intracellular redox mechanisms resulting in impaired glucose oxidation (Nystrom and Rickert 1987). Reproductive effects in male rats were noted 12 hours after intraperitoneal injection of 25 mg/kg body weight 1,3-DNB, and 48-hours after 15 mg/kg (Blackburn et al. 1988).

REFERENCES

AQUATIC INFORMATION RETRIEVAL (AQUIRE). 1990. Computerized database. Chemical Information Systems, Inc. Baltimore, Maryland.

BLACKBURN, D.M., GRAY, A.J., LLOYD, S.C., SHEARD, C.M., and FOSTER, P.M. 1988. A comparison of the effects of the three isomers of dinitrobenzene on the testis in the rat. Toxicol. and Appl. Pharmacol. 92:54-64.

NYSTROM, D.D. and RICKERT, D.E. 1987. Metabolism and excretion of dinitrobenzenes by male Fischer-344 rats. Drug Metabolism and Disposition. 15(6):821-825.

PEARSON, J.G., GLENNON, J.P., BARKLEY, J.J., and HIGHFILL, J.W. 1979. An approach to the toxicological evaluation of a complex industrial wastewater. In: Marking, L.L. and Kimerle, R.A. (eds.). Aquatic Toxicology. Philadelphia, Pennsylvania. ASTM STP 667.

1,4-DITHIANE

AQUATIC TOXICITY

1,4-Dithiane is a thermal degradation product of mustard. Toxicity data for 1,4-dithiane were not available in the literature. However, EPA (1988) has published a methodology which utilizes relationships between a chemical's physico-chemical properties (molecular weight, and octanol water partition coefficient, K_{ow}) and its toxicity. These structure activity relationships (SARs) are regression equations that may be used to derive toxicity estimates for organic chemicals with no available toxicity data. EPA presents SAR equations for 3 broad classes of organic chemicals: (1) neutral organics which are non-reactive and non-ionizable, (2) neutral organics which are reactive and show excess toxicity in addition to narcosis, and (3) surface active organic compounds such as surfactants and polycationic polymers. According to R.G. Clements at EPA[1], 1,4-dithiane may be grouped into the first class of chemicals, neutral organics. For this group, four freshwater and two saltwater species SAR equations are provided in EPA (1988). These SARs and an SAR for tadpoles derived by (Lipnick 1989) are presented below. The aquatic toxicity information that follows was derived using these equations.

1) Fish 96-hour LC_{50} SAR:
   $\log LC_{50} = -0.94 \log K_{ow} + 0.94 \log (0.000068\ K_{ow} + 1) - 1.25$;
   where LC_{50} is in moles/L.

2) Fish 14-day LC_{50} SAR:
   $\log(1/LC_{50}) = 0.871\ \log Kow - 4.87$; where LC_{50} is in umoles/L.

2) Sheepshead Minnow 96-hour LC_{50} SAR:
   $\log (1/LC_{50}) = 0.73\ \log Kow - 3.69$; where LC_{50} is in umoles/L.

3) Daphnid 48-hour LC_{50} SAR:
   $\log (1/LC_{50}) = 0.91\ \log Kow - 4.72$; where LC_{50} is in umoles/L.

4) Mysid 96-hour LC_{50} SAR:
   $\log (1/LC_{50}) = 1.25\ \log Kow - 4.83$; where LC_{50} is in umoles/L.

5) Algae 3-hour EC_{50} SAR:
   $\log EC_{50} = 8.865 - 1.0446\ \log Kow$; where EC_{50} is in umoles/L.

6) Tadpole lowest observed effect concentration (LOEC) SAR:
   $\log (1/LOEC) = 0.909\ \log Kow + 0.727$; where LOEC is in moles/L.

1,4-Dithiane has an octanol-water partition coefficients (Kow) of 70.8 (logKow = 1.85) and a molecular weight of 120.4 g/mole. Molecular weight was used to convert concentrations from moles or micromoles/L to ug/L.

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[1]Clements, Richard G. Personal communication with Richard G. Clements, EPA SAR hotline. December 18, 1990.

Invertebrates

A freshwater daphnid 48-hour LC_{50} of 131,500 ug/L and a saltwater mysid LC_{50} of 40,000 ug/L are calculated using the above SARs. These high values indicate that 1,4-dithiane is not very toxic to aquatic invertebrates.

Fish

The 96-hour LC_{50} derived for freshwater fish is 124,500 ug/L, while that for the saltwater sheepshead minnow (Cyprinodon variegatus) is 26,500 ug/L. A 14-day LC_{50} for freshwater fish of 219,000 ug/L was also calculated.

Amphibians

A LOEC of 4.7×10^6 ug/L is calculated for tadpoles. This high value indicates that 1,4-dithiane is probably not very toxic to tadpoles.

Plants

The estimated 3-hour EC_{50} for algae is 1.1×10^{11} ug/L. This value indicates that 1,4-dithiane is not acutely toxic to aquatic plants.

TERRESTRIAL TOXICITY

Data regarding toxicity to terrestrial wildlife were not available in the literature. Based on a rat oral LD_{50} of 2,768 mg/kg, 1,4-dithiane does not appear to be very toxic. Tremors, ataxia, and dyspnea were noted in animals prior to the onset of death (RTECS 1990).

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Estimating toxicity of industrial chemicals to aquatic organisms using structure activity relationships. Volume 1. Office to Toxic Substances. Washington, D.C. EPA 560/6-88-001.

LIPNICK, R.L. 1989. A quantitative structure-activity relationship study of Overton's data on the narcosis and toxicity of organic compounds to the tadpole, Rana temporaria. Aquatic Toxicol. and Environ. Fate. ASTM STP 1007. 77:468-489.

REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (RTECS). 1990. National Institute of Occupational Safety and Health, U.S. Department of Health and Human Services.

FLUORIDE

Fluoride is a ubiquitous component of the environment. Consequently, animal and plant species have most likely evolved while in contact with small amounts of fluoride and can be assumed to have tolerance for trace amounts of the substance. However, exceeding the "tolerance" level by even small amounts has been shown to induce physiological response in both animal and plants. Below, the effects of fluoride on aquatic and terrestrial species are discussed. Effects on invertebrates, vertebrates and plants are presented.

AQUATIC TOXICITY

Invertebrates

Data regarding toxicity of fluoride to freshwater invertebrate organisms are extremely limited. Fluoride had no effect on the survival of freshwater protozoa and rotifera exposed to up to 450,000 ug/L for up to 9 days (Wantland 1956 in Groth 1975). A 48-hour median effective concentration (EC_{50}) of 225,000 ug/L based on cessation of activity is reported for Daphnia sp. (Anderson 1946 in Groth 1975). A fluoride concentration of 122,000 ug/L is reported to produce acute toxic effects in Daphnia (Bringmann and Kuhn 1959 in Groth 1975). A 48-hour lethal concentration (LC_{50}) for fluoride (as cryolite) of 5,000 ug/L is reported by Sanders and Cope (1966 in Groth 1975) for Daphnia. These authors also report a 48-hour EC_{50} of 5,000-10,000 ug/L based on cessation of activity for Simocephalus.

Fish

Neuhold and Sigler (1960 in Groth 1975) reported an LC_{50} of 75,000-91,000 ug/L for freshwater carp (Carassius auratus) exposed for up to 480 hours in soft water. They also reported 48- and 240-hour LC_{50} values of 2,700 ug/L and 4,700 ug/L respectively for rainbow trout (Salmo gairdneri). Summerfelt and Lewis (1967) reported a 5-minute no-effect level (NOEL) of 20,000 ug/L based on avoidance in the green sunfish (Lepomis cyanella).

Hebert and Shurben (1964 in Groth 1975) reported 5% mortality in rainbow trout exposed for 3 weeks to 4,000 ug/L in soft water. A fluoride concentration of 8,500 ug/L, also in soft water, resulted in 50% mortality. However, in a 3-week study in hard water, concentrations up to 75,000 ug/L resulted in no increased mortality. This indicates that water hardness may have an antagonistic effect on fluoride toxicity, possibly by reducing the amount of fluoride available for uptake by fish. Trout eggs exposed to 1,500 ug/L of fluoride during development experienced a 7-10 day delay in hatching (Ellis et al. 1948 in Groth 1975).

Data regarding toxicity to saltwater species are limited. Fluoride has induced altered behavior in chinook salmon (Oncorhychus tshawytscha), coho salmon (O. kisutch), and chum salmon (O. keta) (Dankaer and Dey 1986). Avoidance behavior was apparent in the adults and juveniles of these three species at a fluoride concentration of 500 ug/L. A fluoride concentration of 200 ug/L is believed to be at or below the threshold level of fluoride sensitivity for chinook and coho salmon. Avoidance behavior may result in decreased reproductive success of salmon if water concentrations are sufficient to inhibit migration to spawning sites.

Groth (1975) presents toxicity data for a variety of aquatic organisms. A concentration of 32,000 ug/L caused 100% mortality in oysters (Crassostrea sp.) exposed for 60 days, while 8,000 ug/L had no effect on survival. Growth in blue crab (Callinectes sapidus) was reduced at a concentration of 20,000 ug/L.

Amphibians

Only limited data are available regarding toxicity of fluoride to amphibians. For frogs immersed in fluoride-containing water for one week, an LC_{50} of 990,000 ug/L was reported (Simonin and Pierron 1937 in Groth 1975). In frogs exposed to 5,000-3,000,000 ug/L for prolonged periods of time, reduced red and white blood cell counts were observed (Kaplan et al. 1964 in Groth 1975). Other effects include premature hatching and retarded development in Northern leopard frog (Rana pipiens) eggs kept in well water containing 1,000 ug/L fluoride (Cameron 1940 in Groth 1975). Tadpoles kept in water at 500 and 4,500 ug/L fluoride showed delayed metamorphosis and abnormal thyroid development (Kuusisto and Telkka 1961 in Groth 1975). Toad eggs and tadpoles kept in water at fluoride concentrations of 13,000-450,000 ug/L experienced premature hatching, delayed metamorphosis, and retarded development (Kawahara and Kawahara 1971 in Groth 1975).

Plants

In the freshwater alga Chlorella, a 37% growth reduction was observed following exposure to 2,000 ug/L for 48 hours. Exposure to concentrations of 200,000 ug/L resulted in a 86%-99% reduction in growth in Chlorella exposed for 48-72 hours (Smith and Woodson 1965 in Groth 1975). For Scenedesmus sp., a 4-day LC_{50} of 43,000 ug/L fluoride (as sodium fluoride) was reported (McKee and Wolf 1963).

Sediment Toxicity

Data regarding toxicity of fluoride in sediment were not found in the literature.

Bioaccumulation

Bioaccumulation of significant amounts of fluoride has been reported in aquatic organisms, primarily in the skeleton (including the gills) and exoskeleton (NRCC 1977). A bioconcentration factor of 1.3 was reported in trout fillets (Groth 1975). Fluoride concentrations of 450-700 ppm have been detected in bone of brown trout (Salmo trutta) living in a stream with a natural fluoride content of 1-14 ppm (Groth 1975). A whole-body concentration of 10 ppm fluoride wet weight basis is reported in fry of brown trout exposed to 500 ug/L fluoride in tap water for 200 hours (Wright 1977 in NRCC 1977).

Some aquatic plants can accumulate fluoride from waters containing about 1 ppm, but magnification is much less than that seen in some terrestrial plant species (Groth 1975). Fluoride concentrations of 40.5 ppm were observed in freshwater plants; however, the fluoride content of the water was not reported (Danilova 1944 in Groth 1975).

Criteria

EPA has not established any criteria for fluoride based on protection of freshwater aquatic life. Adequate toxicity data to establish criteria are not available, and

further research is necessary to accurately determine the toxic effect of fluoride on aquatic organisms.

TERRESTRIAL TOXICITY

Invertebrates

Data on the effects of exposure of invertebrates to fluoride are limited. Lillie (1970 in Groth 1975) reviewed the literature on the toxicity of fluoride to honeybees (Apis mellifera) and concluded that 4-5 mg of accumulated fluoride per bee may be lethal. Gerdes et al. (1971a in NRCC 1977) reported airborne gaseous HF concentrations of 1.3, 2.9, 4.2, and 5.5 ppm were lethal to fruit flies (Drosophila melanogaster) exposed for up to 6 weeks. All flies in the 5.5 ppm group were killed within 3 days. All other dose groups suffered at least 25% mortality in 6 weeks. Effects on reproduction of surviving flies also were observed (Gerdes et al. 1971b in NRCC 1977); statistically significant declines were observed in fecundity and egg hatchability with increasing parental exposure.

Changes in invertebrate populations may have implications for community and ecosystem function. For example, Beyer et al. (1987) reported that concentrations of fluoride in vegetative litter near an aluminum plant were between 400 and 1,000 ppm and were toxic to wood lice (Oniscoidea sp.). Wood lice are important litter decomposers in forested ecosystem. Theoretically, decreases in wood lice populations could affect an entire ecosystem by disrupting energy flow and nutrient cycling. Therefore, in addition to direct effects on forest vegetation, fluoride could indirectly affect forests by inhibiting decomposition of litter.

Birds

There is little data on the effects of fluoride on wild birds. However, fluoride emissions in the vicinity of aluminum smelters were reported to result in elevated yolk and albumen fluoride levels in the eggs of chicken and wild birds (Van Toledo 1978 in Hoffman et al. 1985). Additionally, it has been suggested that house martins (Delichon urbica) and tawney owls (Strix aluco) near an aluminum smelter were adversely affected, with evidence of impaired reproduction in the owls (Hoffman et al. 1985). However, sodium fluoride at a concentration of 50 ppm in the diet of captive American kestrels (Falco sparverius) had few adverse effects on reproductive performance (Bird and Massari 1983 in Hoffman et al. 1985).

Hoffman et al. (1985) exposed screech owls (Otus asio) to chronic dietary sodium fluoride at dietary concentrations of 0, 40, and 200 ppm and reported that fluoride at 40 ppm resulted in significantly smaller egg volume. Dietary concentrations of 200 ppm also resulted in lower egg weights and lengths. Hatching success was impaired by nearly 40% in the 200 ppm groups.

Fleming et al. (1987) exposed nestling European starlings (Sturnus vulgaris) to daily oral doses of distilled water at concentrations of 193 mg sodium/kg bw (sodium control group) or 6, 10, 13, 17, 23, 40, 80, or 160 mg of fluoride as NaF in distilled water per kg of body weight. Dosing began when the starlings were 24-48 hours old and continued for 16 days. The sodium control group did not differ from the water control with respect to any

measured values. The 24-hour LD_{50} of fluoride for the day old starling was 50 mg/kg. The 16-day LD_{50} was 17 mg/kg. Growth rates were significantly reduced in the 13 and 17 mg/kg dose group; growth rates in birds at the higher dose levels were omitted from growth comparisons because of high fluoride-induced mortality.

Mammals

Non-domestic Species. Fluoride has been documented to accumulate in the skeletons of wild animals (NRCC 1977). Although the data currently available are not adequate to assess the toxicological significance of fluoride pollution to wild mammals, some data indicate the effects may be serious. Lameness induced by fluorosis has been observed in wild mules and white-tailed deer (Odocoileus virginianus) (Kay et al. 1975b in NRCC 1977). Impairment of mobility can affect predator-prey relationships such that the affected individual is rapidly eliminated from the population (NRCC 1977). This could lead to decreases in population sizes and disruption of population structures. In fact, Kay et al. (1975b in NRCC 1977) did observe an apparent population age shift in a deer population afflicted by fluoride toxicosis.

Domestic Species. The toxic effects of fluoride on domestic species have been studies extensively. The primary toxic effect of excessive fluoride intake in domestic animals is skeletal fluorosis. Because it is the result of chronic ingestion of fluoride, fluorosis most often is apparent in older animals (NRCC 1977). In the most severe cases, animals become intermittently or permanently lame. Bone exostoses become radiographically or even visually apparent, especially near the leg joint (NRCC 1977). Excessive fluoride intake during tooth development can cause tooth damage and eventually lead to increased tooth wear (NRCC 1977). This can contribute to a decline in the nutritional status of the animal.

Out of all North American domestic species, cattle appear to be the most sensitive to fluoride (NRCC 1977). However, data sufficient to develop a quantitative dose-response relationship are limited. Most of the available data for cows provide bone fluoride content in healthy and unhealthy animals. A study by Forsyth et al. (1972 in NRCC 1977) provides some data useful for determining dose-response relationships in swine. In this study, young swine were fed diets containing 0, 30, 150, or 450 ppm fluoride, as sodium fluoride, for up to 18 weeks. A linear decline in growth rate with increasing dietary fluoride was observed. Growth in the 150- and 450-ppm groups was (statistically) significantly decreased.

These data are of limited usefulness for predicting dose-response in cattle, however, because cattle are more sensitive to the effects of fluoride. Also, the results were reported following only 18 weeks of exposure. Because fluorosis usually occurs following low-level exposure over a period of years, it is possible that a much lower "lowest-effect" level would become apparent if exposure and observation were increased over a period of years.

Plants

Plants absorb and accumulate fluoride from the atmosphere, soil, or water, although exposure to airborne fluoride has been the most extensively studies. Root absorption is a significant route of entry only when fluoride exists in a soluble form (Weinstein and

Alscher-Herman 1982). In most cases, the most important route of entry into the plant, particularly of gaseous fluoride, is through the stomata of leaves. Once inside the plant, fluoride dissolves in plant liquids and moves in the transpirational stream to its principal sites of accumulation at the tips and margins of the leaves (Weinstein and Alscher-Herman 1982).

Fluoride causes toxic effects in plants by disrupting a variety of physiological processes. Fluoride can cause leaf necrosis, reduced growth, decreased apparent photosynthesis, decreased germination, decreased fruit size and seed number, and chromosomal aberrations in terrestrial species (NRCC 1977). Exposure concentrations producing these effects vary with species and a variety of environmental factors.

Long-term exposures of sorghum to relatively low levels of HF (0.7, 1.7, or 3.5 ug/m^3) resulted in reversible inhibition of apparent photosynthesis (McCune et al. 1976 in Weinstein and Alscher-Herman 1982). Other studies have shown an effect of fluoride on apparent photosynthesis (Weinstein and Alscher-Herman 1982), but exposures to high levels of fluoride (>10 ug/m^3) have been reported to reduce apparent photosynthesis in all species tested.

Exposures as low as 0.019 ppm fluoride in water have been observed to cause chromosomal aberrations in barley (Bale and Hart 1973 in NRCC 1977). Conover and Poole (1971 in NRCC 1977) found that cuttings of a horticultural foliage plant suffered serious (approaching 50%) leaf necrosis when set for rooting in water containing 0.5 ppm fluoride.

Conifers appear to be the most susceptible forest species to fluoride toxicity, and the greater toxicity to these species could result in changes to community and ecosystem structure. For example, Sidhu (1977 in NRCC 1977) studies the effects of fluoride on forest vegetation in New Foundland and observed that the softwood tree canopy (balsam fir, black spruce, larch) was being replaced by an undergrowth of hardwoods (white birch, American ash) (NRCC 1977). As the mortality of the original tree cover continued, the shrub layer also showed a significant change in community structure. Based upon the results of this study, Sidhu (1977 in NRCC 1977) concluded that the safe levels of fluoride in air for forest species appear to be between 0.17 and 0.23 ug/m^3.

Bioaccumulation and Biomagnification

Both plants and animals accumulate fluoride. Plants can accumulate fluoride from air, water, or soil, although the fluoride from the soil must be in a solubilized form (Weinstein and Alscher-Herman 1982). The amount accumulated before observable toxicity vary with species sensitivity; highly susceptible species, such as conifers, may exhibit foliar lesions from an accumulation of as little as 10-20 ppm fluoride (Weinstein and Alscher-Herman 1982). In contrast, more resistent species, such as hickories or cotton, can accumulate high concentrations of fluoride (as much as 4,000 ppm in cotton) without foliar injury (Weinstein and Alscher-Herman 1982).
Bioconcentration in wildlife also varies with species. Based on analysis of fluoride content in femurs of 30 wild species, Kay et al. (1975a in NRCC 1977) reported that carnivorous species appear to concentrate more fluoride in the bone than herbivorous species. But these data are insufficient to draw conclusions regarding biomagnification of fluoride.

Criteria

No federal criteria for the protection of terrestrial species from fluoride toxicity have been promulgated. Some U.S. state regulations make it unlawful for an industry to emit fluoride at a level that will cause a fluoride content of locally-grown forage to exceed 30 or 40 ppm on a dry-weight basis (NRCC 1977).

REFERENCES

BEYER, W.N., FLEMING, W.J. and SWINFORD, D. 1987. Changes in litter near an aluminum reduction plant. J. Environ. Qual. 16:246-250.

DANKAER, D.M. and DEY, D.B. 1986. Effects of Water-borne Pollutants on Salmon-Passage at John Day Dam, Columbia River. Final Report of Research. U.S. Corps of Engineers. Contract DACW57-85-H-000.

FLEMING, W.J., GRUE, R.E., SCHULER, G.A, and BUNCK, C.M. 1987. Effects of oral doses of fluoride on nestling European starlings. Arch. Environ. Contam. Toxicol. 16:483-489.

GROTH, D. 1975. An evaluation of the potential for ecological damage by chronic low-level environmental pollution by fluoride. Fluoride. 8(4):224-240.

HOFFMAN, D.J., PATTEE, O.H., and WIEMEYER, S.N. 1985. Effects of fluoride on screech owl reproduction: teratological evaluation, growth, and blood chemistry of hatchlings. Tox. Lett. 26:19-24.

McKEE, J.E. and WOLF, H.W. 1963. Water Quality Criteria. California State Water Quality Control Board. Sacramento, California. Publication No. 3A.

NATIONAL RESEARCH COUNCIL OF CANADA (NRCC). 1977. Environmental Fluoride 1977. Associate Committee on Scientific Criteria for Environmental Quality. Ottawa, Ontario. NRCC No. 16081.

SUMMERFELT and LEWIS. 1967. Data from an AQUIRE literature search. In: Dynamac Corp. Information Review-Cryolite. Working Draft. Prepared for TSCA Interagency Testing Committee. April 1986. Contract No. 68-02-4251.

WEINSTEIN, L.H. and ALSCHER-HERMAN, R. 1982. Physiological responses of plants to fluorine. In: Unsworth, M.H. (ed.). Effects of Air Pollution in Agriculture. Butterworth Press. England. Pp. 139-167.

HEXACHLOROBENZENE

AQUATIC TOXICITY

Invertebrates

Mayer (1987) reports 96-hour EC_{50}s for 3 estuarine invertebrate species. EC_{50}s are reported to be >17 ug/L for grass shrimp Palamonetes pugio, >25 ug/L for pink shrimp Penaeus duorarum, and >1,000 ug/L for the eastern oyster Crassostrea virginica. Laska et al. (1978 in AQUIRE 1991) reported no toxic effects in red swamp crayfish (Procambarus clarkii) exposed to 27.3 ug/L.

Fish

Acute toxicity values for 5 species of fish are reported in Johnson and Finley (1980). A 96-hour LC_{50} of 12,000 ug/L is reported for both bluegill sunfish (Lepomis macrochirus) and largemouth bass (Micropterus salmoides) exposed in hard water. 96-Hour LC_{50}s of 14,000, 22,000, and >50,000 ug/L are reported for channel catfish (Ictalurus punctatus), fathead minnow (Pimephales promelas), and coho salmon (Oncorhyncus kisutch), respectively. No adverse effects were noted in largemeouth bass exposed to 25.8 ug/L for 10 days (Laska et al. (1978 in AQUIRE 1991).

Mayer (1987) reports 96-hour EC_{50}s of >13 and >100 ug/L for the saltwater species sheepshead minnow (Cyprinodon variegatus) and pinfish (Lagodon rhomboides).

Amphibians

Nishiuchi (1980 in AQUIRE 1991) reported a 24-hour LC_{50} of >4,200 ug/L for the frog Bufo bufo japonicus.

Plants

No data regarding hexachlorobenzene toxicity to aquatic plants were located in the literature.

Bioaccumulation

Bioconcentration factors for 4 invertebrate species are reported in the AQUIRE (1991) database. The average of two 33-day bioconcentration factors (BCFs) is 1,800 for the water flea and 1,570 for the ramshorn snail (Helisoma sp.) (Isensee et al 1976 in AQUIRE 1991). A 33-day BCF of 1,247 is reported for the pouch snail (Physa sp.) and a 72-hour BCF of 16 is reported for the mosquito Culex pipiens (Metcalf et al. 1973 in AQUIRE 1991). Johnson and Finley (1980) report that Daphnia magna accumulated residues near 900 times the exposure level of 0.05 to 0.15 ug/L within 48 hours.

Rainbow trout (Salmo gairdneri) accumulated residues 3,800 to 8,900 times the exposure level of 0.1 to 2.0 ug/L within 28 days (Johnson and Finley 1980). An 11-day BCF of 235 is reported for the longnose killifish ( )(Glam et al. 1980 in AQUIRE 1991), and a 72-hour BCF of 1,200 is reported for the golden orfe (Korte et al. 1978).

Sediment Toxicity

Barrick and Beller (1989) reported sediment toxicity values in terms of apparent effect thresholds (AETs). They report AETs of 0.38, 4.5, and 9.6 mg/kg (organic carbon) for benthic invertebrates, amphipods, and oysters, respectively.

Criteria

No ambient water quality criteria have been established for hexachlorobenzene. EPA (1986) states that for the general class of chlrinated benzenes, acute toxicity to freshwater aquatic life occurs at concentrations as low as 250 ug/L and would occur at lower concentrations among species that are mores sensitive than those tested. Chronic toxicity occurs at concentrations as low as 50 ug/L for a fish species exposed for 7.5 days.

EPA (1986) states that for the general class of chlorinated benzenes, aucte and chronic toxicity to saltwater aquatic life occur at concentraitons as low as 160 and 129 ug/L, respectively, and would occur at lower concentraitons among more sensitive species.

TERRESTRIAL TOXICITY

Mammals

Studies using rhesus monkeys indicate that long-term storage of hexachlorobenzene in fat occurs, and metabolism is relatively slow, with only 4.4 % of the dose excreted as metabolites after one year (Menzie 1980). Toxicity data for wild and domestic mammals were not located in the literature.

Birds

Hill et al. (1975) report 8-day LC_{50}s of 617 and > 5,000 mg/kg diet (mortality = 30 % at 5,000 mg/kg diet) for ring-necked pheasant (Phasianus colchicus) and mallard (Anas platyrhynchos), respectively.

Plants

Data regarding toxicity to terrestrial plants were not located in the literature.

REFERENCES

AQUATIC INFORMATION RETRIEVAL (AQUIRE). 1991. Computerized database. Chemical Information Systems, Inc. Baltimore, Maryland.

BARRICK, R.C. AND BELLER, H.R. 1989. Reliability of sediment quality assessment in Puget Sound. In: Oceans '89 Proceedings. Inst. of Electrical and Electronic Engineers. Piscataway, New Jersey. Publication No. 89CH2780-5.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Quality Criteria for Water 1986. Office of Water Regulations and Standards, Washington D.C. May 1, 1986. EPA 440/5-86-001

HILL, E.F., HEATH, R.G., SPANN, J.W., and WILLIAMS, J.D. 1975. Lethal Dietary Toxicities of Environmental Pollutants to Birds. U.S. Fish and Wildlife Service. Special Scientific Report--Wildlife No. 191. Washington, D.C.

JOHNSON, W.W. and FINLEY, M.T. 1980. Handbook of Acute Toxicity of Chemicals to Fish and Aquatic Invertebrates: Summaries of Toxicity Tests Conducted at Columbia National Fisheries Research Laboratory, 1965-1978. U.S. Fish and Wildlife Service. Resource Publication 137. Washington, D.C.

MAYER, F.L., Jr. 1987. Acute toxicity handbook of chemicals to estuarine organisms. Environmental Research Laboratory, Gulf Breeze, FL. EPA 600/8-87-017.

MENZIE, C.M. 1980. Metabolism of Pesticides - Update III. U.S. Fish and Wildlife Service, Washington, DC. Special Scientific Report - Wildlife No. 232.

AQUATIC TOXICITY

Iron is an essential trace element required by both plants and animals. Ferrous (Fe^{+2}) and ferric (Fe^{+3}) iron are the species of concern in aquatic systems, although ferric iron is practically insoluble (EPA 1986).

Invertebrates

Warnick and Bell (1969 in EPA 1976) reported 96-hour LC_{50} values of 320 ug/L for mayflies, stoneflies, and caddisflies. Precipitates of iron can smother bottom-dwelling organisms. Dissolved iron readily precipitates in sea water, where it may adversely affect mussels and other shellfish (EPA 1976).

Fish

Brandt (1948 in EPA 1976) found iron toxic to carp (Cyprinus carpio) at concentrations of 900 ug/L at a water pH of 5.5. Iron concentrations of 1,000-2,000 ug/L were lethal to northern pike (Esox lucius) and trout (species not known) (Doudoroff and Katz 1953 in EPA 1976). In an iron polluted Colorado stream, neither trout nor other fish were found until the waters were diluted or the iron had precipitated to effect a concentration of less than 1,000 ug/L even though other water quality parameters measured were suitable for the presence of trout (FWPCA 1967 in EPA 1976). Precipitates of iron (ferric hydroxide) coat the gills and inhibit oxygen uptake, and also create a smothering effect detrimental to fish eggs. Davies and Goettl (1979 in Lehnertz 1989) have reported that 1,000 ug/L iron at low stream flow can be lethal to eggs and fry via this mechanism.

Amphibians

Data regarding toxicity of iron to amphibians were not located in the literature.

Plants

Concentrations of 100 to 500 ppm soluble iron in soil were shown to be toxic to rice; concentrations above 500 ppm were highly toxic (Foy et al. 1978 in EPA 1985).

Sediment Toxicity

Data regarding toxicity of iron in sediment were not located in the literature.

Bioaccumulation

Data regarding bioaccumulation of iron in aquatic organisms were not located in the literature.

Criteria

The European Inland Fisheries Advisory Commission (1964 in EPA 1976) recommended that iron concentrations not exceed 1,000 ug/L in waters to be managed for aquatic life. The EPA chronic water quality criteria for iron is 1,000 ug/L, based on toxicity to fish (EPA 1986). This level is too high according to Smith et al. (1979), in part because of the reported toxicity to aquatic insects at 320 ug/L. No acute criterion for iron has been established by EPA.

TERRESTRIAL TOXICITY

Wildlife

Birds

No information was found on the toxicity of iron to wild birds. Limited information is available for domestic birds. Chicks were able to tolerate 1,600 ppm of iron when also given adequate amounts copper in the diet. Decreased weight gain and increased mortality occurred at 200 ppm iron and 5 ppm copper (McGhee et al. 1965 in NAS 1980). Rickets were produced in young chicks at 4,500 ppm iron (Deobald and Elvehjem 1935 in NAS 1980). Turkeys shows no adverse effects at 440 ppm iron (Woerpel and Balloun 1964 in NAS 1980). NAS (1980) suggests that the maximum tolerable level of dietary iron 1,000 ppm for poultry.

Mammals

No information on the toxic effects of iron on wildlife species were found in the literature, but a number of investigations into its effects on domestic animals were available.

Acute effects of hepatic congestion due to iron were seen in rabbits at a concentration of 750 mg ferrous sulfate/kg body weight (Luongo and Bjornson 1954 in NAS 1980). While this concentration caused death in 24-48 hours, 2,000 mg ferrous sulfate/kg body weight caused death in all rabbits within a few hours of administration. The acute oral LD_{50} in mice is 306 mg/kg for ferrous sulfate and 429 mg/kg for ferrous gluconate and 200 mg/kg for ferric chloride (NAS 1980). Iron concentrations of 3,000 ug/g in feed resulted in no effect on swine after 56 days, however, at 4,000 ug/g reduced growth was observed (EPA 1985). Iron at exceedingly high concentrations has been reported to be toxic to livestock and to interfere with the metabolism of phosphorous (NAS 1974 in EPA 1976). Cattle fed 477 ug/g iron for 84 days showed a slight decrease in weight gains; at 1,677 ug/g a significant decline in growth rate was observed (EPA 1985).

Plants

Iron can have a variety of detrimental effects on terrestrial plants. Precipitated iron may complex phosphorous and molybdenum making them less available as plant nutrients. In alkaline soils, iron may be so insoluble as to result in chlorosis, an iron deficiency. Wheeler et al. (1985) showed, through field and laboratory experiments, that in addition to producing indirect effects on plants by interacting with other nutrients, iron also may be directly toxic to some plant species.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1976. Quality Criteria for Water. Office of Water and Hazardous Materials. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Environmental Profiles and Hazard Indices for Constituents of Municipal Sludge: Iron. Office of Water Regulations and Standards. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Quality Criteria for Water 1986. Office of Water Regulations and Standards. Washington D.C. EPA 440/5-86-001.

LEHNERTZ, C. 1989. Clear Creek Basin-1989: The Effects of Mining on Water Quality and the Aquatic Ecosystem--Draft. Colorado Division of Wildlife. Denver, Colorado.

NATIONAL ACADEMY OF SCIENCES (NAS). 1980. Mineral Tolerance of Domestic Animals. National Research Council, Subcommittee on Mineral Toxicity in Animals. Washington, D.C.

SMITH, E.J., CHEM, K.Y., HODSON, P.V., PEARCE, J.B., and SWANSON, D.L. 1979. Iron. In: Thurston, R.V., Russo, R.C., Fetterolf, C.M., Jr., Edsall, T.A., and Barber, Y.M., Jr. (eds.). A Review of the EPA Red Book: Qualtiy Criteria for Water. American Fisheries Soc. Bethesda, Maryland. Pp. 121-125.

WHEELER, B.D., AL-FARRAJ, M.M., and COOK, R.E.D. 1985. Iron toxicity to plants in base-rich wetlands: Comparative effects on the distribution and growth of Epilobium hirsutum L. and Juncus subnodulosus Schrank. New Phytol. 100:653-669.

AQUATIC TOXICITY

The primary mechansism of acute toxicity of lead to freshwater orgamisms is unknown. Lead inhibits the formation of heme, adversely affects blood chemistry, and accumulated in hematopoietic organs of aquatic organisms (Eisler 1988). Lead toxicity decreases with increasing water hardness; as water hardness increases, lead precipitates, resulting in lower bioavailability to aquatic organimsms. Below, toxicity to aquatic invertebrates, fish, and plants is discussed.

Invertebrates

Acute toxicity studies reported by Eisler (1988) for freshwater invertebrates range from 28.4 ug/L for the amphipod Gammarus pseudolimnaeus to 117,000 ug/L for the snail Viviparus ater. A lead concentration of 70 ug/L resulted in immobilization of the protozoan Uronema sp. in a 20 hour test (EPA 1985). 96-Hour LC_{50}s for the water flea Daphnia magna were 612, 952, and 1,910 ug/L at water hardnesses of 54, 110, 152 mg/l CaCO3 (EPA 1985). Data reported by Fraser (1980 in Eisler 1988) indicate that some organisms may be less sensitive to toxic effects of lead following exposure to low levels of the metal. For previously unexposed and pre-exposed isopods (Asellus aquaticus), acute toxicity was reported at 330,000 and 794,000 ug/L, respectively.

Acute toxicity to saltwater species from exposure to inorganic lead occurs at concentrations ranging from 476 ug/L for larvae of the blue mussel Mytilus edulis to >500,000 ug/L for adult blue mussel (Maddock and Taylor 1980 in Eisler 1988). Acute toxicity to the amphipod Ampelisca abdita, softshell clam Mya arenaria, and shrimp Crangon crangon is reported at 547, 27,000, and 375,000 ug/L, respectively (EPA 1985, Eisler 1977, and Maddock and Taylor 1980, all as in Eisler 1988).

The data indicate that organic forms of lead such as tetraethyl and tetramethyl lead are more toxic to saltwater invertebrates than inorganic forms (Pb^{2+}); comparable data regarding lead speciation and toxicity to freshwater invertebrate species were not available (Eisler 1988). Acute toxicity from exposure to organic forms of lead is reported at concentrations ranging from 100 ug/L for the blue mussel to 8,800 ug/L for the shrimp C. crangon (Maddock and Taylor 1980 in Eisler 1988).

Chronic toxicity is reported in freshwater invertebrates at concentrations ranging from 19 ug/L for the snail Lymnaea palustris (Borgmann et al. 1978 in Eisler 1988) to 181 ug/L for D. magna (Berglind et al. 1985 in Eisler 1988). Only one study was available reporting chronic effects of lead on saltwater species. Adverse effects were noted at 37 ug/L but not at 17 ug/L for the mysid Mysidopsis bahia (EPA 1985 in Eisler 1988).

Fish

Acute toxicity in freshwater fish is reported at inorganic lead concentrations ranging from 300 ug/L, which was 100% lethal for the three-spined stickleback (Gasterosteus aculeatus) to 2,800 ug/L for the smallmouth bass (Micropterus dolomieui)(Wong et al. 1978, Coughlan et al. 1986 in Eisler 1988). In a study using fathead minnows (Pimephales promelas), acute toxicity is reported at 6,500 ug/L in soft water (20 mg/L as CaCO3) and 460,000 ug/L in very hard water (360 mg/L as CaCO3)(NRCC 1973 in Eisler 1988). As was noted with invertebrates, fish species are more sensitive to organic forms of lead than inorganic. An acute value of 3.5 ug/L tetramethyl lead is reported for rainbow trout (Salmo gairdneri)(Wong et al. 1981 in Eisler 1988).

Saltwater acute values of 315 ug/L inorganic lead for the mummichog Fundulus heteroclitus (EPA 1985) and 180,000 ug/L for the plaice Pleuronectes platessa (Maddock and Taylor 1980 in Eisler 1988) are reported in Eisler (1988). Toxicity of organic forms of lead increase with increasing alkylation; LC_{50}s for plaice ranged from 50 ug/L for tetramethyl lead to 300,000 ug/L for dimethyl lead (Maddock and Taylor 1980 in Eisler 1988).

Chronic toxicity to freshwater species occurs at inorganic lead concentrations ranging from 7.6 ug/L in rainbow trout to 483 ug/L for northern pike (Esox lucius) (Davies et al. 1976 in EPA 1980; Demayo et al. 1982 in Eisler 1988). Chronic values of 120, 136, and 253 ug/L are reported for bluegill sunfish (Lepomis macrochirus), channel catfish (Ictalurus punctatus), and white sucker (Catostomus commersoni), respectively (EPA 1980; Demayo et al. 1982 in Eisler 1988). Chronic toxicity studies for organic lead, and chronic toxicity for saltwater fish species, were not located in the literature.

Amphibians

A 30-day LC_{50} of 105,000 ug/L is reported for leopard frog Rana pipiens (Eisler 1988). In an 8-day toxicity test using marbled salamander (Ambystoma opacum), increased mortality was noted at 1,400 ug/L (EPA 1985 in Eisler 1988). Yeung (1978 in Eisler 1988) reported that 500 ug/L caused a decreased rate of development of Rana utricularia tadpoles.

Plants

Irmer et al. (1986 in Eisler 1988) reported a 3-hour EC_{50} based on reduced photosynthesis of 1,000 ug/L in the freshwater alga Chlamydomonas reinhardii. In a 12-day study using the saltwater diatom Skeletonema costatum, no effects were noted at 0.05 ug/L, but 100% inhibition of growth was observed at 10 ug/L. LC_{50} values for organic lead compounds ranged from 100 to 1,300 ug/L for the alga Phaeodactylum tricornutum, while the LC_{50} of inorganic lead to this species was >5,000 ug/L (Eisler 1988).

Sediment Toxicity

Barrick and Beller (1989) reported sediment values in terms of apparent effect thresholds (AETs). They report AETs of 450 mg/kg (in dry weight) for benthic invertebrates, and 660 mg/kg for both amphipods and oysters.

Bioaccumulation

Bioconcentration factors in freshwater invertebrates range from 1,000 to 9,000 following 28 days of exposure (Demayo et al. 1982 in Eisler 1988). Spehar et al. (1978 in EPA 1985) reported a 28-day whole body bioconcentration factor (BCFs) of 1,120 for stoneflies. BCFs in saltwater invertebrates range from 17.5 for the Quahog clam Mercenaria mercenaria in a 56 day study (Pringle et al. 1968 in EPA 1980) to 6,600 for the American oyster Crassostrea virginica in a 140 day study (Schulz-Baldes 1972 in Eisler 1988). A whole-body BCF of 45 was reported for bluegills (Atchison et al. 1977 in EPA 1980). BCFs for organic forms of lead ranged from 1 to 130 in plaice exposed for 96 hours (Maddock and Taylor 1980 in Eisler 1988). A 28-day BCF range of 26,000 to 92,000 was reported by Vighi (1981 in Eisler 1988) for the freshwater algae Chlamydomonas reinhardii. Wong et al. (1981 in Eisler 1988) reported a 7-day whole body BCF for rainbow trout of 726 for tetramethyl lead.

Criteria

Ambient water quality criteria state that freshwater organisms should not be adversely affected if the 4-day average concentration does not exceed $e^{(1.266[\ln(\text{hardness})]-4.661)}$ and if the 1-hour average concentration does not exceed $e^{(1.266[\ln(\text{hardness})]-1.416)}$ in ug/L more than once every three years (EPA 1986). Using an average water hardness of 100 mg/L, the 4-day and 1-hour criteria correspond to 3.2 and 83 ug/L, respectively.

For protection of saltwater organisms, EPA (1986) has established a continuous concentration criterion for lead of 5.6 ug/L and a 1-hour average concentration criterion of 150 ug/L, not to be exceeded more than once every 3 years.

TERRESTRIAL TOXICITY

Birds

The majority of information on lead toxicity in birds is on body burdens in waterfowl that have ingested spent lead shot and died. However, limited dose-response information is available for a few species. Neurological effects were observed within 24 hours of dosing in mallard ducks that had ingested and absorbed lead shot for a total intake of 423.8 mg/kg bw (Mautino and Bell 1987). In 1-day-old American kestrels fed 125 or 625 mg/kg bw lead for 10 days, growth was seriously depressed by day 6, and hematocrit values were significantly depressed by day 10 (Hoffman et al. 1985). Forty percent of the birds receiving 625 mg/kg lead died within 6 days. No effects were observed in kestrels exposed to 25 mg/kg bw. American kestrels fed 10 or 50 ppm lead in the diet for 7 months experienced no toxic effects with respect to survival, egg laying, initiation of incubation, or egg shell thickness (Pattee 1984). Assuming a kestrel weighs 0.11 kg and ingests 26 g of food each day (USDA 1988), these dietary levels correspond to dosages of 2.4 and 12 mg/kg bw. The 12 mg/kg NOAEL is the highest chronic NOAEL for birds based on the studies reviewed and is used to develop a toxicity criterion for this assessment.

Mammals

Lead adversely affects survival, growth, development, and metabolism of most animal species. Acute toxicosis is often characterized by impairment of the central nervous system, the gastrointestinal tract and the muscular and hematopoietic systems and death. Most data on the toxic effects of lead in mammals are from studies with laboratory and domestic species. In wildlife species, subacute exposure of rabbits (Lepus sp.) to lead dosages of >0.005 mg/kg body weight (bw) reduced blood ALAD activity, and dosages of 0.03 mg/kg bw resulted in elevated blood levels (Eisler 1988). A toxicity criterion of 0.0005 mg/kg is derived by applying an uncertainty factor of 10 (for using a less than chronic study) to the LOAEL of 0.005 mg/kg. An additional uncertainty factor of 10 for extrapolation of a NOAEL from a LOAEL is not used here because the effects (altered blood ALAD) is regarded as minimally severe.

Plants

Lead inhibits plant growth, reduces photosynthesis and reduces mitosis and water absorption (Eisler 1988). Inhibition of photosynthesis is attributed to the blocking of protein sulfhydryl groups and to changes in phosphate levels in cells (Eisler 1988). Lead levels of approximately 500 mg/kg soil reduced pollen germination by greater than 90% in two species of weeds (Eisler 1988). Normal germination rates were observed at soil lead levels of 46 mg/kg but other adverse effects were

observed at lead levels of 12 to 312 mg/kg soil (Eisler 1988). The 12 mg/kg LOAEL is selected for this assessment.

REFERENCES

BARRICK, R.C. AND BELLER, H.R. 1989. Reliability of sediment quality assessment in Puget Sound. In: Oceans '89 Proceedings. Inst. of Electrical and Electronic Engineers. Piscataway, New Jersey. Publication No. 89CH2780-5.

EISLER, R. 1988. Lead Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Department of the Interior, Fish and Wildlife Service. Contaminant Hazard Reviews Report No. 14. Biological Report 85(1.14).

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Lead. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-80-057.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Ambient Water Quality Criteria for Lead-1984. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-84-027.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Quality Criteria for Water-1986. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-86-001.

HOFFMAN, D.J., FRANSON, J.C., PATTEE, O.H., BUNCK, C.M., AND ANDERSON, A. 1985. Survival, growth, and accumulation of ingested lead in nestling American kestrels (Falco sparverius). Arch. Environ. Contam. Toxicol. 14:89-94.

MAUTINO, M. and BELL, J.U. 1987. Hematological evaluation of lead intoxication in mallards. Bull. Environ. Contam. Toxicol. 38:78-85.

PATTEE, O.H. 1984. Eggshell thickness and reproduction in American kestrels exposed to chronic dietary lead. Arch. Environ. Contam. Toxicol. 13:29-34.

U.S. DEPARTMENT OF AGRICULTURE (USDA). 1988. Draft Environmental Impact Statement-Vegetation Management in the Coastal Plain/Piedmont. Vol. II-Appendices.

MANGANESE

AQUATIC TOXICITY

Manganese is an essential nutrient for animals, and it is important to growth and reproduction (NAS 1980). Manganese toxicity can decrease with increased water hardness (Davies 1980, Lewis et al. 1979). Toxicity can also be affected by pH (Lewis et al. 1979). The permanganate forms of manganese are more toxic than the manganous salts (Doudoroff and Katz 1953). However, permanganates are not persistent in aquatic environments and they are rapidly converted to relatively nontoxic substances through the oxidation of organic materials (EPA 1986). Most of the available toxicity information is for manganous salts. Antagonism with nickel toxicity has been reported, as well as synergistic effects with some other metals (Lewis et al. 1979). Limited information is available on the toxicity of manganese to freshwater aquatic organisms.

Invertebrates

Daphnia sp. had 16 percent reproductive impairment after three weeks of exposure to 4.1 mg/L manganese (Biesinger and Christensen 1972 in Lewis et al. 1979). A concentration of 1 mg/L was lethal to river crayfish (McKee and Wolf 1963 in Lewis et al. 1979). Manganese was toxic to zooplankton at 5.7 mg/L after three weeks exposure (Clarke 1974 in Lewis et al. 1979). Other freshwater invertebrates that have been tested show very little sensitivity to manganese. Manganese was lethal to the aquatic worm Tubifex tubifex at 700 mg/L after 7 days of exposure (AQUIRE 1990). Midge larvae (Chironomus thummi) died after 7 days of exposure to 1,000 mg/L manganese (AQUIRE 1990). The caddisfly Anabolia nervosa is the least sensitive to manganese of all the freshwater invertebrates tested. Death occurred after 7 days exposure to 2,000 mg/L manganese (AQUIRE 1990). Manganese was toxic to aquatic beetles after two weeks of exposure to 549 mg/L (Wooldridge and Wooldridge 1967 in Lewis et al. 1979).

Limited information is available on the effects of manganese on marine invertebrates (EPA 1986). The 48-hour LC_{50} for the American oyster (Crassostrea virginica) is 16 mg/L. Fifty percent inhibition of the enzyme glutamine synthetase occurred at 610 mg/L in the marine polychaete worm Eudistylia vancouveri (Brown 1976 in AQUIRE 1990). Watling (1983 in AQUIRE 1990) reported growth reduction in Pacific oyster (Crassostrea gigas) larvae after 14 days exposure to 10 ug/L manganese.

Fish

As previously indicated, the permanganates are more acutely toxic than the manganous salts. In freshwater, eels (Anguilla japonica) have survived more than 2,700 mg/L (as manganese chloride) for 50 hours, but were killed in approximately 8 hours when exposed to 4.1 mg/L manganese as permanganate (Doudoroff and Katz 1953). Goldfish were killed in hard water in 12 to 18 hours when exposed to 3.5 mg/L manganese as permanganate (Doudoroff and Katz 1953). Manganese as permanganate was lethal to brook trout within 24 hours at a concentration of 6.25 mg/L (Doudoroff and Katz 1953).

Toxicity information for the manganous salts is presented below. The LC_{50} for Orizias sp., a freshwater fish, is 6,045 mg/L (as manganese chloride) (McKee and Wolf 1963 in Lewis et al. 1979). Manganese was lethal to sticklebacks within 24 hours at a

concentration of 300 mg/L (Gasterosteus aculeatus). Davies (1980) reported that the acute toxicity of manganese to fish decreases with increased water hardness, as well as increased fish size. The 96-hour LC_{50} for rainbow trout in soft water (hardness = 36 mg/L) was 14.5 mg/L; the 144-hour LC_{50} was 5.7 mg/L (Davies 1980). England and Cummings (1971 in Lewis et al. 1979) reported a 96-hour LC_{50} in young rainbow trout of 16 mg/L manganese. Manganese at 100 mg/L was not lethal to brook trout after 7 days of exposure; at 600 mg/L deaths occurred within 23 hours (Schweiger 1957 in AQUIRE 1990). Carp showed no mortality at 600 mg/L after 7 days, but died within 24 hours at a concentration of 2,000 mg/L (Schweiger 1957 in AQUIRE 1990). Tench (Tinca tinca) were quite tolerant of manganese. No mortality occurred at 1,200 mg/L manganese after 7 days. Lethality occurred at 1,800 mg/L after 96 hours (Schweiger 1957 in AQUIRE 1990).

The 28-day LC_{50} for rainbow trout is 2.91 mg/L (Pickering et al. 1983). Exposure of rainbow trout eggs to 0.37-4.0 mg/L manganese for 29-days resulted in 5-23 percent increased mortality (Lewis 1976 in Lewis et al. 1979). Fry exposed for 72 hours, to these same concentrations, showed no increased mortality.

No toxicity values from saltwater toxicity studies were identified in the literature reviewed for this assessment.

Carp, goldfish, and sticklebacks are the only fish species associated with Aberdeen for which toxicity information is available. Toxicity values for these species are cited above.

Amphibians

No information on the toxicity of manganese to amphibians was identified in the literature reviewed for this assessment.

Plants

No information on the toxicity of manganese to aquatic plants was reported by EPA (1976, 1986). An EC_{50} (based on growth) of 31 mg/L has been reported from a 4-day study with duckweed (Lemna minor) (Wang 1986 in AQUIRE 1990). The species composition of freshwater phytoplankton populations was altered at 0.1 mg/L manganese (Lewis et al. 1979). Concentrations of 0.2 and 0.3 mg/L have been reported to be toxic to some species of marine algae (Guseva 1937, 1939 in Lewis et al. 1979).

Sediment Toxicity

No information was located on the toxicity of manganese in sediments.

Bioaccumulation

BCFs reported by Rodgers et al. (1980 in AQUIRE 1990) for freshwater molluscs range from 800-1,300 for the pouch snail (Physa sp.) to 1,800 (viscera) for the Asiatic clam (Corbicula fluminea). BCFs of 160,000 and 350,000 were reported for the freshwater mussel Unio pictorum and swan mussel Anodonta cygnea, respectively (Salanki et al. 1982 in AQUIRE 1990). BCFs of 88 and 28 have been reported for oligochaetes and insects,

respectively (Greichus et al. 1978 in AQUIRE 1990). A BCF of 3,900 was reported for chironomid larvae (Salanki et al. 1982 in AQUIRE 1990).

BCFs up to 12,000 have been reported for saltwater molluscs (EPA 1986). Van As et al. (1973 in AQUIRE 1990) reported BCFs of 450, 1,400, and 2,800 for three species of marine molluscs from around the Cape of Good Hope. In the same study, a BCF of 370 was reported for the Cape spiney lobster (Jasus lalandei).

Diet is apparently more important than ambient water in the accumulation of manganese in fish (Eisler 1981). The only whole body BCF identified for manganese in freshwater fish is a value of 84 obtained from a study in an African lake (Greichus et al. 1978 in AQUIRE 1990). Salanki et al. (1982 in AQUIRE 1990) determined BCFs in gill, liver, and kidney tissues of bream (Abramis brama) and pike perch (Stizostedion lucioperca). In bream, BCFs were 600 for gill tissues, 240 for liver, and 170 for kidney. Pike perch had BCFs of 260 in gill, 210 in liver, and 70 in kidney.

Eisler (1981) indicates that in marine fish manganese concentrations rarely exceed 0.5 mg/kg (fresh weight) in muscle, 2.0 mg/kg in liver, and 9.0 mg/kg in whole fish. In general, plankton-feeding fish accumulate manganese more than benthos-feeding fish (Petkevich 1967 in Eisler 1981). Van As et al. (1973 in AQUIRE 1990) reported manganese BCFs for eleven species of marine fish around the Cape of Good Hope. The BCF values ranged from 120 to 1,800; the arithmetic mean of the BCFs for the eleven species was approximately 560.

Criteria

No ambient water quality criteria for the protection of freshwater aquatic life have been established for manganese by EPA. McKee and Wolf (1963 in EPA 1976) reported tolerance values of 1.5 mg/L to more than 1,000 mg/L and they suggest that 1 mg/liter is not deleterious to fish and aquatic life. Davies and Goettl (1977 in Lewis et al. 1979) also indicate that 1.0 mg/L is protective of freshwater species. Dawson (1974 in Lewis et al. 1979) has recommended a criterion of 0.1 mg/L. A criterion of 100 ug/L is recommended by EPA for marine waters based on human health concerns from residues in shellfish (EPA 1986).

TERRESTRIAL TOXICITY

Limited data are available relating to toxic effects of manganese in wildlife. Manganese is an essential mineral for birds and mammals (EPA 1986). It is a cofactor for a number of enzymatic reactions (Klaassen et al. 1986). The recommended minimum dietary level for poultry is 60 ppm (Puls 1988). Dietary manganese levels of 40-200 ppm are considered adequate for most livestock (Puls 1988). Toxic levels of manganese can cause decreased feed intake, decreased growth, reduced hemoglobin, and even death.

Birds

A NOEL of 4,080 ppm dietary manganese was determined in a study with young turkeys exposed for 21 days; decreased growth occurred at 4,800 ppm (Vohra and Kratzer 1968 in NAS 1980). The dietary concentration of 4,080 ppm corresponds to an approximate dosage of 510 mg/kg bw assuming a dietary conversion rate of 0.125 mg/kg bw per 1 ppm in the diet (Lehman

1954). There were 10 birds per treatment. A 4,080 ppm level in the diet is approximately equivalent to a dose of 510 mg/kg bw, based on a conversion factor for young chickens in Lehman (1954). Chickens also showed adverse effects at 4,800 ppm dietary manganese; reduced growth and 52 percent mortality occurred (Heller and Penquite 1937 in NAS 1980).

Mammals

Sheep had reduced feed intake at 9,000 ppm dietary manganese (Puls 1988).

The Maximum Tolerable Levels for dietary manganese recommended by NAS (1980) are 1,000 ppm for cattle (15 mg/kg bw) and sheep (40 mg/kg bw), 400 ppm (16 mg/kg bw) for swine, and 2,000 ppm (250 mg/kg bw) for poultry. Puls (1988) recommends a maximum concentration of 0.05 mg/L (50 ug/L) in drinking water for livestock and poultry.

Plants

Manganese is an essential micronutrient for plants and functions in many enzyme systems (EPA 1986, Kabata-Pendias and Pendias 1984). Soil concentrations (dry weight) of manganese of 1,500 and 3,000 mg/kg have been reported as phytotoxic by Kabata-Pendias and Pendias (1984). EPA (1986) indicates that a manganese concentration slightly less than 1 mg/L in irrigation water may be toxic to plants if the soil pH is less than 6.0.

REFERENCES

AQUATIC INFORMATION RETRIEVAL (AQUIRE). 1990. Computerized database. Chemical Information System, Inc.. Baltimore, Maryland.

DAVIES, P.H. 1980. Investigations on the Toxicity of Metals to Fish. Colorado Division of Wildlife. Ft. Collins, Colorado. Water Pollution Studies: Job 2. Job Progress Report F-33-R-15.

DOUDOROFF, P. and KATZ, M. 1953. Critical review of literature on the toxicity of industrial wastes and their components to fish. II. The metals, as salts. Sewage and Industrial Wastes. 25(7):802-839.

EISLER, R. 1981. Trace Metal Concentrations in Marine Organisms. Pergamon Press. New York, New York.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1976. Quality Criteria for Water. Office of Water Planning and Standards. Washington, D.C. EPA 440/9-76-023.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Quality Criteria for Water 1986. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-86-001.

KABATA-PENDIAS, A. and PENDIAS, H. 1984. Trace Elements in Soil and Plants. CRC Press. Boca Raton, Florida.

KLAASSEN, C.D., AMDUR, M.O. and DOULL, J. 1986. Casarett and Doull's Toxicology, The Basic Science of Poisons. Third Edition. Macmillan Publishing Company. New York, New York.

LEHMAN, A.J. 1954. Appraisal of the study of chemicals in food, drugs, and cosmetics. U.S. Association of Food and Drug Officials. 18:66.

LEWIS, M.A., EVANS, D.W., and WIENER, J.G. 1979. Manganese. In: Thurston, R.V., Russo, R.C., Ferrerolf, C.M. Jr., Edsall, T.A., and Barber, Y.M., Jr. (eds.). 1979. A Review of the EPA Red Book: Quality Criteria for Water. American Fisheries Society. Bethesda, Maryland. Pp. 137-144.

NATIONAL ACADEMY OF SCIENCES (NAS). 1980. Mineral Tolerance of Domestic Animals. National Research Council, Subcommittee on Mineral Toxicity in Animals. Washington, D.C.

PICKERING, Q.H., HUNT, E.P., PHIPPS, G.L., ROUSH, T.H., SMITH, W.E., SPEHAR, D.L., STEPHENS, C.E., and TANNER, D.K. 1983. Effects of pollution on freshwater fish and amphibians. JWPCF. 55(6):840-862.

PULS, R. 1988. Mineral Levels in Animal Health. Sherpa International. Clearbrook, British Columbia.

MERCURY

AQUATIC TOXICITY

The aquatic toxicity of mercury has been reviewed by EPA (1985) and Eisler (1987). Mercury is considered one of the most toxic of the heavy metals (EPA 1985). It has no known function in biological systems (Eisler 1987). Elemental mercury and mercury (II) can be converted into methylmercury compounds by aerobic and anaerobic bacteria in both freshwater and saltwater environments. Methylmercury compounds are much more toxic than inorganic mercury forms. Acute toxicity in fish causes loss of equilibrium, increased respiration, and sluggishness (Eisler 1987). Chronic toxicity can result in emaciation, loss of coordination, and lesions on the brain (Eisler 1987). Alkalinity, hardness, ascorbic acid, chloride, dissolved oxygen, pH, organic complexing agents, sediment, and temperature all probably affect the toxicity and bioaccumulation of mercury (EPA 1985). In addition, increased salinity has been shown to reduce the toxicity of mercuric chloride to the freshwater fish Oreochromis mossambicus (Prakasam 1989). At 0.66 mg/L mercury, 50 percent mortality occurred at 0 percent salinity, whereas at 30-50 percent salinity no mortality occurred. There is evidence to indicate that fish can develop some tolerance to mercury exposure. Killifish (Fundulus sp.) previously exposed to mercury are more tolerant than are unexposed fish (Weis 1984).

Invertebrates

Freshwater invertebrate acute toxicity values for mercury(II), for 22 genera, range from 2.2 ug/L (Daphnia pulex) to 2,000 ug/L (stoneflies [Order Plecoptera], mayflies [Order Ephemeroptera], and caddisflies [Order Trichoptera]). Acute toxicity data for organomercury compounds is limited. Based on available information, organomercury compounds are 4 to 31 times more toxic than mercury (II) (EPA 1985). Methylmercury is the most toxic form with respect to chronic aquatic toxicity. Daphnia magna has a chronic value of less than 0.04 ug/L (methylmercury) (EPA 1985). For mercury (II) the lowest available chronic value is 0.96 ug/L for Daphnia magna (EPA 1985).

Acute toxicity values for mercury (II) for 25 genera of saltwater invertebrates range from 3.5 ug/L for mysid shrimp to 400 ug/L for softshell clam (Mya arenaria). No saltwater acute values are available for methylmercury. The only chronic value for saltwater invertebrates is a value of 1.131 ug/L for a mysid exposed to mercury (II) (EPA 1985).

Fish

Freshwater fish acute toxicity values for mercury(II) range from 30 ug/L (guppy, Poecilia reticulata) to 1,000 ug/L (Mozambique tilapia). Methylmercury is more toxic than the inorganic form with respect to chronic toxicity. Brook trout, Salvelinus fontinalis has a chronic value of less than 0.07 ug/L (EPA 1985). For mercury (II) the chronic value for fathead minnow (Pimephales promelas) is 0.26 ug/L (EPA 1985).

Acute toxicity values for saltwater fish range from 36 ug/L for spot (Leiostomus xanthurus) to 1,678 ug/L for winter flounder (Pseudopleuronecthes americanus) (EPA 1985). No chronic values are available for saltwater fish.

Some toxicity values are available for fish species associated with the Aberdeen Proving Ground. For freshwater fish, the species mean acute values for mercury (II) are mosquitofish (Gambusia affinis) -- 180 ug/L and bluegill (Lepomis macrochirus) -- 160 ug/L (EPA 1985). Saltwater species mean acute values for mercury (II) are 1,678 ug/L for winter flounder, 115.7 ug/L for Atlantic silverside (Menidia menidia), 71 ug/L for tidewater silverside (Medinia beryllina), and 36 ug/L for spot (EPA 1985). Spot (Leiostomus xanthurus) was the most sensitive saltwater fish species tested and it ranked 11th in sensitivity out of 29 saltwater species of fish and invertebrates.

Amphibians

Acute toxicity values for inorganic mercury have been reported by Birge et al. (1987 in Eisler 1987) for the embryo-larva stage of a number of amphibian species. The available 96-hr LC_{50}s in ug/L are as follows: narrow-mouthed toad (Gastrophryne carolinensis) 1.3, treefrog (Hyla spp.) 2.4-2.8, leopard frog (Rana pipiens) 7.3, cricket frog (Acris spp.) 10.4, four species of anurans (frogs and toads) 36.8-67.2, and marbled salamander (Ambystoma opacum) 107.5.

Plants

Aquatic plants appear to be less sensitive to mercury than the most sensitive fish or invertebrates. Toxicity values for mercury(II) for freshwater algae reported in EPA (1985) range from 5 ug/L to 1,030 ug/L (based on eight studies). The lowest acute value of 5 ug/L is for the alga Microcystis aeruginosa based on incipient inhibition at eight days of exposure (EPA 1985). LC_{50}s between 100-1,000 ug/L have been reported for the alga Chlorella vulgaris (EPA 1985). The only value available for an aquatic macrophyte is a 32-day EC_{50} (based on root weight) of 3,400 ug/L for Eurasion watermilfoil (Myriophyllum spicatum) (EPA 1985). Three toxicity values for methylmercury for freshwater plants are reported in EPA (1985). The lowest value is a 15-day EC_{50} (based on growth) of 0.8-4.0 ug/L for the alga Chlorella vulgaris. EC_{50}s (based on growth) of 2.8 ug/L and 6.0 ug/L were reported for the alga Anabaena flos-aquae (EPA 1985).

Toxicity values for saltwater plants are in the same range as those reported for freshwater plants. Photosynthesis was affected in a saltwater alga species at a mercury (II) concentration of 10 ug/L (EPA 1985). The 5-day EC_{50} (based on growth) for a diatom was 10 ug/L. Growth EC_{50} values for seaweed and kelp ranged from 45-160 ug/L (EPA 1985).

Sediment Toxicity

Some information is available on the toxicity of mercury-contaminated sediments to aquatic organisms. Pavlou and Weston (1983 in Pavlou 1987) reviewed proposed limits for mercury in sediments and found reported values of 0.3 to 1 mg/kg (dry weight). Using the equilibrium partitioning approach, Pavlou and Weston estimated a safe level for mercury in marine sediments of 0.006 mg/kg (dry weight, assuming 2 percent organic carbon in sediments). Birge et al. (1977 in Birge et al. 1987) reported statistically significant mortality in rainbow trout (Salmo gairdneri) (early eyed-egg stage thru 10-days post-hatch) exposed to mercury-enriched sediment at a measured concentration of 1.050 mg/kg (dry weight). Survival was 45 percent at this concentration, compared to 94 percent survival in the control (0.052 mg/kg mercury). The mercury concentration of the

overlying water column was 0.15 ug/L. Based on their results, Burge et al. (1977 in Birge et al. 1987) estimated a threshold concentration of mercury in sediments of 0.8 mg/kg.

Bioaccumulation

BCFs for the cladoceran Daphnia magna derived from 21-day flow through studies are 23,861 for inorganic mercury and 706,703 for organic mercury (Biesinger et al. 1982). Mercury concentrations in crayfish appear to be a useful indicator of mercury contamination in river systems according to Sheffy (1978). The pattern of mercury concentrations in crayfish (Order Astacidae) was very similar to the distribution of mercury in the sediments of the Wisconsin River. For one sampling station where the sediment mercury concentration was approximately 25 mg/kg, the concentration in crayfish (whole body) was 1.72 mg/kg wet weight.

For the marine copepod Acartia clausi the 24-hour BCFs are 14,360 for inorganic mercury and 181,000 for organic mercury (Hirota et al. 1983 in Eisler 1987).

Whole-body BCF values for rainbow trout are 7,000 for inorganic mercury (based on a 30-day test) (Ribeyre and Boudou 1984 in Eisler 1987) and 85,700 (based on a 75-day test) (Niimi and Lowe-Jinde 1984 in EPA 1985). BCFs for muscle are substantially less than those for whole-body. Ribeyre and Boudou (1984 in Eisler 1987) reported a value for inorganic mercury of 2,300 for muscle of rainbow trout (30-days exposure). A muscle-BCF of 11,000 - 33,000 was reported for organic mercury in brook trout (273-days exposure) (McKim et al. 1976 in EPA 1985).

Marine algae are reported to concentrate inorganic mercury up to 100 times the level in seawater (Goldwater 1971 in Holderness et al. 1975).

Criteria

The freshwater ambient water quality criteria for mercury is 2.4 ug/L for acute exposure and 0.012 ug/L for chronic exposures (EPA 1986). The acute and chronic marine criteria are 2.1 ug/L and 0.025 ug/L, respectively (EPA 1986). It is important to note that the freshwater and saltwater chronic AWQCs for mercury are based on protection of humans that may consume fish rather than on chronic aquatic toxicity values, because the Final Residue Values are lower than the Final Chronic Values. The FDA action level for mercury in fish is 1 ppm. The freshwater chronic criterion of 0.012 ug/L was derived using a BCF of 81,700 based on methylmercury in fathead minnows (Olson et al. 1975 in EPA 1986). The saltwater chronic criterion of 0.025 ug/L is based on a BCF of 40,000 for methylmercury in the eastern oyster (Kopfler 1974 in EPA 1986). These criteria may be over-protective of most aquatic life because the criteria are based on concerns about tissue residues rather than on toxic effects.

TERRESTRIAL TOXICITY

The toxicity of mercury to terrestrial animals has been reviewed by Eisler (1987). In birds and mammals, mercury can adversely affect reproduction, growth and development, blood chemistry, metabolism, and the central nervous system. It is considered mutagenic, teratogenic, and carcinogenic (Eisler 1987).

Birds

Symptoms of mercury poisoning in birds include muscular incoordination, hyporeactivity, and withdrawal (Eisler 1987). Mercury toxicity in birds depends on the form of the element, route of administration, and species, sex, and age of the animal (Fimreite 1979 in Eisler 1987). Organic forms of mercury, such as methylmercury, are more toxic than inorganic mercury based on studies by Hill (1981 in Eisler 1987) and Hill and Soares (1984 in Eisler 1987). Adverse effects did not occur in chickens, turkeys, pheasants, and ducks given dietary levels of 3.3 ppm mercury (NAS 1980). Assuming a dietary conversion factor of 0.125 mg/kg bw per 1 ppm in the diet (Lehman 1954), this corresponds to an approximate dosage of 0.41 mg/kg bw. However, increased mercury levels in tissues were observed at dietary concentrations below this. A mercury level in drinking water of 1 mg/L did not cause adverse effects in Japanese quail. At 5 mg/L, toxicosis and death occurred (NAS 1980).

Acute oral LD_{50}s for methylmercury were 11-27 mg/kg-bodyweight (bw) in coturnix and 14.4-33.7 mg/kg-bw in Japanese quail (Coturnix japonica), whereas for inorganic mercury the acute oral LD_{50}s were 26.0-54.0 mg/kg-bw in coturnix and 31.1 mg/kg-bw in Japanese quail (Eisler 1987). Acute oral LD_{50}s based on tests with five other bird species ranged from 2.2 to 37.8 mg/kg-bw for methylmercury and 11.5 to 75.5 mg/kg-bw for ethyl mercury (Eisler 1987). The LD_{50}s (as mg/kg-bw) for mallard are 2.2-23.5 for methyl mercury, 75.7 for ethyl mercury, and 524.7 for phenyl mercury (Eisler 1987). The LD_{50} for the fulvous whistling duck (Dendrocygna bicolor) for methyl mercury is 37.8 mg/kg-body weight (Eisler 1987). In grey pheasants adverse reproductive effects were observed at doses of organomercury of 640 ug/kg body weight (McEwen et al. 1973 in Eisler 1987). Spann et al. (1972 in Eisler 1987) and Mullins et al. (1977 in Eisler) reported similar results in pheasants at similar dosages. Eisler (1987) recommends that for the protection of birds the daily dose should not exceed 640 ug/kg-bodyweight.

Mammals

In mammals, organomercury compounds are more toxic than the inorganic form. Larger mammals such as the mule deer (Odocoileus hemionus) appear to be more resistant than smaller animals such as cats, dogs, pigs, monkeys, and river otters (Lutra canadensis). This may be related to differences in metabolism and detoxication rates (Eisler 1987). The LD_{50} in mule deer is 17.88 mg/kg-bw (Hudson et al. 1984 in Eisler 1987). Dietary concentrations of 1.0 mg/kg organomercury were fatal to all exposed mink (Mustela vison) within approximately two months (Sheffy and St. Amant 1982 in Eisler 1987). At 5.0 mg/kg in the diet, all exposed mink died within 30-37 days. Increased mercury levels were noted in the kidney and liver. Mink showed signs of mercury poisoning at dietary concentrations of 1.1 mg/kg (Kucera 1983 in Eisler 1987). They had mercury concentrations in brain tissue of 7.1 to 9.3 mg/kg. Organomercury dietary concentrations of greater than 2.0 mg/kg were fatal to river otter (Kucera 1983 in Eisler 1987). Mercury at 0.48 mg/kg-bw was fatal to cattle and sheep within 7-31 days (NAS 1980).

Dairy calves were not adversely affected by 90 days of exposure to approximately 3 ppm in the diet (0.1 mg/kg-bw). At a dose of 0.2 mg/kg-bw toxic effects occurred (NAS 1980). Swine receiving a daily dose (by capsule) of 0.38 mg mercury per kg-bw showed no adverse effects after 60-90 days. At a dose of 0.76 mg/kg-bw toxic effects occurred (NAS 1980).

Yearling sheep showed adveres effects on coordination at a mercury dose of 0.22 mg/kg-bw after 40-50 days (NAS 1980).

NAS (1980) has recommended maximum tolerable dietary mercury levels for livestock and poultry of 2 ppm, for both inorganic and organic forms of mercury. Puls (1988) has recommended a maximum level of mercury in drinking water for livestock of less than 0.003 mg/L. NAS (1974) has recommended a "safe upper limit" of 0.010 mg/L for mercury in drinking water for livestock and poultry.

Plants

Mercury is not known to be readily taken up by plants. Symptoms of toxicity include stunting of seedling growth and root development, and an inhibition of photosynthesis causing yield reductions (Kabata-Pendias and Pendias 1984). Concentrations of mercury in soil (dry weight) of 0.3 to 5 mg/kg (mean of four authors = 3.1) were reported to be phytotoxic (Kabata-Pendias and Pendias 1984).

REFERENCES

BIESINGER, K.E., ANDERSON, L.E. and EATON, J.G. 1982. Chronic effects of inorganic and organic mercury on Daphnia magna: toxicity, accumulation, and loss. Arch. Env. Cont. Toxicol. 11:769-774.

BIRGE, W.J., BLACK, J.A., WESTERMAN, A.G., and FRANCIS, P.C. 1987. Toxicity of sediment-associated metals to freshwater organisms: biomonitoring procedures. In: Dickson, K.L., Maki, A.W., and Brungs, W.A. (eds.). Fate and Effects of Sediment-Bound Chemicals in Aquatic Systems. Proceedings of the Sixth Pellston Workshop, Florissant, Colorado, August 12-17, 1984. SETAC Special Publication Series, Pergamon Press. New York, New York. Pp. 199-218.

EISLER, R. 1987. Mercury Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Fish and Wildlife Service Patuxent Wildlife Research Center. Contaminant Hazard Reviews, Report No. 10. Biological Report 85 (1.10).

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Ambient Water Quality Criteria for Mercury-1984. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-84-026.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Quality Criteria for Water 1986. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-86-001.

HOLDERNESS, J., FENWICK, M.G. and LYNCH, D.L. 1975. The effect of methyl mercury on the growth of the green alga Coelastrum microporum Naeg. strain 280. Bull. Env. Cont. & Tox. 13(3):348-350.

KABATA-PENDIAS, A. and PENDIAS, H. 1984. Trace Elements in Soil and Plants. CRC Press. Boca Raton, Florida.

LEHMAN, A.J. 1954. Appraisal of the study of chemicals in food, drugs and cosmetics. U.S. Assoc. of Food and Drug Officials. Vol. 18:66.

NATIONAL ACADEMY OF SCIENCES (NAS). 1974. Nutrients and Toxic Substances in Water for Livestock and Poultry. National Research Council. Washington, D.C.

NATIONAL ACADEMY OF SCIENCES (NAS). 1980. Mineral Tolerance of Domestic Animals. National Research Council, Subcommittee on Mineral Toxicity in Animals. Washington, D.C.

PAVLOU, S.P. 1987. The use of the equilibrium partitioning approach in determining safe levels of contaminants in marine sediments. In: Dickson, K.L., Maki, A.W., and Brungs, W.A. (eds.). Fate and Effects of Sediment-Bound Chemicals in Aquatic Systems. Proceedings of the Sixth Pellston Workshop, Florissant, Colorado, August 12-17, 1984. SETAC Special Publication Series, Pergamon Press. New York, New York. Pp. 388-412.

PRAKASAM, V.R. 1989. Salinity effect on the toxicity of HgCl2 in Oreochromis mossambicus. Indian J. Env. Hlth. 31(4):339-344.

PULS, R. 1988. Mineral Levels in Animal Health. Diagnostic Data. Sherpa International. Clearbrook, British Columbia.

SHEFFY, T.B. 1978. Mercury burdens in crayfish from the Wisconsin River. Environ. Pollut. 17:219-225.

WEIS, P. 1984. Metallothionein and mercury tolerance in the killifish. Marine Environmental Research. 14:153-166.

MUSTARD GAS

AQUATIC TOXICITY

No information was found regarding the toxicity of mustard gas to aquatic organisms, or for its decomposition products.

TERRESTRIAL TOXICITY

Wildlife

No data were found concerning the toxic effects of mustard gas on wildlife species, but a limited number of studies of its effects on domestic species were located. In one study exposure to 0.001 mg/m^2, 24 hours/day, 5 days/week produced no detectable damage in dogs, rabbits, guinea pigs, rats, and mice. This was approximately 150 times the ground concentration level. Mustard agents decompose forming a variety of compounds for which toxicity is unknown. One such product is thiodiglycol, which has been found to have low toxicity to several terrestrial species (Sax 1979 in DEMIL 1988).

Plant

No plant toxicity data was found in the literature, but indirect observations during World Wars I and II showed defoliation in areas of heavy mustard gas use, and barren patches of ground where soils were contaminated during the manufacture of mustard agents.

REFERENCES

PROGRAM FOR CHEMICAL DEMILITARIZATION (DEMIL). 1988. Chemical Stockpile Disposal Program Final Programmatic Environmental Impact Statement. Program Executive Officer - Program Manager for Chemical Demilitarization. Aberdeen Proving Ground, Maryland.

AQUATIC TOXICITY

The adverse effects of nickel (Ni) in aquatic organisms include alteration of cell membranes, formation of precipitates on gills, hematological effects and reproductive impairment. Toxicity of nickel to freshwater organisms decreases with increasing water hardness.

Invertebrates

Daphnia sp. are the most acutely sensitive species tested with nickel; the genus mean acute value (hardness = 50 mg/L) is 1,500 ug/L (EPA 1986a). Mayflies (Ephemerella subvaria) are the most sensitive aquatic invertebrate, with a mean acute value of 4,636 ug/L. Mean acute values for damselfly, caddisfly, and stonefly (Acroneuria lycorias) are 21,200, 30,200, and 40,460 ug/L, respectively. The mean acute value for amphipods (Gammarus sp.) is 13,000 ug/L (EPA 1986a).

Chronic toxicity values for invertebrates are available only for a caddisfly and a cladoceran. The chronic value for the caddisfly (Clistoronia magnifica) is 128.4 ug/L at a hardness of 54 mg/L (Nebeker et al. 1984 in EPA 1986a). For Daphnia magna the chronic value at a hardness of 51 mg/L is 14.77 ug/L, and at a hardness of 105 mg/L is 123.1 ug/L (Chapman et al. manuscript in EPA 1986a).

Fish

The mean acute value for rainbow trout is 13,380 ug/L and they ranked 12th in sensitivity out of all species tested. Acute toxicity values (48-hour LC50s) for brown and brook trout are 60,290 ug/L and 54,040 ug/L, respectively (Willford 1966 in EPA 1986a). Fathead minnows are more acutely sensitive than trout to nickel; the mean acute value for this species is 8,027 ug/L (ranked 4th) (EPA 1986a).

A chronic value of less than 35 ug/L was reported by Nebeker et al. (1985 in EPA 1986a) for rainbow trout (early life-stage) at a hardness of 53 mg/L. The same author also reported chronic values of 91.15 and 240.3 ug/L for hardness values of 52 and 49 mg/L, respectively (rainbow trout, early life-stage) (EPA 1986a).

Amphibians

Some species of amphibians are quite sensitive to nickel. Embryo-larval narrow-mouthed toads (Gastrophyrne carolinensis) had 7-day EC50s (based on death and deformity) of 50 ug/L at hardness values of 95-103 and 195 mg/L (Birge 1978, Birge et al. 1979, and Birge and Black 1980 all in EPA 1986a). Embryo-larval marbled salamanders (Ambystoma opacum) were less sensitive, with an 8-day EC50 of 420 ug/L at a hardness of 93-105 mg/L (Birge and Black 1980 and Birge et al. 1978 both in EPA 1986a). Least sensitive was Fowler's toad (Bufo fowleri) with a 7-day EC50 of 11,030 ug/L (Birge and Black 1980 in EPA 1986a).

Plants

EC50's (28-days) of 340 ug/L and 2,800 ug/L, have been reported for duckweed (Lemna minor) and the macrophyte Elodea canadensis. Nickel concentrations of 50 to 5,000 ug/L have caused 40-60 percent reductions in growth in green alga. Based on available information, EPA (1986a) indicates that chronic effects may occur in plants at concentrations that result in chronic effects in animals.

Sediment Toxicity

Barrick and Beller (1989) report sediment toxicicty values in terms of apparent effect thresholds (AETs). For nickel, they report an AET of >140 mg/kg dry weight for both amphipods and benthic invertebrates.

Bioaccumulation

Information on bioconcentration in aquatic organisms is limited. Calamari et al (1982 in EPA 1986a) found a BCF of 0.8 for rainbow trout muscle, based on 180 days of exposure. Whole-body BCFs for fathead minnow range from 47 to 106 after 30 days exposure (Lind et al. manuscript in EPA 1986a).

Criteria

Water quality criteria have been developed to reflect the relationship between hardness and toxicity. The Federal EPA (1986b) ambient water quality criteria (AWQCs) for freshwater are as follows: the 4-day average (chronic) concentration is not to exceed the value given by $e^{(0.8460[\ln(\text{hardness})]+1.1645)}$ and the 1-hour average (acute) concentration is not to exceed the value given by $e^{(0.8460[\ln(\text{hardness})]+3.3612)}$. At water hardness of 100 mg/l $CaCO_3$, the corresponding AWQC values are 160 and 1,400 ug/liter, respectively. Acute and chronic AWQC for saltwater organisms are 75 and 8.3 ug/L, respectively.

TERRESTRIAL TOXICITY

Birds

No adverse effects were observed in Japanese quail (Coturnix) at up to 5,000 ppm nickel in the diet for 5 days (Hill and Camardese 1986). Chickens showed no adverse effects at 300 ppm nickel after 4 weeks. At 500 ppm decreased growth was observed (Weber and Reid 1968 in NAS 1980). In a feeding study with mallard ducklings fed 0, 200, 800, or 1,200 mg/kg dietary nickel from day 1 to day 90 of age, neurological effects were observed in the highest dose group within 14 days of dosing (Cain and Pafford 1981). The weights of the ducks in the highest dose group were significantly decreased at 28 days of age, and the weight/length ratio of females in the 800 mg/kg group were significantly different from controls at days 30 and 60. A NOEL of 200 mg/kg can be identified for this study. No information was available on nickel toxicity in raptor species or in other species at a concentration lower than the NOEL for ducks.

Mammals

Mammals have shown a low to moderate toxicity to nickel. They appear to have a mechanism which limits absorption of the element in the intestine (Gough et al. 1979). The oral LD_{50} for nickel is 136 mg/kg in mice and 116 mg/kg in rats (NAS 1980). Death and runting occurred in first and third generation rats given 5 ppm nickel in drinking water during weaning. No adverse effects were reported in cattle fed 50 ppm (approximately 0.75 mg/kg-body weight) nickel for up to 6 weeks (Odell et al. 1970c: 1971 in NAS 1980) and this is the maximum tolerable level for cattle recommended by NAS (1980). At higher levels (100 ppm) decreased food intake was observed in young cattle, and decreased growth rate occurred at 1,000 ppm (NAS 1980).

Plants

Kabata-Pendias and Pendias (1984) reviewed the literature on metal phytotoxicity and identified the total concentrations of selected metals in surface soils that were phytotoxically excessive. The arithmetic mean phytotoxic concentration for nickel, based on results from five authors, was 100 mg/kg (dry weight). USEPA (1985) reports levels of 50 mg/kg (DW) as causing reduced crop yields.

Criteria

The maximum concentration of nickel in drinking water for livestock and poultry, as recommended by NAS (1974), is 1.0 mg/L.

REFERENCES

CAIN, B.W. and PAFFORD, E.A. 1981. Effects of dietary nickel on survival and growth of mallard ducklings. Arch. Environ. Contam. Toxicol. 10:737-745.

GOUGH, L.P., SHACKLETTE, H.T. and CASE, A.A. 1979. Element concentrations toxic to plants, animals and man. Geologic Survey Bulletin #1466, U.S. Government Printing Office. Washington, D.C.

HILL, E.F. and M.B. CAMARDESE. 1986. Lethal Dietary Toxicities of Environmental Contaminants and Pesticides to Coturnix. Fish and Wildlife Technical Report 2. U.S. Department of the Interior, Fish and Wildlife Service. Washington, D.C.

KABATA-PENDIAS, A. and H. PENDIAS. 1984. Trace Elements in Soil and Plants. CRC Press. Boca Raton, Florida.

NATIONAL ACADEMY OF SCIENCES (NAS). 1980. Mineral Tolerance of Domestic Animals. Subcommittee on mineral toxicity in animals. National Research Council, Washington, D.C.

NATIONAL ACADEMY OF SCIENCES (NAS). 1974. Nutrients and toxic substances in water for livestock and poultry. National Research Council. Washington, D.C.

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1986a. Ambient Water Quality Criteria for Nickel - 1986. Office of Water Regulations and Standards, Criteria and Standards Division. Washington D.C. September, 1986. EPA 440/5-86-004.

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1986b. Quality Criteria for Water 1986. Office of Water Regulations and Standards, Washington D.C. May 1, 1986. EPA 440/5-86-001

U.S. ENVIRONMENTAL PROTECTION AGENCY, Office of Water Regulations and Standards. 1985a. Environmental Profiles and Hazard Indices for Constituents of Municipal Sludge: Nickel. June, 1985. Washington, D.C.

AQUATIC TOXICITY

Nitrate, as the nitrate ion No32-, is the physicochemically stable form of combined nitrogen in oxygenated aqueous systems. In dilute systems, nitrate is chemically unreactive (EPA 1986). EPA (1986) summarized aquatic toxicity data for nitrate/nitrites. No data regarding toxicity to invertebrate, amphibian, or aquatic plant species were presented, and none were found in additional literature searches. Fish toxicity data are summarized below.

Nitrate

Acute toxicity values are available for 5 fish species. Westin (1974 in EPA 1986) conducted 96-hour and 7-day toxicity test using chinook salmon (Oncorhyncus tshawyscha) and fingerling rainbow trout (Salmo gairdneri) in fresh and 15 °/oo saline water. LC_{50} values were similar for the two species in both types of water. 96-hour LC_{50} values were 1,310,000 and 1,360,000 ug/L nitrate nitrogen in fresh water, and 990,000 and 1,050,000 ug/L nitrate nitrogen in salt water, for chinook salmon and fingerling rainbow trout, respectively. Seven-day LC_{50} values were 1,080,000 and 1,060,000 ug/L in freshwater for salmon and trout respectively, and 900,000 ug/L for both species in saline water.

Toxicity data for 3 fish species found at APG were presented. Trama (1954 in EPA 1986) reported toxicity in bluegill sunfish (Lepomis macrochirus) exposed to two nitrate salts. 96-Hour LC_{50} values were 2,000,000 ug/L nitrate nitrogen introduced as sodium nitrate, and 420,000 ug/L nitrate nitrogen introduced as potassium nitrate. Knepp and Arkin (1973 in EPA 1986) reported no effects on growth and feeding in largemouth bass (Micropterus salmoides) and channel catfish (Ictalurus punctatus) exposed to 90,000 ug/L nitrate nitrogen.

Nitrite

Westin (1974 in EPA 1986) reported 96-hour LC_{50} values of 900 and 700 ug/L nitrite nitrogen for chinook salmon in freshwater. Smith and Williams (1974 in EPA 1976) reported 55% mortality in yearling rainbow trout exposed to 550 ug/L nitrite nitrogen for 24 hours.

Nitrite toxicity data are available for 6 species found at APG. 48-Hour and 96-hour LC_{50}s were 1,500 ug/L nitrite for mosquitofish Gambusia affinis (Wallen et al. 1957 In EPA 1986). Saeki (1965 in EPA 1986) reported no adverse effects in common carp (Cyprinus carpio) exposed to 1,800 ug/L nitrite. McCoy (1972 in EPA 1986) reported that carp and black bullhead Ictalurus melas survived for at least 48 hours at a nitrite concentration of 40,000 ug/L, and white sucker 100,000 ug/L nitrite for 48 and 36 hours, respectively.

Sediment Toxicity

No data regarding toxicity of nitrates/nitrites in sediment were found in the literature.

Bioaccumulation

No bioaccumulation data were available in the literature.

Criteria

EPA (1986) concludes that nitrate and nitrite concentrations of 90,000 ug/L and 5,000 ug/L respectively should be protective of most warmwater fish, and that nitrite concentrations below 60 ug/L should be protective of salmonid fishes. These levels either are not known to occur or would be unlikely to occur in natural surface waters, and therefore EPA does not recommend restrictive criteria.

TERRESTRIAL TOXICITY

Birds

Data regarding toxicity to wild bird species were not found in the literature. Mugler et al. (1970 in NAS 1984) reported no effect on meat color in immature turkeys exposed to 450 ppm nitrate nitrogen in water. Adams et al. (1967 in NAS 1984) reported reduced growth and mortality in turkeys exposed to 900 ppm nitrate nitrogen in water. In immature chickens exposed to 658 ppm nitrite nitrogen in the diet, decreased vitamin A in the liver and thyroid enlargement were observed (Sells and Roberts 1963 in NAS 1984).

Mammals

In mammals, nitrate can be reduced to nitrite in the gastrointestinal tract under certain conditions. In the bloodstream nitrite reacts directly with hemoglobin to produce methemoglobin, which results in impaired oxygen transport (Schmidt-Nielsen 1983).

No data regarding toxicity of nitrate to wildlife species were located in the literature. However, oral LD_{50} values of 1, 986, 310, and 332 mg/kg have been reported for rats, sheep, and cows, respectively (EPA 1985). The greater acute toxicity of nitrate in sheep and cows is apparently related to the greater conversion of nitrate to nitrite by bacteria in these ruminants.

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1976. Quality Criteria for Water. Office of Water and Hazardous Materials. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Drinking Water Criteria Document on Nitrate/Nitrite. Final Draft. Office of Drinking Water. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Quality Criteria for Water. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-86-001.

NATIONAL ACADEMY OF SCIENCES (NAS). 1984. Nutrient Requirments of Poultry. 8th Revised Edition. Chapter 9: Toxic Levels of Elements. National Academy Press. Washington, D.C.

SCHMIDT-NIELSON, K. 1983. Animal Physiology: Adaptation and Environment. Third Edition. Cambridge University Press. New York, New York.

PHTHALATES

AQUATIC TOXICITY

All chemicals referred to as phthalates are esters of phthalic acid (1,2-benzenedicarboxylic acid). Phthalates are used in the manufacturing of plastics to impart flexibility. The most widely used and extensively studied phthalate is di-2-ethylhexyl phthalate (DEHP). Limited data also exist for di-n-butyl phthalate (DBP), di-n-octyl phthalate (DOP), butylbenzyl phthalate (BBP), and dimethyl phthalate (DMP).

The mechanism of phthalate toxicity has not been identified. In fish, reduced brain levels of epinephrine were observed following exposure to BBP. These results indicate that the mechanism of acute toxicity for BBP may be through its effects on the catecholamines of the central adrenergic nervous system (Ozretich et al. 1983). Similar studies using other phthalates have not been conducted.

Veith et al. (1985) have included phthalates in the group of industrial chemicals which produce toxicity by a nonspecific mode of action called narcosis. Narcosis is a retardation of normal physiological processes caused by the absorption of foreign molecules into biological membranes. However, they also found that for phthalates, acute toxicity often is observed at concentrations below concentrations inducing narcosis. Therefore it is likely that narcosis is not the primary mode of toxic action for all of the phthalates.

When chemicals in a mixture act upon the same organ system or by a smiliar mode of action, EPA recommends that their combined toxic effect be considered additive. However, since it is not known whether the toxic mechansisms are similar among all of the phthalate esters, additivity of toxicity among the different esters cannot be assumed.

Aquatic toxicity data for the phthalate esters are summarized in Table C-1. Toxicity among different classes of organisms is discussed briefly below.

Invertebrates

Phthalate concentrations resulting in acute and chronic toxicity to invertebrates do not differ greatly. Laughlin et al. (1978) found DBP to be the most toxic to grass shrimp (Palaemonetes pugio), followed by DMP and DEHP.

Fish

In acute tests using DEHP, fish appear to be slightly less sensitive to phthalates than invertebrates. Veith et al. (1985) report that the three isomers of DOP (o,m, and p), and DEHP, are not lethal to fish at concentrations exceeding the chemicals' solubilities.

Amphibians

An EC_{50} of 150 mg DEHP/kg sediment was reported for hatchability of moorfrog (Rana arvalis) eggs exposed to DEHP in sediment. Tadpoles which hatched successfully were not adversely affected (Larsson and Thuren 1987). In tests using platanna (Xenopus laevis), 2,000 ug/L of DEHP adversely affected larval development and pigmentation of tadpoles (Dumpert and Zietz 1984).

TABLE C-1

AQUATIC TOXICITY OF PHTHALATES

CHEMICAL	ORGANISM	TYPE	DURATION	EFFECT	CONCENTRATION (ug/L)	REFERENCE
DI-N-BUTYL PHTHALATE						
--acute						
	Rainbow trout	NR	96 h	LC50	6,740	EPA 1980 (a)
	Crayfish	NR	96 h	LC50	> 10,000	Mayer & Sanders 1973 (a)
	Grass shrimp	RE	96 h	NOEC	1,000	Clark et al. 1987
	Midge	NR	48 h	EC50	760	Streufert et al. 1980 (a)
	Amphipod	NR	96 h	LC50	2,100	Mayer & Sanders 1973 (a)
--chronic						
	Fathead minnow	FT	ELS (20d)	LOEC m	1,000	McCarthy & Whitmore 1985
	Fathead minnow	FT	ELS (20d)	NOEC m	560	McCarthy & Whitmore 1985
	Mud crab	NR	ELS	NOEC gr	1,000	Laughlin et al. 1977 (b)
	Grass shrimp	RE	to p-hatch	LOEC m	100	Laughlin et al. 1978
	Brine shrimp	NR	to hatch	LOEC ha	10,000	Sugawara 1974 (c)
	D. magna	RE	15 d	LOEC re	1,800	McCarthy & Whitmore 1985
	D. magna	RE	15 d	NOEC re	560	McCarthy & Whitmore 1985
	Amphipod	FT	2 wk	LOEC	340	Tagatz et al. 1983
	S.water comm.	FT	2 wk	LOEC	3,700	Tagatz et al. 1983
	S.water comm.	FT	2 wk	NOEC	40	Tagatz et al. 1983
	Diatom	NR	4 d	LOEC	5,000	Medlin 1980 (c)
	Gymnodinium	NR	4 d	LOEC	200	Wilson et al 1978 (a)
	Amphipod	FT	14 d	BCF	6,700	Sanders et al. 1973 (a)
	Grass shrimp	SED	96 h	NOEC	10 *	Clark et al. 1987
	S.water comm.	SED	8 wk	NOEC	100 *	Tagatz et al. 1986
	S.water comm.	SED	8 wk	LOEC	1,000 *	Tagatz et al. 1986
	Amphipod	SED	NA	AET	260 *	Barrick & Beller 1989
	Oyster	SED	NA	AET	260 *	Barrick & Beller 1989
	Benthic Invert.	SED	NA	AET	1,700 *	Barrick & Beller 1989
DI-N-OCTYL PHTHALATE						
--chronic						
	D. magna	RE	15 d	NOEC re	320	McCarthy & Whitmore 1985
	D. magna	RE	15 d	LOEC re	1,000	McCarthy & Whitmore 1985
	Fathead minnow	FT	ELS (28 d)	NOEC ha	3,200	McCarthy & Whitmore 1985
	Fathead minnow	FT	ELS (28 d)	LOEC ha	10,000	McCarthy & Whitmore 1985
	Amphipod	SED	NA	AET	58 *	Barrick & Beller 1989
	Oyster	SED	NA	AET	> 57 *	Barrick & Beller 1989
	Benthic Invert.	SED	NA	AET	4,500 *	Barrick & Beller 1989
BUTYLBENZYL PHTHALATE						
--acute						
	Sheepshead minnow	ST	96 h	LC50	440,000	Heitmuller et al. 1981 (a)
	Sheepshead minnow	ST	96 h	LC50	3,000	Gledhill et al. 1980 (a)
	Shiner perch	FT	96 h	LC50	510	Ozretich et al. 1983
	Shiner perch	FT	96 h	LOEC bc,bh	80	Ozretich et al. 1983
--chronic						
	Amphipod	SED	NA	AET	42 *	Barrick & Beller 1989
	Oyster	SED	NA	AET	> 9.2 *	Barrick & Beller 1989
	Benthic Invert.	SED	NA	AET	64 *	Barrick & Beller 1989
DIMETHYL PHTHALATE						
--chronic						
	Grass shrimp	RE	to p-hatch	LOEC m	10,000	Laughlin et al. 1978
	Mud crab	NR	ELS	NOEC gr	1,000	Laughlin et al. 1977 (b)
	Amphipod	SED	NA	AET	53 *	Barrick & Beller 1989
	Oyster	SED	NA	AET	> 22 *	Barrick & Beller 1989
	Benthic Invert.	SED	NA	AET	53 *	Barrick & Beller 1989
DIETHYL PHTHALATE						
--chronic						
	Amphipod	SED	NA	AET	>110 *	Barrick & Beller 1989
	Oyster	SED	NA	AET	> 5.3 *	Barrick & Beller 1989
	Benthic Invert.	SED	NA	AET	61 *	Barrick & Beller 1989

TABLE C-1 (Continued)

AQUATIC TOXICITY OF PHTHALATES

CHEMICAL	ORGANISM	TYPE	DURATION	EFFECT	CONCENTRATION (ug/L)	REFERENCE
DI-2-ETHYLHEXYL PHTHALATE						
--acute						
	rainbow trout	NR	96 h	LC50	19,508	Spehar 1986 (a)
	fathead minnow	NR	A	LC50	1,106,200	Horne et al. 1983 (a)
	channel catfish	NR	96 h	LC50	100,000	Mayer & Ellersiech 1986
	bluegill sunfish	NR	96 h	LC50	770,000	Mayer & Ellersiech 1986
	coho salmon	NR	96 h	LC50	100,000	Mayer & Ellersiech 1986
	sheepshead minnow	NR	J	NOEC	550,000	Heitmuller et al. 1981 (a)
	D.magna	NR	48 h	LC50	11,000	LeBlanc 1980 (a)
	D.magna	NR	48 h	LC50	2,000	Adams & Heidolph 1985 (a)
	D.Magna	ST	48 h	EC50	133	Passino & Smith 1987
	amphipod	NR	NR	NOEC	400	Stephenson 1983 (a)
	copepod	NR	NR	NOEC	300,000	Linden et al 1979 (a)
	grass shrimp	NR	L	NOEC	> 450	Laughlin et al. 1978
--chronic						
	Rainbow trout	NR	ELS (24 d)		8	Mehrle & Mayer 1976 (a)
	Rainbow trout	NR	ELS (90 d)	NOEC	> 502	Spehar 1986 (a)
	fathead minnow	NR	ELS (32 d)		31,770	Horne et al. 1983 (a)
	D. magna	RE	LC	LOEC re	3	Mayer & Sanders 1973 (a)
	D. magna	RE	LC	NOEC re	107	Brown & Thompson 1985 (a)
	D. magna	RE	LC	LOEC	1,300	Adams & Heidolph 1985 (a)
	D. magna	RE	LC	NOEC	640	Adams & Heidolph 1985 (a)
	D. magna	RE	LC	CHRONIC	912	Adams & Heidolph 1985 (a)
	D. magna	FT	21 d	LC54	811	Knowles et al. 1987 (a)
	D. magna	FT	21 d	NOEC m	158	Knowles et al. 1987 (a)
	D. magna	FT	21 d	CHRONIC	358	Knowles et al. 1987 (a)
	Grass shrimp	RE	to p-hatch	NOEC m	1,000	Laughlin et al. 1978
	Midge	NR	35 d	NOEC	> 360	Streufert et al. 1980 (a)
	Green algae		5 d	EC50	> 410	Richter 1982 (a)
	Duckweed		NR	EC50	2,060,000	Davis 1981 (a)
	Diatom		96 h	EC50	31,000,000	Wilson et al. 1978 (a)
	Rainbow trout	NR	36 h	BCF	113	Mehrle and Mayer 1976 (a)
	Fathead minnow		56 d	BCF	155-886	Mayer 1976 (a)
	Scud		14-21 d	BCF	3,600	Sanders et al. 1973 (a)
	Sowbug		21 d	BCF	14	Sanders et al. 1973 (a)
	Clam M. edulis		28 d	BCF	2,366-2,627	Brown & Thompson 1982 (a)
	Amphipod	SED	NA	AET	78 *	Barrick & Beller 1989
	Oyster	SED	NA	AET	60 *	Barrick & Beller 1989
	Benthic Invert.	SED	NA	AET	60 *	Barrick & Beller 1989

(a) This reference is as in EPA (1987).
(b) This reference is as in Laughlin et al. (1978).
(c) This reference is as in McCarthy and Whitmore (1985).

* = Sediment concentrations in mg/kg.

ST = static
RE = Renewal
FT = flow through
BCF = bioconcentration factor (whole body)
AET = apparent effects threshold. Obtained using field sediment concentrations and observed biological adverse effects in the organism.
LC50 = median lethal concentration (lethal to 50% of test population)
EC50 = median effect concentration (effect seen in 50% of test pop.)
LOEC = lowest observed effect concentration
NOEC = no observed effect concentration
re = reproduction
m = mortality
gr = population growth
ha = hatchability
bc = biochemical
bh = behavioral

NA = not available
h = hour
d = day
E-L = embryo-larval stage organism
ps = photosynthesis
im = immobilization
ph = physiological
bl = blood chemistry

Aquatic Plants

Data from tests using species of green algae and diatoms were available for two of the phthalates. Four day lowest observed effect concentration (LOEC) values of 200 and 5000 ug/L were reported for the diatom Skeletonema sp. (Medlin et al. 1980 in McCarthy and Whitmore 1985) and Gymnodinium sp. (Wilson et al. 1978 in EPA 1987) exposed to DBP. EC_{50} concentrations for the diatom Skeletonema sp. and duckweed (Lemna minor) were 2,060,000 and 31,000,000 ug/L, respectively (Davis 1981 and Wilson et al. 1978 in EPA 1987).

Sediment Toxicity

Barrick and Beller (1989) reported sediment toxicity values in terms of apparent effect thresholds (AETs) for 6 phthalate esters. Amphipod AETs range from 42 mg/kg (BBP) to 260 (DBP) mg/kg, and oyster AETs range from 5.3 mg/kg (Diethyl phthalate) to 260 (DBP) mg/kg. AETs for benthic invertebrates ranged from 53 (DMP) to 4,500 (DOP) mg/kg. Clark et al. (1987) reported a NOEC for grass shrimp (Palaemonetes pugio) exposed to a DBP sediment concentration of 10 mg/kg for 96 hours. Tagatz et al. (1986) reported NOEC and LOEC concentrations of 100 and 1,000 mg/kg DBP for saltwater communities exposed for 8 weeks. Sediment toxicity data are included in Table X-1.

Bioaccumulation

Bioconcentration factors (BCFs) for invertebrates range from 14 (DEHP) to 6700 (DBP)(Sanders et al. 1973 in EPA 1987). BCFs for fish were available only for DEHP. They range from 113 to 886 (Mehrle and Mayer 1976; Mayer and Sanders 1976 in EPA 1987).

Criteria

EPA has proposed ambient water quality criteria (AWQC) for di-2-ethylhexyl phthalate (DEHP)(EPA 1987). For freshwater organisms, the 4-day average concentration criterion is 360 ug/L, and the 1-hour average concentration criterion is 400 ug/L. Criteria could not be derived for saltwater organisms using the avaialble data. However, DEHP does not ionize in water, and therefore EPA believes it reasonable to assume that the toxicity of DEHP in saltwater is equal to that in freshwater. Therefore, the freshwater criteria stated above are also adopted as saltwater criteria. Criteria for other phthalate esters have not been established.

TERRESTRIAL WILDLIFE

Data regarding toxicity of phthalates to terrestrial wildlife are extremely limited. Available data are presented below. Data from tests using laboratory mammals are presented when no other data were located.

Tagatz et al. (1983) report that DBP is used as an insect repellant, indicating insecticidal properties; however, no toxicity data for insects were located in the literature.

Birds

Schafer et al. (1983) report a LD_{50} of > 100 mg/kg diet for red-winged blackbirds (Agelaius phoeniceus) exposed to DMP.

Mammals

Data indicate that phthalate esters induce abnormal lipid metabolism in mammals (Bell 1976 and Bell and Nazir 1976 in Bell and Buthala 1983). Bell and Buthala (1983) found that in rats fed 1% DEHP, inhibition of microsomal ACAT (acylCoA:cholesterol acyltransferase) occurred. ACAT is the principle cholesterol-esterifying enzyme in the liver. Reported LD_{50}s for DMP in mice, rats, and guinea pigs are 7.2, 6.9, and 2.4 ml/kg (Budavari et al. 1989). An oral LD_{50} in rats of 8000 mg/kg is reported for DBP by Fishbein and Albro (1972 in Shea et al. 1982).

Plants

Shea et al. (1982) conducted the uptake and toxicity of DBP in corn. At a soil concentration of 2000 mg/kg, plant height and shoot weight were significantly reduced from that in controls. At 20,000 mg/kg, corn growth was severely inhibited.

REFERENCES

BARRICK, R.C. AND BELLER, H.R. 1989. Reliability of sediment quality assessment in Puget Sound. In: Oceans '89 Proceedings. Inst. of Electrical and Electronic Engineers. Piscataway, NJ. Publ. No. 89CH2780-5.

BELL, F.P. and BUTHALA, D.A. 1983. Biochemical changes in liver of rats red the plasticizer di(2-ethylhexyl)phthalate. Bull. Environ. Contam. Toxicol. 31:177-182.

BUDAVARI, S., O'NEIL, M., SMITH, A, and HECKLEMAN, P. 1989. The Merck Index. Merck & Co., Inc. Rahway, NJ.

CLARK, J.R., PATRICK, J.M., Jr., MOORE, J.C., and LORES, E.M. 1987. Waterborne and sediment-source toxicities of six organic chemicals to grass shrimp (Palaemonetes pugio) and amphioxus (Branchiostoma caribaeum). Arch. Environ. Contam. Toxicol. 16:401-407.

DUMPERT, K. and ZIETZ, E. 1984. Platanna (Xenopus laevis) as a test organism for determining the embryotoxic effects of environmental chemicals. Ecotox. and Environ. Safety. 8:55-74.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Ambient Aquatic Life Water Quality Criteria for di-2-Ethylhexyl phthalate - Draft. Office of Research and Development, Environmental Research Laboratories. Duluth MN, Narragansett, RI.

LARSSON, P. and THUREN, A. 1987. Di-2-ethylhexylphthalate inhibits the hatching of frog eggs and is bioaccumulated by tadpoles. Environ. Toxicol and Chem. 6:417-422.

LAUGHLIN, R.B., Jr., NEFF, J.M., HRUNG, Y.C., GOODWIN, T.C., and GIAM, C.S. 1978. The effects of three phthalate esters on the larval development of the grass shrimp Palaemonetes pugio. Water, Air, and Soil Pollut. 9:323-336.

MAYER, F.L., Jr., and ELLERSIECH, M.R. 1986. Manual of acute toxicity: Interpretation and database for 410 chemicals and 66 species of freshwater animals. U.S. Fish and Wildlife Service. Washington, D.C. Resource Publication No. 160. p. 394.

McCARTHY, J.F., and WHITMORE, D.K. 1985. Chronic toxicity of di-n-butyl and di-n-octyl phthalate to Daphnia magna and the fathead minnow. Environ. Toxicol. and Chem. 4:167-179.

OZRETICH, R.J., RANDALL, R.C., BOESE, B.L., SCHROEDER, W.P., and SMITH, J.R. 1983. Acute toxicity of butylbenzylphthalate to shiner perch (Cymatogaster aggregata). Arch. Environ. Contam. Toxicol. 12:655-660.

PASSINO. D.R., and SMITH, S.B. 1987. Acute bioassays and hazard evaluation of representative contaminants detected in Great Lakes fish. Environ. Toxicol. Chem. 6(11):901-907.

SCHAFER, E.W., BOWLES, W.A., JR., and HURLBUT, J. 1983. The acute oral toxicity, repellancy, and hazard potential of 998 chemicals to one or more species of wild and domestic birds. Arch. Environ. Contam. Toxicol. 12:355-382.

SHEA, P.J., WEBER, J.B., and OVERCASH, M.R. 1982. Uptake and phytotoxicity of di-n-butyl phthalate in corn (Zea mays). Bull. Environ. Contam. Toxicol. 29:153-158.

TAGATZ, M.E., DEANS, C.H., MOORE, J.C., and PLAIA, G.R. 1983. Alterations in composition of field- and laboratory-developed estuarine benthic communities exposed to di-n-butyl phthalate. Aquatic Toxicol. 3:239-248.

TAGATZ, M.E., PLAIA, G.R., and DEANS, C.H. 1986. Toxicity of dibutyl phthalate-contaminated sediment to laboratory- and field-colonized estuarine benthic communities. Bull. Environ. Contam. Toxicol. 37:141-150.

VEITH, G.D., DEFOE, D., and KNUTH, D. 1985. Structure-activity relationships for screening organic chemicals for potential ecotoxicity effects. Drug Metabolism Reviews. 15(7):1295-1303.

POLYCHLORINATED BIPHENYLS (PCBs)

AQUATIC TOXICITY

In fish, PCBs have caused death, behavioral abnormalities, increased locomotor activity, neurochemical alterations, disrupted osmoregulation, and liver and thyroid effects. Reproductive effects also have been linked to PCB exposure. Eisler (1986) has compiled toxicity data for a wide range of organisms including aquatic invertebrates and fish. In general, the toxicity of PCBs to aquatic organisms decreases as the degree of chlorination increases (Eisler 1986).

Invertebrates

Among the freshwater invertebrates, the scud Gammarus pseudolimnaeus and crayfish Orconectes nais appear to be among the most sensitive. 96-hour LC_{50}s for the scud were 10, 52, and 2,400 ug/L for Aroclor 1242, 1248, and 1254, respectively. For crayfish, 7-day LC_{50}s of 30 and 80-100 ug/L were reported for Aroclors 1242 and 1254, respectively. A 7-day LC_{50} of 3 ug/L is reported for the glass shrimp (Palaemonetes kadiakensis) exposed to Aroclor 1254. A 21-day LC_{50} of 1.3 ug/L is reported for Daphnia magna exposed to Aroclor 1260. Stoneflies (Pteronarcella sp.), dragonflies (Macromia sp.), and damselflies (Ischnura sp.) are the least sensitive invertebrates tested. Four and 5-day LC_{50}s for these organisms range from 200 to 878 ug/L for the Aroclors 1016, 1242, and 1254 (Eisler 1986).

Based on data for three saltwater shrimp species (Palaemonetes pugio, Penaeus aztecus, and Penaeus duorarum), saltwater invertebrates may be more sensitive to PCBs than freshwater species. 48-Hour LC_{50}s range from 6 to 12.5 ug/L for Aroclors 1016 and 1254 (Eisler 1986).

Chronic no observed effect concentrations (NOECs) and lowest observed effect concentrations (LOECs) for freshwater invertebrates range from 0.5 ug/L (NOEC-midge Tanytarsus dissimilis) to 8.7 ug/L (LOEC-amphipod Gammarus pseudolimnaeus) (Eisler 1986). No chronic values were available for saltwater invertebrates.

Fish

In 96-hour acute assays using largemouth bass (Micropterus salmoides) and redear sunfish (Lepomis microlophus) exposed to Capacitor 21 (a PCB mixture), LC_{50} values of 2.3 and 19 ug/L, respectively were reported (EPA 1980).

Acute toxicity in yellow perch (Perca flavescens) occurred only at concentrations exceeding 100 ug/L for Aroclors 1242, 1248, and 1260. Toxicity in channel catfish (Ictalurus punctatus) is reported at Aroclor concentrations ranging from 110 ug/L for Aroclor 1242 to 740 ug/L for Aroclor 1248. Bluegill sunfish (Lepomis macrochirus) appear to be slightly more sensitive than the two species mentioned above. LC_{50}s range from 10 to 15 ug/L for Aroclors 1248, 1254, and 1260. Aroclor 1016 was least toxic, with LC_{50}s ranging from 240 to 560 ug/L in species occurring at APG (yellow perch, bluegill and channel catfish).

Based on data for three saltwater fish species, saltwater fish may be more sensitive to acute toxic effects of PCBs than freshwater species. In 12 to 38-day tests using Aroclor 1254, LC_{50}s ranging from 0.1 to 0.9 ug/L are reported for sheepshead minnows (Cyprinodon variegatus), spot (Leiostomus xanthurus), and pinfish (Lagodon rhomboides).

Chronic NOEC and LOEC values for brook trout (Salvelinus fontinalis) are 0.7 and 1.2 ug/L for Aroclor 1254. Fathead minnow (Pimephales promelas) NOECs and LOECs range from 0.1 (1248) to 5.4 ug/L (1242) and from 0.4 (1248) to 15 ug/L (1242), respectively. Based on data for invertebrates and fish species, the chronic toxicity of Aroclor 1248 appears to be higher than either of the two more highly chlorinated Aroclors (1254 and 1260). Reproductive effects have been observed in fish at PCB tissue concentrations between 0.1 to > 24 mg/kg body weight (fresh weight) (Eisler 1986). Field and Dexter (1988) cite studies that show reproductive impairment in fish when gonad concentrations exceeded 1 ppm.

Amphibians

Data regarding PCB toxicity to amphibians were not found in the literature.

Plants

Toxicity in sensitive freshwater algae species occurred at concentrations as low as 0.1 ug/L (Aroclor 1254) (Eisler 1986).

Sediment Toxicity

Sediments can act as a source of PCBs. Field and Dexter (1988) reviewed the available literture on PCB sediment toxicity and suggested that a PCB sediment level of 0.1 mg/kg may be protective of aquatic life. The level was based on observed toxicity in aquaitc life at organism PCB tissue concentrations between 0.1 and 1.0 mg/kg and an assumed 1:1 sediment-to-organism concentration ratio. However, they state the 0.1 mg/kg sediment level may not be protective of aquatic organisms in higher trophic levels.

Barrick and Beller (1989) reported sediment toxicity values in terms of apparent effect thresholds (AETs). They report AETs of > 46, 65, and 190 mg/kg (organic carbon) for oysters, benthic invertebrates, and amphipods, respectively.

Bioaccumulation

Four-day whole body bioconcentration factors (BCFs) for freshwater invertebrates exposed to Aroclor 1254 range from 60 for the protozoan Tetrahymena pyriformis to 47,000 for the water flea D. magna (EPA 1980). BCFs for 2 saltwater invertebrate species are 85,000 for the oyster Crassostrea virginica (soft parts only) following 168 days of exposure and 340,000 for the rotifer Brachionus plicatilis (lipid) following 45 days of exposure (EPA 1980).

Whole body bioconcentration factors of 60,000 to 270,000 in fish have been reported in EPA (1980). A 3-day muscle BCF of 164 is reported for a freshwater cichlid fish Cichlasoma facetum exposed to Aroclor 1254. Whole body BCFs of 21,800 and 27,800 are reported for the pinfish Lagodon rhomboides and spot Leiostomus xanthurus, following 35 and 56 days of exposure to Aroclor 1254 (Eisler 1986).

Willford et al. (1987) reported sediment:organism bioaccumulation factors in the range of 0.7 to 12.2 for Oligochaeta and 1.1 to 9.7 for fathead minnows exposed for 10 days to PCBs in Great Lakes sediments. The average values were 4.9 for Oligochaeta and 4.3 for fathead minnows.

Criteria

EPA (1980) has established 4-day and 1-hour concentration criteria for PCBs in freshwater of 0.014 and 2.0 ug/L not to be exceeded more than once every three years. These criteria are protective of fish but were designed specifically to protect mink (Mustela vison) that eat fish from PCB-contaminated waters. In marine waters, EPA has established 4-day and 1-hour average concentrations of 0.03 and 10 ug/L, respectively.

ICF-Clement (Clement 1988) developed a PCB action level of 0.1 mg/kg for sediments of Pennsylvania surface water based on toxicity and exposure in aquatic wildlife.

TERRESTRIAL TOXICITY

Birds

PCBs can cause death and a variety of sublethal effects in birds exposed via the diet. Sublethal effects following chronic exposures include enzyme induction, porphyria, altered vitamin A metabolism, alteration of the thyroid, and cardiac, behavioral, hormonal, and reproductive effects. There is significant variability in the species sensitivity to the reproductive effects of PCBs. No reproductive effects were observed in mallards (Anas platyrhynchos) exposed for 12 weeks during egg laying to 150 ppm (mg/kg feed) Aroclor 1252 in the diet (Haseltine and Prouty 1980), or in mallards exposed to 25 ppm Aroclor 1254 in the diet for at least a month before egg laying (Custer and Heinz 1980). However, mourning doves (Zeinada macroura) exposed to 10 ppm Aroclor 1254 in the food for 28 days experienced delayed reproduction and a decrease in the number of eggs laid (Koval et al. 1987). American kestrel (Falco sparverius) exposure to l0 ppm Aroclor 1254 in food potentiated increased egg shell thinning and subsequent breakage of eggs caused by DDE (Lincer 1972). No reproductive effects were observed in screech owls (Otus asio) exposed for 8 weeks before the onset of egg laying to 3 ppm Aroclor 1248 in the diet (McLane and Hughes 1980).

This 3 ppm level is the highest NOAEL found in the literature. Assuming a screech owl (by analogy to burrowing owls) weighs approximately 0.166 kg and consumes an estimated 0.026 kg of food per day (Welty 1982), the 3 ppm dietary level corresponds to an estimated dosage of approximately 0.5 mg/kg bw-day. Applying an uncertainty factor of 100 to this value (10 for subchronic exposures and 10 for interspecies differences) results in a toxicity value of 0.005 mg/kg/day.

Mammals

Reproductive toxicity following chronic or subchronic exposures appears to be the most sensitive toxic endpoint of PCBs exposure in mammals. Mink (Mustela vison) are particularly susceptible to PCBs' reproductive effects. Mink fed contaminated PCB (Aroclor 1254) contaminated beef developed reproductive complications at dietary residue levels as low as 0.64 ppm (Platanow and Karstad 1973). A dietary level LC_{50} of 6.7 ppm was reported for an exposure period of 9 months for Aroclor 1254 (Ringer 1983). Other species may be less sensitive to PCBs' toxic effects. White-footed mice (Peromyscus leucopus) exposed to PCBs at a concentration of 10 ppm in the diet through the second generation exhibited poor reproductive success, growth, and development of reproductive organs, but no increased mortality (Linzey 1988).

This dietary level is used to develop a toxicity value for shrews instead of the mink dietary information because shrews are believed to be more similar to mice than to mink. Assuming that a white-footed mouse weighs approximately 30 g and eats 13 g of food daily (by analogy to laboratory mice, EPA

1985), the 10 ppm dietary level corresponds to a daily dosage of approximately 4 mg/kg bw/day. Applying an uncertainty factor of 100 (10 for interspecies differences and 10 for using a LOAEL) to this value, gives a toxicity value of 0.04 mg/kg/day.

Criteria

ICF-Clement (Clement 1988) developed a PCB action level of 0.5 to 2.0 mg/kg for terrestrial wildlife based on toxicity of PCBs to mink that eat PCB contaminated fish.

REFERENCES

BARRICK, R.C. AND BELLER, H.R. 1989. Reliability of sediment quality assessment in Puget Sound. In: Oceans '89 Proceedings. Inst. of Electrical and Electronic Engineers. Piscataway, New Jersey. Publ. No. 89CH2780-5.

CUSTER, T.W. and HEINZ, G.H. 1980. Respiratory success and nest attentiveness of mallard ducks fed Aroclor 1254. Environ. Pollution (Series A). 21:313-318.

EISLER, R. 1986. Polychlorinated Biphenyl Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Department of the Interior, Fish and Wildlife Service. Contaminant Hazard Reviews Report No. 7. Biological Report 85(1.7).

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Polychlorinated Biphenyls. Office of Water Regulations and Standards. EPA 440/5-80-068. PB81-117798

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Reference Values for Risk Assessment--Draft. Environmental Criteria and Assessment Office. Cincinnati, Ohio. ECAO-CIN-477.

FIELD, L.J. and DEXTER, R.N. 1988. A Discussion of PCB Target Levels in Aquatic Sediments. National Oceanic and Atmospheric Administration, Ocean Assessments Division and EVS Consultants, Inc. Seattle, Washington.

HASELTINE, S.D. and PROUTY, R.M. 1980. Aroclor 1242 and reproductive success of adult mallards (Anas platyrhynchos). Environ. Res. 23:29-34.

IFC-CLEMENT (CLEMENT). 1988. Technical Development Document for Action Levels of Polychlorinated Biphenyls in Sediments of Pennsylvania Waters. Revised Draft. Prepared for Commonwealth of Pennsylvania, Department of Environmental Resources. Fairfax, Virginia.

KOVAL, P.J., PETERLE, T.J. and HARDER, J.D. 1987. Effects of polychlorinated biphenyls on mourning dove reproduction and circulation progesterone levels. Bull. Environ. Contam. Toxicol. 39:663-670.

LINCER, J. 1972. The Effects of Organochlorines on the American Kestrel (Falco sparverius). Cornell University, New York. PhD. Thesis.

LINZEY, A.V. 1988. Effects of chronic polychlorinated biphenyls exposure on growth and reproduction of second generation white-footed mice (Peromyscus leucopus). Arch. Environ. Contam. Toxicol. 17:39-45.

McLANE, A.R. and HUGHES, D.L. 1980. Reproductive success of screech owls fed Aroclor 1248. Arch. Environ. Contam. Toxicol. 9:661-665.

PLATANOW, N.S. and KARSTAD, L.H. 1973. Dietary effects of polychlorinated biphenyls on mink. Can. J. Comp. Med. 37:391-400.

RINGER, R.K. 1983. Toxicology of PCBs in minks and ferrets. In: D'Itri, F.M. and Kamrin, M.A. (eds.). PCBs: Human and Environmental Hazards. Ann Arbor Science Book. Butterworth Publishers, Boston, Massachusetts.

WELTY, J.C. 1982. The Life of Birds. Third Edition. CBS College Publishing. New York.

WILLFORD, W.A., MAC, M.J., and HESSELBERG, R.J. 1987. Assessing bioaccumulation of contaminants from sediments by fish and other aquatic organisms. Hydrobiologia. 149:107-111.

AQUATIC TOXICITY

The aquatic toxicity of polycyclic aromatic hydrocarbons (PAHs) has been reviewed by Eisler (1987). PAHs are widespread environmental pollutants. These compounds vary considerably in their toxicity to aquatic organisms, but, in general, toxicity increases with molecular weight (Eisler 1987). PAHs have been associated with the increased occurrence of tumors, neoplasms, and other growth abnormalities in fish in polluted waters (EPA 1988). The toxicity of some PAHs are influenced by sunlight or ultraviolet light (EPA 1988) and dissolved humic materials (McCarthy et al. 1985).

Invertebrates

Freshwater invertebrate acute toxicity data for PAHs is limited. For the PAHs in which there is data, aquatic crustaceans are normally more sensitive than fish to toxic effects of PAHs (Eisler 1987). Based on the acute toxicity values for 4 PAHs (flourene, naphthalene, trimethylnaphthalenes, and phenanthracene) in 5 species of freshwater invertebrates, naphthalene was the most acutely toxic of the PAHs tested.

The 96-hour LC_{50}s for freshwater invertebrates range from 300 ug/L (1-methyphenanthrene) to 5,600 ug/L (fluorene); however toxicities varied depending on both the species and PAH tested. For example, the lowest freshwater invertebrate acute value (30% mortality within 10 days of exposure) for the PAHs is 50 ug naphthalene/L for the copepod (Eurytemora affinis); whereas, a 24-hour LC_{50} of 320 ug/L was obtained for trimethylnaphthalenes in the same species (Eisler, 1987); and the 96-hour LC_{50} is 2,680 ug/L (naphthalene) for the amphipod (Elasmopus pectenicurus). The only freshwater invertebrate chronic toxicity value is 96.39 ug/L for phenathracene in Daphnia magna (EPA 1988).

Similar to freshwater invertebrates, acute toxicity of PAHs to saltwater invertebrates varies within and between species and PAHs. Phenanthracene is the most toxic of the PAHs tested in saltwater invertebrates. Acute toxicity values (96-hour LC_{50}s) for PAHs for 11 genera of saltwater invertebrates range from 17.7 ug/L (phenanthracene) for the mysid (Mysid bahia) to 3,800 ug/L (naphthalene) for the sandworm (Neanthes arenceodentata) (Eisler 1987; EPA 1988). Larval mud crab (Rithropanopeus harrissi) exposed up to 24 days to phenanthracene at 200 ug/L exhibited a decreased developmental rate (EPA 1988). A chronic toxicity value of 8.129 ug/L for phenanthracene was reported for the saltwater invertebrate mysidopsis bahia (EPA 1988).

Fish

The acute toxicity values for PAHs for seven species of freshwater fish range from 234 ug/L (phenanthracene) for bluegill (Lepomis macrochirus) to 150,000 ug/L (naphthalene) for the fathead minnow (Pimephales promelas) of (Eisler 1987, EPA 1988). Bluegill and rainbow trout (Salmo gairdneri) are among the most sensitive of the freshwater fish species tested with PAHs. Benz(a)anthracene at a concentration of 1 ug/L caused 87% mortality in bluegill in 6 months (Harris et al. 1982). Acute 96-hour LC_{50}s (for phenanthracene) reported for juvenile bluegill and rainbow trout are 375 ug/L and 234 ug/L, respectively (EPA 1988). Fathead minnows are relatively tolerant to the PAHs tested with acute LC_{50}s exceeding the solubility limits for both phenanthracene and fluorene (Eisler 1987, EPA 1988). The 96-hour EC_{50} (loss of equilibrium) for juvenile bluegill and rainbow trout exposed to phenanthracene is 50 ug/L and 49 ug/L, respectively (EPA 1988). A 27-day EC_{50} (based on death and deformity) for rainbow trout embryo-larva exposed to phenanthracene is 30 ug/L (EPA 1988). Chronic effects values of 250 ug/L (phenanthracene) and 620 ug/L (naphthalene) were

reported for embryo-larva of the largemouth bass (Micropterus salmoides) and the fathead minnow, respectively (Harris et al. 1982, EPA 1988). The chronic value for phenanthracene is 6.325 ug/L based on an early life-stage test in rainbow trout (EPA 1988). Carcinogenic PAHs can produce cancer-like growths in fish. Schultz and Schultz (1982 in Eisler 1987) reported liver neoplasms in two species of minnows (Poeciliopsis spp.) exposed 6 hours per week for 5 weeks to an aqueous suspension of 5,000 ug/L of 7,12-dimethylbenz(a)pyrene. High dietary levels of benzo(a)pyrene (1,000 mg/kg) produced liver tumors in rainbow trout (Eisler 1987).

Acute toxicity values in saltwater fish have been reported for five PAHs in three species. The acute LC_{50} values range from 108 ug/L (phenanthracene) for the atlantic silverside (Menidia menidia) to 3,400 ug/L for the sheepshead minnow (Cyprinodon variegatus) (Eisler 1987, EPA 1988). No chronic toxicity values are available for saltwater fish in Eisler (1987) or EPA (1988). Sand sole (Psettichthys melanostichus) eggs exposed to an environmental level of benzo(a)pyrene at 0.1 ug/L for 5 days displayed a decline in hatching success and a higher incidence of developmental anomalies (Hannah et al. 1982).

Toxicity information is limited for PAHs and fish species that occur at or near the Aberdeen Proving Ground (APG). The LC_{50}s for freshwater species are: 234 ug/L (phenanthracene) and 910 ug/L (flourene) for bluegill, and 150,000 ug/L (naphthalene) for the mosquitofish (Gambusia affinis) (Eisler 1987). An EC_{50} of 50 ug/L (phenanthracene) was reported for juvenile bluegill based on loss of equilibrium (EPA 1988). Chronic exposure of bluegill to benz(a)anthracene at 1 ug/L resulted in 87% mortality (Harris et al. 1982). For saltwater fish species, PAH toxicity information is available for only one species, the atlantic silverside (Menidia menidia). The species mean acute value for the silverside is 108 ug/L (phenanthracene) (EPA 1988).

Amphibians

Edmisten and Bantle (1982) reported a 96-hour LC_{50} for naphthalene of 2.1 mg/L for larvae of the frog Xenopus laevis. In the same study, 6-hour EC_{50}s for based on the absence of swimming and depigmentation were 2.3 to 1.7 mg/L and 3.7 mg/L, respectively.

Plants

Information on the toxicity of PAHs to aquatic plants is limited to phenathrene. The production of new fronds in the duckweed (Lemna minor) was reduced by 7 to 36% by concentrations from 198 to 658 ug/L following 4 days of exposure to phenathrene (EPA 1988). Acute toxicity values for phenathrene for several species of alga have been reported. The blue-green alga (Microcystis aeruginosa) is the most sensitive based on a 4-hour EC_{20} of 150 ug/L for reduction in photosynthesis. The green alga (Selenastrum capricarnutum) was the least sensitive species tested. A 24% reduction in photosynthesis was reported following 4 hours of exposure to 1,212 ug/L (EPA 1988).

Sediment Toxicity

Limited information is available on the toxicity of PAH-contaminated sediments. Hannah et al. (1982) found that rainbow trout alevins (Salmo gairdneri) reared on sand experimentally coated with 5 ppm benzo(a)pyrene (produced an aqueous concentration of 0.21 ug/L) for 36 days exhibited an increase in morphological abnormalities. A number of studies have reported an increase incidence of tumors and hyperplastic diseases in feral fish associated with PAH contaminated sediments (Eisler 1987)

Chapman et al. (1987) reported a 10-day LC_{50} in Rhepoxynius abronius (anamphipod) of 2.0 mg/kg for PAHs.

Barrick and Beller (1989) report sediment toxicity in terms of apparent effect thresholds (AETs). They divided PAHs into low molecular weight PAHs (LMW-PAHs) and high molecular weight PAHs (HMW-PAHs). AETs for LMW-PAHs are generally lower than those for HMW-PAHs. For LMW-PAHs, AETs for benthic invertebrates, amphipods, and oysters are 780, 2,200, and 370 mg/kg (organic carbon), respectively. For HMW-PAHs, AETs are 7,600, 5,300, and 960 mg/kg, for invertebrates, amphipods and oysters.

Bioaccumulation

PAHs can be accumulated in aquatic organisms from water, sediments, and diet. In general, bioconcentration is greater for the higher molecular weight compounds than for the lower molecular weight compounds (Eisler 1987). Bioconcentration tends to be rather species-specific being higher in the lower forms of aquatic organisms which have a reduced capability of metabolizing PAHs (Eisler 1987).

Bioconcentrations factors (BCFs) of PAHs in freshwater invertebrates range from 10.6 to 134,248 (Eisler 1987). A BCF value of 200 was reported for the cladoceran Daphnia magna after 6 months exposure to anthracene, whereas a BCF for benzo(a)pyrene was 2,837 after 6 hours (Eisler 1987). A BCF for naphthalene of 131 was reported for Daphnia pulex after 24 hours, but after 3 days of exposure to benzo(a)pyrene, a BCF of 134,248 was obtained (Eisler 1987). BCF values for other freshwater invertebrates are: 10.6 (for phenanthracene) in the alga (Selnastrum capricornutum) after 8 hours; 3,500 (for anthracene) in the mayfly (Hexania sp.) following 28 hours of exposure; 166 (for benzo(a)pyrene) in larvae of the midge (Chironomus riparius) after 8 hours exposure. BCFs of 5,258, 11,536, and 82,231 were reported for the alga (Oedogonium cardiacum), the mosquito (Culex pipiens), and the snail (Physa sp.), respectively, following 3 days of exposure to benzo(a)pyrene (Eisler 1987, EPA 1988).

Bioconcentration factors of PAHs reported in saltwater invertebrates species range for 6 to 236 (Eisler 1987). BCFs of less than 40 were reported for the clam Rangia cuneata from 24-hour exposures to chrysene, naphthalene, and phenanthracene; and in the sandworm (Neanthes arenaceodenta) exposed 3 to 24 hours to naphthalene (Eisler 1987). The highest BCF for PAHs reported in saltwater invertebrates was 242 for the oyster Crassostrea virginica after 14 days exposure to benzo(a)pyrene (Eisler 1987).

PAHs have shown bioconcentration factors of 69 to 9,200 in freshwater fish (Eisler 1987). BCFs of 619 (liver), 213 (gills), and less than 55 (kidney) were reported for the northern pike (Esox lucius) following 23 days of exposure to benzo(a)pyrene (Balk et al. 1984). BCFs for the rainbow trout, based on liver concentrations, were: 182 to 920 following 10 days of exposure to benzo(a)pyrene; and 379 and 69 after 21 days of exposure to fluoranthrene and pyrene, respectively (Gerhardt and Carlson 1978 in Eisler 1987). Three-day BCFs (whole body) of 485 (anthracene), 930 (benzo[a]pyrene), and 4,400 to 9,200 were determined for the fathead minnow (Pimephales promelas), mosquitofish (Gambusia affinis), and rainbow trout (Salmo gairdneri) (Eisler 1987). A BCF of 200 to 1,800 was reported for the bluegill after 30 days of exposue to fluorene (Fingers et al. 1985 in Eisler 1987).

For marine fish, a (liver) BCF of 258 to 367 was determined for the mangrove snapper (Lutjanus griseus) after 20 days of exposure to chrysene (Miller et al. 1982 in Eisler 1987). In two studies with eggs of saltwater fish, BCFs of 11,536 and 44 to 83 were reported for the sand sole (Psettichthys

melanostictus) after 6 days of exposure to benzo(a)pyrene and atlantic salmon (Salmo salar) following 168 hours of exposure to naphthalene (Eisler 1987).

The potential of PAHs to bioaccumulate in aquatic organisms is partially determined by the capability of organisms to metabolize PAHs and thus, reduce the concentration of the parent PAH and result in far lower bioconcentration factors (Eisler 1987). Experiments performed with radiolabeled compounds have demonstrated that estimation of bioconcentration factors for some PAHs may overstate the bioconcentration of parent compounds. Spacie et al. (1983) estimated BCFs of 900 for anthracene and 4,900 for benzo(a)pyrene in bluegills (whole body) based on total radiolabeled carbon (^{14}C) activity. The estimated BCFs based only on the parent compounds were 675 and 490, respectively. Thus, biotransformation of the parent compounds occurred in addition to bioconcentration.

Fish and crustaceans readily assimilate PAHs from contaminated food, whereas assimilation in molluscs and polychaete worms is limited (Eisler 1987). Lu et al. (1977) found that the mosquitofish (Gambusia affinis) accumulated virtually no benzo(a)pyrene from water after 3 days of exposure, whereas the mosquito larvae (Culex pipens) and snail (Physa sp.) had reported BCFs of 37 and 2177. However, using a laboratory model aquatic ecosystem, these researchers determined BCFs following 3 days of exposure to benzo(a)pyrene of: mosquitofish, 930; alga (Oegonium cardiacum), 5258; mosquito, 11,536; snail, 82,231; and daphnia (Daphnia magna), 134,248. Thus, the fish accumulated benzo(a)pyrene primarily from its diet with negligible accumulations from the medium.

Sediment-associated PAHs can be accumulated by bottom-dwelling invertebrates and fish (Eisler 1987). Great Lakes sediments contaminated with elevated levels of PAHs were reported by Eadie et al. (1983 in Eisler 1987) to be the source of body burdens of the compounds in bottom-dwelling invertebrates. Lake et al (1985 in Eisler 1987) found that teh marine mussel (Mytilus edulis) and the annelid (Nereis virens) exposed for 28 days to sediments heavily contaminated with PAHs accumulated up to 1000 times more than controls.

Environmental factors such as suspended particulate matter and exposures to complex mixtures can influence the bioavailability of some PAHs to aquatic organisms. McCarthy and Jimenez (1985) reported BCFs of that dissolved humic material (DHM) reduced the accumulation of benzo(a)pyrene by 90% in bluegill (Lepomis macrochirus) (BCFs of 225 and 2,657 after 48 hours exposure with and without DHM, respectively), but had little effect on naphthalene (BCFs of 305 and 310 with and without DHM, respectively). BCFs of 9,200 and 6,400 were obtained in rainbow trout (Salmo gairdneri) exposed for 72 hours to anthracene alone and anthracene in oil shale retort water (Linder et al. 1985).

Criteria

The freshwater ambient water quality criteria (AWQC) proposed for phenanthracene is 30 ug/L for acute exposure and 6.3 ug/L for chronic exposures (EPA 1988). For saltwater organisms, the acute and chronic criteria are 7.7 ug/L and 4.6 ug/L, respectively (EPA 1988). The available data are not sufficient to develop AWQCs for other PAHs.

TERRESTRIAL TOXICITY

Few data are available on the toxicity of PAHs to terrestrial species and has been reviewed by Eisler (1987). The primary effect of PAH exposure in mammalian laboratory species is tumor development. The tumorigenic or carcinogenic potential of the PAHs appears to be essentially related to the number of unsubstituted aromatic rings (Eisler 1987).

Birds

Chronic dietary exposure of to 4,000 mg PAHs/kg (primarily as naphthalenes, naphthenes, and penanthrene) caused increased liver weights and increased blood flow to the liver in mallards (Eisler 1987). No overt signs of toxicity were observed. Some PAHs have been shown to be embryotoxic and teratogenic when applied to the surface of mallard eggs (Eisler 1987); however, the significance of these concentrations to realistic environmental exposures is not known. No other information was found on the acute and chronic toxicity of PAHs for avian wildlife.

Mammals

Information on the toxicity of PAHs to mammalian wildlife species is quite limited. Food consumption in deer mice decreased following oral exposure for 5 days to 825 mg/kg body weight of 2-methoxynaphthalene, a noncarcinogenic PAH (Eisler 1987).

Plants

Plants can absorb PAHs from soils through the roots, and from air through the leaves. Under laboratory conditions, some plants concentrated selected PAHs above their immediate surroundings, but this has not been conclusively been demonstrated in field-grown crops or other vegetation (Eisler 1987). The phytotoxic effects of PAHs have been examined in only a few studies and based on this limited data, PAHs do not appear to be toxic to plants.

REFERENCES

BALK, L.J., MEIJER, J.W., DePIERRE, and APPELGREN, L.E. 1984. The uptake and distribution of [^{3}H]benzo(a)pyrene in the northern pike (Esox lucius); examination by whole-body autoradiography and scintillation counting. Toxicology and Applied Pharmacology. 74:430-449.

BARRICK, R.C. and BELLER, H.R. 1989. Reliability of sediment quality assessment in Puget Sound. In: Oceans '89 Proceedings. Inst. of Electrical and Electronic Engineers. Piscataway, NJ. Publ. No. 89CH2780-5.

CHAPMAN, P.M., BARRICK, R.C., NEFF, J.M., and SWARTZ, R.C. 1987. Four independent approaches to developing sediment quality criteria yield similar values for model contaminants. Env. Tox. Chem. 6:723-725.

EDMISTEN, G.E., and BANTLE, J.A. 1982. Use of Xenopus laevis larvae in 96-hour, flow-through toxicity tests with naphthalene. Bulletin of Environmental Contamination and Toxicology. 29:392-399.

EISLER, R. 1987. Polyaromatic Hydrocarbon Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Department of the Interior, Fish and Wildlife Service. Laurel, Maryland. Contamination Hazard Reviews Report No. 11. Biological Report 85(1.11).

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Ambient Water Quality Criteria for Phenanthracene--Draft. Office of Water Regulations and Standards. Washington, D.C.

HANNAH, J.R., HOSE, J.E., LANDOL, M.L., MILLER, B.S., FELTON, S.P. and IWAOKA, W.T. 1982. Benzo(a)pyrene-induced morphologic and developmental abnormalities in rainbow trout. Archives of Environmental Contamination and Toxicology. 11:727-734.

HARRIS, J., PERWAK, J. and COONS, S. 1982. An exposure and risk assessment for benzo(a)pyrene and other polycyclic aromatic hydrocarbons: Volume I. Summary--Final Draft Report. Arthur D. Little, Inc. Prepared for the U.S. Environmental Protection Agency, Office of Water Regulations and Standards. Washington, D.C. Contract No. 68-01-6160.

LINDER, G., BERGMAN, H.L and MEYER, J.S. 1985. Anthracene bioconcentration in rainbow trout during single-compound and complex-mixture exposures. Environmental Toxicology and Chemistry. 4:549-558.

LU, P-Y., METACALF, R.L., PLUMMER, N. and MANDEL, D. 1977. The environmental fate of three carciongens: benzo(a)pyrene, benzidine, and vinyl chloride evaluated in laboratory model ecosystems. Archives of Environmental Contamination and Toxicology. 6:129-142.

McCARTHY, J.F., JIMENEZ, B.D. and BARBEE, T. 1985. Effect of dissolved humic material on accumulation of polycyclic aromatic hydrocarbons: structure-activity relationships. Aquatic Toxicology. 7:15-24.

SPACIE,-A., LANDRUM, P.F., and LEVERSEE, G.J. 1984. Uptake, depuration, and biotransformation of anthracene and benzo(a)pyrene in bluegill sunfish. Ecotoxicology and Environmental Safety. 7:330-341.

SELENIUM

AQUATIC TOXICITY

The aquatic toxicity of selenium has been reviewed by EPA (1987) and Eisler (1985). Three oxidation states can occur at pH 6.5-9.0 in aerobic surface waters: selenide, selenite (Se[IV]), and selenate (Se[VI]) (EPA 1987). Selenate is the predominant form in aerobic alkaline water. The conversion from selenite to selenate is quite slow. Elemental selenium occurs in sediments, but is insoluble in water (EPA 1987). Based on 11 species mean acute values, the acute toxicity of Se(IV) is 1.6-6.3 times more toxic than Se(VI) (EPA 1987). Some studies indicate that toxicity increases in softer water and that toxicity increases with increasing temperature. There is evidence that selenium protects aquatic and terrestrial life from the toxic effects of arsenic, cadmium, mercury, silver, and the herbicide paraquat (EPA 1987).

Invertebrates

Genus mean acute values for Se(IV) for 10 genera of freshwater invertebrates range from 340 ug/L for the amphipod Hyallela azteca to 203,000 ug/L for the leech Nephelopsis obscura (EPA 1987). For Se(VI), genus mean acute values for 11 genera range from 65.38 ug/L for the amphipod Gammarus pseudolimnaeus to 442,000 ug/L for the leech Nephelopsis obscura (EPA 1987). The only freshwater invertebrate chronic toxicity value is 1,999 ug/L (Se[IV]) for Daphnia magna (EPA 1987).

Species mean acute values for saltwater invertebrates for Se(IV) range from 1,040 ug/L for dungeness crab (Cancer magister) to greater than 10,000 ug/L for blue mussel (Mytilus edulis), based on eight species (EPA 1987). The only available chronic value for a saltwater invertebrate species exposed to Se(IV) is a value of 211.7 ug/L for mysid (EPA 1987).

Fish

Acute values for freshwater fish for Se(IV) range from 1,601 ug/L for fathead minnow (Pimephales promelas) to 35,000 ug/L for carp (based on 12 genera). The chronic toxicity values for Se(VI) in freshwater are 565.5 ug/L for fathead minnow and 2,891 ug/L for rainbow trout. Parental exposure of bluegill (Lepomis macrochirus) to dietary selenium resulted in teratogenesis and reduced survival in larvae (Woock et al. 1987). Larva from parents given 30 ug/g (selenomethionine) all died and all showed deformities. The controls had 95 percent survival and 2 percent with terata. Parent bluegill had significantly increased mortality at dietary selenium concentrations of 30 ug/g; mortality was not significant at the next highest concentration of 13 ug/g. Woock et al. (1987) found dietary organoselenium was more toxic than inorganic selenium.

The genus mean acute values for Se(IV) in saltwater range from 599 ug/L for haddock (Melanogrammus aeglifinus) to 17,350 ug/L for the fourspine stickleback (Apeltes quadracus) (based on 15 genera) (EPA 1987). No saltwater mean acute values for Se(VI) were reported in EPA (1987). The only chronic value for a saltwater fish species exposed to Se(IV) is 675.2 ug/L for the sheepshead minnow (Cyprinodon ariegatus) (EPA 1987). No chronic studies have been performed with saltwater organisms exposed to Se(VI) (EPA 1987).

Species mean acute values for some fish species associated with Aberdeen Proving Ground are available in EPA (1987). For Se(IV) the acute values for freshwater species are: carp (Cyprinus carpio) 35,000 ug/L, white sucker (Ictalurus catus) 30,180 ug/L, bluegill 28,500 ug/L, goldfish (Carassius auratus) 26,100 ug/L, channel catfish (Ictalurus puntatus) 13,600 ug/L, mosquitofish (Gambusia affinis)

12,600 ug/L, yellow perch (Perca flavescens) 11,700 ug/L, and striped bass (Morone saxatilis) 1,783 ug/L. The values for Se(VI) are: channel catfish 66,000 ug/L and bluegill 63,000 ug/L. For saltwater fish the values for Se(IV) are: winter flounder (Pseudopleuronecthes americanus) 14,650 ug/L, Atlantic silverside (Menidia menidia) 9,725 ug/L, and striped bass 1,550 ug/L.

Amphibians

Browne and Dumont (1979 in Eisler 1985) reported a 96-hour LC_{50} of 4,000 ug/L for embryo of the frog Xenopus laevis. The 7-day LC_{50} for the tadpole of the same species is 1,500 ug/L.

Plants

Toxicity values for freshwater plants for Se(IV) range from 522 ug/L for the green alga Scenedesmus quadricauda (based on the 8-day incipient inhibition level) to 30,000 ug/L for the blue-green alga Anacystis nidulans (based on the 10-18 day LC_{50} (EPA 1987). For Se(VI) the highest no effect level is 10 ug/L; this level did not reduce growth in the green alga Ankistrodesmus falcatus during 14 days of exposure (EPA 1987). The lowest effect level is 100 ug/L; reduced growth occurred in the green alga Scenedesmus obliquus during 14 days of exposure (EPA 1987). The blue-green alga Anacystis nidulans was the least sensitive species tested (also the case with Se[IV] as indicated above). The estimated 6-18 day EC_{50} for this species is 39,000 ug/L (EPA 1987).

Toxicity values for saltwater plants are available only for diatom Skeletonema costatum with a 4-day EC_{50} (reduction in chlorophyll a) of 7,930 ug/L (EPA 1987).

Sediment Toxicity

No information was found on the toxicity of selenium in sediments.

Bioaccumulation

Laboratory closed-system microcosm studies indicated that selenomethionine was bioaccumulated much more than selenite or selenate (Besser et al. 1989). Bioconcentration factors were derived for three forms of selenium for zooplankton (daphnids), periphyton (algae and other attached organisms), and macrophytes (rooted plants and filamentous algae). The bioconcentration factors after 28 days were as follows:

	Selenate	Selenite	Selenomethionine
zooplankton	351	1,087	28,870
periphyton	141	755	16,836
macrophytes	72	363	3,266

A BCF of 800 has been reported for the marine invertebrate euphasiid (Meganyctiphanes norvegica), based on a 28-day study (Fowler and Benayoun 1976c in EPA 1987).

The whole-body BCF for bluegill exposed for 120 days is 470 (hardness = 25 mg/L as $CaCO_3$) (Lemly 1982 in EPA 1987). For the fathead minnow a muscle-BCF of 11.6 has been derived, based on 96 days of exposure (Adams 1976 in EPA 1987).

A whole-body BCF of 11.78 has been reported for striped bass (starved juveniles) exposed for 60 days (Klauda 1985 in EPA 1987).

Criteria

EPA has established acute and chronic ambient water quality criteria for freshwater of 20 ug/L and 5 ug/L, respectively (EPA 1987). The acute and chronic criteria for marine environments are 300 ug/L and 71 ug/L, respectively (EPA 1987).

TERRESTRIAL TOXICITY

The toxicity of selenium to terrestrial animals has been reviewed by Eisler (1985) and Wilber (1980). Selenium is an essential trace mineral. Recommended dietary concentrations for livestock are 0.1-0.3 mg/kg (dry weight) (NAS 1980). NAS (1980) indicated that toxic levels occur at only about 10-50 times the recommended dietary levels. Eisler (1985) also emphasized that there is a relatively narrow difference between dietary concentrations associated with selenium deficiency and selenium poisoning. Dietary sulfate can reduce the toxicity of selenium as selenate. However, sulfate is not effective in reducing the toxicity of selenite or organic selenium (NAS 1980).

Birds

Domestic chickens are extremely sensitive to selenium (Eisler 1985). Reduced hatching has been reported at dietary concentrations of 7-9 mg/kg. Hatching has also been adversely affected in Japanese quail (Coturnix japonica) at 6-12 mg/kg selenium in the diet (Eisler 1985). Mallards (Anas platyrhynchos) are apparently somewhat less sensitive to selenium than chickens and Japanese quail (Smith et al. 1988). Poor egg hatchability was observed in mallards at 25 mg/kg dietary selenium (as sodium selenite, SeL). At 10 mg/kg dietary selenium, reduced hatching in mallards was reported when introduced as selenomethionine (SeM) but not when introduced as SeL (Eisler 1985).

Selenium in agricultural drainage water has been reported as the cause of severe adverse effects in aquatic birds at Kesterson National Wildlife Refuge in California (Ohlendorf et al. 1988, Williams et al. 1989, Hoffman et al. 1988). Only one of 250 black-necked stilts (Himantopus mexicanus) that hatched during 1984 or 1985 survived beyond age class 2 (Williams et al. 1989). None of the 90 American avocets (Recurvirostra americana) that hatched in this same time period survived beyond age class 2. Concentrations of selenium in aquatic invertebrates, the main food items of these birds, ranged from 45-215 mg/kg. Assuming avocets and stilts weight 166 g and 316 g, respectively (Dunning 1984) and ingest food at a rate (g/day) of 0.648 ($Wt^{0.651}$), where wt is body weight (Nagy 1987), this dietary concentration corresponds to dosages in the range of 4.9 to 23 mg/kg bw for avocets and 3.8 to 19 mg/kg bw for stilts.

Heinz et al. (1989) has reported adverse reproductive effects in mallards at 8 mg/kg dietary selenium as SeM. At this dietary concentration, decreased numbers of offspring and increased numbers of malformations were reported. The offspring showed reduced survival and growth. The most common malformations involved the eyes, bill, legs, and feet. No effects on egg laying, egg size, shell thickness, fertility, or sex ratios occurred at 8 mg/kg. At 4 mg/kg no adverse effects were reported. In the same study, selenocystine caused no adverse effects at the highest concentration tested of 16 mg/kg.

In a study with mallard ducklings (from hatching to age 6 weeks), decreased growth and food consumption occurred at 20 mg/kg dietary selenium (as SeL or SeM) (Heinz et al. 1988). Significantly increased mortality occurred at 40 mg/kg dietary selenium (as SeL or SeM). No adverse effects were reported at 10 mg/kg SeM. At 10 mg/kg SeL (lowest dose tested) increased liver size was reported (Heinz et al. 1988).

Mallards given 2.2 mg/L selenium in drinking water (as SeM, only concentration tested) had delayed-type hypersensitive response to tuberculin M. bovis and increased levels of plasma glutathione peroxidase (GPX) and serum alanine aminotransferase (ALT) (Fairbrother and Fowles 1990). At 3.5 mg/L SeL in drinking water, mallards had increased ALT. No changes were observed at 0.5 mg/L SeL (Fairbrother and Fowles 1990).

Black-crowned night herons (Nycticorax nycticorax) are apparently less sensitive to selenium than mallards (Smith et al. 1988). Egg hatching was not affected in Black-crowned night herons fed 10 mg/kg dietary selenium as SeM. There were also no effects on organ weight, hemoglobin, hematocrit, and eggshell thickness, and there were no malformations. The only effects reported at this concentration were reduced radius-ulna and femur lengths and increased liver malondialdehyde (Smith et al. 1988). Assuming a night heron weighs 883 g (Dunning 1984) and ingests food at a rate (g/day) of 0.648 ($wt^{0.651}$), where wt is body weight (Nagy 1987), the 10 mg/kg dietary level corresponds to a dosage of 0.61 mg/kg bw.

Mammals

Signs of acute selenium poisoning in livestock include abnormal movements, diarrhea, rapid pulse, and labored breathing. Death is caused by respiratory failure. The lowest lethal oral doses for livestock are 3.3 mg/kg body weight for horses and mules, 11 mg/kg-body weight for cattle, and 15 mg/kg body weight for swine (Eisler 1985).

In livestock three levels of selenium poisoning are recognized in the field: acute, low level of the "blind staggers" type, and low level of the "alkali disease" type. Acute poisoning in livestock occurs at about 400-800 mg/kg selenium in plants. Doses of about 3.2 to 12.8 mg/kg-body weight are lethal to sheep. Livestock with blind staggers have impaired vision, some respiratory failure, and incoordination. There is some evidence that these symptoms are caused by alkaloids in the plants rather than selenium (NAS 1980). The alkali disease type poisoning is associated with feeds with 5 to 40 ppm selenium being consumed over a period of weeks or months. Signs of this type of poisoning include lameness, hoof malformations, loss of hair from mane or tail, and emaciation (NAS 1980, Eisler 1985).

NAS (1980) suggested a maximum tolerable level for dietary selenium of 2 mg/kg for livestock and poultry. This corresponds to a dose of 0.06 mg/kg bw for rabbits and 0.25 mg/kg bw for birds (based on dietary conversion factors presented in Lehman 1954). Puls (1988) recommended a maximum level of selenium in drinking water for livestock of less than 0.01 mg/L.

Plants

Solubilized selenium has been found to be readily absorbed by plants. Phytotoxic symptoms include leaf chlorosis, root thickening, and growth reduction. Phytotoxic concentrations of selenium reported by Kabata-Pendias and Pendias (1984), based on four studies, were 5 to 10 mg/kg (arithmetic mean = 8.75 mg/kg).

REFERENCES

BESSER, J.M., HUCKINS, J.N., LITTLE, E.E. and LaPOINT, T.W. 1989. Distribution and bioaccumulation of selenium in aquatic microcosms. Env. Pollut. 62:1-12.

DUNNING, J.B. 1984. Body Weights of 686 Species of North American Birds. Western Bird Banding Assoc. Monograph No. 1.

EISLER, R. 1985. Selenium Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. Department of the Interior, U.S. Fish and Wildlife Service. Biological Report 85(1.5).

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Ambient Water Quality Criteria for Selenium-1987. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-87-006. PB88-142237.

FAIRBROTHER, A. and FOWLES, J. 1990. Subchronic effects of sodium selenite and selenomethionine on several immune functions in mallards. Arch. Env. Contam. Toxicol. 19:836-844.

HEINZ, G.H, HOFFMAN, D.J. and GOLD, L.G. 1988. Toxicity of organic and inorganic selenium to mallard ducklings. Arch. Env. Contam. Toxicol. 17:561-568.

HEINZ, G.H, HOFFMAN, D.J. and GOLD, L.G. 1989. Impaired reproduction of mallards fed an organic form of selenium. J. Wildl. Management. 53(2):418-428.

HOFFMAN, D.J., H.M. OHLENDORF, T.W. ALDRICH. 1988. Selenium teratogenesis in natural populations of aquatic birds in central California. Arch. Env. Cont. & Tox. 17:519-525.

KABATA-PENDIAS, A. and PENDIAS, H. 1984. Trace Elements in Soil and Plants. CRC Press. Boca Raton, Florida.

LEHMAN, A.J. 1954. Appraisal of the study of chemicals in food, drugs and cosmetics. U.S. Assoc. of Food and Drug Officials. 18:66.

NAGY, K.A. 1987. Field metabolic rate and food requirement scalining in mammals and birds. Ecol. Mono. 57:111-128.

NATIONAL ACADEMY OF SCIENCES (NAS). 1980. Mineral Tolerance of Domestic Animals. National Research Council, Subcommittee on Mineral Toxicity in Animals. Washington, D.C.

OHLENDORF, H.M. 1988. Selenium toxicosis in wild aquatic birds. J. Tox. Env. Health. 24:67-92.

PULS, R. 1988. Mineral Levels in Animal Health. Diagnostic Data. Sherpa International. Clearbrook, British Columbia.

SMITH, G.J., HEINZ, G.H., HOFFMAN, D.J., SPANN, J.W. and KRYNITSKY, A.J. 1988. Reproduction in black-crowned night-herons fed selenium. Lake and Reservoir Management. 4(2):175-180.

WILLIAMS, M.L., HOTHEM, R.L. and OHLENDORF, H.M. 1989. Recruitment failure in American avocets and black-necked stilts nesting at Kesterson Reservoir, California, 1984-1985. The Condor. 91:797-802.

WILBER, C.G. 1980. Toxicology of selenium: a review. Clinical toxicology. 17(2):171-229.

WOOCK, S.E., GARRETT, W.R., PARTIN, W.E. and BRYSON, W.T. 1987. Decreased survival and teratogenesis during laboratory selenium exposures to bluegill, Lepomis macrochirus. Bull. Env. Cont. & Tox. 39:998-1005.

AQUATIC TOXICITY

Silver (Ag) is one of the most toxic metals to aquatic organisms, and was particularly toxic to development stages of rainbow trout (Birge et al. 1981). Although silver can exist in several oxidation states, only the silver (I) occurs in substantial concentrations in natural waters (EPA 1987). The toxicity of silver in aquatic organisms is primarily observed as separation and disruption of the gill epithelium (EPA 1987). Katz (1979 in EPA 1987) suggested that this effect may be the result of silver ions reacting directly at the gill membrane or as the indirect result of a hematological osmotic balance. Toxicity to silver appears to be pH independent, however water hardness has an antagonistic effect on the acute toxicity of silver (EPA 1985 in EPA 1987).

Invertebrates

Based on available information, the five most sensitive genera to silver are freshwater arthropods. Daphnia magna are the most acutely sensitive animal species tested; the lowest estimated species mean acute value of 0.9 ug/L and the lowest acute value reported is 0.6 ug/L at a hardness of 38-40 ug/L as $CaCO_3$ (EPA 1987). The acute mean species values for the other four genera are the mayfly (Leptophlebia sp.) 2.2 ug/L; the cladoceran (Ceriodaphnia reticulata), 3.924 ug/L; the amphipod (Gammarus pseudolimnaeus) 4.5 ug/L; and the amphipod (Crangonyx pseudogracilis) 5 ug/L (EPA 1987). All values were determined at a hardness of 50 mg/L as $CaCO_3$ or lower, except for the cladocerans (EPA 1987). The 14-day LC_{50} for the mayfly, Ephemerella grandis, is less than 1 ug/L (hardness = 30-70 mg/L as $CaCO_3$) (Nehring 1976 in EPA 1987). The stonefly, Pteronarcys californica, is nearly as sensitive with a 14-day LC_{50} of 4 to 9 ug/L (hardness = 30-70 mg/L as $CaCO_3$) Nehring (1973, 1976 in EPA 1987). Chronic toxicity values of 2.6 to 28.6 ug/L have been reported for Daphnia magna (EPA 1987). No chronic values for aquatic insects or amphipods were reported by EPA (1987).

Marine invertebrates are generally more resistent to acute toxicity from zinc than freshwater invertebrates. The genus Crassostrea, the oyster, is the most sensitive genus to silver of the 21 marine animal species tested (EPA 1987). Species mean acute values for the eastern oyster (Crassostrea virginica) and the Pacific oyster (Crassostrea gigas) are 14.15 ug/L and 14.21 ug/L, respectively (EPA 1987). For the seven other saltwater invertebrates mean species values range from 13.3 ug/L for the copepod (Acartia clausi) to greater than 838 ug/L for the sand shrimp (Crangon sp.) (EPA 1987). Nelson et al. (1988) determined 96-hour LC_{50}s of 2,250 ug/L, 2,950 ug/L, and 159 ug/L for juvenile bay scallops (Agropecten irradians), surf clams (Spisula solidissima), and blue mussels (Mytilus edulis), respectively. A 5-day EC_{50} (development of larvae to pluteus stage) of 14.9 ug/L was reported for the sea urchin (Strongylocentratus purpuratus) (EPA 1987). Chronic toxicity values of 15 to 87.8 ug/L have been reported for Mysidopsis bahia (EPA 1987). No other chronic values were found for marine invertebrates (EPA 1987).

Fish

Freshwater fish are sensitive to the toxic effects of silver. The genus mean acute values for silver in freshwater fish range from 8.163 ug/L for the speckled dace(Rhinichthys osculus) to 17.3 ug/L for the channel catfish (Ictalurus punctatus), based on 7 genera (EPA 1987). Goetti and Davies (1978 in EPA 1987) estimated acute values for speckled dace of 4.9 ug/L for soft water (hardness = 30 mg/L as $CaCO_3$) and 13.6 ug/L for hard water (hardness = 250 mg/L as $CaCO_3$). The second most sensitive freshwater fish species is the mottled sculpin (Cottus bairdi) based on the species mean acute values

for 7 genera. Acute values of 5.3 ug/L for soft water (hardness = 30 mg/L as $CaCO_3$) and 13.6 ug/L for hard water (hardness = 250 mg/L as $CaCO_3$) have been reported for this species by Goetti and Davies (1978 in EPA 1987). The acute values for the fathead minnow (Pimephales promelas), based on 30 studies, ranged from 3.9 ug/L for soft water (hardness = 33 mg/L as $CaCO_3$) to 150 ug/L for hard water (hardness = 250 mg/L as $CaCO_3$) (EPA 1987). A similar range of toxicity to silver has been reported for the rainbow trout (Salmo gairdneri) from 32 studies (EPA 1987). The chronic maximum acceptable toxicant concentration (MATC) for rainbow trout, based on mortality after 18 months exposure, was 0.09 to 0.17 ug/L in soft water (hardness = 27.5 mg/L as $CaCO_3$) (Davies et al. 1978). At 0.17 ug/L premature hatching of eggs occurred and fry showed reduced rates of growth (Davies et al. 1978). Survival of rainbow trout and fathead minnow fry was reduced as a result of exposure to similar concentrations of silver in two early life-stage studies, at 0.51 ug/L (hardness = 36 mg/L as $CaCO_3$) and 0.65 ug/L (hardness = 45.1 mg/L as $CaCO_3$), respectively (EPA 1987). No effects were observed in these two species at 0.36 ug/L and 0.37 ug/L, respectively (EPA 1987). Chronic values from these studies are: 0.12 ug/L and 0.43 ug/L for rainbow trout, and 0.49 ug/L for the fathead minnow.

The most sensitive saltwater fish tested is the summer flounder (Paralichthys dentatus) with a species mean acute value for silver of 18.08 ug/L (EPA 1987). The quachog is the second most sensitive to silver with a species mean acute value of 21 ug/L (EPA 1987). For nine other saltwater fish the species mean acute values range from 110.1 ug/L for the Atlantic silverside (Menidia) to 2,700 ug/L for the mummichog (Fundulus heteroclitus) (EPA 1987). A significant increase in mortality of winter flounder larvae (Pseudopleuronectes americanus) was observed after 18 days exposure to 92 ug/L silver (Klein-MacPhee 1984 in EPA 1987). Growth was significantly reduced in these larvae by 180 ug/L; no effect on growth was observed at 54 ug/L. No chronic values are available for saltwater fish in the EPA (1987) review.

Toxicity data are available for several of the fish species found at or near the Aberdeen Proving Grounds. Species mean acute values for freshwater fish are 13 ug/L for the bluegill and 17.3 ug/L for the channel catfish. Acute toxic effects values, based on death and deformity, of 111 ug/L (hardness = 93-107) and 30 ug/L (hardness = 195) have been reported for embryo-larvae of the largemouth bass (Micropterus salmoides) and the goldfish (Carassus auratus) (Birge et al. 1978 in EPA 1987). The species mean acute values for saltwater fish species are Atlantic silverside (Menidia menidia) 110.1 ug/L; winter flounder (Psedopleuronectes americanus) 196.3 ug/L; four-spine stickleback, (Apeltes quadracus), 546.6 ug/L; mummichog, (Fundulus heteroclitus), 2,700 ug/L. The Atlantic silverside was ranked 7th out of 19 saltwater animal species reported, and it is the 3rd most sensitive saltwater fish, whereas the mummichog was the most resistent to silver of the saltwater animal species tested (EPA 1987). Winter flounder larvae exhibited a significant increase in mortality from 92 ug/L silver for 18 days (EPA 1987).

Amphibians

Toxicity information for amphibians is limited. An 8-day LC_{50} of 240 ug/L has been reported for the marbled salamander (Ambystoma opacum) at a hardness of 93-105 mg/L as $CaCO_3$ (Birge et al. 1978 in EPA 1980). The 7-day EC_{50}s (death and deformity) are 240 ug/L (for hardness = 93-105 mg/L as $CaCO_3$) and 10 ug/L (for hardness = 195 mg/L as $CaCO_3$) for embryo-larvae of the marble salamander and the narrow-mouthed toad (Castrophryne carolinensis). The same authors reported that the salamander was less sensitive than embryo-larvae of rainbow trout (Salmo gairdneri) and largemouth bass (Micropterus salmoides).

Plants

In general, aquatic plants are less sensitive to silver than the most sensitive aquatic animals, thus plants will be protected if the most sensitive animals are protected (EPA 1987). Based on tests with 22 species, concentrations causing adverse effects (including growth inhibition and death) ranged from 1.9 to 7,500 ug/L.

Sediment Toxicity

Barrick and Beller (1989) reported sediment toxicity in terms of apparent effect thresholds (AETs). They report AETs of > 0.56, 6.1, and >6.1 mg/kg for oysters, amphipods, and benthic invertebrates, respectively.

Bioaccumulation

Studies by Nehring (1976 in EPA 1987) provide the only bioconcentration data for freshwater invertebrates. BCFs of 17.4 to 84.4 for mayfly (Ephemerella grandis) and 14.4 to 36.6 for stonefly (Pteronarcys californicus) were determined in naiads postmortem following 1 to 14 days of exposure to silver and hardnesses of 30 to 70 mg/L as $CaCO_3$.

The highest soft tissue-bioconcentration factor (BCF) reported in studies with the blue mussel (Mytilus edulis) was 6,500 during 12 to 21 months of exposure to silver (as silver nitrate) (Calabrese et. al. 1984 in EPA 1987). The BCF values decreased with increasing concentrations of silver in water and achieved a maximum value after 12 months of exposure. Fisher et al. (1984 in EPA 1987) determined BCFs for two marine invertebrate species exposed to silver cyanide for 12 hours: 13,000 for the green alga Dunaliella tertiolecta), and 34,000 for the diatom (Thalassiosira pseudonana).

EPA (1987) reported BCFs of 11 and 19 for the largemouth bass (Micropterus salmoides) muscle tissue after a 120-day exposure to 1 and 10 ug/L (silver nitrate). Whole-body BCFs of 15 and 150 have been reported for bluegill (Lepomis macrochirus) exposed to concentrations of 10 and 100 ug/L for 180 days (Cearley 1971 in EPA 1987).

Bioconcentration factors for aquatic plants ranged from less than detectable to 150 (EPA 1987).

Criteria

The EPA (1987) concluded that although water hardness may influence silver toxicity to aquatic animals, there is insufficient data available at medium and high hardnesses (greater than 75 mg/L as $CaCO_3$) upon which to derive a national freshwater criterion based upon hardness. Therefore, the freshwater criterion is based on weighted toxicity data from soft waters and might be overly protective of aquatic organisms in hard waters (EPA 1987). For freshwater, EPA (1987) has proposed that the 4-day average concentration criterion (chronic criterion) for silver is 0.12 ug/L (not to be exceeded more than once every three years on the average) and the 1-hour average concentration (acute criterion) is 0.92 ug/L. For saltwater aquatic organisms, the acute and chronic criteria are 7.2 ug/L and 0.92 ug/L, respectively. EPA considers the acid-soluble measurement of silver to provide the more toxicologically and practical measurement for aquatic life criteria; however, because there is no EPA-approved method for this measurement, EPA suggests that measurement of acid-soluble and total recoverable silver be evaluated (EPA 1987).

TERRESTRIAL TOXICITY

Information on the toxicity of silver to wildlife is not available, however poultry and rats have been relatively well studied. Silver is not known to have any biological functions in animals (NAS 1980). Silver causes multiple deficiencies of vitamin E, selenium, and copper, and the symptoms associated with these deficiencies are manifested (NAS 1980).

Birds

Turkeys showed decreased weight gain at 300 ppm (Jensen et al. 1974 in NAS 1980). In the same study at 900 ppm toxic symptoms included enlarged heart, dystrophic gizzard musculature, and severely depressed weight gain. In another study with turkeys no adverse effects were observed at 100 ppm (Bunyan et al. 1968 in NAS 1980). Chickens also showed no adverse effects at diets up to 100 ppm silver, but at 200 ppm increased mortality was observed (Hill et al. 1964 in NAS 1980). NAS (1980) has recommended a maximum tolerable level of dietary silver for poultry of 100 ppm.

Mammals

Toxic effects in rats include decreased weight gain, liver necrosis, and increased mortality (NAS 1980). The maximum tolerable level of silver in the diet for swine is 100 ppm (NAS 1980).

Plants

The National Research Council (1977) found no adverse effects to plants concentrations as high as 100 mg/kg in the soil. Kabata-Pendias and Pendias (1984) report a phytotoxic concentration for silver of 2 mg/kg.

REFERENCES

BARRICK, R.C. and BELLER, H.R. 1989. Reliability of sediment quality assessment in Puget Sound. In: Oceans '89 Proceedings. Inst. of Electrical and Electronic Engineers. Piscataway, NJ. Publ. No. 89CH2780-5.

BIRGE, W.J., BLACK, J.A. and RAMEY, B.A. 1981. The reproductive toxicology of aquatic contaminants. In: Hazard Assessment of Chemicals: Current Developments. Volume I. Arndemire Press. New York.

DAVIES, P.H., GOETTL, J.P., and SINLEY, J.R. 1978. Toxicity of silver to rainbow trout (Salmo gairdneri). Water Research. 12:113-117.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Silver. Office of Water Regulations and Standards. Washington, D.C. EPA-440/5-80-071.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Ambient Water Quality Criteria for Silver - Draft. Office of Water Regulations and Standards. Washington, D.C.

KABATA-PENDIAS, A. and PENDIAS, H. 1984. Trace Elements in Soil and Plants. CRC Press. Boca Raton, Florida

NATIONAL ACADEMY OF SCIENCES (NAS). 1980. Mineral Tolerance of Domestic Animals. National Research Council, Subcommittee on Mineral Toxicity in Animals. Washington, D.C.

NATIONAL RESEARCH COUNCIL (NRC). 1977. Drinking Water and Health. National Academy of Sciences. Washington, D.C.

NELSON, D.A., MILLER, J.E. and CALABRESE, A. 1988. Effect of heavy metals on bay scallops, surf clams, and blue mussels in acute and long-term exposures. Archives of Environmental Contamination and Toxicology. 17:595-600.

SULFATE

AQUATIC TOXICITY

Patrick et al. (1968) reported a LC_{50} of 1,900 mg SO_4^{2-}/L in diatoms, and LC_{50} of 13,500 mg SO_4^{2-}/L in bluegill sunsfish (Lepomis macrochirus).

TERRESTRIAL TOXICITY

No information was found on the toxicity of sulfate to wildlife. Toxicity information for domestic species is presented below.

Birds

NAS (1984) reports that sulfate concentrations as low as 8,100 mg/kg diet resulted in reduced egg production in chickens. A sulfate concentration of 14,000 mg/kg diet is associated with reduced growth in immature chickens.

Mammals

In domestic grazing animals, sulfate toxicity is indirect. Copper deficiency diseases have been reported in lambs following the consumption of herbage naturally high in sulfate or from grazing in mining areas rich in sulfur materials (Underwood 1971 in Gough et al. 1979). Cattle drinking water containing 3590 mg/L and 2104 mg/L of SO_4^{2-} eventually weakened and died (Allison 1930). However, cattle weighing 275 kg exposed for 30 days to 1,260 mg SO_4^{2-} /liter in drinking water showed no adverse effects on milk production (NAS 1980). Assuming a cow drinks 58 liters water per day (APHIS 1987), this drinking water concentration corresponds to a dosage of approximately 270 mg/kg bw. A toxicity value of 2.7 mg/kg is derived by applying an uncertainty factor of 100 (for interspecies differences and for subchronic exposures) to the NOAEL for cattle.

REFERENCES

ALLISON, I.S. 1930. The problem of saline drinking waters. Science. 71:559.

ANIMAL AND PLANT HEALTH INSPECTION SERVICE (APHIS). 1987. Rangeland Grasshopper Cooperative Management Program. Final Environment Impact Statement - 1987. U.S. Department of Agriculture. APHIS FEIS 87-1.

GOUGH, L.P., SHACKLETTE, H.T., AND CASE, A.A. 1979. Element concentrations toxic to plants, animals, and man. U.S. Government Printing Office. Washington, DC. Geological Survey Bulletin 1466.

NATIONAL ACADEMY OF SCIENCES (NAS). 1980. Mineral Tolerance of Domestic Animals. National Research Council, Subcommittee on Mineral Toxicity in Animals. Washington, D.C.

NATIONAL ACADEMY OF SCIENCES (NAS). 1984. Nutrient Requirements of Poultry. 8th Revised Edition. Chapter 9: Toxic levels of elements. National Academy Press. Washington, DC.

AQUATIC TOXICITY

The toxicity of thallium to aquatic organisms has been reviewed by EPA (1980). No information was reported in EPA (1980) on the mechanism of toxicity. In addition, insufficient information is available to determine the effects of water quality (such as water hardness and pH) on the toxicity of thallium (EPA 1980).

Invertebrates

Acute EC_{50}s of 910 and 2,180 ug/L have been reported for Daphnia magna; the species mean acute value is 1,400 ug/L (EPA 1980). The chronic value for Daphnia magna, determined from a life-cycle test, is 130 ug/L (EPA 1980).

The only acute toxicity value reported in EPA (1980) for marine invertebrates is the acute value for mysid (Mysidopsis bahia) of 2,130 ug/L. Cessation of development (blastula) of sea urchin (Paracentrotus lividus) eggs occurred at 41,000 ug/L (EPA 1980).

Fish

Acute values (based on 96-hour LC_{50}s) for fathead minnow and bluegill are 1,800 ug/L and 126,000 ug/L, respectively (EPA 1980). A 28-day LC_{50} of 180 ug/L was reported for rainbow trout (Pickering et al. 1983). Juvenile Atlantic salmon, exposed for approximately 108 days, had forty percent mortality at 20 ug/L (EPA 1980). In the same study, seventy percent mortality occurred at 45 ug/L. Two embryo-larval studies with fathead minnows reported chronic toxicity values of 57 ug/L (Kimball manuscript in EPA 1980) and less than 40 ug/L (EPA 1978 in EPA 1980).

Acute values are available for two species of marine fish: sheepshead minnow (Cyprinodon variegatus) 20,900 ug/L and Tidewater silverside (Menidia beryllina) 24,900 ug/L (EPA 1980). The chronic value for the sheepshead minnow is 6,000 ug/L, based on an embryo-larval study (EPA 1980). No other chronic data are available for saltwater fish.

The only fish species associated with Aberdeen for which there are toxicity values are bluegill and Tidewater silverside. The toxicity values for these species have been cited above.

Amphibians

Very little information is available on the toxicity of thallium to amphibians. Mortality occurred in frog embryos after 56 days of exposure to 409 ug/L thallium (Dilling and Healey 1926 in EPA 1980).

Plants

Toxicity information for aquatic plants exposed to thallium is limited. The 96-hour EC_{50} (based on cell numbers) for the alga Selenastrum capricornutum is 100 ug/L (EPA 1980).

Fifty percent inhibition of photosynthesis was reported in the saltwater algae Dunaliella tertiolecta and Phaeodoactylum tricornutum at thallium concentrations of 4,080 and 51,200 ug/L, respectively (EPA 1980).

Sediment Toxicity

No information is available on the toxicity of thallium in sediments.

Bioaccumulation

A whole body BCF of 34 has been reported for bluegill exposed to thallium for 28 days (EPA 1980). A BCF of 130 was determined for muscle of Atlantic salmon exposed for 12.5 days (EPA 1980).

No BCFs are available for marine fish. BCFs of 12 and 18 have been reported for edible tissues of blue mussel (Mytilus edulis) and soft shell clam (Mya arenaria), respectively (EPA 1980). Exposure periods were 40 days for blue mussel and 88 days for soft shell clam.

Criteria

Insufficient data are available to develop ambient water quality criteria for thallium according to EPA (1986). For freshwater, the LOEL from acute studies is 1,400 ug/L and the LOEL from chronic studies is 40 ug/L (EPA 1986). EPA indicates that the lowest LOEL for a fish species is 20 ug/L based on approximately 108 days of exposure. Acute toxicity to marine life occurs at approximately 2,130 ug/L (EPA 1986). No chronic marine studies are available (EPA 1986).

TERRESTRIAL TOXICITY

Information on the toxicity of thallium to wildlife is limited. Thallium is not known to be an essential trace element in animals (Puls 1988); it is however, one of the more toxic metals (Klaassen et al. 1986). Thallium poisoning causes neural, hepatic, and renal effects, and it can also cause deafness and blindness (Klaassen et al. 1986).

Birds

Signs of acute poisoning include regurgitation, ataxia, hypoactivity, immobility, and loss of righting reflex (Hudson et al. 1984). The LD_{50} for thallium sulfate in starlings is 34.6-56.6 mg/kg (Schafer et al. 1983). Mallards (Anas platyrhynchos) are similar to starlings in their acute sensitivity to thallium. The LD50 for mallards, based on a study with 12 three month old birds, is 36.7 mg/kg (as thallium sulfate, 80.2% thallium) (Hudson et al. 1984). Ring-necked pheasants (Phasianus colchicus) are more acutely sensitive than starlings or mallards. The LD50 for pheasants is 23.7 mg/kg (as thallium sulfate), based on a study with 12 three to four month old birds (Hudson et al. 1984). An LD50 of 60-120 mg/kg (as thallium sulfate) was determined from a study with four immature golden eagles (Aquila chrysaetos) (Hudson et al. 1984).

Mammals

Acute toxicity values (LD_{50}s and LD_{LO}s) for thallium for five mammalian species range from 15-50 mg/kg-body weight (Stokinger 1981). The LD_{50} for rats is 30 mg/kg (Klaassen et al. 1986). The LD_{LO} for guinea pigs is 5 mg/kg (Stokinger 1981). The approximate lethal dose (ALD) for the deer mouse is 42 mg/kg and the LD_{50} is less than 654 mg/kg (Schafer and Bowles 1985). The minimum lethal dose for dogs is approximately 8 mg/kg (Puls 1988). Livestock given a single oral dose of 50 mg/kg died within 4 days (Puls 1988). In the same study, death occurred in 13 days at a single oral dose of 25 mg/kg. At 15 mg/kg the animals were sick, but the dose was not fatal.

Signs of chronic toxicity in rats include hair loss, cataracts, renal lesions, hindleg paralysis, liver degeneration, and brain injury (Klaassen et al. 1986). Death in rats occurred after four months of daily oral doses of 0.45 mg/kg (Browning 1969). Signs of toxicity included loss of appetite and weight. Dwarfism has been reported in rats exposed to thallium (Klaassen et al. 1986).

Plants

Carson and Smith (1977 in Adriano 1986) indicate that many crops show signs of toxicity at thallium concentrations in soil of 7 ppm. Tobacco plants are quite sensitive to thallium; toxic effects have been shown at 1 ppm in soil (Adriano 1986). Wheat is also relatively sensitive to thallium; adverse effects occurred at 1.4 ppm in soil, death occurred at 28 ppm (Adriano 1986). Plants may accumulate thallium up to 10 times the ambient concentration in soil (based on plant and soil dry weights) (Adriano 1986).

REFERENCES

ADRIANO, D.C. 1986. Trace Elements in the Terrestrial Environment. Springer-Verlag. New York.

BROWNING, E. 1969. Toxicology of Industrial Metals. 2nd edition. Appleton-Century-Crofts. New York.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Thallium. Washington, D.C. EPA 440/5-80-074.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Quality Criteria for Water. Office of Water Regulations and Standards. Washington, D.C.

HUDSON, R.H., TUCKER, R.K. and HAEGLE, M.A. 1984. Handbook of Toxicity of Pesticides to Wildlife. U.S. Department of the Interior, Fish and Wildlife Service. Washington, D.C. Resource Publication 153.

KLAASSEN, C.D., AMDUR, M.O. and DOULL, J. 1986. Casarett and Doull's Toxicology, The Basic Science of Poisons. Third Edition. Macmillan Publishing Company. New York.

PICKERING, Q.H., HUNT, E.P., PHIPPS, G.L., ROUSH, T.H., SMITH, W.E., SPEHAR, D.L., STEPHENS, C.E., and TANNER, D.K. 1983. Effects of pollution on freshwater fish and amphibians. JWPCF. 55(6):840-862.

PULS, R. 1988. Mineral Levels in Animal Health. Sherpa International. Clearbrook, British Columbia.

SCHAFER, E.W. Jr., BOWLES, W.A. Jr., and HURLBUT, J. 1983. The acute oral toxicity, repellency, and hazard potential of 998 chemicals to one or more species of wild and domestic birds. Arch Environ. Contam. Toxicol. 12:355-382.

SCHAFER, E.W. Jr., and BOWLES, W.A. Jr. 1985. Acute oral toxicity and hazard potential of 933 chemicals of house and deer mice. Arch. Environ. Contam. Toxicol. 14:111-129.

STOKINGER, H.E. 1981. The Metals. In: Clayton G.D. and Clayton, F.E. Patty's Industrial Hygiene and Toxicology. Volume 2A. 3rd Revised Edition. John Wiley and Sons. New York. Pp. 1493-2060.

AQUATIC TOXICITY

Thiodiglycol is a degradation product of mustard agent. Information regarding toxicity to aquatic life was not found in the literature. However, EPA (1988) has published a methodology which utilizes relationships between a chemical's physico-chemical properties (molecular weight, and octanol water partition coefficient, K_{ow}) and its toxicity. These structure activity relationships (SARs) are regression equations that may be used to derive toxicity estimates for organic chemicals with no available toxicity data. EPA presents SAR equations for 3 broad classes of organic chemicals: (1) neutral organics which are non-reactive and non-ionizable, (2) neutral organics which are reactive and show excess toxicity in addition to narcosis, and (3) surface active organic compounds such as surfactants and polycationic polymers. According to R.G. Clements at EPA[1], thiodiglycol may be grouped into the first class of chemicals, neutral organics. For this group, four freshwater and two saltwater species SAR equations are provided in EPA (1988). These SARs and an SAR for tadpoles derived by (Lipnick 1989) are presented below. The aquatic toxicity information that follows was derived using these equations.

1) Fish 96-hour LC_{50} SAR:
$\log LC_{50} = -0.94 \log K_{ow} + 0.94 \log (0.000068\ K_{ow} + 1) - 1.25$;
where LC_{50} is in moles/L.

2) Fish 14-day LC_{50} SAR:
$\log(1/LC_{50}) = 0.871$ logKow - 4.87; where LC_{50} is in umoles/L.

2) Sheepshead Minnow 96-hour LC_{50} SAR:
$\log (1/LC_{50}) = 0.73$ log Kow - 3.69; where LC_{50} is in umoles/L.

3) Daphnid 48-hour LC_{50} SAR:
$\log (1/LC_{50}) = 0.91$ logKow - 4.72; where LC_{50} is in umoles/L.

4) Mysid 96-hour LC_{50} SAR:
$\log (1/LC_{50}) = 1.25$ logKow - 4.83; where LC_{50} is in umoles/L.

5) Algae 3-hour EC_{50} SAR:
$\log EC_{50} = 8.865 - 1.0446$ logKow; where EC_{50} is in umoles/L.

6) Tadpole lowest observed effect concentration (LOEC) SAR:
$\log (1/LOEC) = 0.909$ log Kow + 0.727; where LOEC is in moles/L.

Thiodiglycol has an octanol-water partition coefficients (Kow) of 0.83 (logKow = -0.08) and a molecular weight of 122.2 g/mole. Molecular weight was used to convert concentrations from moles or micromoles/L to ug/L.

Invertebrates

A freshwater daphnid 48-hour LC50 of 7,580,000 ug/L and a saltwater mysid LC50 of 10,400,000 ug/L are calculated using the above SARs. These high values indicate that thiodiglycol is not very toxic to aquatic invertebrates.

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[1]Clements, Richard G. Personal communication with Richard G. Clements, EPA SAR hotline. December 18, 1990.

Fish

The 96-hour LC_{50} derived for freshwater fish is 82,000,000 ug/L, while that for the saltwater sheepshead minnow (Cyprinodon variegatus) is 684,300 ug/L. A 14-day LC_{50} for freshwater fish of 10,600,000 ug/L was also calculated.

Amphibians

A LOEC of $2.7x10^{8}$ ug/L is calculated for tadpoles. This high value indicates that thiodiglycol is probably not very toxic to tadpoles.

Plants

The estimated 3-hour EC_{50} for algae is $1.1x10^{11}$ ug/L. This value indicates that thiodiglycol is not acutely toxic to aquatic plants.

TERRESTRIAL TOXICITY

Mammals

Data regarding toxicity of thiodiglycol to terrestrial wildlife are not available. It is a mild skin and eye irritant in rabbits. Unlike mustard agent, thiodiglycol has no effect on the cardiovascular system after intravenous injection in rabbits or dogs (Anslow and Houck 1946). Based on oral LD_{50} values of 3,960 and 6,610 mg/kg for the guinea pig and rat respectively, it does not appear to be acutely toxic. Signs of toxicity in these animals resemble those of the glycols (Smyth et al. 1941 in Baronian 1988). Thiodiglycol sulfoxide and thiodiglycol sulfone fed to mice at 1,000 ppm in drinking water had no adverse effects (Cates and Moore 1946 in Baronian 1988).

Plants

A biological screen for herbicidal activity of thiodiglycol at 0.1 and 1 pound per acre conducted at Fort Detrick (Wiswesser and Frank 1975) found no effect in plants sprayed with thiodiglycol; plants tested were beans, oats, rice, soybeans, radishes, and morning glories.

Sax (1979 in Baronian 1988) reports that thiodiglycol exhibits low toxicity in several terrestrial species.

REFERENCES

ANSLOW, W.P. and HOUCK, C.R. 1946. Systemic pharmacology and pathology of sulfur and nitrogen mustards. In: Chemical Warfare Agents and Related Chemical Problems. Chapter 22, Summary Technical Report of Division 9. NDRC. Pp. 440-478 and 731-737.

BARONIAN, C. 1988. Chemical Stockpile Disposal Program - Final Programmatic Environmental Impact Statement. Program Executive Officer - Program Manager for Chemical Demilitarization. Aberdeen Proving Ground, Maryland.

CATES, M., and MOORE, S. 1946. Mustard gas and other sulfur mustards. In: Chemical Warfare Agents and Related Chemical Problems. Part I. Summary Technical Report of Division 9. NDRC. Pp. 30-58 and 664-672.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Estimating toxicity of industrial chemicals to aquatic organisms using structure activity relationships. Volume 1. Office to Toxic Substances. Washington, DC. EPA 560/6-88-001.

LIPNICK, R.L. 1989. A quantitative structure-activity relationship study of Overton's data on the narcosis and toxicity of organic compounds to the tadpole, Rana temporaria. Aquatic Toxicol. and Environ. Fate. ASTM STP 1007. 77:468-489.

SMYTH, H.F., JR., SEARTON, J., and FISCHER, L. 1941. The single dose toxicity of some glycols and derivatives. J. Indust. Hyg. Toxicol. 23: 259-268.

WISWESSER, W., and FRANK, J.R. 1975. Fort Detrick Screening Test for Herbicide Activity. Ft. Detrick, Maryland.

2,4,6-TRICHLOROANILINE

AQUATIC TOXICITY

Aquatic toxicity data for 2,4,6-trichloroaniline are extremely limited. Toxicity data presented below were obtained from the CIS-AQUIRE database (AQUIRE 1991).

Invertebrates

Knie et al. (1983 in AQUIRE 1991) report an EC_{50} of 6,000 ug/L for the water flea Daphnia magna. This is the only invertebrate species for which toxicity data were available.

Fish

Barnhart and Campbell (1972 in AQUIRE 1991) report a 96-hour LC_{50} of 1,000 to 10,000 ug/L, and a 30-day no-observed-effect concentration (NOEC) of 135 ug/L, for fathead minnow Pimephales promelas. Knie et al. (1983 in AQUIRE 1991) reported an LC_{50} of 2,300 ug/L for the golden orfe, Leuciscus idus.

Plants

Knie et al. (1983 in AQUIRE 1991) report an EC_{10} of >12,000 ug/L for green alga Haematococcus pluvialis.

Data regarding toxicity of 2,4,6-trichloroaniline to amphibians were not located in the literature.

Criteria

Due to lack of toxicity data, criteria have not been established for 2,4,6-trichloroaniline.

TERRESTRIAL TOXICITY

No data regarding toxicity of 2,4,6-trichloroaniline were located in the literature

REFERENCES

AQUATIC INFORMATION RETRIEVAL (AQUIRE). 1991. Computerized database. Chemical Information Systems, Inc. Baltimore, Maryland.

N-N'-BIS-(2,4,6-TRICHLOROPHENYL)UREA
(TCPU)

AQUATIC TOXICITY

No data were available regarding toxicity of TCPU to aquatic organisms. General estimates on the relative toxicity of TCPU may be inferred by examining physico-chemical properties of the chemical. TCPU has an octanol-water partition coefficient (Kow) of greater than 5, indicating that the chemical is persistent in aquatic environment and is likely to be resistant to chemical and biological alteration (Harvey et al. 1990). Although TCPU binds to sediment, the potential exists for benthic organisms to bioaccumulate this hydrophobic compound directly from the sediment.

Although no aquatic toxicity data currently exist for TCPU, structure-activity relationships (SARs) have been developed which relate the toxicological properties of chemicals to their lipophilicity or other chemical parameters such as molecular size or weight. Veith et al. (1983), Abernethy et al. (1988), and others have reviewed a large portion of the aquatic toxicity data base and have shown that increased toxicity can be correlated quantitatively with increasing lipophilicity. Specifically, they plot toxicity as a function of the log of the octanol-water partition coefficient (Kow) of a chemical .

EPA (1988) has published a methodology which utilizes SARs to derive toxicity estimates for organic chemicals with no available toxicity data. Specifically, relationships between chemical-specific Kows, molecular weight, and available toxicity data (LC_{50}s) were determined based on regression analysis. For the group of chemicals referred to as substituted ureas which includes TCPU, a SAR developed by Hansch (1969 in EPA 1988) is presented which predicts an algal 4-hour EC_{50} based on inhibition of photosynthesis. The SAR applies to substituted ureas with log Kow values up to and including 3.9. For chemicals with log Kow values between 3.9 and 7.9, the SAR does no apply, and for chemicals with log Kow values greater than 8.0, no adverse effects are expected at saturation.

Unfortunately, log Kow values for TCPU estimated using the methods of Hansch and Leo's (1979 in Dennis 1983) fragment constant (log Kow = 7.46) and Chiou et al.'s (1977 in Dennis 1983) solubility equation (log Kow = 5.47), are between 3.9 and 7.9, and therefore the SAR equation does not apply. However, data presented in EPA (1988) in support of this SAR present 4-hour EC_{50} values for 25 other substituted ureas. Four-hour EC_{50} values range from $2x10^{-4}$ ug/L for 3-(3,4,5-trichlorophenyl)-1,1-dimethylurea to 3 ug/L for 3-(4-acetylaminophenyl)-1,1-dimethylurea. These data indicate that if TCPU acts with the same mode of toxic action as these substituted ureas, toxic effects may be observed in algae at relatively low environmental concentrations.

TERRESTRIAL TOXICITY

Few data are available regarding the toxicity of TCPU to terrestrial organisms. Metker et al. (1987) investigated the acute toxicity of TCPU to rats and guinea pigs from a single oral dose. Animals dosed orally with the maximum concentration of 1,000 mg/kg TCPU (in drinking water) or 1,500 mg/kg in corn oil (by gavage) experienced no adverse effects during the 14 days of observation following exposure. Karel (1948 in Dennis 1983) reported results of a screening toxicity test on Wistar albino rats. At an oral dosage of 100 mg/kg, no animals died. They conclude that TCPU should not be included among those chemicals classified to possess extreme acute toxicity. No data regarding toxicity to birds or other terrestrial organisms were located in the literature.

Plants

No data are available regarding toxicity of TCPU to plants. However, in a study of 500 urea derivatives, Bruce and Zwar (1966 in Dennis 1983) found that about half of the N,N'-disubstituted type possessed cytokinin activity. Diphenylurea, a coconut milk cytokinin, stimulates cell division in the tobacco pith at levels as low as 0.1 ppm, and several substituted diphenylureas at levels as low as 0.1 ppm. Increased chlorination appears to result in loss of activity. N,N'-bis(3,4-dichlorophenyl)urea was effective at 2 ppm.

REFERENCES

ABERNETHY, S.G., MACKAY, D., and McCARTY, L.S. 1988. "Volume fraction" correlation for narcosis in aquatic organisms: the key role of partitioning. Environ. Toxicol. and Chem. 7:469-481.

DENNIS, W.H.,JR. 1983. An investigation of the presence of n,n'-bis(2,4,6-trichlorophenyl)urea in estuarine sediments of Aberdeen Proving Ground, MD (Edgewood Area). U.S. Army Medical Bioengineering Research and Development Laboratory. Fort Detrick, Maryland.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Estimating Toxicity of Industrial Chemicals to Aquatic Organisms Using Structure Activity Relationships. Volume 1. Office to Toxic Substances. Washington, D.C. EPA 560/6-88-001.

HARVEY, S.D., DAUBLE, D.D., McVEETY, B.D., FELLOWS, R.J., BEAN, R.M., CATALDO, D.A., CAMPBELL, J.A., and WENTSEL, R.S. 1990. Fate of n,n-bis-(2,4,6-trichlorophenyl)-urea in a freshwater sediment. U.S. Armament Munitions Chemical Command, Chemical Research, Development and Engineering Center. Aberdeen Proving Ground, Maryland. 21010-5423.

METKER, L.W., BALCZEWSKI, L.M., and HARVEY, J.G. 1987. Determination of the Acute Toxicity of n,n'-Bis-(2,4,6-trichlorophenyl) Urea in Male and Female Rats and Guinea Pigs. U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground, Maryland. Study No. 75-51-0523-87.

VEITH, G.D., CALL, D.J., and BROOKE, L.T. 1983. Structure-toxicity relationships for the fathead minnow, Pimephales promelas: narcotic industrial chemicals. Can. J. Fish. Aquat. Sci. 40:743-748.

AQUATIC TOXICITY

Aquatic toxicity data summarized in the AQUIRE database (AQUIRE 1990) were collected for the volatile organic chemicals (VOCs) at APG. The majority of the aquatic toxicity studies for VOCs to date are freshwater acute bioassays with mortality as the endpoint. Toxicity values are most frequently reported as a median lethal concentration (LC_{50}), which is the concentration in water which is lethal to 50 % of a test population. Other studies report median effect concentrations (EC_{50}s), lowest observed effect concentrations (LOECs), or no observed effect concentrations (NOECs). Toxicity data for each chemical are summarized in Table C-2.

Invertebrates

The majority of invertebrate toxicity data are from studies using the water flea Daphnia magna, a common invertebrate test organism. LeBlanc (1980) conducted toxicity tests using D. magna for a number of VOCs, and reported NOEC and LC_{50} concentrations. These values are shown under each chemical in Table C-2.

Fish

Tests using saltwater fish were not located in the literature. LC50 values for freshwater fish do not differ greatly from those for D. magna. Fish toxicity data are shown in Table C-2.

Amphibians

Toxicity data for amphibians were available for some VOCs; these data are included in Table C-2.

Aquatic Plants

Species of green algae are the most commonly used plant in aquatic toxicity tests. EC_{50} values range from 4,600 ug/L for ethylbenzene (Galassi et al. 1988 in AQUIRE 1990) to 2,290,000 ug/L for methylene chloride (Hutchinson et al. 1980) (see Table C-2). Based on this limited data, VOCs do not appear to be very toxic to aquatic plants.

Sediment Toxicity

Barrick and Beller (1989) present sediment toxicity values in terms of apparent effect thresholds (AETs). For tetrachloroethene, AETs for aquatic invertebrates are > 22 mg/kg organic carbon. For ethylbenzene, the AETs for these organsisms are > 3.8 mg/kg.

Bioaccumulation

Bioconcentration factors (BCFs) for bluegill sunfish (Lepomis macrochirus) exposed to 5 VOCs are reported in Barrows et al. (1980). BCFs range from 6 (chloroform) to 49 (trichloroethene), indicating that the potential for significant bioaccumulation is probably very low. The BCF data are included in Table C-2.

TABLE C-2

SUMMARY OF TOXICITY DATA FOR VOCS AT APG

CHEMICAL	ORGANISM	TYPE	DURATION	EFFECT	CONC.(ug/L)		REFERENCE (a)
BENZENE							
--acute							
	bluegill sunfish	ST	96 h	LC50	100,000		Johnson & Finley 1980
	channel catfish	ST	96 h	LC50	425,000		Johnson & Finley 1980
	fathead minnow	FT	96 h	LC50	15,100		Degraeve et al. 1982
	guppy	RE	96 h	LC50	28,600		Galassi et al. 1988
**	rainbow trout	FT	96 h	LC50	5,300		Degraeve et al. 1982
	medaka	NR	48 h	LC50	250,000		Slooff et al. 1983
	striped bass	FT	4 h	LOECbl	113		Macfarlane & Benville 1986
	crab	ST	96 h	LC50 in ML/L	6.61	*	Rao et al. 1988
	mussel	ST	24 h	LOEC	10,000		Sabourin & Tullis 1981
	great pond snail	ST	48 h	LC50	230,000		Slooff et al. 1983
**	clawed toad	ST	48 h	LC50	190,000		DeZwart & Slooff 1987
	salamander	ST	48 h	LC50	370,000		Slooff & Baerselman 1980
	water flea	ST	24 h	EC50im	18,000		Galassi et al. 1988
	water flea	ST	48 h	LC50	200,000		Leblanc 1980
	mayfly	ST	48 h	LC50	34,000		Slooff 1983
**	brine shrimp	ST	24 h	LC50	1,630		Abernethy et al. 1986
	green algae	ST	72 h	EC50gr	29,000		Galassi et al. 1988
--chronic							
	guppy	RE	14 d	LC50	63,500		Koneman 1981
**	rainbow trout	FT	to p-hatch	LC50	8,250		Black et al. 1982
	rainbow trout	FT	to hatch	LC50	8,640		Black et al. 1982
	crab	ST	30 d	LOECbc IN ML/L	0.725		Rao et al. 1988
**	leopard frog	FT	to p-hatch	LC50	3,660		Black et al. 1982
	salamander	FT	to p-hatch	LC50	5,120		Black et al. 1982
	green algae	ST	7 d	NOECgr	1,400,000		Bringmann & Kuhn 1980
	red algae	RE	14 d	LOECgr	34,260		Thursby et el. 1985
CARBON TETRACHLORIDE							
--acute							
	bluegill sunfish	ST	96 h	LC50	27,000		Buccafusco et al. 1981
	water flea	ST	48 h	LC50	35,000		Leblanc 1980
	water flea	ST	48 h	NOEC	7,700		Leblanc 1980
--chronic							
	guppy	NA	14 d	LC50	67,100		Koneman 1981
	green algae	ST	7 d	LOEC	600,000		Bringmann & Kuhn 1980
--BCF							
	bluegill sunfish	FT	21 d	BCF	30		Barrows et al. 1980
CHLOROBENZENE							
--acute							
	bluegill sunfish	ST	96 h	LC50	,000		Buccafusco et al. 1981
	fathead minnow	ST	96 h	LC50	,600	*	Mayes et al. 1983
**	rainbow trout	FT	96 h	LC50	-,700		Dalich et al. 1982
	zebrafish	ST	48 h	LC50	-,500		Calamari et a.. 1983
	brine shrimp	NA	24 h	LOECre	25,000		Kerster & Schaeffer 1983
	water flea	ST	48 h	NOEC	10,000		Leblanc 1980
**	water flea	ST	48 h	LC50	10,850	*	Cowgill et al. 1985
	green algae	ST	96 h	EC50gr	12,500		Galassi & Vighi 1981
--chronic							
**	guppy	RE	14 d	LC50	19,100		Koneman 1981
	rainbow trout	FT	30 d	LOEC	2,100		Dalich et al. 1982
	rainbow trout	FT	30 d	LOECbc	2,900		Dalich et al. 1982
	leopard frog	FT	to p-hatch	LC50	1,200		Black et al. 1982
	salamander	FT	to p-hatch	LC50	1,150		Black et al. 1982
	water flea	RE	14 d	EC50re	2,500		Calamari et al. 1983
	green algae	ST	7 d	NOECgr	390,000		Bringmann & Kuhn. 1980

TABLE C-2 (Continued)

SUMMARY OF TOXICITY DATA FOR VOCS AT APG

CHEMICAL	ORGANISM	TYPE	DURATION	EFFECT	CONC.(ug/L)		REFERENCE
CHLOROFORM							
--acute							
	largemouth bass	FT	96 h	LC50	51,000	*	Anderson & Lusty 1980
	channel catfish	FT	96 h	LC50	75,000	*	Anderson & Lusty 1980
**	bluegill sunfish	FT	96 h	LC50	17,900	*	Anderson & Lusty 1980
	rainbow trout	FT	96 h	LC50	18,000	*	Anderson & Lusty 1980
**	Chlorella sp.	ST	3 h	EC50 ps	382,000		Hutchinson et al. 1980
	Chlamydomonas sp.	ST	3 h	EC50 ps	406,000		Hutchinson et al. 1980
--chronic							
**	rainbow trout	FT	28 d E-L	LC50	1,240		Black and Birge 1980
	bluegill sunfish	FT	7 d E-L	LC50	2,030		Black and Birge 1980
	guppy	NA	14 d	LC50	102,000		Koneman 1981
	Scenedesmus sp.	ST	7 d	LOECgr	1,100,000		Bringmann & Kuhn 1980
--BCF							
	bluegill sunfish	FT	14 d	BCF	6		Barrows et al. 1980
1,2-DICHLOROETHANE							
--acute							
	bluegill sunfish	ST	96 h	LC50	430,000		Buccafusco et al. 1981
	coho salmon	ST	1 h	LC100	100		Reid et al. 1983
	fathead minnow	FT	96 h	LC50	116,000		Walbridge et al. 1983
	orfe	NR	NR	LC50	1,800		Knie et al. 1983
**	rainbow trout	ST	96 h	LC50	225,000		Johnson & Finley 1980
	brine shrimp	ST	24 h	EC50im	69,900	*	Foster & Tullis 1984
	scud	ST	96 h	NOEC	100,000		Johnson & Finley 1980
	water flea	ST	48 h	EC50im	169,000	*	Call et al. 1983
	water flea	ST	48 h	LC50	220,000		Leblanc 1980
--chronic							
	coho salmon	RE	5 d	LOECre	124,000		Reid et al. 1983
	fathead minnow	FT	28 d	LOECgr	29,000		Benoit et al. 1982
	guppy	RE	7 d	LC50	106,000		Koneman 1981
**	rainbow trout	FT	28 d	LC50	34		Black et al. 1982
	leopard frog	FT	to p-hatch	LC50	4,400		Black et al. 1982
**	salamander	FT	to p-hatch	LC50	2,540		Black et al. 1982
	water flea	RE	28 d	LOEC	20,700		Call et al. 1983
	flagellate euglenoid	ST	72 h	PGR	1,127,000		Bringmann & Kuhn 1980
	green algae	NR	NR	EC50	130,000		Knie et al. 1983
	green algae	ST	7 d	LOECgr	710,000		Bringmann & Kuhn 1980
--BCF							
	bluegill sunfish	FT	14 d	BCF	95.6		Barrows et al. 1980
1,1-DICHLOROETHENE							
--acute							
**	bluegill sunfish	ST	96 h	LC50	74,000		Buccafusco et al. 1981
	fathead minnow	FT	96 h	LC50	108,000		Dill et al. 1980
**	water flea	ST	48 h	LC50	79,000		LeBlanc 1980
	water flea	ST	48 h	NOEC	2,400		LeBlanc 1980
--chronic							
**	fathead minnow	FT	7 d	LC50	29,000		Dill et al. 1980
1,2-DICHLOROETHENE							
--acute							
**	water flea	ST	48 h	LC50	220,000		Leblanc 1980
	water flea	ST	48 h	NOEC	110,000		Leblanc 1980

TABLE C-2 (Continued)

SUMMARY OF TOXICITY DATA FOR VOCS AT APG

CHEMICAL ORGANISM	TYPE	DURATION	EFFECT	CONC.(ug/L)	REFERENCE
ETHYLBENZENE					
--acute					
bluegill sunfish	ST	96 h	LC50	88,000	Johnson & Finley 1980
channel catfish	ST	96 h	LC50	210,000	Johnson & Finley 1980
fathead minnow	FT	96 h	LC50	12,100	Geiger et al. 1986
guppy	RE	96 h	LC50	9,600	Galassi et al. 1988
** rainbow trout	RE	96 h	LC50	4,200	Galassi et al. 1988
water flea	ST	48 h	LC50	75,000	Leblanc 1980
water flea	ST	24 h	EC50im	2,200	Galassi et al. 1988
** brine shrimp	ST	24 h	LC50	145	Abernethy et al. 1986
flagellate euglenoid	ST	72 h	PGR	140,000	Bringmann & Kuhn 1980
** green algae	ST	72 h	EC50gr	4,600	Galassi et al. 1988
METHYLENE CHLORIDE					
--acute					
** bluegill sunfish	ST	96 h	LC50	220,000	Buccafusco et al. 1981
** water flea	ST	48 h	LC50	220,000	Leblanc 1980
water flea	ST	48 h	NOEC	68,000	Leblanc 1980
** Chlorella sp.	ST	3 h	EC50ps	1,480,000	Hutchinson et al. 1980
Chlamydomonas sp.	ST	3 h	EC50ps	2,290,000	Hutchinson et al. 1980
--chronic					
guppy	NA	14 d	LC50	294,000	Koneman 1981
1,1,2,2-TETRACHLOROETHANE					
--acute					
bluegill sunfish	ST	96 h	LC50	21,000	Buccafusco et al. 1981
** fathead minnow	FT	96 h	LC50	20,300	Veith et al. 1983
water flea	ST	48 h	EC50im	23,000	Call et al. 1982
water flea	ST	48 h	LOEC	1700	Leblanc 1980
** water flea	ST	48 h	LC50	9,300	Leblanc 1980
--chronic					
** guppy	RE	7 d	LC50	36,700	Koneman 1981
water flea	RE	28 d	LOEC	6,900	Richter et al. 1983
** water flea	ST	28 d	LOEC	14,400	Call et al. 1983
--BCF					
bluegill sunfish	FT	14 d	BCF	8	Barrows 1980
TETRACHLOROETHENE					
--acute					
** bluegill	ST	96 h	LC50	13,000	Buccafusco et al. 1981
** water flea	ST	48 h	LC50	18,000	Leblanc
water flea	ST	48 h	NOEC	10,000	Leblanc 1980
--BCF					1980
bluegill	FT	21 d	BCF	49	Barrows et al. 1980
TOLUENE					
--acute					
bluegill sunfish	ST	96 h	LC50	13,000	Buccafusco et al. 1981
channel catfish	ST	96 h	LC50	240,000	Johnson & Finley 1980
coho salmon	FT	96 h	LC50	6,805 *	Moles et al. 1981
fathead minnow	FT	96 h	LC50	39,400 *	Devlin et al. 1982
guppy	RE	96 h	LC50	28,200	Galassi et al. 1988
** rainbow trout	RE	96 h	LC50	5,800	Galassi et al. 1988
zebra mussel	FT	NR	LOECbh	610	Slooff et al. 1983
** brine shrimp	ST	24 h	LC50	641	Abernethy et al. 1986
brine shrimp	NR	24 h	LOECtr	25,000	Kerster & Schaeffer 1983
water flea	ST	48 h	LC50	310,000	Leblanc 1980
water flea	ST	24 h	EC50im	7,000	Galassi et al. 1988
flagellate euglenoid	ST	72 h	LOECgr	456,000	Bringmann & Kuhn 1980
green algae	ST	72 h	EC50gr	12,500	Galassi et al. 1988

TABLE C-2 (Continued)

SUMMARY OF TOXICITY DATA FOR VOCS AT APG

CHEMICAL	ORGANISM	TYPE	DURATION	EFFECT	CONC.(ug/L)	REFERENCE
TOLUENE (Continued)						
--chronic						
	coho salmon	FT	40 d	LOECgr	2085 *	Moles et al. 1981
	fathead minnow	FT	32 d	LOECgr	6,000	Devlin et al. 1982
	guppy	RE	14 d	LC50	68,300	Koneman 1981
**	rainbow trout	FT	to p-hatch	LC50	25 *	Black et al. 1982
**	leopard frog	FT	to p-hatch	LC50	390	Black et al. 1982
	salamander	FT	to p-hatch	LC50	850	Black et al. 1982
	green algae	ST	7 d	LOECgr	400,000	Bringmann & Kuhn 1980
1,1,1-TRICHLOROETHANE						
--acute						
	bluegill sunfish	ST	96 h	LC50	40,000	Buccafusco et al. 1981
	water flea	ST	48 h	NOEC	530,000	Leblanc 1980
	Chlorella sp.	ST	3 h	EC50 ps	280,000	Hutchinson et al. 1980
	Chlamydomonas sp.	ST	3 h	EC50 ps	153,000	Hutchinson et al. 1980
--chronic						
	guppy	NA	7 d	LC50	133,000	Koneman 1981
--BCF						
	bluegill sunfish	FT	28 d	BCF	9	Barrows et al. 1980
TRICHLOROETHENE						
--acute						
**	bluegill sunfish	ST	96 h	LC50	45,000	Buccafusco et al. 1981
**	zebra mussel	FT	NA	LOEC	8,000	Slooff et al. 1983
**	salamander	ST	48 h	LC50	48,000	Slooff & Baerselman 1980
	clawed toad	ST	48 h	LC50	45,000	Slooff & Baerselman 1980
**	water flea	ST	48 h	LC50	18,000	Leblanc 1980
	water flea	ST	48 h	NOEC	2,200	Leblanc 1980
	green algae	ST	7 d	LOEC	1,000,000	Bringmann & Kuhn 1980
--BCF						
	bluegill sunfish	FT	14 d	BCF	17	Barrows et al. 1980

(a) References are as in AQUIRE (1990) except for chloroform references, which are as in AQUIRE (1985).

* = This value is the arithmetic mean of several values from the same study.
** = This is the lowest acute or chronic value for a class of organisms.

NA = not available
FT = flow through
ST = static
RE = Renewal
h = hour
d = day
E-L = embryo-larval stage organism
LC50 = median lethal concentration (lethal to 50% of test population)
EC50 = median effect concentration (effect seen in 50% of test pop.):
ps = photosynthesis
im = immobilization
gr = population growth
ph = physiological
bh = behavioral
bl = blood chemistry
bc = biochemical
re = reproduction
NOEC = no observed effect concentration
LOEC = lowest observed effect concentration
BCF = bioconcentration factor

Criteria

Ambient water quality criteria have not been established for any volatile organic chemicals, due to inadequate toxicity data. Although chemical-specific toxicity data are lacking, it is believed that volatile organic chemicals induce toxic effects in aquatic organisms via a non-specific narcotic or anesthetic mode of action. The actual narcotic mechanisms and site of action remain unclear; however, studies on a variety of industrial chemicals indicate that the site of toxic action has nonpolar properties, supporting the lipid solubility theories of anesthesia. It has been suggested that narcosis results from changes in the structure of lipid bilayers in nerve cell membranes due to either an increase in volume caused by the dissolved toxicant or by a binding of the toxicant to the nerve membrane (Franks and Lieb 1978, 1984, 1985). In either case, toxicity is believed to be directly related to the ability of the toxicant to penetrate the lipid components of cells membranes. The effective potency is controlled by the organism-water partitioning properties of the chemical, which in turn, are controlled by the affinity of the chemical for the water and lipid phases.

Structure-Activity Relationships

Structure activity relationships (SARs) have been developed which relate the narcotic properties of chemicals to their lipophilicity. Veith et al. (1983) and others have reviewed a large portion of the aquatic toxicity data base and have shown that increased toxicity can be correlated quantitatively with increasing lipophilicity. Specifically, they plot toxicity as a function of the log of the octanol-water partition coefficient (Kow) of a chemical. More recently, Abernethy et al. (1988) have correlated narcosis with lipophilicity at the action site (i.e., cell membrane), as well as with the size of the molecule (i.e., the molar volume of the chemical in the cell membrane), with toxicity increasing along with lipophilicity at the action site and chemical size.

EPA (1988) has published a methodology which utilizes SARs to derive toxicity estimates for organic chemicals with no available toxicity data. Specifically, relationships between chemical-specific Kows, molecular weight, and available toxicity data (LC_{50}s) were determined based on regression analysis. SAR equations were developed for 3 broad classes of organic chemicals: (1) neutral organics which are non-reactive and non-ionizable, (2) neutral organics which are reactive and show excess toxicity in addition to narcosis, and (3) surface active organic compounds such as surfactants and polycationic polymers. The VOCs at APG fall under the first category, Neutral Organics. For this group, three freshwater and two saltwater species SAR equations are presented in EPA (1988). These SARs were used by EPA (1988) to develop 96-hour LC_{50} values. They present the LC_{50} values in tables, ordered by $\log(K_{ow})$ and molecular weight.

Using these tables, LC_{50}s for fresh- and saltwater fish and invertebrates were estimated for the organic chemicals of concern at APG. Additionally, a SAR developed by Lipnick (1989) for tadpoles was used to develop LOEC concentrations for amphibians. The six SAR equations are given in Table C-3. The calculated LC_{50} values along with the molecular weight and Kow used to select them, are presented in Table C-4.

Toxicity of VOCs as a Mixture

The VOCs at APG are present in water as a mixture. When chemicals in a mixture act upon the same organ system or by a similar mode of action (e.g., narcosis), EPA recommends that their combined toxic effect be considered additive. The American Conference of Governmental Industrial Hygienists (ACGIH) uses a formula to calculate Threshold Limit Values (TLVs) for chemical mixtures (ACGIH

TABLE C-3

STRUCTURE ACTIVITY RELATIONSHIPS (SAR) USED TO ESTIMATE VOC TOXICITY

Organism	SAR Equation	Reference (a)
Freshwater fish (b)	log LC50 = -0.94 log Kow + 0.94 log (0.000068 Kow + 1) - 1.25	Veith et al. 1983
Sheepshead minnow (c)	log (1/LC50) = 0.73 log Kow - 3.69	Zaroogian et al. 1985
Freshwater fish 14-day (c)	log (1/LC50) = 0.871 log Kow - 4.87	Koneman 1981
Daphnid 48-hour LC50 (c)	log (1/LC50) = 0.91 log Kow - 4.72	Hermans et al. 1984
Mysid shrimp 96-hour LC50 (c)	log (1/LC50) = 1.25 log Kow - 4.83	Zaroogian et al. 1985
Tadpole LOEC (b)	log (1/LOEC) = 0.909 log Kow + 0.727	Lipnick 1989

(a) All as cited in EPA 1988 except Lipnick 1989.
(b) LC50 concentration in moles per liter. Multiply by molecular weight and 1E+06 to obtain concentration in micrograms per liter.
(c) LC50 concentration in micromoles per liter. Multiply by molecular weight to obtain concentration in micrograms per liter.

TABLE C-4

CHEMICAL PROPERTIES AND ESTIMATED TOXICITY VALUES FOR VOLATILE ORGANIC CHEMICALS AT APG
BASED ON STRUCTURE-ACTIVITY RELATIONSHIPS

Chemical	Molecular Weight (g/mole)(a)	Log Kow	Calculated Fish 96-hour LC50 (ug/liter)		Calculated Freshwater Fish 14-day LC50 (ug/liter) (h)	Calculated Invertebrate LC50 (ug/liter) (h)		Calculated Tadpole LOEC (ug/liter)(i)
			Freshwater	Saltwater		Freshwater (48-hour)	Saltwater (96-hour)	
Benzene	78.11	2.13 b	46,000	10,500	85,200	52,000	12,800	170,000
Carbon tetrachloride	153.84	2.73 c	25,000	8,000	60,500	27,000	4,300	95,000
Chlorobenzene	112.56	2.84 d	15,000	5,000	31,000	14,500	2,500	55,000
Chloroform	119.39	1.97 e	90,000	20,000	161,000	95,000	25,700	362,000
1,2-Dichloroethane	98.96	1.45 c	246,000	43,000	410,000	226,000	90,000	892,000
1,1-Dichloroethene	96.95	1.48 f	232,000	38,000	360,000	220,000	88,000	821,000
1,2-Dichloroethene	96.95	NA	NA	NA	NA	NA		NA
Ethylbenzene	106.16	3.15 e	7,400	27,000	15,600	6,800	710	27,000
Methylene chloride	84.94	1.51 b	186,000	33,000	311,000	192,000	76,500	675,000
1,1,2,2-Tetrachloroethane	167.86	2.39 c	53,000	15,000	100,000	58,000	11,300	212,000
Tetrachloroethene	165.85	2.53 c	42,500	12,000	81,500	46,000	8,300	156,000
Toluene	92.13	2.69 d	15,000	5,000	30,800	16,000	2,800	62,000
1,1,2-Trichloroethane	133.42	2.07 f	78,000	19,000	146,000	86,000	20,800	329,000
Trichloroethene	131.4	2.42 c	41,000	11,000	78,000	45,000	8,800	156,000
Trichlorofluoromethane	137.4	2.53 j	33,000	10,000	64,000	36,000	6,600	129,000
Vinyl chloride	62.5	1.38 g	163,000	28,000	280,000	168,000	75,000	652,000

(a) Budavari 1989 (Merck Index)
(b) Hansch and Leo 1979
(c) Banerjee et al. 1980
(d) Chiou and Schmedding 1982
(e) Leo et al. 1971
(f) Mabey et al. 1982
(g) EPA 1984
(h) EPA 1988
(i) Lipnick 1989
(j) Callahan et al. 1979
NA = Not available

1986). This formula is presented by Abernethy et al. (1988) as a method to calculate the LC_{50} of a chemical mixture:

$$1/LC50_{mix} = C_1/LC50_1 + C_2/LC50_2 + ...$$

$$= \sum C_N/LC50_i$$

where C_i is the concentration of each chemical in the mixture containing N chemicals. This relationship holds true only for chemicals of similar molar volume, and therefore molar volumes of the VOCs of concern at APG were analyzed to determine whether the chemicals should be divided into groups of low or high molar volumes. The molar volumes (shown in Table C-5) ranged from 64 to 123 ml/mole, which is similar to the range of molar volumes for chemicals used by Abernethy et al. (1988). Therefore VOCs were kept as a single group.

Using the above formula, an LC_{50} for the mixture of VOCs in surface water at each APG area may be calculated by summing the ratio of concentration over LC_{50} for each chemical, and taking the inverse.

TERRESTRIAL TOXICITY

Data regarding toxicity of VOCs to terrestrial organisms are scarce, and are generally restricted to studies using laboratory mammals to evaluate potential effects in humans. No data regarding toxicity to birds, plants, or other terrestrial organisms were located in the literature.

REFERENCES

ABERNETHY, S.G., MACKAY, D., and McCARTY, L.S. 1988. "Volume fraction" correlation for narcosis in aquatic organisms: the key role of partitioning. Environ. Toxicol. and Chem. 7:469-481.

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Threshold Limit Values and Biological Exposure Indices for 1986-1987. ISBN: 0-936712-69-4.

AQUATIC INFORMATION RETRIEVAL (AQUIRE). 1985. Computerized database. Chemical Information Systems, Inc. Baltimore, Maryland.

AQUATIC INFORMATION RETRIEVAL (AQUIRE). 1990. Computerized database. Chemical Information Systems, Inc. Balitmore, Maryland.

BARRICK, R.C. and BELLER, H.R. 1989. Reliability of sediment quality assessment in Puget Sound. In: Oceans '89 Proceedings. Inst. of Electrical and Electronic Engineers. Piscataway, New Jersey. Publ. No. 89CH2780-5.

BARROWS, M.E., PETROCELLI, S.R., MACEK, K.J., and CARROLL, J.J. 1980. Bioconcentration and elimination of selected water pollutants by the bluegill sunfish (Lepomis macrochirus). Dyn., Exposure Hazard Assess. Toxic Chem., (Paper Symposium 1978). Ann Arbor Science. Ann Arbor, Michigan.

BUCCAFUSCO, R.J., ELLS, S.J., and LeBLANC, G.A. 1981. Acute toxicity of priority pollutants to bluegill (Lepomis macrochirus). Bull. Environ. Contam. Toxicol. 26(4):446-452.

TABLE C-5

CHEMICAL PROPERTIES FOR VOLATILE ORGANIC CHEMICALS AT APG

Chemical	Molar Volume (ml/mole)		Density (a)
Benzene	89.41	(a)	0.879
Carbon tetrachloride	97.09	(a)	1.589
Chlorobenzene	101.74	(a)	1.107
Chloroform	80.67	(a)	1.484
1,2-Dichloroethane	79.43	(a)	1.257
1,1-Dichloroethene	NA		1.213
1,2-Dichloroethene	77.26	(a)	NA
Ethylbenzene	123.08	(a)	0.866
Methylene chloride	64.50	(a)	1.326
1,1,2,2-Tetrachloroethane	105.79	(a)	1.587
Tetrachloroethene	103.23	(a)	1.623
Toluene	106.86	(a)	0.886
1,1,2-Trichloroethane	92.55	b	1.442
Trichloroethene	90.53	(a)	1.465
Vinyl chloride	68.64	b	0.911

(a) Budavari 1989 (Merck Index)
(b) Calculated (molecular wt/density)

CALLAHAN, M.A., SLIMAK, M.W., GABEL, N.W., MAY, I.P., FOWLER, C.F., FREED, J.R., JENNINGS, P., DURFEE, R.L., WHITMORE, F.C., MAESTRI, B., MABEY, W.R., HOLT, B.R., and GOULD, C. 1979. Water-Related Environmental Fate of 129 Priority Pollutants, VOlumes I and II. Office of Water and Waste Management, Office of Water Planning and Standards, U.S. Environmental Protection Agency. Washington, D.C. EPA Contract No. 68-03-3116.

COWGILL, U.M., TAKAHASHI, I.T., and APPLEGATH, S.L. 1985. A comparison of the effect of four benchmark chemicals on Daphnia magna and Ceriodaphnia dubia affinis tested at two different temperatures. Environ. Toxicol. Chem. 4(3):415-422.

DALICH, G.M., LARSON, R.E., and GINGERICH, W.H. 1982. Acute and chronic toxicity studies with monochlorobenzene in rainbow trout. Aquat. Toxicol. 2:127-142.

DeGRAEVE, G.M., ELDER, R.G., WOODS, D.C., and BERGMAN, H.L. 1982. Effects of naphthalene and benzene on fathead minnows and rainbow trout. Arch. Environ. Contam. Toxicol. 11:487-490.

DEVLIN, E.W., BRAMMER, J.D., and PUYEAR, R.L. 1982. Acute toxicity of toluene to three age groups of fathead minnows (Pimephales promelas). Bull. Environ. Contam. Toxicol. 29(1):12-17.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Estimating Toxicity of Industrial Chemicals to Aquatic Organisms Using Structure Activity Relationships. Volume 1. Office to Toxic Substances. Washington, D.C. EPA 560/6-88-001.

FOSTER, G.D., and TULLIS, R.E. 1984. A quantitative structure-activity relationship between partition coefficients and the acute toxicity of naphthalene derivatives in artemia. Aquat. Toxicol. 5(3):245-254.

FRANKS, N. and LIEB, W. 1978. Where do general anesthetics act? Nature. 274:339-342.

FRANKS, N. and LIEB, W. 1984. Do general anesthetics act by competitive binding to specific receptors? Nature. 310:599-601.

FRANKS, N. and LIEB, W. 1985. Mapping of general anesthetic target sites provides a molecular basis for cutoff effects. Nature. 316:349-351.

HERMANS, J., CANTON, H., JANSSEN, P., and deJONG, R. 1984. Quantitative structure-activity relationships and toxicity studies of mixtures of chemicals with anaesthetic potency: acute lethal and sublethal toxicity to *Daphnia magna*. Aquatic Toxicol. 5:143-154 (as cited in EPA 1988).

HUTCHINSON, T.C., HELLEBUST, J.A., TAM, D., MACKAY, D., MASCARENHAS, R.A. and SHIU, W.Y. 1980. The correlation of the toxicity to algae of hydrocarbons and halogenated hydrocarbons with their physical-chemical properties. Environ. Sci. Res. 16:577-586.

KONEMAN, H. 1981. Quantitative structure-activity relationships in fish toxicity studies. Part 1: relationship for 50 industrial pollutants. Toxicology. 19(3):209-221.

LeBLANC, G.A. 1980. Acute toxicity of priority pollutants to water flea (Daphnia magna). Bull. Environ. Contam. Toxicol. 24(5):684-691.

LIPNICK, R.L. 1989. A quantitative structure-activity relationship study of Overton's data on the narcosis and toxicity of organic compounds to the tadpole, Rana temporaria. Aquatic Toxicol. and Environ. Fate. 77:468-489.

MOLES, A., BATES, S., RICE, S.D., and KORN, S. 1981. Reduced growth of coho salmon fry exposed to two petroleum components, toluene and naphthalene, in fresh water. Trans. Am. Fish. Soc. 110(3):430-436.

VEITH, G.D., CALL, D.J., and BROOKE, L.T. 1983. Structure-toxicity relationships for the fathead minnow, Pimephales promelas: narcotic industrial chemicals. Can. J. Fish. Aquat. Sci. 40:743-748.

ZAROOGIAN, G., HELLSHE, J.F., and JOHNSON, M. 1985. Estimation of toxicity to marine species with structure activity models developed to estimate toxicity to freshwater fish. Aquatic Toxicol. 6:251-270.

AQUATIC TOXICITY

Data regarding aquatic toxicity of this chemical agent are extremely limited. Existing research efforts focused on acute toxicity in the event of a spill; no chronic data are available. Based on test data by Weiss and Botts (1957 in Baronian 1988) and Weimer et al. (1970 in Baronian 1988), an equation was derived to estimate toxicity in terms of median lethal time, LT_{50}, which is the amount of time required for a given concentration of chemical to kill half of the test population. The equation, presented in Baronian (1988) is:

$$\log(LT_{50}) = a - b \log(C)$$

where LT_{50} is the median lethal time in minutes, C is the nerve agent concentration in ppm (mg/l), and a and b are constants. For striped bass (Morone saxitalis) exposed to VX, $a = 1.52 \pm 0.06$, and $b = 0.63 \pm 0.07$ (Baronian 1988).

Aquatic toxicity data are typically described in terms of median lethal concentration, LC_{50}, which is the concentration of chemical which is lethal to half of the test population over a standard period of time, usually 24 to 96 hours. Using the above equation, 24-, 48-, and 96-hour LC_{50}s of 2.5, 0.8, and 0.3 ug/L were estimated for striped bass.

LT_{50} values for fathead minnow Pimephales promelas are also presented in Baronian (1988); however, since the precise equation used to derive the values is not presented, calculation of standard-exposure time LC_{50}s is not possible. A concentration of 2 ug/l was lethal to half the population in 31.2 hours, and concentrations of 0.7 and 0.3 ug/L were lethal to half the population in 52.8 and 91.2 hours, respectively. These may be considered a close approximation of toxicity for the standard exposure times of 24, 48, and 96 hours, respectively.

Data regarding toxicity to invertebrate organisms and saltwater species were not found in the literature. Information regarding bioaccumulation potential was not located.

TERRESTRIAL TOXICITY

Wildlife

Data regarding VX toxicity to terrestrial wildlife species are scarce. Studies have been conducted using domestic and some laboratory species. These data are discussed below.

Birds

Data indicate that birds and insects may be particularly sensitive to toxic effects of VX (Baronian 1988).

Predictions of probable effects of VX on mallards (Anas platyrhynchos) and ring-necked pheasants (Phasianus colchicus) were extrapolated from data on rats by regression analysis. Avian LD_{50} values for organophosphate pesticides, which are similar in structure and biological acitivity (AChE inhibition) to VX, were regressed against rat LC50 values for the same chemicals. Based on this regression analysis, LD50s of 0.012 and 0.18 ppm were predicted for mallards and pheasants, respectively (U.S. Army 1974 in Baronian 1988).

Studies indicate that this compound is probably not accumulated through the food chain.

Mammals

Toxicity data from ingestion of VX are presented in Baronian (1988) for a number of laboratory mammals and domestic livestock. These data indicate that ingested VX is extremely toxic, with reported oral LD50 values ranging from 0.026 mg/kg diet for young steer (Sutton and Salomon 1975 in Baronian 1988) to 0.25 mg/kg diet for the common mouse Mus musculus (Ballard et al. 1968 in Baronian 1988). Sensitivity of sheep rat, and rabbit were between these two values.

Plants

A number of studies of the effects of VX on terrestrial plants were located in the literature. In general it has been determined that plants are susceptible to this compound; upon exposure to airborne chemical, plants become flaccid, a blue/black coloration appears in the leaves, and they eventually disintegrate into jellylike masses (Worthley 1970 in Baronian 1988). A concentration of 10 ppm in soil (Ross et al. 1983 in Baronian 1988) or aqueous solution (Worthley 1971 in Baronian 1988) resulted in the death of "some" of the plants.

Evidence exists that plants absorb and retain VX and its breakdown products. VX appears to be relatively persistent in soils, and plants treated with an aqueous solution of this compound's breakdown products accumulated these substances in the leaves, flowers, and fruit (Worthley 1971 in Baronian 1988). VX did not prevent germination of seeds (Worthley 1970 and Ross et al. 1983 in Baronian 1988), and plants in contaminated areas of Aberdeen Proving Ground, MD showed the same viability as those from a pristine area (McNamara and Leitnaker 1971 in Baronian 1988).

REFERENCES

BARONIAN, C. 1988. Chemical Stockpile Disposal Program - Final Programmatic Environmental Impact Statement. Program Executive Officer - Program Manager for Chemical Demilitarization. Aberdeen Proving Ground, Maryland.

WHITE PHOSPHOROUS

AQUATIC TOXICITY

Laboratory and field studies indicate that white phosphorous is quite toxic to aquatic organisms. Aquatic toxicity data for invertebrates and fish are discussed below.

Invertebrates

Field studies have documented white phosphorous related-alterations in benthic community structure resulting from decreases in diversity and selective mortality. Aquatic macroinvertebrates are more resistent to white phosphorous, with 48-hour EC_{50} values ranging form 30 ug/L (Daphnia magna) to greater than 560 ug/L (Ascellus militaris) (Davidson et al. 1987)

Fish

Massive fish kills have been reported after white phosphorous release from manufacturing areas. Bluegill are the most sensitive aquatic species with a 96-hour LC_{50} of 2.4 ug/L (Davidson et al. 1987). Chronic studies with fathead minnows indicate that white phosphorous can stunt growth and sexual maturity and decrease survival at concentrations in the range of 1.5 to 3.4 ug/L (Davidson et al 1987). Hatchability in this species is reduced at water concentrations as low as 0.4 ug/L (Davidson et al. 1987). Bioaccumulation of white phosphorous is rapid and extensive, but depuration is also rapid. A maximum bioconcentration factor of 2,000 has been reported in fathead minnows (Davidson et al. 1987).

Plants

No data was found on the toxic effects of white phosphorous on aquatic plants.

Criteria

Davidson et al. (1987) proposed an acute criterion of 1.15 ug/L based on the available data.

TERRESTRIAL TOXICITY

Wildlife

No data relating toxicity to terrestrial wildlife species were located in the literature.

Toxicity and toxicokinetic data from humans and laboratory animals indicate that white phosphorous is absorbed following oral exposure. Once absorbed, white phosphorous is distributed primarily to the liver and blood. No data are available which indicate that white phosphorous is absorbed from the lungs or skin. The primary target organs of elemental phosphorous in humans and experimental animals following acute oral exposure are the gastrointestinal tract, brain, liver, kidney, and cardiovascular systems. In animals, longterm exposure has been associated with liver necrosis, weight loss, growth retardation, and bone thickening and growth alteration. White phosphorous also has been shown to reduce survival, viability, and lactation indices in rats pups.

Oral LD_{50} values for white phosphorous are in the range of 3 to 4 mg/kg for rats and mice potential surrogates for wildlife species) (Davidson et al. 1987). Monsanto (1985 in Davidson et al. 1987)

conducted a one generation reproductive toxicity study of yellow phosphorous. Observations of weights and a complete histopathological evaluation, including bone and liver, did not reveal any effects in exposed males, females or pups. The 0.075 mg/kg/day level was identified as a "frank effect level" and the 0.015 mg/kg/day was identified as the NOAEL. A chronic criterion (RfD) of 0.00015 mg/kg/day can be derived by applying an uncertainty factor of 100 to the NOAEL.

Plants

No data were found on the toxic effects of white phosphorous on terrestrial plant species.

REFERENCES

DAVIDSON, K.A., HOVATTER, P.S., SIGMON, C.F. 1987. Water Quality Criteria for White Phosphorous. Oak Ridge National Laboratory. Oak Ridge, Tennessee. AD ONRL-6336.

AQUATIC TOXICITY

Zinc is an essential trace element for animals, and is important to cell growth and differentiation and in the formation of a number of metalloenzymes (Rand and Petrocelli 1985, NAS 1980). The toxicity of zinc to aquatic organisms has been reviewed by EPA (1987). Acute toxicity to fish results from gill destruction and hypoxia (Rand and Petrocelli 1985). Exposure of fish to sublethal concentrations of zinc can cause extensive edema and necrosis of liver tissue (Rand and Petrocelli 1985). Zinc toxicity decreases as water hardness increases (EPA 1986), thus, water quality criteria reflecting this relationship have been developed. There is some evidence of zinc tolerance in fish (trout) and amphibians that are previously exposed to sublethal concentrations of zinc (Woodall et al. 1988, Anadu et al. 1989). This tolerance is correlated with an increase in the level of the metal-binding protein metallothionein in the liver (Woodall et al. 1988, Anadu et al. 1989).

Invertebrates

Cladocerans are the most sensitive freshwater aquatic animal species tested with zinc (EPA 1987). The genus Ceriodaphnia was the most sensitive of 35 genera reported; the mean acute value (hardness = 50 mg/L as $CaCO_3$) is 93.95 ug/L. The genus Daphnia was the fourth most sensitive genus; the mean acute value is 299.8 ug/L. Damselflies (Argia sp.) were the most tolerant species tested, with an acute value of 88,960 ug/L (for hardness = 50 mg/L) (EPA 1987). Acute toxicity values for stoneflies (Order Plecoptera), mayflies (Order Ephemeroptera), and caddisflies (Order Trichoptera) were not reported in EPA (1987). Nehring (1976 in EPA 1987) reported 14-day LC_{50}s of greater than 9,200 ug/L and greater than 13,900 ug/L, for mayfly (Ephemerella grandis) and stonefly (Pteronarcys californica), respectively (hardness = 30-70 mg/L). In chronic studies the lowest Maximum Acceptable Toxicant Concentration (MATC) reported for a freshwater invertebrate was 47 ug/L for Daphnia magna (EPA 1986, 1987), at a hardness of 104 mg/L.

Species mean acute values for 26 species (22 genera) of saltwater invertebrates range from 195 ug/L for the quahog clam (embryos) to 320,400 ug/L for the clam (Macoma balthica) (EPA 1987). Acute values were less than 10,000 ug/L for all of the species tested except for starfish Asterias forbesii, mud snail Nassarius obsoletus, and the clam mentioned above. The only chronic value reported in EPA (1987) for a saltwater invertebrate is a value of 166.5 ug/L for mysid (salinity = 30 ppt).

Fish

For 21 species of freshwater fish the species mean acute values (hardness = 50 mg/L as $CaCO_3$) range from 119.4 ug/L for the striped bass (Morone saxatilis) to 17,940 ug/L for the banded killifish (Fundulus diaphanus) (EPA 1987). The mean acute value for rainbow trout is 689.3 ug/L based on numerous tests reported by EPA (1987). The mean acute value for brook trout (Salvelinus fontinalis) is 2,100 ug/L (EPA 1987). Nehring and Goettl (1974) evaluated the toxicity of zinc to four trout species and reported 14-day LC_{50}s of 410 ug/L for rainbow trout (Salmo gairdneri) (hardness = 20-51 mg/L), 640 ug/L for brown trout (Salmo trutta) (hardness = 22-55 mg/L), 670 ug/L for cutthroat trout (Salmo clarki) (hardness = 22-58 mg/L), and 960 ug/L for brook trout (alkalinity = 34-54 mg/L, hardness not measured). The relative sensitivities of the trout species were evaluated in light of the above results and accounting for differences in the average size of the fish used in each test. Based on this analysis (Nehring and Goettl (1974) determined the following order of sensitivity to zinc: brook trout (least sensitive) < brown trout < cutthroat trout < rainbow trout (most sensitive). Davies (1980)

has reported 96-hour LC_{50}s for rainbow trout (length = 170 mm) of 105 ug/L (hardness = 36.7 mg/L) and 186 ug/L (hardness = 39.2 mg/L) in aerated and non-aerated tests, respectively.

The flagfish (Jordanella floridae) had an MATC of 36.4 ug/l (hardness = 44 mg/L) and was the most chronically sensitive of seven freshwater fish species tested (EPA 1986). Chronic values for the other species range from 106.3 ug/L for the fathead minnow to 854.7 ug/L for the brook trout (EPA 1987). Trout are apparently not quite as sensitive to the chronic effects of zinc as the flagfish, guppy (Poecilia retuculata), or fathead minnow (Pimephales promelas). The chronic value (based on a life-cycle test) for brook trout is 854.7 ug/L (hardness = 45.9 mg/L) (Holcombe et al. 1979 in EPA 1987). Chronic values based on early life cycle tests with rainbow trout are 276.7 ug/L (hardness = 26 mg/L) reported by Sinley et al. (1974 in EPA 1987) and 603.0 ug/L (hardness = 25 mg/L) reported by Cairns et al. (1982 in EPA 1987).

The most acutely sensitive saltwater fish tested is cabezon (Scorpaenichthys marmoratus) with a species mean acute value of 191.4 ug/L (EPA 1987). For six other saltwater fish the species mean acute values range from 430 ug/L for the striped bass (Morone saxitilis) to 38,000 ug/L for the spot (Leiostomus xanthurus) (EPA 1987). No chronic values are available in EPA (1987) for saltwater fish.

Toxicity values are available for a number of fish species that occur at or near the Aberdeen Proving Ground. Species mean acute values for freshwater species (based on a hardness of 50 mg/L as $CaCO_3$) are: pumpkinseed (Lepomis gibbosus) 18,790 ug/L, American eel (Anguilla rostrata) 13,630 ug/L, goldfish (Carassius auratus) 10,250 ug/L, carp (Cyprinus carpio) 7,233 ug/L, golden shiner (Notemigonus crysoleucas) 6,000 ug/L, bluegill (Lepomis macrochirus) 5,937 ug/L, white sucker (Catostomus commersoni) 5,228 ug/L, and striped bass 119.4 ug/L (EPA 1987). The striped bass was the second most acutely sensitive fish or invertebrate species reported in EPA (1987); only the cladoceran Ceriodaphnia reticulata was more sensitive (species mean acute value = 50.7 ug/L) (based on 35 species). The species mean acute values for saltwater fish species are spot 38,000 ug/L, winter flounder (Pseudopleuronecthes americanus) 9,467 ug/L, tidewater silverside (Menidia beryllina) 5,600 ug/L, Atlantic silverside (Menidia menidia) 3,640 ug/L, and striped bass 430 ug/L (EPA 1987). Striped bass was ranked 6th out of 28 saltwater animal species reported, and it was the most sensitive saltwater fish (EPA 1987).

Amphibians

Acute toxicity values (EC_{50}s based on death and deformity) have been reported for embryo larva of two species of amphibians. The 7-day EC_{50} for narrow-mouthed toad (Gastrophryne carolinensis) is 10 ug/L (hardness = 195 mg/L as $CaCO_3$) (Birge 1978 and Birge et al. 1979 in EPA 1987). The marbled salamander (Ambystoma opacum) is apparently much less sensitive than the narrow-mouthed toad. The 8-day EC_{50} for the marbled salamander is 2,380 ug/L (hardness = 93-105 mg/L as $CaCO_3$) (Birge et al. 1978 in EPA 1987). Woodall et al. (1988) reported that tadpoles of Xenopus laevis had 50 percent mortality after 30 hours of exposure to 20 mg/L zinc.

Plants

Adverse effects have been reported for freshwater plants (20 species) at concentrations ranging from 30 to greater than 200,000 ug/L (EPA 1987). Very limited information is available on the influence of hardness on the toxicity of zinc to aquatic plants. An EC_{50} of 50.9 ug/L was reported for the green alga Selenastrum capricornutum based on effects on biomass during 14 to 21 days of exposure (Turbak et al. 1986 in EPA 1987). A 4-day EC_{50} of 7,100 ug/L was reported by Rachlin et al. (1982 in EPA 1987) for the green alga Chlorella saccharophila. The 28-day EC_{50} for duckweed (Lemna minor)

based on tissue damage and death is 67,700 ug/L (Brown and Rattigan 1979 in EPA 1987). A 4-day EC_{50} of 10,000 ug/L was reported for duckweed based on growth effects (Wang 1986a in EPA 1987).

The lowest toxicity values for saltwater plant species are 4-day EC_{50}s (for growth) of 19 ug/L for the diatom Schroederella schroederi and 271 ug/L for the diatom Nitzschia closterium (EPA 1987). No long term studies are available for saltwater plants (EPA 1987).

Sediment Toxicity

Limited information is available on the toxicity of zinc-contaminated sediments. Pavlou and Weston (1983 in Pavlou 1987) reviewed proposed limits for zinc in sediments and found values of 75-100 mg/kg (dry weight). Using the equilibrium partitioning approach (assuming 2 percent organic matter in the sediments), Pavlou and Weston estimated a safe concentration for zinc in marine sediments of 380 mg/kg (dry weight). Birge et al. (1977 in Birge et al. 1987) reported statistically significant mortality in rainbow trout (early eyed-egg stage thru 4-days post-hatch) exposed to zinc-enriched sediment with a measured concentration of 121.4 mg/kg (dry weight). The overlying water had 21.2 ug/L zinc. Based on these results, they estimated a threshold-concentration for zinc of 760 mg/kg for sediments containing 4% organic carbon. Midge larvae have shown lower survival rates, reduced size, and decreased rates of emergence after exposure to sediments contaminated with zinc, cadmium, and chromium (Wentsel et al. 1977b, 1978 in Francis et al. 1984). Midge larvae have also shown avoidance behavior to sediments contaminated with more than 8,330 mg/kg zinc and more than 422 mg/kg cadmium (Wentsel et al. 1977a in Francis et al. 1984).

Bioaccumulation

For freshwater invertebrates, Nehring (1976 in EPA 1987) reported BCFs of 1,130 for mayfly and 106 for stonefly, after 14 days exposure.

A BCF of 23,820 has been reported for the eastern oyster (Crassostrea virginica), based on 126 days of exposure (Schuster and Pringle 1968 in EPA 1987).

Zinc has shown bioconcentration factors of 51 to approximately 1,000 in freshwater fish (EPA 1986, EPA 1987), although limited information is available. A whole body bioconcentration factor of 417.3 was reported for the flagfish (Jordanella floridae) following 100 days of exposure (Spehar 1978 in EPA 1987). Similar values were reported for guppy (Poecilla reticulata); whole body BCFs of 466.3 to 965.5 were determined from three tests (each of 134 days) by Pierson (1981 in EPA 1987). A BCF (whole body) of 51 was reported for Atlantic salmon (Salmo salar) exposed in freshwater for 80 days (Farmer et al. 1979 in EPA 1987).

For marine fish, a whole body BCF of 18.1 was determined for juvenile mummichog (Fundulus heteroclitus) after 56 days of exposure (Sauer and Watabe 1984 in EPA 1987).

BCFs for green algae of 133 and 210 were reported by Coleman et al. (1971 in EPA 1987). Coleman et al. also reported a BCF of 144 for the freshwater plant, euglena (Euglena viridis).

The marine diatom Thalassiosira psuedonana bioconcentrated zinc up to 12,000 times the concentration in water after 1/2 day of exposure (Fisher et al. 1984 in EPA 1987).

Criteria

For freshwater, EPA (1987) has proposed that the one-hour concentration should not exceed the value in ug/L given by $e^{(0.8473[\ln(\text{hardness})]+0.8604)}$ and the 4-day average should not exceed the value in ug/L given by $e^{(0.8473[\ln(\text{hardness})]+0.7614)}$. At a water hardness of 100 mg/liter (as $CaCO_3$), the 1-hour (acute) and 4-day (chronic) criteria are 120 and 110 ug/L, respectively. EPA considers the acid soluble measurement of metals to provide a more scientifically appropriate basis for establishing criteria for metals. However, no EPA-approved methods are available for acid-soluble measurements, and until methods are available, EPA recommends applying the criteria using the total recoverable method (EPA 1986). The acute and chronic criteria for marine environments are 95 ug/L and 86 ug/L, respectively (EPA 1987).

TERRESTRIAL TOXICITY

The toxicity of zinc to terrestrial animals has been reviewed by Taylor et al. (1982) and NRC (1979). In general, livestock and poultry are quite tolerant of dietary zinc. Concentrations that are 100 times greater than those required in the diet do not result in adverse effects (NRC 1979). Information on livestock and poultry are discussed below. Information for wildlife is quite limited.

Birds

NAS (1980) reported results for a number of studies conducted with chickens, turkeys, ducks, and Japanese quail (Coturnix japonica). In general these studies indicate that decreased weight gain is first observed at zinc dietary concentrations of 270 ppm in Japanese quail, at 800 ppm in chickens, and at 4,000 ppm in turkeys. The NOEL for chickens is 1,000 ppm (one day old, exposed for four weeks); at 1,500 ppm decreased growth occurred (NAS 1980). In this study, there were 40 birds per treatment. NAS recommended a maximum tolerable dietary zinc level of 1,000 ppm for poultry. Decreased hemoglobin concentrations and hematocrit were observed in young Japanese quail at doses as low as 125 ppm after 2 weeks of treatment (NAS 1980). No significant adverse effects were observed in the quail at 62.5 ppm. Assuming a dietary conversion factor of 0.125 mg/kg bw per 1 ppm diet (Lehman 1954), this corresponds to a dosage of 7.8 mg/kg bw. Severe effects were observed in ducks after 60 days at the lowest concentration tested of 3,000 ppm, including decreased food consumption, decreased body weight, paralysis of the legs, low hemoglobin and hematocrit levels, and decreased pancreas and gonad weights (NRC 1979, NAS 1980).

Mammals

Zinc poisoning has been reported in cattle. In one outbreak, poisoning was caused by food contaminated with zinc at a concentration of 20 g/kg. An estimated intake of 140 g of zinc per cow per day for about 2 days was reported. This is approximately 280 mg/kg per day assuming a cow weighs 500 kg (Lehman 1954). The exposed cows exhibited severe enteritis, and some died or had to be slaughtered. Postmortem findings showed severe pulmonary emphysema with changes in the myocardium, kidneys, and liver. Dietary levels of approximately 1,000 ppm approach toxic levels for lambs and feeder cattle (NRC 1979). In pigs given dietary zinc at concentrations greater than 1,000 mg/kg, decreased food intake and weight gain were observed. At dietary levels greater than 2,000 mg/kg, deaths occurred as soon as 2 weeks after exposure. Severe gastrointestinal changes and brain damage, both of which were accompanied by hemorrhages, were observed, as well as changes in the joints.

NAS (1980) recommends the following maximum tolerable dietary levels of zinc: 300 ppm for sheep, 500 ppm for cattle, 500 ppm for rabbits, and 1,000 ppm for swine. The maximum recommended level in drinking water for poultry and livestock is 25 mg/L according to NAS (1974). Puls (1988) has recommended maximum concentrations for drinking water of 5.0 mg/L for livestock and 2.5 mg/L for poultry.

Plants

Zinc is an essential nutrient for plants and functions in both carbohydrate and protein metabolism (Kabata-Pendias and Pendias 1984). Kabata-Pendias and Pendias (1984) reported phytotoxic soil concentrations of zinc, based on results from six authors, of 70 to 400 mg/kg (arithmetic mean = 270 mg/kg).

REFERENCES

ANADU, D.I., CHAPMAN, G.A., CURTIS, L.R. and TUBB, R.A. 1989. Effect of zinc exposure on subsequent acute tolerance to heavy metals in rainbow trout. Bull. Env. Cont. Tox. 43:329-336.

BIRGE, W.J., BLACK, J.A., WESTERMAN, A.G. and FRANCIS, P.C. 1987. Toxicity of sediment-associated metals to freshwater organisms: biomonitoring procedures. In: Dickson, K.L., Maki, A.W., and Brungs, W.A. (eds.). Fate and Effects of Sediment-Bound Chemicals in Aquatic Systems. Proceedings of the Sixth Pellston Workshop, Florissant, Colorado, August 12-17, 1984. SETAC Special Publication Series, Pergamon Press. New York, New York. Pp. 199-218

DAVIES, P.H. 1980. Investigations on the toxicity of metals to fish. Colorado Division of Wildlife. Ft. Collins, Colorado. Water Pollution Studies: Job 2. Job Progress Report F-33-R-15.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Quality Criteria for Water 1986. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-86-001.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987. Ambient Water Quality Criteria for Zinc-1987. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-87-003. PB87-153581.

FRANCIS, P.C., BIRGE, W.J. and BLACK, J.A. 1984. Effects of cadmium-enriched sediment on fish and amphibian embryo-larval stages. Ecotoxicology and Environmental Safety. 8:378-387.

KABATA-PENDIAS, A. and PENDIAS, H. 1984. Trace Elements in Soil and Plants. CRC Press. Boca Raton, Florida.

LEHMAN, A.J. 1954. Appraisal of the study of chemicals in food, drugs, and cosmetics. U.S. Association of Food and Drug Officials. 18:66.

NATIONAL ACADEMY OF SCIENCES (NAS). 1980. Mineral Tolerance of Domestic Animals. National Research Council, Subcommittee on Mineral Toxicity in Animals. Washington, D.C.

NATIONAL ACADEMY OF SCIENCES (NAS). 1974. Nutrients and Toxic Substances in Water for Livestock and Poultry. National Research Council. Washington, D.C.

NATIONAL RESEARCH COUNCIL (NRC). 1979. Zinc. National Research Council, Division of Medical Sciences, Committee on Medical and Biological Effects of Environmental Pollutants, Subcommittee on Zinc. University Park Press. Baltimore, Maryland.

NEHRING, R.B., and GOETTL, J.P., JR. 1974. Acute toxicity of a zinc-polluted stream to four species of salmonids. Bulletin of Environmental Contamination and Toxicology. 12(4):464-469.

PAVLOU, S.P. 1987. The use of the equilibrium partitioning approach in determining safe levels of contaminants in marine sediments. In: Dickson, K.L., Maki, A.W., and Brungs, W.A. (eds.). Fate and Effects of Sediment-Bound Chemicals in Aquatic Systems. Proceedings of the Sixth Pellston Workshop, Florissant, Colorado, August 12-17, 1984. SETAC Special Publication Series, Pergamon Press. New York, New York. Pp. 388-412.

PULS, R. 1988. Mineral Levels in Animal Health. Sherpa International. Clearbrook, British Columbia.

RAND, G.M., and PETROCELLI, S.R. 1985. Fundamentals of Aquatic Toxicology. Hemisphere Publishing Corp. Washington, D.C.

TAYLOR, M.C., DEMAYO, A., and TAYLOR, K.W. 1982. Effects of zinc on humans, laboratory and farm animals, terrestrial plants, and freshwater aquatic life. CRC Critical Reviews in Environmental Control. 12:113-181.

WOODALL, C., MACLEAN, N. and CROSSLEY, F. 1988. Responses of trout fry (Salmo gairdneri) and Xenopus laevis tadpoles to cadmium and zinc. Comp. Biochem. Physiol. 89C(1):93-99.

APPENDIX D

SPECIES PROFILES FOR SELECTED RECEPTOR SPECIES

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GREEN FROGS
(*Rana clamitans*)

General Description

Green frogs are amphibians in the order Salientia. The subspecies found in the Maryland-APG area is *Rana clamitans melanota*. These are green, bronze, or brown frogs with large external eardrums and prominent bilateral ridges on the dorsum; the ridges do not reach the pelvic girdle. The belly is white with spots. Reports of their weight vary depending on the study. Various reported averages were 29, 49.1, and 70 grams, and values ranged from 25.5 to 103.5 grams (McAlpine and Dilworth 1989; Pough and Kamel 1984); they are approximately 5-10 cm long. They are primarily nocturnal.

Green frogs are widespread throughout North America. The home range of a green frog is approximately 50 square meters. They live in loosely knit communities except during breeding season when they congregate at breeding sites. Males establish territories by vocalization and fighting. A high proportion (approximately 50%) of adult frogs die yearly, although green frogs can live >2 years in captivity. The population at APG is abundant.

Habitat Use

Their optimal habitat is close to shallow water, springs, marshes, brooks or at the edges of ponds and lakes. They generally stay on land close to the water and when alarmed, leap into the water and swim for cover at the bottom. They hibernate in the bottom mud in winter. They can also be found among rotting debris of fallen trees. APG has large areas of wetlands, as well as old bombing ranges with intermittent ponds, that provide habitat for green and other frog populations.

Diet and Foraging Behavior

Green frogs, when they are on land, feed on flying insects which they snap from the air. As a rule they do not forage in water, but may consume some aquatic insects. In one study the diet of green frogs was composed of 10.8% plant material and 89.2% animal matter by volume (Stewart and Sandison 1973). The animal matter was almost exclusively insects, and the orders consumed in the greatest amounts were Coleoptera (32.8%), Hymenoptera (14.4%), Hemiptera (12.9%), and Araneae (12.1%). Most of these were terrestrial varieties.

Life Cycle Characteristics

Green frogs reach sexual maturity in 1-2 years. Green frogs breed in March through August. They are oviparous. A female may lay up to 5,000 eggs per year. Their eggs are deposited in 3-4 small clutches attached to submerged vegetation. Eggs hatch into tadpoles which feed on aquatic vegetation, but later become carnivorous. Terrestrial areas must be present at the time of metamorphosis.

Ecological and Societal Significance

Frogs are prey of various carnivorous mammals, birds such as the great blue heron, reptiles, and fish. Tadpoles also are prey of numerous vertebrates.

References

COCHRAN, D.M. 1961. Living Amphibians of the World. Doubleday, New York

DULLEMAN, W.E. and TRUEB, L. 1986. Biology of Amphibians. McGraw-Hill, New York

FRAZER, J.F.D. 1973. Amphibians. Wykeham Publications, London.

McALPINE, D.F. and DILWORTH, T.G. 1989. Microhabitat and prey size among three species of Rana (Anura: Ranidae) sympatric in eastern Canada. Can. J. Zool. 67:2241-2252.

POUGH, F.H. and KAMEL, S. 1984. Post-metamorphic change in activity metabolism of anurans in relation to life history. Oecologia (Berl.) 65:138-144.

NIERING, W.A. 1985. Wetlands. Alfred A. Knopf, New York

STEWART, M.M. and SANDISON, P. 1973. Comparative food habits of sympatric mink frogs, bullfrogs, and green frogs. J. Herpetol. 6:241-244.

BALD EAGLE
(Haliaeetus leucocephalus)

General Description

The bald eagle (Haliaeetus leucocephalus), with its long rounded wings, large hooked bill, and sharp talons, is the largest raptorial bird in the United States. Adult wingspan ranges from 178 to 229 cm (5.8 to 7.5 ft), and length from tip to tail averages 81 cm (2.7 ft). Females (6.3 kg) are significantly larger than males (4.0 to 4.6 kg), but otherwise the sexes are similar in appearance (Brown and Amadon 1968). In a study done on a lake in Saskatchewan, at 60 days of age wild males averaged 4066 g (3575-4500 g) and females averaged 5172 g (4800-5600 g) (Bortolotti 1984).

Our national symbol, the bald eagle is a federally-designated endangered species and has disappeared from much of its range in the lower 48 states.

Habitat Use

The U.S. Fish and Wildlife Service has prepared a habitat suitability index model for the bald eagle during the breeding season (Peterson 1986). Three general categories of habitat characteristics are important for bald eagles during the breeding season: food supply, nesting habitat, and human disturbance. Each of these components is discussed briefly below.

Food. The principal component of food availability for the bald eagle is adequate foraging territory (i.e., open water). Because of its largely piscivorous diet during the breeding season, suitable bald eagle habitat must include at least eight hectares of open water and associated emergent scrub-shrub wetlands. However, lakes between eight and 1,000 ha in size with opposing shorelines less than 0.5 km apart are not most efficiently used because territoriality may preclude use of opposing shores by more than one pair.

Nesting. Although they will nest in second-growth timber in deforested areas, bald eagles prefer areas of undisturbed, old-growth timber with an open and discontinuous canopy, and optimum nesting areas contain at least 75 percent mature timber cover. Logged areas in which 5-10 percent of the old growth has been left standing also may support significant eagle populations. Nests are placed in tall, dominant trees of any species with stable limbs and an open structure for easy approach. Dense, even-aged stands do not provide these attributes. Nearly all bald eagle nests are found within 1.5 km of open water.

Disturbance. Bald eagles reach maximum densities in areas of minimal human activity and are almost never found in areas of heavy human use. The minimum distance eagles will tolerate from human residences is 1.0 km, and they will nest away from shore to avoid disturbance.

A large portion of the land area of Aberdeen Proving Ground can be classified as optimum breeding habitat for bald eagles. More than 80 percent (66,000) of the 79,000 total acres of land area on the installation is unimproved and designated as test range (Pottie 1986). Active nests have been recorded on Aberdeen Proving Ground since 1936; more recent data suggest that from 0 to 3 nests may be active in a given year (Millsap et. al. 1983, Buehler et al. 1987). Traditional nest sites are located on the Edgewood Peninsula, near Romney Creek, near the Trench Warfare Area, and on Spesutie Island. Active nests have been found in numerous areas within the installation.

Aberdeen Proving Ground also is an important foraging and roosting area for the bald eagle population of the northern Chesapeake Bay. Chesapeake Bay provides habitat for over 200 wintering bald eagles; the installation may be used by as much as 17 percent of these birds (Millsap et al. 1983). High-use areas on Aberdeen Proving Ground were identified during two ground and aerial tracking studies (Millsap et al. 1983, Buehler et al. 1987). These include communal roost areas (see below), Romney Creek, Mosquito Creek, Spesutie Island, the lower half of the Edgewood Peninsula, and Aberdeen Proving Ground's Chesapeake Bay shoreline. Several communal roosting areas are located on the Aberdeen Peninsula near Romney Creek, the Michaelsville Landfill, and Mosquito Creek. Locations of nesting sites, communal roosting areas, and high-use areas are dynamic, varying both seasonally and over several years (Buehler et al. 1987). Therefore, much of the land area of Aberdeen Proving Ground can be considered important to the bald eagle population.

Diet and Foraging Behavior

Bald eagles are primarily fish-eaters and prefer fish over other foods when available; however this species is an opportunist, taking advantage of whatever food source is most plentiful seasonally. Based on direct observation and pellet analysis, Mersmann (1989) found that bald eagles in the northern Chesapeake Bay feed on live and dead fish almost exclusively from April through October and on mammal and bird carrion from November through March. Shifts in prey correlate well with shifts in the abundance of fish. Food is obtained either by foraging or kleptoparasitism (i.e., stealing food from other birds).

Fish species preyed upon most often included gizzard shad, catfish, menhaden, white perch, American eel, yellow perch, and striped bass. Gizzard shad, the most commonly identified species, were preyed upon mostly from late fall through winter. More were taken dead than alive. Catfish, preyed upon from spring through fall, were always taken dead. Menhaden, preyed upon during spring and summer, usually were taken alive. White and yellow perch were preyed upon during their spring spawn, and usually were taken dead. American eel were preyed upon from late spring through summer (Mersmann 1989).

Birds and mammals commonly scavenged included Canada geese, mallard, lesser scaup, and white-tailed deer. Other food items observed incidentally included black racer, opossum, and grey squirrel. Avian and mammalian species found in bald eagle pellets collected beneath communal roosts included gulls, passerine birds, raccoons, muskrats, rabbits, turtles, and a blue crab, although waterfowl and white-tailed deer were most abundant. Gizzard shad and carp were the most common fish species found in pellets. Most birds and mammals consumed by bald eagles in the northern Chesapeake Bay probably are killed or crippled by hunters. Pellets from the Aberdeen peninsula contained more deer, raccoon, and muskrat remains than waterfowl, probably due to the extensive forests and marshes of this area (Mersmann 1989).

Measured daily food consumption rates for captive bald eagles ranged from 50 to 80 g dry weight in one study (Mersmann 1989) and from 65 to 92 g in a second study (Stalmaster and Gessaman 1982). Estimated daily intake rates for free-living birds ranged from 410 to 552 g (Stalmaster and Gessaman 1982). Estimated winter consumption rates for three food types at -10, 5, and 20 C were calculated and the average values were: salmon - 0.092 g/g-day, black-tailed rabbit - 0.0748 g/g-day, and mallard duck - 0.0651 g/g-day (Stalmaster and Gessaman 1982). Another study found that the minimum consumption of salmon required for wintering bald eagles, averaged over all ages and sexes was 0.1087 g/g-day (Stalmaster and Gessaman 1984)

Based on the energy requirements of various species and the energy content of food, Nagy (1987) calculated food ingestion rates (FI) for birds (in grams dry matter per day). For all birds, FI(g/d) = 0.648 ($Wt^{0.651}$) where wt is body weight. For the bald eagle this gives FI = 0.648 (5,450 $g^{0.651}$) = 175g/d. Based on measured body weights and drinking water values from Calder (1981) and Skadhauge (1975), Calder and Braun (1983) determined an allometric equation for water ingestion (WI) for birds: WI(L/d) = 0.059 ($Wt^{0.67}$) where wt is average body weight in kilograms. For the bald eagle this gives WI = 0.059 (5.45 $kg^{0.67}$) = 0.18 L/d.

At Aberdeen Proving Ground, most fish captured live were taken in relatively shallow water (Mersmann 1989). All fish captured live were taken in water less than 1.2 m in depth (average = 0.69 m). Dead fish were taken in water significantly deeper, ranging from 0.3 to 5 m in depth (average = 1.6 m). In non-winter months, more than 75% of strikes occurred within 500 m of the shore in bay and river habitats, although eagles made strikes as far as 5 km from the shore on the Bay. Half of the strikes made more than 1 km from shore were made by eagles attracted by flocks of foraging gulls. Strikes were most frequent in the period directly preceding sunrise; a smaller peak in strike frequency also occurred in early afternoon.

The communal roosts of bald eagles may serve as "information centers" (Ward and Zahavi 1973) where inexperienced or unsuccessful individuals can learn the whereabouts of good foraging areas by following experienced or successful individuals. Bald eagles are known to converge on areas in which food resources are particularly abundant (Green 1985, Mersmann 1989).

Life Cycle Characteristics

Bald eagles have been observed to nest successfully at four years of age, but most do not breed until at least their fifth year (Nye 1983 cited in Green 1985). Breeding pairs remain together as long as both are alive (Brown and Amadon 1968). Large stick nests are built near water, most often in a large tree. Timing of breeding varies by as much as eight weeks within sites and occurs later in more northerly portions of their range (Green 1985). The breeding season at Aberdeen Proving Ground lasts from January to June. Most nestlings fledge in late June (Buehler 1990), suggesting that most eggs are laid in February and March. Clutch size ranges from one to three eggs; replacement clutches are rare (Hoxie 1910 cited in Green 1985). Incubation ranges from 34 to 38 days (Maestrelli and Wiemeyer 1975). Both sexes take responsibility for feeding the young (Brown and Amadon 1968). Age at fledging ranges from 70 to 98 days (Green 1985). Fledging weight of captive eagles reared in Maryland ranged from 3.6 to 4.7 kg (Maestrelli and Wiemeyer 1975). In one study in Canada, average nestling growth rates ranged from 67 to 83 g/day for females and from 76 to 80 g/day for males (Bortolotti 1989).

In Florida, average fledging success from 1973 to 1976 was 1.14 young per active nest (n = 109) and 1.59 young per successful nest (n = 78, McEwan and Hirth 1979). Breeding success at Aberdeen Proving Ground has varied in recent years. The number of active nests has ranged from 0 to 3 per year, the number of successful nests has ranged from 0 to 2 per year, and the total number of young fledged has ranged from 0 to 3 per year. Nesting success appears to have increased in recent years. Only two young fledged from 1977 to 1982; nine fledged from 1983 to 1985 (Millsap et al. 1983, Buehler et al. 1987).

Bald eagles have lived for up to 36 years in captivity, and one wild eagle from Alaska was estimated to be nearly 22 years old (Cain 1986). Typically, 50-70 percent of fledglings die during their first year,

and fewer than 10 percent of fledglings survive to attain adult plumage. Adult mortality estimates range from 5 to 10 percent annually (Sherrod et al. 1977 cited in Green 1985).

Population Dynamics

Buehler et al. (1987) summarized information on the population dynamics of bald eagles in the northern Chesapeake Bay. Of 402 eagles aged by plumage characteristics, 40% were adults, 7% were subadults, and 53% were immatures. Age structure varied seasonally, with adults comprising 53% of the population during winter but only 25% during fall. There appears to be an influx of adults from northern populations in winter but not a comparable influx of adults in summer. Although little is known about eagle dynamics, the eagle population appears to be expanding based on observations of an increasing number of nesting pairs on the Bay and the large percentage of immature birds observed in population counts. Recent recruitment into the population has been well documented.

Home range sizes of eagles radio-tracked at Aberdeen Proving Ground ranged from 21 km^2 to 66 km^2 in late spring and summer. Home ranges from autumn to early spring were much more variable, ranging from 63 km^2 to more than 17,000 km^2, indicating that some birds remained in a relatively small area throughout the year while others ranged more widely (Buehler et al. 1987).

Population density of bald eagles measured in Ontario was 0.84 eagles per 100 km^2 overall and 0.57 eagles per 100 km^2 in breeding areas (Grier 1977). Using the 66,000 acres (267 km^2) of unimproved land as the baseline areal measure, bald eagle population density at Aberdeen ranged from 6.1 eagles per 100 km2 in spring to 9.6 eagles per 100 km^2 in winter, with density in summer and autumn averaging 6.5 eagles per 100 km^2. With 0 to 3 active nests per year, breeding bird density ranges from 0 to 2.2 eagles per 100 km^2.

Grier (1980) determined through modeling that adult survival is much more important than reproductive rate in regulating bald eagle populations. In past years, bioaccumulation of organochlorine pollutants reduced the reproductive success of bald eagles, but in many areas this species now reproduces at rates similar to those prior to the use of these chemicals (Green 1985).

Ecological and Societal Significance

Bald eagles are a federally designated endangered species. This species has particular societal significance because it is our national symbol. Bald eagles are highly valued aesthetically because of their large size, distinctive plumage, and powerful appearance. Bald eagles also play a prominent ecological role as a top carnivore and scavenger in riparian, lacustrine, estuarine, and bay ecosystems.

References

BORTOLOTTI, G.R. 1984. Criteria for determining age and sex of nestling bald eagles. J. Field Ornithol. 55: 467-481.

BORTOLOTTI, G.R. 1989. Factors influencing the growth of bald eagles in north central Saskatchewan. Can. J. Zool. 67: 606-611.

BROWN, L. and AMADON, D. 1968. Eagles, Hawks, and Falcons of the World. New York, NY: McGraw-Hill Book Company.

BUEHLER, D.A. 1990. Bald eagle distribution, abundance, roost use and response to human activity on the northern Chesapeake Bay, Maryland. Ph.D. dissertation, Virginia Polytechnic Institute and State University, Blacksburg, Virginia, January, 1990.

BUEHLER, D.A., FRASER, J.D., and CHASE, J.D. 1987. Bald eagle movements, distribution, and abundance on the northern Chesapeake Bay. Report prepared for the U.S. Army Chemical Research and Development Center, Aberdeen Proving Ground, Maryland, January 15, 1987.

CAIN, S.L. 1986. A new longevity record for the bald eagle. J. Field Ornithol. 57: 173.

CALDER, W.A. III. 1981. Scaling of physiological processes in homeothermic animals. Ann. Rev. Physiol. 43:301-322

CALDER, W.A. and BRAUN, E.J. 1983. Scaling of osmotic regulation in mammals and birds. Am. J. Physiol. 244:R601-R606

GREEN, N. 1985. The bald eagle. Audubon Wildl. Rep. 508-531.

GRIER, J.W. 1977. Quadrant sampling of a nesting population of bald eagles. J. Wildl. Manage. 41: 438-443.

GRIER, J.W. 1980. Modeling approaches to bald eagle population dynamics. Wildl. Soc. Bull. 8: 316-322.

HOXIE, W.J. 1910. Notes on the bald eagle in Georgia. Auk 27: 454.

MAESTRELLI, J.R. and WIEMEYER, S.N. 1975. Breeding bald eagles in captivity. Wilson Bull. 87: 45-53.

McEWAN, L.C. and HIRTH, D.H. 1979. Southern bald eagle productivity and nest site selection. J. Wildl. Manage. 41: 438-443.

MERSMANN, T.J. 1989. Foraging ecology of bald eagles on the northern Chesapeake Bay with an examination of techniques used in the study of bald eagle food habits. M.S. thesis, Virginia Polytechnic Institute and State University, Blacksburg, Virginia, November, 1989.

MILLSAP, B.A., CLINE, K.W., and GILROY, M.J. 1983. Distribution and movements of bald eagles on Aberdeen Proving Ground, Maryland. Report prepared for the U.S. Army, Aberdeen Proving Ground, Maryland, March, 1983.

NAGY, K.A. 1987. Field metabolic rate and food requirement scaling in mammals and birds. Ecol. Mono. 57:111-128

PETERSON, A. 1986. Habitat suitability index models: bald eagle (breeding season). U.S. Fish Wildl. Serv. Biol. Rep. 82(10.126). 25 pp.

POTTIE, J.J. 1986. U.S. Army Aberdeen Proving Ground eagle plan. Report prepared for the U.S. Army, Aberdeen Proving Ground, Maryland, December, 1986.

SHERROD, S.K., WHITE, W.M., and WILLIAMSON, F.S.L. 1977. Biology of the bald eagle on Amchitka Island, Alaska. Living Bird 15: 143-182.

SKADHAUGE, R. 1975. Renal and cloacal transport of salt and water. Symp. Zool. Soc. London 35:97-106

STALMASTER, M.V. and GESSAMAN, J.A. 1982. Food consumption and energy requirements of captive bald eagles. J. Wildl. Manage. 46:646-654.

STALMASTER, M.V. and GESSAMAN, J.A. 1984. Ecological energetics and foraging behavior of overwintering bald eagles. Ecol. Monograph. 54: 407-428.

WARD, P. and ZAHAVI, A. 1973. The importance of certain assemblages of birds as "information centres" for food finding. Ibis 115: 517-634.

GREAT BLUE HERON
(Ardea herodias)

General Description

The great blue heron is a large gray bird with a yellowish bill. It is 99-132 cm long with a wingspan of 1.8 m; adults range in mass from 2.3-3.6 kg (Oberholser as cited in Palmer 1962).

Great blue herons are a common species in North America. The population is considered to be stable. They are considered common at APG during the breeding season and the winter. Great blue herons are solitary territorial birds except during the breeding season.

Habitat Use

Great blue herons are found on the shallow shores of marshy pools, ponds, lakes or rivers where they hunt fish or frogs. They require tall trees for nesting. They winter mainly in coastal areas with snow-free ground and open water.

Appropriate habitat for breeding and wintering is present at APG. There are at least three major nesting colonies (heronries) at APG. One, which is in an extensive marsh created by beavers, has 200-300 nesting pair. Heronries are also present on Pooles Island and the Grenade Range. APG also has coastal areas, adequate slow moving streams and marsh-forest edges for great blue heron habitat, although freshwater ponds are considered inadequate in some areas.

Diet and Foraging Behavior

Fish and frogs are the principal food of great blue herons. Occasionally, they will eat reptiles, birds, aquatic and terrestrial insects and crustaceans. Their behavior when hunting aquatic species is to stand at or near the shore edge and wait or walk slowly. Active pursuit is infrequent. They may pound struggling prey against a rock before swallowing it. Great blue herons will sometimes visit hillsides, meadows, and fields to hunt pocket gophers, ground squirrels, and field mice. During the winter, they may forage at tidal flats. When food is available, the birds will gorge themselves. Home range size is approximately a 20 km radius (Pieffer 1979 in Hancock and Kushlan 1984).

Based on the energy requirements of various species and the energy content of food, Nagy (1987) calculated food ingestion rates (FI) for birds (in grams dry matter per day). For all birds, FI (g/d) = 0.648 ($Wt^{0.651}$) where Wt is body weight in grams. For the great blue heron, this gives a food ingestion rate of FI (g/d) = 0.648 (3,600 $g^{0.651}$) = 134 g/d. Based on measured body weights and drinking water values from Calder (1981) and Skadhauge (1975), Calder and Braun (1983) determined an allometric equation for water ingestion (WI) for birds: WI (L/d) = 0.059 ($Wt^{0.67}$) where Wt is average body weight in kilograms. For the great blue heron this gives a WI of 0.059 (3.6 $kg^{0.67}$) = 0.14 L/d.

Life Cycle Characteristics

Great blue herons reach sexual maturity in 2 years. The birds are solitary except during breeding season, when heronries are formed. The birds prefer to nest in tall trees, particularly along river

banks, and dozens of nests may be build in the crown of a single tree. They can also build nests on the ground, in shrubs, on rock ledges or on sea cliffs. Some nests are used for more than one season. Gibbs et al. (1987) found nest densities in Maine to be approximately 6.03 nests/acre. The nests may be miles from the herons' food source but Towry (1984) states that the feeding areas must be within 2.5 miles of nest sites. The eggs are laid from April through June. The incubation period is 28 days. There is one brood per year. The clutch size is 3-7, typically 4.

Ecological and Societal Significance

These birds have few natural enemies. Their presence at the top of a largely aquatic food chain places them at potential risk from insult due to water contamination.

References

BULL, J. and FARRAND, J., Jr. 1977. The Audubon Society Field Guide to North American Birds. Eastern Region. Alfred A. Knopf, New York

CALDER, W.A. III. 1981. Scaling of physiological processes in homeothermic animals. Ann. Rev. Physiol. 43:301-322

CALDER, W.A. and BRAUN, E.J. 1983. Scaling of osmotic regulation in mammals and birds. Am. J. Physiol. 244:R601-R606

GIBBS, J.P., WOODWARD, S., and HUNTER, M.L., et al. 1987. Determinants of great blue heron colony distribution in coastal Maine. Auk. 104:38-47

HANCOCK, J. and KUSHLAN, J. 1984. The Herons Handbook. Harper and Row. New York, New York

HARRISON, H.H. 1975. A Field Guide to Birds Nests of 285 Species Found Breeding in the United States East of the Mississippi River. Houghton Mifflin Co., Boston

HENNY, C.J. 1972. An analysis of the population dynamics of selected avian species. With special reference to changes during the modern pesticide era. Wildlife Research Report 1. USDI, FWS, Washington, D.C.

NAGY, K.A. 1987. Field metabolic rate and food requirement scaling in mammals and birds. Ecol. Mono. 57:111-128

NIERING, W.A. 1985. Wetlands. Alfred A. Knopf, New York

PALMER, 1962. Handbook of North American Birds.

PEARSON, T.G., ed. 1936. Birds of America. Doubleday, New York

SKADHAUGE, R. 1975. Renal and cloacal transport of salt and water. Symp. Zool. Soc. London 35:97-106

TOWRY, R.K., Jr. 1984. Wildlife Habitat Requirements - Great Blue Heron. In: Hoover, R.L. and Wills, D.L. (eds.). Managing Forested Lands for Timber and Wildlife. Colorado Division of Wildlife. Denver, Colorado. P. 137.

NORTH AMERICAN MALLARD
(*Anas platyrhynchos*, Linnaeus)

General Description

North American mallards are approximately 18 to 27 inches (46-68 cm) in length. Males of the species have a green head, white neck ring, chestnut breast, and grayish body, while females are a mottled brown. In both the male and female, the inner feather wings (speculum) are metallic purplish-blue, bordered with white in front and back (Audobon Society 1977). Mallards of both sexes average 1,092 g body weight, with a range of 720 to 1,580 g (Owen and Cork 1977, as cited in Dunning 1984).

Habitat Use

Their preferred habitat is the slow moving or still water of ponds, lakes, and marshes. Semi-domesticated birds may be found on almost any body of water (Audubon Society 1977). It is reported that mallards prefer shallow estuarine bays that have agricultural land adjacent for field feeding (Nichols and Hines 1987).

Although not presently quantified, a wealth of high-quality waterfowl habitat exists at the Aberdeen Proving Ground (APG). This is in large part due to the vast complex of relatively undisturbed estuarine and fresh water emergent wetlands, bottomland hardwood wetlands, and uplands. The activity of beavers on APG has resulted in myriad permanently flooded wetlands mixed with saturated wetlands and upland forests (Rewa 1989).

Wintering mallard numbers appear to be related to abundance of submerged aquatic vegetation in some areas of the Chesapeake Bay, and it is possible that declines in Bay mallard numbers are similarly associated with declines of submerged aquatic vegetation (SAV) (Nichols and Hines 1987). SAV provides food for mallards, and food and cover for many aquatic fish and invertebrates. SAV influences the trophic structure of the aquatic community of the Bay (Rewa 1989). SAV was once abundant throughout Chesapeake Bay. Like many important living resources in the Bay, SAV distribution and abundance has been greatly reduced in recent decades in response to water quality degradation. Efforts to reestablish SAV in various aquatic sites around Aberdeen have met with varying degrees of success (Rewa 1989).

Diet and Foraging Behavior

The mallard is omnivorous in regard to its food. Its diet consists of approximately 17 % animal food and 83 % vegetable food (Pearson 1936). The animal food consists of small frogs, tadpoles, toads, lizards, newts, small fish, fish fry, snails, mussels, leeches, earthworms, mice, and similar small game that it finds about the pond and in the edges of the woods (Pearson 1936). The mallard's vegetable food includes grass, many species of seeds and aquatic plants, grain, nuts, acorns, fruits and wild rice (Pearson 1936). Home range size is an average of 630 acres (Gilmer 1975 and Gilmer et al. 1975 in Krby et al. 1985).

Seeds, rootlets and tubers of vegetation indigenous to flooded bottomland forests and other wetlands provide an important winter food source for the mallard. The seeds of native wetland plant are generally more nutritious than those of agricultural grains. In addition, wetland microhabitats generally

support more invertebrates eaten by mallards than upland areas. Common winter food consumed include acorns of pin oak (*Quercus palustris*), willow oak (*Q. phellos*), and red oak (*Q. falcata*); seeds of barnyard grass (*Echinochloa* spp.), rice cutgrass (*Leersia oryzoides*), beggarticks (*Bidens* spp.), and smartweeds (*Polygonum* spp.); and invertebrates including snails, crustaceans, spiders, and beetles (Rewa 1989).

Based on the energy requirements of various species and the energy content of food, Nagy (1987) calculated food ingestion rates (FI) for birds (in grams dry matter per day). For all birds, FI (g/d) = 0.648 ($Wt^{0.651}$) where Wt is body weight in grams. For the average mallard this gives a food ingestion rate of FI (g/d) = 0.648 (1,090 $g^{0.651}$) = 62 g/d. Based on measured body weights and drinking water values from Calder (1981) and Skadhauge (1975), Calder and Bruan (1983) determined an allometric equation for water ingestion (WI) for birds: WI (L/d) = 0.059 ($Wt^{0.67}$) where Wt is average body weight in kilograms. For the North American mallard this gives a WI of 0.059 (1.090 $kg^{0.67}$) = 0.063 L/d.

Reproduction, Growth, and Mortality

The breeding habitat of mallards ranges from Alaska and Greenland south to Virginia, Texas and northern Mexico (Audubon Society 1977). Their nests are well concealed in tall grass, thick dead reeds, or cultivated fields (alfalfa), typically located on dry ground in a depression built up with cattails, reeds, grasses, and other locally available material. The eggs, generally numbering from 8-12 are incubated by female alone for 23-29 days (Harrison 1975). The brood is large with a high percentage of survival (Pearson 1936).

Waterfowl noted to breed at APG include mallards, although wood ducks constitute the majority of production (Rewa 1989).

An age-specific difference in survival probability during fall migration and early winter appears to happen, with higher mortality among young than adult mallards (Nichols and Hines 1987). It was also observed a strong evidence of sex-specific difference in survival rates of winter mallards (Nichols and Hines 1987). Survival rates of males were consistently higher than those of females from the same area (Nichols and Hines 1987).

It appears that mallards exhibit some year-to-year variation in survival rates, however, in many instances survival rates can be modeled as a constant (Nichols and Hines 1987).

Population Dynamics

Mallard breed and winter in the mid-Atlantic region. Mallards are adapted to wintering in dynamic wetland conditions that provide a variety of wetland types and sizes in relatively close proximity (Rewa 1989).

Winter survey data (1950-1978) indicate that the Mid-Atlantic Flyway reference area, which includes New Jersey, Delaware, Maryland, Virginia, North Carolina, West Virginia, and most of Pennsylvania, winters 1-2% of North American mallards (Nichols and Hines 1987). Within this reference area, the Chesapeake Bay is one of the most important and famous traditional waterfowl wintering areas. It is reported that January mallard populations in the Upper Chesapeake Bay during 1953-58 varied from about 16,000 to 151,000 and averaged 69,000 (Nichols and Hines 1987). Recent data on

Chesapeake Bay indicated that the number of mallards wintering in Maryland during 1972-80 (average 26,000) was considerably lower than that during 1956-71 (average 42,000) (Nichols and Hines 1987).

Mallards exhibit some temporal variation in wintering grounds in response to weather and perhaps food supply. However, such variation is relatively small and mallards do indeed exhibit a tendency to return to general wintering areas year after year (Nichols and Hines 1987). That trend is particularly true for adult mallards. In fact, results indicate greater temporal variation in winter distribution patterns of subadults versus adult males (Nichols and Hines 1987).

Mallards (and black ducks) are the main dabbling ducks wintering at APG (Rewa 1989). Approximately 600 pen-reared eight-week old mallards are released at APG each year, in accordance with the State of Maryland mallard release program. Pen-reared mallards are generally released in outlying marsh habitats (Rewa 1989).

Ecological and Societal Significance

The mallard is the most important duck to humans (Pearson 1936). The wild mallard provides food and is the chief waterfowl of most game preserves. In the South the Mallard destroys the scattered rice or volunteer rice of the field, which, if left to grow, would greatly reduce the value of the crop. It is also serviceable to the southern people in another way, as it feeds very largely upon crayfish, which burrow into and undermine the levees and dikes (Pearson 1936).

References

THE AUDUBON SOCIETY. 1977. Field guide to North American birds, Eastern Region. Alfred A. Knopf, New York, pp. 454.

CALDER, W.A. III. 1981. Scaling of physiological processes in homeothermic animals. Ann. Rev. Physiol. 43:301-322

CALDER, W.A. and BRAUN, E.J. 1983. Scaling of osmotic regulation in mammals and birds. Am. J. Physiol. 244:R601-R606

HARRISON, H.H. 1975. A field guide to birds' nests in the United States east of the Mississippi River. The Peterson Field Guide Series. Houghton Mifflin Company. Boston, pp. 24.

KIRBY, R.E., RIECHMANN, J.H., and COWARDIN, L.M. 1985. Home range and habitat use of firest-dwelling mallards in Minnesota. Wilson Bull. 97:215-219

NAGY, K.A. 1987. Field metabolic rate and food requirement scaling in mammals and birds. Ecol. Mono. 57:111-128

NICHOLS, J.D. and HINES, J.E. 1987. Population ecology of the mallard: Winter distribution patterns and survival rates of winter-banded mallards. US Department of the Interior. Fish and Wildlife Service/ Resources Publication 162, 155 pp.

PEARSON, G.T. 1936. Birds of America. Doubleday and Company, Inc., New York. pp. 114-116.

REWA, C.A. 1989. Waterfowl management supplement. Fish and Wildlife Management Section. Natural resources management plan. Aberdeen proving grounds, 43 pp.

SKADHAUGE, R. 1975. Renal and cloacal transport of salt and water. Symp. Zool. Soc. London 35:97-106

PEREGRINE FALCON
(Falco perigrinus)

General Description

The peregrine falcon, or duck hawk, is a raptor with bluish ash coloring above, pinkish to dark rufous with blackish spotting and barring below, a black head, and a blackish macular stripe. It is 47 cm long with a 110 cm wing span. Males weight approximately 700 g while females are larger, in the 1,220 g range.

Habitat Use

Optimal habitat for peregrine falcons in the breeding season is a high cliff or ledge for nesting, with a clear view of surroundings, a ready supply of prey species and water within 0.5-1 mile of the nesting site. Frequently, cliffs are chosen that are along rivers or coastal bays. There have also been reports of peregrine falcons nesting in cities on roofs or ledges of skyscrapers. Winter habitat is less specific and largely predicated on the presence of prey species. APG has excellent winter habitat with estuaries, tidal creeks, and wetlands with an abundance of avian species that are desirable prey for peregrines.

Diet and Foraging Behavior

The peregrine falcon consumes approximately 11-12% of its body weight in food during warn weather and 15-16% in cold weather. Average daily food consumption is 80-100 grams. Based on the energy requirements of various species and the energy content of food, Nagy (1987) calculated food ingestion rates (FI) for birds (in grams dry matter per day). For all birds, FI (g/d) = 0.648 ($Wt^{0.651}$) where Wt is body weight in grams. For the peregrine falcon this gives a food ingestion rate of FI (g/d) = 0.648 (960 $g^{0.651}$) = 57 g/d. Based on measured body weights and drinking water values from Calder (1981) and Skadhauge (1975), Calder and Bruan (1983) determined an allometric equation for water ingestion (WI) for birds: WI (L/d) = 0.059 ($Wt^{0.67}$) where Wt is average body weight in kilograms. For the peregrine falcon this gives a WI of 0.059 (0.96 $kg^{0.67}$) = 0.057 L/d.

The falcon's diet is primarily other birds: medium-sized passerines, icterids, shorebirds, pigeons, and small-to-medium sized waterfowl. Occasionally small mammals or dead fish are eaten. The falcon has two methods of obtaining prey: grasping it in midair (hawking) or striking the prey in midair and recovering it on the ground. The falcon's hunting range is extensive; falcons frequently like to hunt along a river or over a large valley.

Life Cycle Characteristics

Peregrine falcons reach sexual maturity at 2-3 years and mate for life. They build little or no nests, depositing eggs in a scraped depression on a rocky shelf on the side of a cliff. Eggs are laid from March through May. Clutch size is 2-6, typically 4. The incubation period is 33-35 days. There is one brood per year unless the first clutch is destroyed; then, a smaller second clutch may be laid. Breeding success may be as low as 15% of eggs laid. Fledgling age at first flight is 5-7 weeks. Adult birds show strong territorial behavior in defence of their nest. Peregrine falcons were released at APG but are rarely seen during the breeding season.

Population Dynamics

Mortality of immature peregrine falcons is 70% while average adult mortality is 25%. The life span of peregrine falcons is approximately 20 years.

Peregrines have been extirpated in the eastern and mid-USA and in southern Canada. There are still scattered breeding areas in the western USA and Mexico. Peregrines, despite their release, are rare at APG. The primary reason for the severe decline of the population was related to the effects of DDT and other organochlorine insecticides on eggshell thickness.

Ecological and Societal Significance

Perigrine falcons have few natural predators. Each pair of breeding birds has a wide home range, average 20 square miles.

The perigrine falcon is an endangered species because of the previously discussed population decline. Their major societal significance is because they became the focal point of inquiries into the deleterious effects of insecticides and other environmental contaminants. They have great historical significance because of their use in falconry.

References

BULL, J. and FARRAND, J., Jr. 1977. The Audubon Society Field Guide to North American Birds. Eastern Region. Alfred A. Knopf, New York

CALDER, W.A. III. 1981. Scaling of physiological processes in homeothermic animals. Ann. Rev. Physiol. 43:301-322

CALDER, W.A. and BRAUN, E.J. 1983. Scaling of osmotic regulation in mammals and birds. Am. J. Physiol. 244:R601-R606

CHAMBER, E.B. 1974. Rare and Endangered Birds of the Southern National Forests. USDA. Forest Service, Southern Region

CRAIGHEAD, J.J. and CRAIGHEAD, F.O., Jr. 1969. Hawks, Owls, and Wildlife. Dover Publications, New York

EVANS, D.L. 1982. Status Report on Twelve Raptors. USDA, FWS Special Scientific Report - Wildlife No. 238. Washington, D.C.

NAGY, K.A. 1987. Field metabolic rate and food requirement scaling in mammals and birds. Ecol. Mono. 57:111-128

NIERING, W.A. 1985. Wetlands. Alfred A. Knopf, New York

PEARSON, T.G., ed. 1936. Birds of America. Doubleday, New York

SKADHAUGE, R. 1975. Renal and cloacal transport of salt and water. Symp. Zool. Soc. London 35:97-106

SPOTTED SANDPIPER
(Actitis macularia)

General Description

The spotted sandpiper, is a robin-sized (19 cm) shorebird with a long beak. In Minnesota, adult male mass averages about 40 g, while the larger females average approximately 48 g (Maxson and Oring 1980; Oring and Lank 1986). In the breeding season, it is olive brown above with black spots below and in winter it is all brown.

Habitat Use

The sandpiper has relatively few specific habitat requirements except for the presence of open water nearby, although it can also be found in cultivated fields. There is no documentation of the presence of this bird at APG although habitats exist that would support it.

Diet and Foraging Behavior

The spotted sandpiper's diet consists principally of insects, although marine worms, small crustaceans and small mollusks are sometimes eaten. It forages, with a characteristic swaying motion, among pebbles on banks and shoals of bodies of water.

Based on the energy requirements of various species and the energy content of food, Nagy (1987) calculated food ingestion rates (FI) for birds (in grams dry matter per day). For all birds, FI (g/d) = 0.648 ($Wt^{0.651}$) where Wt is body weight in grams. For the spotted sandpiper this gives a food ingestion rate of FI (g/d) = 0.648 (42.5 $g^{0.651}$) = 7.5 g/d. Based on measured body weights and drinking water values from Calder (1981) and Skadhauge (1975), Calder and Bruan (1983) determined an allometric equation for water ingestion (WI) for birds: WI (L/d) = 0.059 ($Wt^{0.67}$) where Wt is average body weight in kilograms. For the spotted sandpiper this gives a WI of 0.059 (0.0425 $kg^{0.67}$) = 0.0071 L/d.

Life Cycle Characteristics

The spotted sandpiper breeds throughout North America and winters south to Brazil and Peru. The bird is highly adaptable in choice of nesting site but always seeks proximity to water. Miller and Miller (1948 in DeGraaf and Rudis 1987) found a density of 43 pairs/17.6 acres of dry meadow-rocky shore-sandy beach habitat in Michigan. The nest, constructed of grass, leaves, and weed stems, is built in a depression in the ground. Breeding usually occurs at the end of May, with one brood per year. Females are often polyandrous. Clutch size is 4. The incubation period is 15 days. There is no documentation of breeding success at APG. The young sandpipers are precocious, being able to run and swim shortly after hatching.

Ecological and Societal Significance

Spotted sandpipers are one of the most common shorebirds in North America. Apparently, the population is quite stable. No documentation was available for populations - if present - at APG.

These sandpipers are solitary and do not gather in large flocks. Spotted sandpipers are potential prey of raptors although they elude them because of their shifty flight patterns.

References

BENT, A.C. 1962. Life Histories of North American Shore Birds. Part II. Dover Publications, New York

BULL, J. and FARRAND, J., Jr. 1977. The Audubon Society Field Guide to North American Birds. Eastern Region. Alfred A. Knopf, New York

CALDER, W.A. III. 1981. Scaling of physiological processes in homeothermic animals. Ann. Rev. Physiol. 43:301-322

CALDER, W.A. and BRAUN, E.J. 1983. Scaling of osmotic regulation in mammals and birds. Am. J. Physiol. 244:R601-R606

DeGRAAF, R.M. and RUDIS, D.D. 1987. New England Wildlife: Habitat, Natural History, and Distribution. U.S. Department of Agriculture, Forest Service, Northeastern Forest Experiment Station. General Technical Report NE-108.

HARRISON, H.H. 1975. A Field Guide to Birds Nests of 285 Species Found Breeding in the United States East of the Mississippi River. Houghton Mifflin Co., Boston

MAXSON, S.J. and ORING, L.W. 1980. Breeding season time and energy budgets of the polyandrous spotted sandpiper. Behaviour 74:200-263

NAGY, K.A. 1987. Field metabolic rate and food requirement scaling in mammals and birds. Ecol. Mono. 57:111-128

NIERING, W.A. 1985. Wetlands. Alfred A. Knopf, New York

ORING, L.W. and LANK, D.B. 1986. Polyandry in spotted sandpipers: The impact of environment and experience. In: Ecological Aspects of Social Evolution - Birds and Mammals, D.I. Rubenstein, and R.W. Wrangham (eds.).

PEARSON, T.G., ed. 1936. Birds of America. Doubleday, New York

SKADHAUGE, R. 1975. Renal and cloacal transport of salt and water. Symp. Zool. Soc. London 35:97-106

FISH

ALEWIFE

(*Alosa pseudoharengus*, Wilson)

General Description

The alewife is found from Newfoundland to South Carolina with some landlocked populations found in freshwater lakes. In the Chesapeake Bay male alewives reach a length of 229-259 mm (FL) and females reach 239-282 mm (FL).

Habitat Use

Young of the year are found in fresh and low salinity estuarine waters in the summer and early fall. They prefer 0-2.0 ppt salinity, 15.5°C, and a dissolved oxygen level of at least 3.5 ppm. Subadults and adults inhabit ocean waters unless spawning and can tolerate salinities of 0-35 ppt. They also require a minimum dissolved oxygen concentration of 5.0 ppm and are most abundant at depths of 56-110 m.

Larvae are present in the northern Chesapeake Bay from hatching until June when they show slight downstream movement no further than areas with salinities of 12 ppt. Juveniles show vertical diel movement in the water column and move from the fresh estuarine nursery areas in the spring of their second year (migration takes two to three days). This move is stimulated by heavy rainfall, high water, and a sharp decline in water temperature. During the spring adults spawn in fresh or brackish water, migrate downriver soon thereafter, and spread out over the continental shelf (contained in the study area of NMFS catch data). The same study found that in the summer and fall adults are found north of 40 degrees N. latitude in three major areas: Nantucket Shoals, Georges Bank, and the perimeter of the Gulf of Maine. In the winter adult Alewives are found between 40 and 43 degrees N. latitude. In cold weather adults are found in deep bay waters or have returned to the sea.

Young alewives and blueback herring are found in the same habitats but there is a spatial separation between the species in the same area.

Adult distribution is similar to that of the shad and they are probably found throughout APG waters. Alewife and blueback herring both spawn in a similar locale to the shad. Spawning is most likely to take place north of Canal Creek from the mouth of Bird river to 5 miles upstream, similarly north of the mouth of and including Otter Point Creek. According to the Environmental Sensitivity Map, alewife and blueback herring spawn in the Gunpowder River near Canal Creek. Larvae and juveniles will be present at APG until they emigrate to the sea in the fall.

Diet and Foraging Behavior

Alewives feed in schools and are zooplanktivorous. Larvae and juveniles concentrate on small cladocerans and copepods. Young of the year in Cape Fear River, N.C. consumed more ostracods, insect eggs, and insect parts than Blueback herring however, both had stomach contents of over 80% crustacean eggs. The size and range of adult prey increases with fish size. Along with zooplankton adults eat various types of eggs (fish, crustacean, insect) and small fish.

Alewives link zooplankton and top piscivores in estuarine and marine food webs. Both alewife and blueback herring are important prey for riverine, estuarine, and marine piscivores such as gulls, terns, weakfish, bluefish, and striped bass. Since the clupeids are schooling they tend to be preyed upon by schooling predators.

Life Cycle Characteristics

The optimum temperature for hatching is 18-20.8°C. The yolk sac/larvae stage lasts 2-5 days. By August of the first year alewives reach a mean FL of 44-47.8 mm and in the lower Chesapeake young grow an average of 102 mm TL between hatching and emigration. Growth rate levels off at sexual maturity.

Growth rates are affected by both increased or decreased temperature. An upper lethal temperature of 29.7°C is found for Hudson River Alewife eggs and egg mortality is directly correlated with incubation temperature (22% mortality at 3.5-6.0°C, 66% mortality at 25.5-28.5°C). Larval growth rate also increases with increasing temperature. Hudson River larvae have a maximum growth rate of 0.084 g/day at 29.1°C and an upper lethal temperature of 31°C.

Alewives are anadromous fish spawning late March through April beginning when the water reaches 10.5°C and ceasing when temperatures exceed 27°C. Spawning occurs in the northern Chesapeake Bay and all major tributaries in a variety of habitats ranging from standing water (although MDNR (1988) states that moving water is required) to fast moving or mid river sites. Salinity can range 0-6.0 ppt but most spawning is in water with salinities below 1 ppt. Temperature of the spawning site ranges 10-21°C with a minimum requirement for dissolved oxygen of 5 ppm and a pH range of 6.5-7.8.

Population Dynamics

The percentage of males in the population decreases with age class as seen in a study of Connecticut and Thames River spawning alewife populations.

age	% male
4	72.3
5	63.7
6	49.7
7	33.8
8	0

Ecological and Societal Significance

There is a significant commercial and recreational fishery for the river herrings, alewife and blueback herring.

All ages of alewives are sensitive to low dissolved oxygen and young are sensitive to changes in temperature. River herring eggs and larvae suffer high mortalities below pH 6.5.

References

LIPPSON, A.J., ed. 1985. The Chesapeake Bay in Maryland - An Atlas of Natural Resources. Natural Resources Institute of the University of Maryland.

MARYLAND DEPARTMENT OF NATURAL RESOURCES AND VIRGINIA MARINE RESOURCES COMMISSION (MDNR). 1988. Chesapeake Bay Fisheries: Status, Trends, Priorities and Data Needs. Annapolis, MD. September 1988.

U.S. DEPARTMENT OF THE INTERIOR. 1983. Species Profiles: Life Histories and Environmental Requirements of Coastal Fishes and Invertebrates (Mid-Atlantic): Alewife/Blueback Herring. Fish and Wildlife Service. Coastal Ecology Group, Waterways Experiment Station. FWS/OBS-82/11.9. October 1983

VIRGINIA INSTITUTE OF MARINE SCIENCE (VIMS). No Date. Environmental Sensitivity Map. College of William and Mary.

AMERICAN SHAD
(*Alosa sapidissima*, Wilson)

General Description

The shad is an anadromous fish present all along the Atlantic Coast from NewFoundland to Florida. It is most concentrated from Connecticut to North Carolina. On the Pacific Coast it occurs from Southern California to Cook Inlet, Alaska and also occurs around the Kamchatka Peninsula in Asia.

Average total length for the shad is 380 mm and it generally weighs up to 2.7 kg for males and 3.6 kg for females.

Habitat Use

Young of the year are found in fresh and low salinities through the early fall. At this time they inhabit areas with salinities of 0-7.5 ppt and a minimum temperature of 15.6°C. Young avoid changes in temperature of 4°C or more. The minimum allowable DO concentration is 5.0 ppm and the preferred pH range for young is 5-9. Larvae are more susceptible to suspended sediment concentrations than eggs. Larvae can not handle sediments above 0.1 g/l but eggs can handle concentrations up to 1 g/L Once the young leave the estuary for the sea in the fall they must have a greater tolerance to salinity and temperature. Adults can be found in salinities from 0-35 ppt but probably spend a few days in the estuary between the sea and freshwater while migrating upstream to adjust to the salinity changes. They have a temperature range of 7.2-17.8°C and a requirement of 5 ppm DO. Substrate is not important to the adults because eggs can be laid anywhere as they are just carried downstream soon after.

American shad spend most of their life at sea. All ages form large schools and undertake extensive migrations in the ocean preferring to follow bottom water temperatures between 7 and 13°C. In the spring they follow the Gulf Stream back down the coast and enter the Chesapeake Bay primarily in March, April, and May. Adults spawn in low salinity to fresh water in their river of origin. Natal streams are found by olfaction and rheotaxis (detection of water current) and fish can even find the stream if major changes in water flow have occurred (such as a dam). Young of the year spend the summer in fresh and brackish water and when triggered by a decrease in water temperature in the fall they emigrate to the ocean where they will stay until they reach sexual maturity.

APG serves as a nursery ground for juveniles and possibly, though unlikely, parts also serve as a spawning area. Shad are located at APG as far north as the mouth of Bird river, which drains into Gunpowder River, and just south of Otter Point Creek which drains into the Bush River. The eggs will only be found in the freshwater tidal portions of the rivers and spawning grounds are indicated as being north of the mouth of Bird River as well as north of and including Otter Point Creek. The Environmental Sensitivity Map (VIMS, no date) suggests spawning may take place further south in the Gunpowder River near Canal Creek. Spawning occurs in late spring and larvae and juveniles will be present in the area until late October and November when they migrate to the lower Bay and ocean.

Diet and Foraging Behavior

The American shad eats a wider range of food than the alewife and blueback herring. The juveniles spend their first summer in the river feeding on crustaceans and aquatic insets at the surface or in the water column. Juveniles and adults at sea feed on small crustaceans (many of which are benthic)

and to a lesser extent feed on small fishes, euphausiids, fish eggs and amphipods. They exhibit daily vertical migrations to follow the zooplankton. When the adults are in freshwater they feed on mayflies and small fish.

In freshwater shad have a variety of predators. However, in seawater they are preyed on by seals but have few other worries than man. Lampreys may attach to adult fish.

Life Cycle Characteristics

The rate of development of eggs is linked to temperature. No development occurs below 7°C and above 24°C. Eggs develop in 8-12 days at 11-15 C, 6-8 days at 17°C, and 3 days at 24°C although deformities are likely to occur at this high of a temperature. Young grow at a rate of 100 mm/yr until sexual maturity at which time growth slows.

Most spawning in the Chesapeake Bay occurs from April to June in tidal freshwater areas dominated by extensive flats. Spawning is triggered by temperature, photoperiod, flow velocity, and water turbidity. Salinity is limited to 0-2.0 ppt and depth can range from 1 to 10 m but most spawning occurs in water less than 3 m deep. DO must be at least 5 ppm and pH can range from 6.5 to 7.8. Shad spawn far enough upstream so that eggs can drift downstream and hatch before reaching saltwater. Tidal or fluvial movement must be 0.5 to 3.0 ft/sec so that eggs do not settle to the bottom and get smothered.

Population Dynamics

In 1989, the estimated American shad population size in the Upper Chesapeake Bay was 75,329 to 79,973. In the same year the sex ratio of the spawning population was 1:0.51 (M:F). This spawning population was dominated by 4 and 5 year olds and was composed of 3, 4 and 5 year old males and 4, 5 and 6 year old females. The population of shad was down to dangerously low levels in the late seventies but since the fishery was closed in Maryland in 1980 the population size has been increasing and is now fairly stable. Recruitment depends on the size of the spawning stock and environmental factors that govern spawning success and survival. Approximately 85% of the variation in the numbers of shad that spawn in a year depend on the spawners 5, 4 and 1 year ago. About 64% of recruitment is related to the number of spawners and 22% is attributed to environmental factors such as water temperature and river flow.

Ecological and Societal Significance

The American shad is an important commercial fish. Although the fishery in Maryland has been closed since 1980, it is still fished in several other states. The average annual catch at APG from 1975-1980 is 2,486 lbs/yr - 47% of this catch was in the Susquehanna Flats area.[1]

Acid deposition and stream acidification is a problem for all anadromous fish. Although no direct studies have been performed on the shad, the closely related river herring suffers mortality of eggs and larvae at pH below 6.5.

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[1]Personal communication. Jim Pottie. Aberdeen Proving Ground Office of Environmental Management.

References

LIPPSON, A.J., ed. 1985. The Chesapeake Bay in Maryland - An Atlas of Natural Resources. Natural Resources Institute of the University of Maryland.

MARYLAND DEPARTMENT OF NATURAL RESOURCES AND VIRGINIA MARINE RESOURCES COMMISSION (MDNR). 1988. Chesapeake Bay Fisheries: Status, Trends, Priorities and Data Needs. Annapolis, MD. September 1988.

U.S. DEPARTMENT OF THE INTERIOR. 1985. Species Profiles: Life Histories and Environmental Requirements of Coastal Fishes and Invertebrates (Mid-Atlantic): American Shad. Fish and Wildlife Service. Coastal Ecology Group, Waterways Experiment Station. Biological Report 82(11.37). April 1985

U.S. DEPARTMENT OF THE INTERIOR FISH AND WILDLIFE SERVICE. 1990. Investigation of Anadromous Alosids in Chesapeake Bay. Federal Aid Annual Report. Project No. F-37-R. Annapolis, MD

VIRGINIA INSTITUTE OF MARINE SCIENCE (VIMS). No Date. Environmental Sensitivity Map. College of William and Mary.

ATLANTIC MENHADEN
(*Brevoortia tyrannus*, Latrobe)

General Description

The Atlantic menhaden has a mid-Atlantic range in the temperate coastal waters from Nova Scotia to Jupiter Inlet, Florida. In 8 to 10 years the fish reach lengths of 500 mm (TL) and weights of over 1500 g.

Habitat Use

Atlantic menhaden depend on the estuarine and nearshore systems of the eastern seaboard and shelf waters throughout their whole life cycle. Chesapeake young of the year less than 2 inches long stay in estuarine waters until at least the end of the summer when some migrate to coastal waters while the rest overwinter in the Bay. Young can tolerate salinities from 0 to 35 ppt and have a temperature tolerance of 6-30°C. Adults can also tolerate wide salinities from less than 1 to 36 ppt. They prefer temperatures from 15.6 to 21.1°C and require at least 5 ppm dissolved oxygen.

Subadults and adults are generally found in the continental shelf waters adjacent to major estuaries. Extensive north-south migrations and in-offshore movements are made in the spring and fall. Chesapeake adult menhaden migrations coincide with the 10°C isotherm with northern inshore migrations occurring in the spring and southern return (to North Carolina) in the fall. Many age 0 fish who have recently emigrated from the estuaries will migrate southward along the North Carolina coast in late fall and early winter. From November to March all ages congregate offshore in North Carolina. In the summer the fish are distributed along the coast by age and size with the larger fish in the North and the smaller fish towards the Southern Atlantic.

Adult menhaden general distribution relative to APG is primarily south of the tip of Gunpowder Neck and around Poole Island and towards the eastern shore of the Bay. The juveniles, however, are found throughout APG waters. According to the Environmental Sensitivity Map (VIMS, no date), menhaden are common local species in the Gunpowder Neck area during spring, summer, and fall. The Management Plan says that larvae arrive in winter. They are most likely to occur in tidal marshes with salinities less than 5 ppt and could occur in Watson Creek and Canal Creek areas.

Diet and Foraging Behavior

Larvae are zooplanktivores extremely selective in the size and species they eat favoring specific taxa of copepods and copepodites. Larvae eat to capacity and will starve if the food concentration is too low because they are unable to move themselves to areas of higher food concentration. This is mostly a problem when they are spawned too far offshore or swept offshore after spawning. When larvae metamorphose they switch from capturing individual zooplankton to filter feeding. Juveniles and adults filter feed on zooplankton and phytoplankton - especially chain forming diatoms, and may also eat organic detritus. Diet changes with food availability and swimming speed increases with food concentration.

Life Cycle Characteristics

Growth begins in the spring and ends in the fall as the water approaches the 15°C isotherm and the rates vary with year and locality. Metamorphosis of larvae is rarely successful outside of a food rich/low salinity environment. Three stanzas of growth occur in young fish with changes occurring at 30 mm (70 mg) and 38 mm (469 mg).

Spawning occurs almost every month in some part of the species range but there are two spawning peaks off Chesapeake Bay in the spring and fall. It takes place in Continental Shelf waters at salinities around 25 ppt or higher. Spawning temperatures range from 4.4 to 23.3°C but peak spawning occurs from 15 to 18°C. Eggs hatch in 2-3 days at approximately 15.5°C and hatching time decreases with increasing temperature. Because there is little parental care there is a critical period during the first 4 to 5 days after hatching while the fish feed on the yolk and finish development. Soon after larvae migrate to estuaries via Ekman transport and occur in tributaries of the Chesapeake Bay during May, June and November at salinities of 1 to 3 ppt. Young are sensitive to salinity, rapid temperature changes, and food concentration. Also, pollution stress may greatly reduce first year survival rate.

Population Dynamics

Schools consist of fish of similar size and age except for migrating schools which consist of all ages of fish.

Ecological and Societal Significance

Menhaden are very abundant. They have extensive migration patterns, and are an important prey species. Therefore they influence the conversion and exchange of energy and organic matter throughout their range. They are a direct pelagic link between detritus and plankton and top predators and are an important forage species for bluefish, striped bass, bluefin tuna and sandbar sharks.

Atlantic menhaden are important commercially, making up 25-40% of the combined annual landings of all Atlantic coast and Gulf of Mexico species. They made up the largest commercial fishery by weight and eighth largest by dollar in the United States in 1984. The annual average catch at APG is 29,350 lbs and 76% of this catch is from the Chesapeake Bay.

References

FERRARO, S.P. 1981. Eggs and Larvae of Atlantic Menhaden (*Brevoortia tyrannus*) in the Peconic Bays, New York in 1972-74. Rapp. P. -v. Reun. Cons. int. Explor. Mer. 178:181-182

LIPPSON, A.J., ed. 1985. The Chesapeake Bay in Maryland - An Atlas of Natural Resources. Natural Resources Institute of the University of Maryland.

MARYLAND DEPARTMENT OF NATURAL RESOURCES AND VIRGINIA MARINE RESOURCES COMMISSION (MDNR). 1988. Chesapeake Bay Fisheries: Status, Trends, Priorities and Data Needs. Annapolis, MD. September 1988.

MILLER, WIHRY, AND LEE, INC. 1981. Natural Resources Management Plan. Part IV: Fish and Wildlife Management, Aberdeen Proving Ground, MD. Washington, DC

U.S. DEPARTMENT OF THE INTERIOR. 1989. Species Profiles: Life Histories and Environmental Requirements of Coastal Fishes and Invertebrates (Mid-Atlantic): Atlantic Menhaden. Fish and Wildlife Service. Coastal Ecology Group, Waterways Experiment Station. Biological Report 82(11.108). August 1989

VIRGINIA INSTITUTE OF MARINE SCIENCE (VIMS). No Date. Environmental Sensitivity Map. College of William and Mary.

BAY ANCHOVY
(*Anchoa mitchilli*, Valenciennes)

General Description

The bay anchovy ranges all along the Atlantic and Gulf of Mexico coasts from Cape Cod, Massachusetts to Yucatan, Mexico except for the Florida Keys. In the mid-Atlantic they are abundant off Massachusetts, Rhode Island, New York, New Jersey, and the Chesapeake Bay. The average size of a Bay anchovy is 75 mm.

Habitat Use

The anchovy can tolerate wide ranges of temperatures. They have been collected in the Hudson Estuary, NY at temperatures of 2.2-27.1°C. Salinity, substrate type, and vegetation are of little significance. In the mid-Atlantic they have been found in shallow and moderately deep offshore water, nearshore water off sand beaches, open bays and muddy coves, grass areas along beaches, waters around mouths of rivers, bayous and coastal waters, seagrass beds, and freshwater rivers.

When the estuary waters start to warm juveniles and some adults move to freshwater to feed. The mature move downstream to spawn when water reaches 12 C and salinities greater than 10 ppt. The newly hatched larvae move upstream to less than 10 ppt to feed until the fall when the larvae and juveniles move to more saline areas. By late November anchovies occur only in salt water. Chesapeake Bay is a major spawning site where the eggs tend to be close to the surface in salinities of 8-15 ppt. The larvae are found in highest densities at salinities of 4.2-6.0 ppt at or soon after the time of maximum water temperature.

The bay anchovy is seasonally abundant around Carroll Island (Speir 1972). According to the Environmental Sensitivity Map, the bay anchovy is a resident estuarine species at APG in the Gunpowder River and Bush River. They do not spawn at APG, but the Gunpowder and Bush Rivers are used by larvae and juveniles as nursery areas and adult habitat with the exception of the waters surrounding Carroll Island and Gunpowder Neck in the vicinity of J-field and O-field, these are less likely to be suitable nursery habitats. Anchovy could be present at APG from spring through late fall in all habitats.

Diet and Foraging Behavior

Young principally eat copepods while adults primarily eat mysids. However, the range of foods reported to have been eaten by anchovies is quite varied including other crustaceans such as amphipods and ostracods, mollusks, larval fish, and detritus.

Bay anchovies are important links between zooplankton and piscivores. Their predators include piscivorous birds, bluefish, young weakfish, striped bass, and other commercially important fish.

Anchovies require high and stable food densities to survive. If food densities are low they spend too much energy to get the minimum required food level and are susceptible to predation and starvation.

Life Cycle Characteristics

Spawning occurs from May to September in the Chesapeake Bay when the temperature is at least 12°C and the salinity is over 10 ppt. Spawning occurs when temperature is above 20°C and salinity is 13-15 ppt. Larvae hatch in 24 hours at temperatures of 27.2-27.8°C. Newly hatched larvae must feed within 2.5 days of hatching or abnormal development may occur. If larvae hatch early enough they may become sexually mature by the end of their first summer.

Ecological and Societal Significance

Anchovies are an important food source for commercial fish, because of their abundance and widespread distribution. They have some limited use as anchovy paste and bait.

References

LIPPSON, A.J., ed. 1985. The Chesapeake Bay in Maryland - An Atlas of Natural Resources. Natural Resources Institute of the University of Maryland.

SPEIR, H.J. 1972. Fish Populations Around Edgewood Arsenals Chemical Agent Test Area. Prepared for Department of the Army, Edgewood Arsenal, Maryland. Technical Report EATR 4609

U.S. DEPARTMENT OF THE INTERIOR. 1989. Species Profiles: Life Histories and Environmental Requirements of Coastal Fishes and Invertebrates (Mid-Atlantic): Bay Anchovy. Fish and Wildlife Service. Coastal Ecology Group, Waterways Experiment Station. Biological Report 82(11.97). February 1989

VIRGINIA INSTITUTE OF MARINE SCIENCE (VIMS). No Date. Environmental Sensitivity Map. College of William and Mary.

BLUEBACK HERRING
(*Alosa aestivalis*, Mitchill)

General Description

Blueback herrings range from Nova Scotia to the St. John's River in Florida. Males from Albemarle Sound, N.C. reach 231-270 mm (FL) and females reach 245-307 mm (FL).

Habitat Use

Physical parameters are similar to those which govern the alewife. Young of the year are found in fresh and low salinity (0-2.0 ppt throughout mid-summer) estuarine waters in the summer and early fall. A minimum of 15.6°C has been reported and dissolved oxygen must be 3.6 ppm or greater. Adults are found in the ocean except during spawning (mid April - mid May) and can tolerate varied salinity from 0 to 35 ppt and must have at least 5.0 ppm dissolved oxygen.

Juvenile blueback herring show vertical diel movements with a sensitivity to the sky opacity index or light intensity. In the fall juveniles move towards the bottom from the surface. Young of the upper Chesapeake Bay move rapidly from fresh/estuarine nurseries early spring of their second year. This migration is stimulated by heavy rainfall, high water, and a sharp decline in water temperature.

Adult distribution is similar to that of the shad and they are probably found throughout APG waters. Alewife and Blueback herring both spawn in a similar locale to the shad. Spawning is most likely to take place north of Canal Creek from the mouth of Bird river to 5 miles upstream, similarly north of the mouth of and including Otter Point Creek. According to the Environmental Sensitivity Map, alewife and blueback herring spawn in the Gunpowder River near Canal Creek. Larvae and juveniles will be present at APG until they emigrate to the sea in the fall.

Diet and Foraging Behavior

Blueback herring mostly feed in schools during the daytime. All ages are zooplanktivores. Young concentrate on cladocerans and copepods. Cape Fear River young of the year ate more copepods and dipteran larvae than young alewife did and like the alewife the blueback herring young were found to have over 80% of their stomach contents consist of crustacean eggs. Adult bluebacks eat eggs and small fish - the size and range of the prey increases with the size of the fish.

Bluebacks are important forage species for riverine, estuarine and marine piscivores such as gulls, terns, bluefish, weakfish, and striped bass. They are important links between zooplankton and top piscivores.

Life Cycle Characteristics

The yolk sac/larvae stage for blueback herring lasts 2-3 days. By November 15th bluebacks reach 35.6 mm and 3.68 g. Growth rate levels off at sexual maturity.

Temperature affects growth rates of various stages. Incubation time shortens with increasing temperature. Lab data from New Brunswick, Canada shows that the larval deformity rate with a 10

degree C increase in temperature is 0-25% and with a 15 degree C increase is 100%. The deformities were such that in nature the larvae would not survive.

Blueback herring are anadromous fish spawning from the last half of April through the first half of May beginning when the water reaches 14°C and ceasing when temperatures exceed 27°C. Spawning habitat varies from streams to large rivers in areas with hard substrates and strong currents. The Bluebacks are more specialized than Alewives and will be more affected by changes in currents or substrates. Salinity in the spawning habitat can range from 0-6 ppt but is mostly below 1 ppt. Temperature range for spawning is 13.8-26.7°C with a minimum requirement of 5.0 ppm dissolved oxygen and pH range of 6.5-7.8.

Population Dynamics

The percentage of males in the population decreases with age class.

age	% males
3	80
4	79.4
5	64.5
6	36.9
7	22.6

Ecological and Societal Significance

There is a significant commercial and recreational fishery for the river herrings, alewife and blueback herring. The annual average catch for APG from 1975-87 is 27,885 lbs.; 91% of this catch is from the Susquehanna flats.

The Blueback is sensitive to temperature changes, light intensity, and changes in substrate or current flows. River herring eggs and larvae suffer high mortalities below pH 6.5.

References

LIPPSON, A.J., ed. 1985. The Chesapeake Bay in Maryland - An Atlas of Natural Resources. Natural Resources Institute of the University of Maryland.

MARYLAND DEPARTMENT OF NATURAL RESOURCES AND VIRGINIA MARINE RESOURCES COMMISSION (MDNR). 1988. Chesapeake Bay Fisheries: Status, Trends, Priorities and Data Needs. Annapolis, MD. September 1988.

U.S. DEPARTMENT OF THE INTERIOR. 1983. Species Profiles: Life Histories and Environmental Requirements of Coastal Fishes and Invertebrates (Mid-Atlantic): Alewife/Blueback Herring. Fish and Wildlife Service. Coastal Ecology Group, Waterways Experiment Station. FWS/OBS-82/11.9. October 1983

VIRGINIA INSTITUTE OF MARINE SCIENCE (VIMS). No Date. Environmental Sensitivity Map. College of William and Mary.

CHANNEL CATFISH
(*Ictalurus punctatus*)

General Description

Channel catfish are a primarily freshwater species found in the southern portions of the Canadian Prairie Provinces south to the Gulf states, west to the Rockies, and east to the Appalachians. They are found in all of the Pacific and Atlantic drainages in the 48 contiguous states.

Habitat Use

The optimum habitat for channel catfish is deep pools and littoral areas with warm temperatures, low salinity, a diversity of velocities, and structural features for food and cover. The best areas are 5 m deep or less with 40% or greater suitable cover. DO should be 7 mg/l or more and the best temperature range is 26-29°C. Turbidities between 25 and 100 ppm may alleviate some of the need for fixed cover.

The Chesapeake Bay young of the year must have cover and prefer shallow water sandbars with submerged trees and rocks. In streams young inhabit shallow riffles and turbulent areas near sandbars. They are found in 0-1 ppt salinity, 28-30°C, and at least 5 ppm DO however, all ages are resistant to low DO and turbid waters.

Subadults and adults of the Chesapeake Bay are found in channels of large rivers and prefer sluggish to swift currents over sand. They are also found in areas with a gravel or rubble bottom which may be mixed with mud or in areas downstream from sandbars in deep quiet water. They are seldom found in areas of dense vegetation and are generally freshwater although they may be found in salinities up to 5.0 ppt. Although they usually require at least 5.0 ppm DO they can stand as low as 3.0 ppm for short periods.

Channel catfish are the predominant species in the Upper Bay and Susquehanna region although brown bullheads are also found there (white catfish are unlikely to be in this region). The channel catfish is a common species at APG and may be found in the fresh water or slightly brackish water. Generally, larger fish are found near the mouths of creeks and in the larger rivers.

Diet and Foraging Behavior

Larvae and juveniles eat plankton and aquatic insects whereas adults are opportunistic feeders with a varied diet. Adults may eat terrestrial and aquatic insects, crayfish, detrital and plant material, and mollusks. Catfish greater than 50 cm have fish as a major part of their diet. Although bottom feeding is more characteristic adults may feed throughout the water column. Feeding is done primarily at night using vision and chemosenses.

Life Cycle Characteristics

Optimum hatching is 6-7 days at 27°C and the best temperature for the growth of fry is 29-30°C.

Sexual maturity for southern fish occurs at age V or less. Northern males mature at age VI or greater whereas female do not mature until age VIII or greater.

Spawning occurs in May and June in the fresh and tidal fresh areas of the Northern Chesapeake Bay and all major tributaries. The male makes (and guards) the nest in a protected area in weeds or under rock ledges. He may also make the nest in small streams and sometimes in swift water. Spawning is inhibited if suitable nesting cover is not available. Salinities are less than 2.0 ppt in the spawning area and there must be at least 5.0 ppm DO. Temperature can range 21-29°C. Catfish fecundity ranges from 1,052 to 70,000 eggs/female.

Ecological and Societal Significance

Channel catfish are caught by both commercial and recreational fisherman. The annual average catch at APG is 39,913 lbs - 32% of which comes from the Susquehanna flats.

References

HILDEBRAND, S.F. and SCHROEDER, W.C. 1928. Fishes of the Chesapeake Bay. U.S. Bur. Fish. Bull. 43(1) (for 1927):1-366

LIPPSON, A.J., ed. 1985. The Chesapeake Bay in Maryland - An Atlas of Natural Resources. Natural Resources Institute of the University of Maryland.

MARYLAND DEPARTMENT OF NATURAL RESOURCES AND VIRGINIA MARINE RESOURCES COMMISSION (MDNR). 1988. Chesapeake Bay Fisheries: Status, Trends, Priorities and Data Needs. Annapolis, MD. September 1988.

McMAHON, T.E., and TERRELL, J.W. 1982. Habitat Suitability Index Models: Channel Catfish. U.S. Fish and Wildlife Service. FWS/OBS-82/10.2 February 1982

POTTIE, J. 1990. Personal communication. Spoke with Tanya Yudelman-Bloch on June 14 and 18, 1990

MUMMICHOG

(*Fundulus heteroclitus*, Linnaeus)

General Description

The mummichog ranges from the Gulf of St. Lawrence to northeastern Florida. The adults range from 51-102 mm (TL) with the males smaller than the females (after the first year).

Habitat Use

Mummichogs remain in water deeper than 10 cm but are seldom found deeper than 3.66 m. They are eurythermal and euryhaline so they can stand rapid temperature and salinity changes although mummichogs have been seen to prefer 20 ppt over 8 ppt in the lab. In the Chesapeake Bay mummichogs are found in freshwater more often than striped killifish and are rarely taken in full seawater. Mummichogs may stay in the low salinity areas to avoid competition with striped killifish and other higher salinity fishes. Salinity stress will reduce tolerance to temperature changes or stress. Mummichogs prefer vegetated areas to hide, may prefer mud as a substrate, and tend to face into the water current.

This fish has been labelled as "one of the most stationary of fishes" as it carries out no breeding migrations. In the summer fish over 60 mm long have home ranges of 36-38 m. In the winter fish migrate to the mouth of the tidal channel where they are living and migrate back up the same channel when the water temperatures reach 15 C in the spring. During the growing season young remain in the intertidal zone for 6-8 weeks. The mummichog is probably ubiquitous in these habitats at APG.

Diet and Foraging Behavior

Mummichogs are omnivorous and feed opportunistically. To ensure normal growth larvae need to feed at least part of the time at the marsh surface and may be food limited if high tide is not long enough for them to feed. Digestion is alkaline as opposed to acidic because mummichogs have no stomach. They swallow their prey intact so mouth gape limits their prey size. Their mouth is adapted for surface feeding but they also feed mid water and on the bottom. Grass shrimp swimming above the fish are easy prey. The species is attracted to GABA (gamma aminobutyric acid) but will not swallow it. Foods which have been found in their gut include crustaceans, insects, algae, mollusks, fish eggs (including their own), and more. They can not subsist on a diet of plant material or detritus alone.

An important ecological role of mummichogs is to move organic material within and out of salt marsh ecosystems. Visual predation increases when males gain their sex specific coloring and their predators include wading birds, aerial searching birds, piscivorous ducks and predatory fish. Some examples of these are herons, egrets, terns, gulls, striped bass, bluefish, eels, white perch, summer flounder, red drum, and crabs.

Life Cycle Characteristics

Incubation occurs in 7-8 days at 22-34°C and both sexes grow fastest during the first two growing seasons. Mid summer during the first growth season the fish may grow an average of 5% of their body weight per day.

In the laboratory embryos incubated in 20 ppt are longest and those incubated in freshwater are the shortest.

Mummichogs spawn in fresh or brackish water at depths only reached by high spring tides in temperatures of 16.5-25°C, but eggs can develop in water 12-27°C. Spawning is stimulated by temperature, tide, moonlight, and salinity; however, semilunar oogenesis persists without lunar or tidal clues. It occurs beginning in March-May and lasts through July-September. The eggs are laid in empty shells, dead leaves of cordgrass, on algal mats, or in shallow pits covered by the female. Eggs incubate in the air and hatch when covered by the next high tide. They will not develop in water with a DO less than 1 mg/l, but low DO is a necessary hatching stimulus.

Environmental stress reduces fecundity, and low temperatures prevent gonadal development. Lower food densities result in less egg production.

Population Dynamics

In autumn, winter, and spring the population is dominated by members of one growing season. In August all age classes are present but 60% have not overwintered yet and less than 8% have completed two growing seasons.

Ecological and Societal Significance

Mummichog have no commercial fishing importance but are sold as bait for sport fisheries. They are also primary predators in the Open Marsh Water Management Mosquito Control Program currently being tested in NJ, MD, and DE.

References

BIGELOW, H.B., and SCHROEDER, W.C. 1953. Fishes of the Gulf of Maine. U.S. Fish and Wildlife Service Fisheries Bulletin #174. First Revision

U.S. DEPARTMENT OF THE INTERIOR. 1985. Species Profiles: Life Histories and Environmental Requirements of Coastal Fishes and Invertebrates (Mid-Atlantic): Mummichog and Striped Killifish. Fish and Wildlife Service. Coastal Ecology Group, Waterways Experiment Station. Biological Report 81(11.40). June 1985

STRIPED BASS
(Morone saxatilis)

General Description

Striped bass range all along the Atlantic Coast from the St. Lawrence River, Canada, west to Montreal and south to the St. Johns River, Florida. They are also found in the Gulf of Mexico and have been introduced along the Pacific coast, Russia, France, Portugal, and as landlocked populations in North America. The species is very abundant in the Chesapeake Bay.

The female of this species is larger than the male and most fish which are over 13.6 kg. are females.

Habitat Use

Young of the year are found in estuarine waters mostly at depths of 10 feet or less. They are tolerant of a wide range of salinities of 0-20 ppt and need a DO concentration of at least 5.0 ppm and a pH of 7-9. Subadults and adults are even more tolerant of salinities ranging from 0-35 ppt and also have a DO requirement of at least 5.0 ppm. Adults are found in both estuarine and ocean waters.

The larvae tend to stay in or near the spawning area. Post yolk sac stages stay mid channel and the highest concentration of them are found near the bottom. During the first summer the young of the year begin to move downstream and shoreward. In the winter juveniles move to deeper water downstream but most do not reach coastal waters until at least their second year. Starting at age 2 or 3 the fish migrate north in the summer and south in the fall and winter although some of the population does not migrate. The Chesapeake Bay stock is composed of two types of fish: premigratory fish ages 5 and under and coastal migratory fish ages 2-20. Mature fish migrate up the rivers to spawn and the males arrive at the spawning grounds first. Shortly after spawning adults return to the ocean and in the late fall or early winter they migrate south to overwinter off North Carolina.

Striped bass are sensitive to temperature and water flow. A sharp rise in temperature such as thermal discharge can cause premature spawning in unsuitable habitat. Likewise a sudden drop in temperature can cause spawning to stop. Adequate water flow is necessary to keep the eggs and larvae from being smothered. The survival of the larval stage is crucial for the future population abundance of the mid-Atlantic stock.

The Atlas of Natural Resources indicates that striped bass probably spawn slightly north and east of APG in high velocity areas of the Bay during April through June. Larvae and juveniles remain in these high velocity areas or move slightly downstream. Striped bass nursery areas encompass the southern end of APG waters - the shore zone of Carroll Island and Gunpowder Neck in the vicinity of J-field and southward. Juveniles leave the nursery grounds at the age of 2 or 3. Adults and juveniles have major summer concentrations around the whole Edgewood Area. The Environmental Sensitivity Map (VIMS, no date) indicates that striped bass is a nursery species in the Bush River between Lauderick Creek and Otter Point Creek.

Diet and Foraging Behavior

Striped bass are generalists. The prey selected by the young varies with salinity and food item availability. Juveniles eat copepods, amphipods, mysids, insects, and shrimp. Larger juveniles may

eat small fish. All ages school by size when they feed and often follow schooled prey. Adults are piscivorous and opportunistic. In the summer and fall they eat bay anchovy and atlantic menhaden. In the winter they have been seen to eat spot and atlantic croaker. They also eat other species such as blueback herring, mummichog, striped mullet, rainbow smelt, weakfish, white perch, silver hake, American eel, American lobster, squid, clam, crab and mussel depending what is available. Feeding drops off in late spring and early summer corresponding to spawning.

Striped bass larvae are eaten by white perch and copepods. Small striped bass are eaten by bluefish and weakfish. Competition among striped bass adults, bluefish, and weakfish may occur for schooling forage species. Larvae and juveniles share nursery habitat with white perch so competition for food may occur.

Life Cycle Characteristics

Eggs hatch 29-80 hours after fertilization depending on the temperature. This relationship is described by the equation $I = -4.6T + 131.6$ (I=incubation time, T=temperature in °C). Young of the year grow 0.272-0.433 mm/day during June to November (in North Carolina) and every age growth rates are affected by temperature. In the Chesapeake Bay growth rates for October and November are 8.8% and 3.8% respectively, for May and June they are 4.6% and 9.1% respectively (% of what is not given). There is no growth between December and April or below 10°C and maximum growth is at 20°C. When females mature at age 4 they begin to grow faster than males.

Spawning in the Northern Chesapeake Bay and tributaries occurs from April to early June within the first 25 miles of tidal freshwater. The peak location is from Turkey Point to Worton Point (in the Bay to the east of the Proving Grounds). Adults spawn over a long time in a variety of environmental gradients so only a few eggs are deposited in suitable habitat. Therefore, the size of the spawning stock does not matter in terms of determining the next year's class size. Spawning is triggered by an increase in water temperature and usually begins when the water reaches 14°C. Peak spawning temperature is 15.5 to 21.1°C. For successful spawning and larval development salinity can not be above 3.0 ppt and DO must be at least 5 ppm. Optimum flow is 0.3-2.0 ft/sec (generally a higher flow means a more successful year class) and pH must be greater than 6.5. Both eggs and yolk sac larvae need turbulence to keep them off the bottom so that they will not be smothered. Larvae tend to stay close to where they were spawned and post yolk sac larvae stay mid channel and near the bottom.

Population Dynamics

The sex ratio in the summer and fall is 1:9 (M:F). In the Potomac River estuary the age structure in terms of % males is as follows:

AGE	% MALES
3	97
4	94
5	81
6	31
7	19

Ecological and Societal Significance

The striped bass is a very important commercial and recreational fish - especially in the Chesapeake Bay area. Since 1930 Maryland and Virginia have produced the highest commercial landings. The Chesapeake is the principal spawning area for the Atlantic Coast and 50-90% of the catch originates from spawning in the Bay. Around APG the annual average catch is 115,883 lbs/yr and 55% of that catch is from the Chesapeake Bay.

References

LIPPSON, A.J., ed. 1985. The Chesapeake Bay in Maryland - An Atlas of Natural Resources. Natural Resources Institute of the University of Maryland.

MARYLAND DEPARTMENT OF NATURAL RESOURCES AND VIRGINIA MARINE RESOURCES COMMISSION (MDNR). 1988. Chesapeake Bay Fisheries: Status, Trends, Priorities and Data Needs. Annapolis, MD. September 1988.

U.S. DEPARTMENT OF THE INTERIOR. 1983. Species Profiles: Life Histories and Environmental Requirements of Coastal Fishes and Invertebrates (Mid-Atlantic): Stripped Bass. Fish and Wildlife Service. Coastal Ecology Group, Waterways Experiment Station. FWS/OBS-82/11.8. October 1983

VIRGINIA INSTITUTE OF MARINE SCIENCE (VIMS). No Date. Environmental Sensitivity Map. College of William and Mary.

STRIPED KILLIFISH
(*Fundulus majalis*, Walbaum)

General Description

The striped killifish ranges from New Hampshire to northeastern Florida.
Adult size is generally 152-178 mm and the males are smaller than the females after the first year.

Habitat Use

Striped killifish occur mainly in saltwater and sometimes in brackish waters but never in freshwater. They occur more over sandy sediments and are the most important killifish of the unvegetated flats of North Carolina. They may be found in water a few centimeters deep and concentrate along shorelines during flood tides. Like the mummichog the striped killifish faces into a water current.

In August and September the striped killifish are slightly more abundant than the mummichog in a heterotypic school in a Maryland estuary. The striped killifish is probably ubiquitous in the more brackish areas of APG.

Diet and Foraging Behavior

Striped killifish eat everything but detritus. Crustaceans and polychaetes are found to be the most frequent food. Like the mummichog they have a superior mouth and feed readily on grass shrimp but also feed mid water and on the bottom. Striped killifish eat more benthic invertebrates and polychaetes than mummichog who eat more epiphytic algae and plant material.

Mummichog and striped killifish have the same predators including wading birds, aerial searching birds, piscivorous ducks and predatory fish. Examples of these predators include herons, egrets, terns, gulls, striped bass, bluefish, eels, white perch, summer flounder, red drum, and crabs.

Life Cycle Characteristics

Incubation time has been shown to decrease with increasing temperature.

In the Chesapeake Bay spawning occurs from April to September. The spawn in still, shallow water close to the shore and in small ponds. Females may actively bury the eggs. In the laboratory 7-29% mortality in embryos occurred when they were transferred from water at 22-26°C to water 6 degrees higher or lower.

Ecological and Societal Significance

Striped killifish are not important commercially but are used in biological experimental research.

References

U.S. DEPARTMENT OF THE INTERIOR. 1985. Species Profiles: Life Histories and Environmental Requirements of Coastal Fishes and Invertebrates (Mid-Atlantic): Mummichog and Striped Killifish. Fish and Wildlife Service. Coastal Ecology Group, Waterways Experiment Station. Biological Report 81(11.40). June 1985

WHITE PERCH
(Morone americana)

General Description

The white perch ranges from Nova Scotia to North Carolina and is most common from the Hudson River to the Chesapeake Bay. It is uncommon north of Cape Cod. Females are generally larger than males and adults are 9-10" long weighing 1.5 lbs.

Habitat Use

White perch tend to remain in or near their river of origin. The young of the year need salinities of 3 ppt or less in the summer but can handle higher salinities after that. They require at least 5 ppm DO and a pH between 7 and 9. Subadults and adults are found in tidal fresh and estuarine water. Salinities required are usually 18 ppt or less and DO must be at least 5 ppm.

White perch are considered to be semi-anadromous because their spawning migration to freshwater begins in the estuary instead of the ocean. Young of the year are concentrated in low salinity waters in the summer but between August and November they move downriver to deeper estuary waters. Adults overwinter in deep estuary waters and make extensive upstream migrations to spawn in the spring. In the summer adult movements are local and random and in the fall adults move downstream closer to the overwintering deeper waters.

White perch are likely to be found at APG most of the year though in winter they may move more towards the open bay to depth contours of 30-40 feet. Spawning probably occurs further upstream than APG - north of the mouth of Bird river and north of and including Otter Point Creek. APG waters do serve as a substantial nursery area.

Diet and Foraging Behavior

The white perch has ecological importance in cycling nutrients within estuarine food webs. The juvenile are prey for yearling and older striped bass, adult white perch, and the bluefish.

Life Cycle Characteristics

First year growth is correlated with light (probably because increased light means increased phytoplankton production) and the amount of time that the water temperature is between 10 and 15°C. The size of the fish is inversely related to population density and heavy rainfall adversely affects first year growth.

The maximum age reached by this species is 9 for males and 10 for females. Males reach sexual maturity by age group II. Females are all sexually mature by age group IV although larger fish in age groups II and III are mature.

White perch spawn from April to June in fresh, tidal fresh, and low salinity waters of large rivers. Physical parameters of spawning are salinities of 0-1.5 ppt and DO concentrations of 5 ppm. Spawning temperatures range 12.2-20°C and pH can range 6.5-8.5. Adults spawn once a year and give no parental care to the eggs or young.

Fecundity of females can be anywhere from 50,000 to 150,000 eggs/female. A regression equation which explains fecundity in relation to size is $Y=1697.08e^{0.02X}$ where Y is the # of eggs and X is standard length in mm.

Some annual mortality rates for Patuxent Estuary, Maryland perch are 0.56 for females and 0.50 for males with a combined value of 0.55. However, these values are more from fishing than natural mortality.

Population Dynamics

Males are the first to arrive at the spawning grounds and are the last to leave so the peak percent of the spawning population which is female is 35%. At the same time in the feeding area of the same estuary the population is dominated by females ranging from 44.2-87.1%.

The abundance of white perch has decreased since 1960 but the population is not depressed.

Ecological and Societal Significance

This species is an important commercial and sport fish - especially since the closure of the fisheries for American and hickory shad and striped bass. The average annual catch in APG waters from 1975-87 is 56,844 lbs - 40% of this catch is from the Gunpowder River in Baltimore County.

References

BATH, D.W. and O'CONNOR, J.M. 1982. "The Biology of the White Perch, *Morone americana*, in the Hudson River Estuary". Fishery Bulletin: Vol. 80, No. 3, 1982. 599-610

MANSUETI, R.J. 1961. "Movement, Reproduction, and Mortality of the White Perch, *Roccus americanus*, in the Patuxent Estuary, Maryland". Chesapeake Science. 2:142-205

MARYLAND DEPARTMENT OF NATURAL RESOURCES AND VIRGINIA MARINE RESOURCES COMMISSION (MDNR). 1988. Chesapeake Bay Fisheries: Status, Trends, Priorities and Data Needs. Annapolis, MD. September 1988.

POTTIE, J. 1990. Personal communication. Spoke with Tanya Yudelman-Bloch on June 14 and 18, 1990

MAMMALS

MUSKRAT
(*Ondatra zibethicus*)

General Description

An aquatic rodent, the muskrat has a stocky body, a broad head, and small ears and eyes. Its short legs are modified for aquatic life and its tail is scaly, naked, and laterally flattened. The sought-after fur of muskrats is soft and velvety and consists of thick waterproof underfur overlaid by long, glossy guard hair. The dorsal coat is rusty red to dark brown or almost black. The fur is lighter on sides of body and on the ventral surface. The sexes are colored similarly. Muskrats have characteristic rodent incisors, which are a single pair of chisel-like teeth separated from the cheek teeth by diastema. The dental formula is 1/1, 0/0, 0/0, 3/3 = 16. Published masses for adult muskrats range from 1030 g for males and 962 g for females in Maryland (Dozier et al. 1968 as cited in Boutin and Birkenholz 1987) to 1370 g for males and 1323 g for females in New York (Erickson 1963 as cited in Perry 1982).

The name, muskrat, stems from the animal's musk glands which produce an oil formed from a mixture of cyclopentadecanol, cycloheptadecanol and corresponding ketones. Musk glands are possessed by both males and females, but are more actively used by males who deposit musk secretions via urination as territorial markings.

Muskrat weights vary, with males usually being somewhat heavier. Maximum weights reported in Maryland, Delaware and North Carolina were 1,814 grams, 1,248 grams, and 1,956 grams respectively. Muskrats collected at Aberdeen in the winter and spring of 1979-80 weighed from 590 to 1,540 grams. The average weight of adult females trapped at Aberdeen was 1040 grams; the average weight of adult males was 1090 grams. Muskrats are generally 406 to 641 mm in total length, with the tail accounting for 177 to 295 mm. The muskrats at Aberdeen are probably from the subspecies *macrodon*, which is found along the Middle Atlantic Coast.

Habitat Use

Muskrats prefer vegetated lentic or slightly lotic water environments such as coastal marshes and marshy areas around streams and lakes. An adaptable animal, muskrats also inhabit other various aquatic habitats such as ponds, sloughs, ditches, canals and pits. Muskrats seem to prefer denning in banks if locations are available and suitable; muskrats will also build houses and other structures.

The presence of humus in muskrat habitats seems to be an important factor in determining muskrat abundance, probably because humus is a medium from which roots and burrows can be easily unearthed. Based on various studies along the Atlantic Coast, the presence of *Scirpus sp.* and *Typha spp.* seems to be important to marsh habitat suitability. Observations on stream habitat suitability include the following: slow-running streams with numerous aquatic plants yield higher populations, whereas deep or swift-running streams are typically unused; nearby corn fields may increase the attractiveness of a stream environment; stream habitats often vary throughout the year, and semipermanent retreats for muskrats are necessary. Some researchers found no correlation between pond muskrat populations and the type or kind of vegetation present, however, according to other studies, the suitability of a pond for muskrat habitat depends upon the availability of food plants.

Due to the distribution and quantity of marsh areas at Aberdeen, all eight study areas could most likely support suitable muskrat habitats. Muskrats have been documented as occurring in various slow-moving streams, cattail marshes and sedge marshes on site.

Diet and Foraging Behavior

Muskrats are chiefly herbivores and eat the shoots, roots, tubers, stems, and leaves of aquatic plants. Muskrats in the mid-Atlantic have also been recorded to eat carp, mussels, turtles, blue crabs, and dead birds. Bulrush and cattail (*Scirpus sp.* and *Typha sp.*) were found by some researchers to be the most important muskrat foods throughout the United States. It has been stated that 80 percent of the foods of muskrats in Maryland marshes consisted of *S. olneyi, S. americanus, T. latifolia, and T. angustifolia*. However, the adaptable muskrat will utilize whatever food items are most available.

Many of the plants that muskrats in the Mid-Atlantic region are known to eat have been documented at Aberdeen. Those recorded as abundantly occurring include the Narrow-leaved Cattail (*Typha angustifolia*) and Reed (*Phragmites communis*). Those commonly occurring include Cord Grass (*Spartina cynosuroides*), Spike Rush (*Eleocharis obtusa*), Three square (*Scirpus americanus*), and Great Bulrush (*Scirpus validus*). Those documented as infrequently occurring include the Common Cattail (*Typha latifolia*) and Doghair (*Eleocharis tenuis*). Olney Bulrush (*Scirpus olneyi*) is documented as uncommonly occurring.

Some data on muskrat food consumption is available. Muskrat stomachs have been found to contain over 63 grams of moist food, and the dry stomach contents of some muskrats in Delaware ranged from 0.1 to 17.7 grams (the average amount was 2.4 grams). One researcher determined that a muskrat in Louisiana consumes one third of its weight in food each day (Svihla as cited in Perry 1982).

Based on energy requirements of herbivores and the energy available in food, Nagy (1987) developed a food ingestion rate (FI) equation (in grams of dry matter per day) for herbivores of FI (g/d) = 0.577 ($Wt^{0.727}$) where Wt is body weight in grams. For the muskrat this gives an FI of 0.577 (1,350 $g^{0.727}$) = 110 g/d. Based on measured body weights and drinking water values from Calder (1981) and Skadhauge (1975), Calder and Braun (1983) determined an allometric equation for water ingestion (WI) for mammals: WI = 0.099 ($Wt^{0.90}$) where Wt is average body weight in kilograms. For the muskrat, water ingestion is 0.099 (1.35 $kg^{0.90}$) = 0.130 L/d.

The foraging habits of the muskrat depend on season and availability. The muskrat is generally nocturnal, but may be out in the daytime in summer and spring. A wary animal, the muskrat relies upon concealment as a primary defense. In the summer, muskrats usually consume their food at the water's edge in an area that affords protective cover. In the winter, especially during periods of below freezing temperatures, food is restricted to underground plant parts and plants that can be reached under the ice. In colder climates, muskrats commonly build feeding huts which are temporary structures that provide protection from the elements and predators and access to oxygen.

The home range of the muskrat is dependent on many factors including the nature of the aquatic habitat, social pressure, age, sex and environmental conditions. For example, muskrats inhabiting a marsh will likely have a circular home range, and muskrats inhabiting a stream edge will have an elongated home range. In general, muskrats forage close to home. One researcher reported that radio-tagged muskrats were within 15 meters of their primary dwelling for at least 50 percent of the time. Most foraging was within 5 to 10 meters of the home, and the muskrat observed rarely ranged to a distance greater than 150 meters. Movements of a greater distance can occur if muskrats

disperse to a new area; such movements are believed to be triggered by physiological cycles and population or food pressures. Proulx and Gilbert (1983) give a summer home range of 0.49 acres for muskrat in a marsh habitat.

Life Cycle Characteristics

Breeding behavior in muskrats is probably brought on by photoperiodism, with actual mating triggered by weather. Males and females are promiscuous or loosely monogamous, and laboratory findings have indicated that the reproductive rate of the muskrat depends on the sexual activity of the male. Muskrat litter size and other breeding parameters vary geographically. One researcher determined that above the 37° N latitude an average of 4 to 7 young are produced per litter, with three or fewer litters produced per year. South of the 37° N latitude, litters were found to be smaller (three to four young), but more litters were produced per year. Breeding was found to generally occur year-round in the southern areas, and from March through October in colder regions.

Young muskrats are weaned and independent four weeks after birth. By three and a half months, they resemble small-to-medium adults in appearance and size. Sexual maturity is typically seen at one year, and the average weight and length at one year are 1,100 grams and 550 mm respectively.

Muskrats generally have a short life span and a high reproductive potential. Although one muskrat in Missouri reportedly lived 4 years in the wild, lifespans are generally of a shorter duration. Adult survivorship appears to be most influenced by latitude, but other factors affecting population size are diseases, parasites, predators, accidents, climatic factors, food, fighting and exploitation. Data reported on the survivability of offspring are quite variable and range from about 40 to 87 percent mortality during the first year of life.

Population Dynamics

Muskrats are territorial and usually live alone or with a mate. They readily fight each other, especially when a demand for food exists. Over-population may lead to eat-outs, which result in the decimation of nearby vegetation and root systems, then muskrat starvation. Under such extreme population pressures, muskrats may be forced into new areas. However, intolerance to over-crowding, and the resulting fighting, decreased breeding and killing of young, may control population expansion more quickly than decreasing food resources. The effects of the above factors are reflected in the cyclic changes in muskrat populations. Mortality factors such as disease, parasites, predation and accidents appear to be less significant in controlling long-term population levels, since an increased loss from one factor is usually accompanied by a compensatory decreased loss from another factor.

The population densities that have been reported for muskrats are quite variable since many parameters can affect density including phase of the population cycle, habitat type and condition, social pressures, competition, harvest, predation and geographical area. Beshears (1951 in Perry 1982) reported a population density of 1.13/acre in Alabama. Butler (1940 in Perry 1982) found 3 raccoons/acre in a sedge habitat and 26 raccoons/acre in a common reed habitat in Manitoba, Canada. Muskrats are known to occur abundantly at Aberdeen, and reportedly may exist in much higher concentrations than in neighboring and nearby areas. Although, trapping for these animals has been carried on at Aberdeen for many years, there has not been a noticeable adverse effect upon the population. Based on current management practices, the abundance of muskrats at Aberdeen in not expected to vary in the future.

Ecological and Societal Significance

The muskrat has been of economic importance in the fur trade since colonial times, and in 1975/76, over 8 million muskrats were harvested, yielding a value exceeding 29 million dollars. Quantities such as these have made the muskrat the most valuable fur animal in North America. Muskrats have also historically been used for food or musk production, but the value of these products is insignificant compared to fur production.

Muskrats can have negative impacts such as damage to structures and ponds due to burrowing and loss of crops. Eat-outs may damage marshes and cause build-ups of stagnant water (although eat-outs may on occasion be beneficial to certain shorebirds, wading birds and waterfowl).

References

ABERDEEN PROVING GROUND, MARYLAND. Natural Resources Management Plan, Part IV: Fish and Wildlife Management. 1987 and 1981

BOUTIN, S. and BIRKENHOLZ, D.E. 1987. Muskrat and round-tailed muskrat. In: Wild Furbearer Management and Conservation, M. Novak, J.A. Baker, M.E. Obbarel, and B. Malloch (eds.).

CALDER, W.A. III. 1981. Scaling of physiological processes in homeothermic animals. Ann. Rev. Physiol. 43:301-322

CALDER, W.A. and BRAUN, E.J. 1983. Scaling of osmotic regulation in mammals and birds. Am. J. Physiol. 244:R601-R606

NAGY, K.A. 1987. Field metabolic rate and food requirement scaling in mammals and birds. Ecol. Mono. 57:111-128

PERRY, H.R., Jr. 1982. Muskrats. In: Wild Mammals of North America. J.A. Chapman and G.A. Feldhamer, eds. The Johns Hopkins University Press, Baltimore

PROULX, G., and GILBERT, F.F. 1983. The ecology of the muskrat, Ondatra zibethicus, at Luther Marsh, Ontario. Can. Field-Nat. 97:377-390

SKADHAUGE, R. 1975. Renal and cloacal transport of salt and water. Symp. Zool. Soc. London 35:97-106

RACCOON
(*Procyon lotor*)

General Description

The raccoon is stocky carnivore, with a broad head, pointed snout, and bushy tail. The animal is generally colored gray to black dorsally, with lighter fur occurring ventrally. Most characteristic is the raccoon's mask of black hair and its multi-ringed tail. The raccoon possesses sensitive front fingers, which are opposable to some degree. Its dental formula is 3/3, 1/1, 4/4, 2/2 = 40

Raccoon body lengths range from 600 to 1050 mm, with the tail accounting for 200 to 400 mm. Adult body weights are generally between 3.6 kg to 9 kg, and males are usually 10 to 15 percent heavier than females. The heaviest weights on record were 25.4 and 28.3 kg and were from large males taken in Autumn. It has been found that animals in the north are generally heavier than those in south. Adult raccoons randomly trapped at Aberdeen in the winter of 1979/80 had weights ranging from 2.6 to 9.1 kg. The average weight for adult males was 6.7 kg, and for adult females was 5.3 kg.

Habitat Use

The raccoon is very adaptable and is distributed across Canada and throughout most of the United States. Habitat suitability is largely determined by the availability of water. Raccoons are abundant in hardwood swamps, mangroves, flood plain forests, and fresh and salt marshes. They are common in mesic hardwood stands, in farmlands (cultivated and abandoned) and suburban areas. In dry upland woodlands and in southern pine forests, raccoons tend to be scarce.

Raccoons take shelter in a variety of structures. In colder climates (central U.S. to Canada), raccoons undergo a period of winter dormancy that is not true hibernation. For this period, raccoons may take shelter in a variety of locations including tree dens, ground burrows, caves, drains, and buildings. Daytime sleep in the summer is spent in the above shelters as well as in a variety of less protected places such as tree limbs, mashed-down squirrel nests and in vegetation on the ground. Dens of any kind are located near water.

Since raccoons are able to adapt to a variety of habitats, they can be expected to occur in all study areas of the Aberdeen Site. Raccoons are known to utilize the slow-moving streams, sloughs, cattail and sedge marshes and den trees on site.

Diet and Foraging Behavior

Raccoons are omnivorous and opportunistic; they utilize hundreds of different types of plant and animal foods, proportions of which vary with season and location. In most habitats, plant foods are generally more important than animal foods in the raccoon diet. Plant foods include fleshy fruits, cultivated fruits, nuts, corn and other grains, seeds, buds and grasses. Animal foods eaten most often consist of invertebrates such as crayfish, insects, shellfish and other marine foods, snails and worms. Vertebrate animal foods included rodents (most important), rabbits, larger animals (usually as carrion), birds, bird eggs and sometimes fish. Garbage is commonly eaten in areas inhabited by humans.

Foraging and other activities typically occur from sunset to sunrise. Home and foraging ranges of the raccoon are variable. Factors affecting range include sex and age of raccoon, differences in population density, habitat quality, and the season and nature of the observational study. In general, raccoons restrict their short-term movements to relatively small areas within a larger range. Areas away from water are less frequented. Long movements are made in order to utilize temporary food sources such as orchards or fields. Reported maximum home range diameters usually fall between 1 and 3 km. Stuewer (1943) shows an average home range of 287 acres in a riverine habitat in Michigan. Some juvenile males and females will disperse from their birthplace in the fall or winter; dispersions be delayed until spring or summer of the next year in colder climates. Dispersions range from a few kilometers to record movements of 264 kilometers.

Based on energy requirements of mammals and the energy available in food, Nagy (1987) developed a food ingestion rate (FI) equation (in grams of dry matter per day). For mammals the equation is FI (g/d) = 0.235 ($Wt^{0.822}$) where Wt is body weight in grams. For the raccoon FI is 0.235 (9,100 $g^{0.822}$) = 422 g/d. Based on measured body weights and drinking water values from Calder (1981) and Skadhauge (1975), Calder and Braun (1983) determined an allometric equation for water ingestion (WI) for mammals: WI = 0.099 ($Wt^{0.90}$) where Wt is average body weight in kilograms. For the raccoon, water ingestion is 0.099 (9.1 $kg^{0.90}$) = 0.722 L/d.

Life Cycle Characteristics

It has been demonstrated that reproductive activity in the raccoon is largely initiated by an increasing photoperiod. Throughout most of their range in North America, raccoons mate from January to March, with a peak in February, and most litters arrive in May. If after the first estrus in the spring, a female raccoon does not become pregnant, a second cycle two to four months later may occur and result in a late litter. Females typically breed their first year (60 percent of wild and captive females have been found to produce litters when they are one year old), but wild males usually have no opportunity to breed until their second year since they come into breeding condition after most females are pregnant. Males typically mate with several females each spring, and may temporarily expand their ranges to visit several females during the mating period.

One litter a year is typical for raccoons. One to eight young in a litter have been reported, with mean litter sizes ranging from two to five.

Full growth in the raccoon is obtained by two years, however in some areas few raccoons survive for this duration. The average longevity for raccoons in both Missouri and Manitoba was calculated to be 1.8 years, while in Alabama, where the climate is milder and hunting pressure less, the average longevity was found to be 3.1 years. The main causes of mortality in raccoons is starvation and malnutrition during the winter and early spring, and the activities of man (hunting, trapping, automobiles). Deaths due to predators, parasites, and diseases are less significant. Juveniles are especially prone to the effects of malnutrition as they possess less expendable body tissue.

Population Dynamics

Population densities vary widely with habitat. Sample densities include: 0.5 to 3.2 racoons per square kilometer on the prairies of North Dakota and Manitoba; up to 20 raccoons per km^2 in marshes and bottomlands in the midwestern and eastern U.S.; 49 per km^2 in a beaver swamp in Alabama; and 68.7 per km^2 in a suburb in Ohio. Slate (1980 in Sanderson 1987) found a density of 0.05 raccoons/acre

in New Jersey. At Aberdeen, raccoons are abundant and may occur in higher concentrations than in neighboring and nearby areas. Hunting and trapping for these animals, which have been carried on for many years, do not appear to have adverse effects upon the raccoon population. Under the current management plan, the abundance of raccoons is not expected to vary greatly.

With a few exceptions, adult raccoons are generally solitary. The most common social interaction is between a mother and her young of the year. Raccoons demonstrate no or little evidence of territoriality, and the ranges of both males and females usually overlap.

Raccoon predators include pumas (*Felis concolor*), bobcats (*Felis rufus*), wolves (*Canis lupus*), coyotes (*Canis latrans*), foxes (*Vulpes vulpes* and *Urocyon cinereoargenteus*), fishers (*Martes pennanti*), great-horned owls (*Bubo virginianus*), and alligators. However, historically these predators have had little influence on raccoon populations, and few are expected to occur at Aberdeen.

Ecological and Societal Significance

The raccoon is an important fur species although its pelt value has varied over time. In 1982, the value was $20 to $30. Most U.S. pelts are exported to Canada and Europe. The raccoon is also valued as a popular game species, and is primarily hunted with dogs at night.

Raccoons may cause damage to agriculture and to managed wildlife populations such as sea turtles, wood ducks and other marsh waterfowl. Generally, however, this damage is inconsequential, temporary or localized.

References

ABERDEEN PROVING GROUND, MARYLAND. Natural Resources Management Plan, Part IV: Fish and Wildlife Management. 1987 and 1981.

CALDER, W.A. III. 1981. Scaling of physiological processes in homeothermic animals. Ann. Rev. Physiol. 43:301-322

CALDER, W.A., AND BRAUN, E.J. 1983. Scaling of osmotic regulation in mammals and birds. Am. J. Physiol. 244:R601-R606.

KAUFMANN, J.H. 1982. Raccoon and Allies. In: Wild Mammals of North America. J.A. Chapman and G.A. Feldhamer, eds. The Johns Hopkins University Press, Baltimore.

NAGY, K.A. 1987. Field metabolic rate and food requirement scaling in mammals and birds. Ecol. Mono. 57:111-128

SANDERSON, G.C. 1987. Raccoon. In: Novak, M., Baker, J.A., Obbard, M.E., and Malloch, B. (eds.). Wildlife Furbearer Management and Conservation.

SKADHAUGE, R. 1975. Renal and cloacal transport of salt and water. Symp. Zool. Soc. London 35:97-106

STUEWER, F.W. 1943. Reproduction of raccoons in Michgan. J. Wildl. Management. 7:60-73

WHITE-TAILED DEER
(*Odocoileus virginianus*)

General Description

The white-tail deer has long legs, pointed hooves, and antlers if male. Its characteristic long, bushy tail is white on the underside and is carried up when the deer is alarmed. The color of the white-tail varies with season; in summer the coat is usually reddish brown, in winter it is blue-grey. Fawns have a reddish brown coat with white dorsal spots. All underparts of the body are white, and adults have a white band across the nose and a white eye ring.

The bodyweight of the white-tailed deer varies greatly, though bucks generally weigh more than does. Some subspecies (O. v. *borealis*) can attain a live weight of 192 kg (160 kg dressed weight), whereas another subspecies has an adult weight of as little as 25 kg (O. v. *clavium*, or Key Deer). At Aberdeen, one and one half-year old male white-tailed deer had an average dressed weight of 80 pounds (36 kg) in 1987. This is low regionally, as deer of similar age and condition in Kent County (across the Chesapeake Bay) had an average dressed weight of 130 pounds (59 kg). At Aberdeen the subspecies would be O. v. *virginianus*.

Habitat Use

White-tailed deer are extremely adaptable; this is illustrated by their range which extends across North America. The species inhabits tropical, desert, and subartic areas, and since they are very tolerant of people, white-tailed deer may live very close to developed areas. The ideal habitat for white-tailed deer is a subclimax, or 'temporary', community consisting of a variety of habitat types. Generally, this is an area that contains cut and overgrown fields intermingled with uneven-aged forest woodlots. Clear-cutting and other cutting practices can also contribute to habitat suitability by increasing food productivity.

White-tailed deer utilize all of the study areas at Aberdeen. They have been documented in the sloughs, saw hardwood forests, pole hardwood forests, sapling hardwood forests, and shrub/forest edges on site. Extirpirated by the 1850's, deer were reintroduced into the area in 1931, when five or six deer were released onto Aberdeen Proving Ground. The deer multiplied to such an extent that they became pests by World War II. In 1948, a trapping program was initiated by the Maryland Wildlife Administration and 2,000 deer were removed to other parts of the state over the following 15 years. Also, hunting has been permitted at Aberdeen since 1951, and this, coupled with the over-consumption of food and the maturation of the forests has caused a decline in deer numbers. However, large numbers of deer and overcrowded conditions remain.

Diet and Foraging Behavior

The diet of the ruminant white-tailed deer is very diverse and reflects the adaptability of the animal. Studies have shown that the deer has the ability to discern and select more nutritious foods when the opportunity to be selective exists. Foods that the white-tailed deer eats is dependent on what is seasonally and regionally available; for example, in agricultural areas, deer may prefer cultivated crops over traditional wild food sources.

White-tailed deer utilize tree, shrub, grassy and herbaceous species. In the spring and summer, dominant food items are green succulent leaves and stems of woody and herbaceous species. Yellow poplar flowers (*Liriodendron tulopifera*), mushrooms and acorns may also be significant. In fall, acorns, mushrooms, and wild fruits are generally the dominant food items. Acorns, grasses, are Japanese honeysuckle (*Lonicera japonica*) are commonly eaten in the winter. Browsing of hardwood twigs in winter is common in the colder climates, though minor in the Southeast. As previously stated, deer are tolerant of people, and will take advantage of cultivated crops. Examples of crops utilized are apples, soybeans, grapes in vineyards, cauliflower, beans and forest species.

Many of the species generally found to be important to the white-tailed deer diet are common at Aberdeen. These include species such as maples (*Acer* spp.), dogwood (*Cornus florida*), yellow poplar (*Liriodendron tulipifera*), oaks (*Quercus* spp.), sumac (*Rhus* spp.), *Vaccinium* spp., *Viburnum* spp. and grapes (*Vitis* spp.).

Some data on the daily energy requirements of white-tailed deer exists. In one study it was found that a 23-27 kg deer requires 0.9 kg (3,600 calories) of air-dry feed daily; a 46 kg deer requires 1.5-1.7 kg (6,300 calories); and a 69 kg deer requires 2.5 kg (9,300 calories). 2.5 kg air-dry feed is equivalent to about 3.0 to 3.4 kg of deer browse with usual moisture content.

Based on energy requirements of herbivores and the energy available in food, Nagy (1987) developed a food ingestion rate (FI) equation (in grams of dry matter per day) for herbivores of FI (g/d) = 0.577 ($Wt^{0.727}$) where Wt is body weight in grams. For the white-tailed deer at Aberdeen this gives a FI is 0.577 (36,000 $g^{0.727}$) = 1,190 g/d. Based on measured body weights and drinking water values from Calder (1981) and Skadhauge (1975), Calder and Braun (1983) determined an allometric equation for water ingestion (WI) for mammals: WI = 0.099 ($Wt^{0.90}$) where Wt is average body weight in kilograms. For the white-tailed deer, water ingestion is 0.099 (36 $kg^{0.90}$) = 2.5 L/d.

Activity and foraging patterns depend on a range of factors including habitat parameters, season of the year, proximity to humans, weather, and population pressures. In northern regions, deer exhibit migratory movements and will "yard up", or concentrate, in a protected area as a method of saving energy.

Life Cycle Characteristics

White-tailed deer are generally Autumn breeders; the specific dates in which breeding occurs appear to depend on latitude. In northern states and Canadian provinces, breeding occurs in early November. Estrus lasts about 24 hours, and will recur in approximately 28 days if the doe is not bred. Gestation is 201 days.

Nutritional condition is important to successful reproduction; females may breed during their first year if inhabiting a good range. On a poor or overstocked range, fawn females do not breed. Moreover, healthy, mature females almost always bear twins (10 to 15 percent have triplets), but mature females on very poor ranges may not even ovulate. Does at Aberdeen had a low reproductive rate of 0.6 in 1981, which reflects the overcrowded conditions.

Males are sexually mature and may possibly breed their first year if range conditions are excellent and adult male competition very low. Bucks become very aggressive as the rutting season approaches. They create and defend breeding season territories which are marked by scrapes and probably urination. Males breed polygamously and track and locate females by smell.

Males achieve maximum growth at four to five years of age, females a year earlier. Survivability of offspring and lifespan vary throughout the range. Mortality factors include legal and illegal harvest, traumas (mostly car collisions), predation, nutritional deficiencies, toxicity, parasites and disease. Deer at Aberdeen have been recorded to have a heavier parasite load than deer in other parts of Maryland, however the effect of this on mortality rates is unknown.

Population Dynamics

Overcrowding of deer at Aberdeen, reflected by the low average weight of the deer, has occurred for many years. However, based on observations of increased body fat content, the general condition of the deer seems to have improved. This may be attributed to the increased emphasis on harvesting female deer. Future trends in populations will be determined by management and to a lesser extent, degree of harvest. Temporary reductions in the form of die-offs may occur from time to time due to a combination of malnutrition, severe winters, diseases and parasites. Temporary population increases could occur following extensive cutting of timber (which stimulates the growth of young trees, shrubs and forbs).

White-tailed deer are gregarious outside the breeding season, and families generally group together. During the breeding season, males become very aggressive and compete for mating opportunities. Females drive away their fawns and become coy and solitary.

Ecological and Societal Significance

Throughout their range, white-tailed deer have caused great damage to cars (through collisions), to crops and nurseries, and to forest production. However, billions of dollars are made annually from hunting and related industries (venison sales, leather making, butchers, hotels, deer-hunting right sales).

Deer have had a tremendous impact at Aberdeen. Deer are so overly abundant that they seriously hinder growth and development of natural vegetation, including forests. No reproduction of pine trees occurs, and herbaceous and woody shrubs have been reduced or eliminated due to browsing. Deer may have an effect on other wildlife populations since they eat foods (i.e. acorns) that may be important to other species.

Aberdeen has an extensive deer hunting season. Most hunting is done by base personnel. Deer kill numbers were 1000 to 1200 per year for years 1980 to 1986.

References

ABERDEEN PROVING GROUND, MARYLAND. Natural Resources Management Plan, Part IV: Fish and Wildlife Management. 1987 and 1981

CALDER, W.A. III. 1981. Scaling of physiological processes in homeothermic animals. Ann. Rev. Physiol. 43:301-322

CALDER, W.A. and BRAUN, E.J. 1983. Scaling of osmotic regulation in mammals and birds. Am. J. Physiol. 244:R601-R606

HESSELTON, W.T and HESSELTON, R.M. 1982. White-tailed Deer. In: Wild Mammals of North America. J.A. Chapman and G.A. Feldhamer, eds. The Johns Hopkins University Press, Baltimore

NAGY, K.A. 1987. Field metabolic rate and food requirement scaling in mammals and birds. Ecol. Mono. 57:111-128

SKADHAUGE, R. 1975. Renal and cloacal transport of salt and water. Symp. Zool. Soc. London 35:97-106

APPENDIX E

LISTS OF SPECIES OCCURRING AT ABERDEEN PROVING GROUND

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TABLE E-1

BIRDS OBSERVED DURING BREEDING SEASON AT ABERDEEN
(JUNE 11 - JULY 18, 1980)[a]

Common Name	Scientific Name	Local Abundance
Acadian Flycatcher	*Empidonax virescens*	Common
American Kestrel	*Falco sparverius*	Common
American Goldfinch	*Cardeulis tristis*	Common
American Redstart	*Setophaga ruticilla*	Common
American Robin	*Turdus migratorius*	Common
American Woodcock	*Philohela minor*	Fairly common
Bald Eagle	*Haliaeetus leucocephalus*	Rare
Barn Swallow	*Hirundo rustica*	Common
Barred Owl	*Strix varia*	Uncommon
Belted Kingfisher	*Megaceryle alcyon*	Common
Blue-gray Gnatcatcher	*Polioptila caerulea*	Common
Blue Grosbeak	*Guiraca caerulea*	Fairly common
Blue Jay	*Cyanocitta cristata*	Common
Bobwhite	*Colinus virginianus*	Common
Brown-headed Cowbird	*Molothrus ater*	Common
Brown Trasher	*Toxostoma rufum*	Common
Cardinal	*Richmondena cardinalis*	Common
Carolina Chickadee	*Parus carolinensis*	Fairly common
Carolina Wren	*Thryothorus ludovicianus*	Common
Catbird	*Dumetella carolinensis*	Common
Chimney Swift	*Chaetura pelagica*	Common
Chipping Sparrow	*Spizella passerina*	Common
Common Crow	*Corvus brachyrhynchos*	Common
Common Flicker	*Colaptes auratus*	Common
Common Grackle	*Quiscalus quiscula*	Common
Downy Woodpecker	*Dendrocopos pubescens*	Common
Eastern Bluebird	*Sialia sialis*	Fairly common
Eastern Kingbird	*Tyrannus tyrannus*	Common
Eastern Meadowlark	*Sturnella magna*	Common
Eastern Phoebe	*Sayornis phoebe*	Common
Eastern Wood Pewee	*Contopus virens*	Common
Field Sparrow	*Spizella pusilla*	Common
Fish Crow	*Corvus ossifragus*	Common
Grasshopper Sparrow	*Ammodramus savannarum*	Common
Great Crested Flycatcher	*Myiarchus crinitus*	Common
Great Blue Heron	*Ardea herodias*	Common
Great Horned Owl	*Bubo virginianus*	Uncommon
Green Heron	*Butorides striatus*	Common
Hairy Woodpecker	*Dendrocopos villosus*	Fairly common
House Sparrow	*Passer domesticus*	Common
House Wren	*Troglodytes aedon*	Common
Indigo Bunting	*Passerina cyanea*	Common
Kentucky Warbler	*Oporornis formosus*	Uncommon
Laughing Gull	*Larus articilla*	Rare
Long-billed Marsh Wren	*Telmatodytes palustris*	Common

TABLE E-1 (Continued)

BIRDS OBSERVED DURING BREEDING SEASON AT ABERDEEN
(JUNE 11 - JULY 18, 1980)[a]

me	Scientific Name	Local Abundance
	Mimus polyglottos	Common
ove	*Zenaidura macroura*	Common
ole	*Icterus glabula*	Uncommon
ole	*Icterus spurius*	Common
	Pandion haliaetus	Uncommon
	Seiurus aurocapillus	Uncommon
ler	*Parual americana*	Uncommon
alcon	*Falco peregrinus*	Rare
Woodpecker	*Centurus carolinus*	Common
reo	*Vireo olivaceus*	Common
ered Hawk	*Buteo lineatus*	Uncommon
lawk	*Buteo jamaicensis*	Common
Blackbird	*Agelaius phoeniceus*	Common
d Pheasant	*Phasianus colchicus*	Common
d Towhee	*Pipilo erythrophthalmus*	Common
ager	*Piranga olivacea*	Common
ow	*Melospiza melodia*	Common
	Porzana carolina	Uncommon (seldom seen)
	Sturnus vulgaris	Common
nager	*Piranga rubra*	Uncommon
atcher	*Empidonax traillii*	Uncommon
w	*Iridoprocne bicolor*	Common
ouse	*Parus bicolor*	Common
	Meleagris gallopavo	Fairly common
ure	*Cathartes aura*	Common
ireo	*Vireo gilvus*	Uncommon
sted Nuthatch	*Sitta carolinensis*	Common
Vireo	*Vireo griseus*	Common
<	*Aix sponsa*	Uncommon
sh	*Hylocichla mustelina*	Common
asted Chat	*Icteria virens*	Fairly common
d Cuckoo	*Coccyzus americanus*	Common
ped Warbler	*Dendroica coronata*	Uncommon
at	*Geothlypis trichas*	Common
ated Vireo	*Vireo flavifrons*	Uncommon
rbler	*Dendroica petechia*	Common

Aberdeen Proving Ground. 1987. Natural Resources Management Plan, Part IV Fish and Management. Aberdeen Proving Ground Environmental Management Office. Aberdeen Proving , Maryland.

TABLE E-2

WINTER BIRDS OBSERVED AT ABERDEEN
(DECEMBER 1979 - MARCH 1980)[a]

Common Name	Scientific Name	Local Abundance
American Coot	*Fulica americana*	Uncommon
American Goldfinch	*Cardeulis tristis*	Common
American Kestrel	*Falco sparverius*	Common
American Robin	*Turdus migratorius*	Common
Bald Eagle	*Haliaeetus leucocephalus*	Rare
Barn Owl	*Tyto alba*	Uncommon
Barred Owl	*Strix varia*	Uncommon
Belted Kingfisher	*Megaceryle alcyon*	Common
Black Duck	*Anas rubripes*	Common
Black Vulture	*Coragyps atratus*	Uncommon
Blue Jay	*Cyanocitta cristata*	Common
Blue-winged Teal	*Anas discors*	Uncommon
Bobwhite	*Colinus virginianus*	Common
Brown Creeper	*Certhia familiaris*	Common
Brown-headed Cowbird	*Molothrus ater*	Common
Canada Goose	*Branta canadensis*	Common
Canvasback	*Aythya valisineria*	Uncommon
Cardinal	*Richmondena cardinalis*	Common
Carolina Chickadee	*Parus carolinensis*	Fairly common
Carolina Wren	*Thryothorus ludovicianus*	Common
Cedar Waxwing	*Bombycilla cedrorum*	Common at times
Chipping Sparrow	*Spizella passerina*	Common
Common Crow	*Corvus brachyrhynchos*	Common
Common Flicker	*Colaptes auratus*	Common
Common Gallinule	*Gallinula chloropus*	Uncommon
Common Goldeneye	*Bucephala clangula*	Uncommon
Common Grackle	*Quiscalus quiscula*	Common
Common Loon	*Gavia immer*	Uncommon
Common Merganser	*Mergus merganser*	Uncommon
Dark-eyed Junco	*Junco hyemalis*	Common
Downy Woodpecker	*Dendrocopos pubescens*	Common
Eastern Bluebird	*Sialia sialis*	Fairly common
Eastern Meadowlark	*Sturnella magna*	Common
Eastern Phoebe	*Sayornis phoebe*	Common
Field Sparrow	*Spizella pusilla*	Common
Fish Crow	*Corvus ossifragus*	Common
Fox Sparrow	*Passerella iliaca*	Common
Golden Eagle	*Aquila chrysaetos*	Rare
Golden-crowned Kinglet	*Regulus satrapa*	Uncommon
Great Black-backed Gull	*Larus marinus*	Common
Great Blue Heron	*Ardea herodias*	Common
Greater Scaup	*Aythya marila*	Uncommon

TABLE E-2 (Continued)

WINTER BIRDS OBSERVED AT ABERDEEN
(DECEMBER 1979 - MARCH 1980)[a]

Common Name	Scientific Name	Local Abundance
Hairy Woodpecker	*Dendrocopos villosus*	Fairly common
Herring Gull	*Larus argentatus*	Common
Horned Grebe	*Podiceps auritus*	Uncommon
House Finch	*Carpodacus mexicanus*	Uncommon
House Sparrow	*Passer domesticus*	Common
Killdeer	*Charadrius vociferus*	Common
Loggerhead Shrike	*Lanius ludovicianus*	Uncommon
Mallard	*Anas platyrhynchos*	Common
Marsh Hawk	*Circus cyaneus*	Common
Mockingbird	*Mimus polyglottos*	Common
Mourning Dove	*Zenaidura macroura*	Common
Oldsquaw	*Clangula hyemalis*	Uncommon
Osprey	*Pandion haliaetus*	Uncommon
Red-bellied Woodpecker	*Centurus carolinus*	Common
Red-shouldered Hawk	*Buteo lineatus*	Uncommon
Red-tailed Hawk	*Buteo jamaicensis*	Common
Red-winged Blackbird	*Agelaius phoeniceus*	Common
Ring-billed Gull	*Larus delawarensis*	Common
Ring-necked Pheasant	*Phasianus colchicus*	Common
Rock Dove	*Columba livia*	Common
Rufous-sided Towhee	*Pipilo erythrophthalmus*	Common
Rusty Blackbird	*Euphagus carolinus*	Uncommon
Savannah Sparrow	*Passerculus sandwichensis*	Uncommon
Song Sparrow	*Melospiza melodia*	Common
Starling	*Sturnus vulgaris*	Common
Tree Sparrow	*Spizella arborea*	Common
Tufted Titmouse	*Parus bicolor*	Common
Turkey	*Meleagris gallopavo*	Fairly common
Turkey Vulture	*Cathartes aura*	Common
Whistling Swan	*Olor columbianus*	Common
White-breasted Nuthatch	*Sitta carolinensis*	Common
White-throated Sparrow	*Zonotrichia albicollis*	Common
Wood Duck	*Aix sponsa*	Uncommon
Yellow-bellied Sapsucker	*Sphyrapicus varius*	Uncommon
Yellow-rumped Warbler	*Dendroica coronata*	Uncommon
Yellow Warbler	*Dendroica petechia*	Common

[a] Source: Aberdeen Proving Ground. 1987. Natural Resources Management Plan, Part IV Fish and Wildlife Management. Aberdeen Proving Ground Environmental Management Office. Aberdeen Proving Ground, Maryland.

TABLE

ANNOTATED LIST OF MAMMALS[a]

Common Name	Scientific Name	Remarks
* Beaver	*Castor canadensis*	Introduced in 1961. They have greatly increased flooding in some sections, creating habitat for waterfowl, bald eagles, herons and other birds, turtles and frogs. Beaver have become troublesome in some locations by blocking drainage ways and flooding roads.
* Big Brown Bat	*Eptesicus fuscus*	One specimen collected.
Bobcat	*Lynx rufus*	Rare; if it does exist at present. None have been reported on the Proving Ground, but rare individuals have been recorded in the eastern portion of Maryland in recent years.
Bog Lemming	*Synaptomys cooperi*	
* Eastern Chipmunk	*Tamias striatus*	Common. Frequently captured in Sherman traps.
* Eastern Cottontail	*Sylvilagus floridanus*	Abundant. Frequently seen along roads over the entire installation.
Eastern Mole	*Scalapus aquaticus*	Mole tunnels are seen in wooded portions of the Proving Ground but none have been captured. The tunnels could have been made either by the Eastern Mole, the Star-nosed Mole or both.
* Eastern Pipistrelle	*Pipistrellus subflavus*	Probably abundant. Three collected.
Eastern Harvest Mouse	*Reithrodontomys humulis*	
Evening Bat	*Nycticeius humeralis*	
* Fox Squirrel	*Sciurus niger vulpinces*	A small colony occurs on the artillery impact area at Abbey Point. (Another colony is said to occur near Michaelsville but this has not been corroborated.) Several animals have been captured and released from artificial nest boxes along Abbey Point Road.

Common Name	Scientific Name	Remarks
* Gray Fox	*Urocyon cinereoargenteus*	Several specimens examined. One was killed in January, 1980, when it entered a building. The animal showed no fear of people and allowed itself to be petted. It may have been infected with canine distemper virus. Less abundant than Red Fox; only two reported taken in 1980 by trappers.
* Gray Squirrel	*Sciurus carolinensis*	Abundant in all mature timber. Also seen in wooded portions of residential and Headquarters areas.
* Hoary Bat	*Lasiurus cinereus*	Two specimens collected.
* House Mouse	*Mus musculus*	Occurs in and around buildings but also captured in traps set in wooded and grassy areas.
Keen's Bat	*Myotis keenii*	
Least Shrew	*Crytotis parva*	
* Little Brown Bat	*Myotis lucifugus*	Abundant. Several collected.
Long-tailed Weasel	*Mustela frenata*	
* Masked Shrew	*Sorex cinereus*	Probably abundant. Three specimens taken in mouse traps in January, 1980.
Meadow Jumping Mouse	*Zapus hudsonius*	
* Meadow Vole	*Microtus pennsylvanicus*	Only two specimens captured. Probably abundant in some grasslands. Their true abundance probably much more than that represented by trapping.
Mink	*Mustela vison*	
* Muskrat	*Ondatra zibethicus*	Abundant and supports annual harvest by commercial trappers. According to records supplied by the Rod and Gun Club, the harvest was 200 in 1978, 354 in 1979, and 4,345 in 1980.

TABLE E-3 (Continued)

ANNOTATED LIST OF MAMMALS[a]

Common Name	Scientific Name	Remarks
* Norway Rat	*Rattus norvegicus*	Found mostly around buildings.
* Opossum	*Didelphis virginiana*	Abundant. The Rod and Gun Club reported that 86 were trapped in 1980.
Pigmy Shrew	*Mirosorex hoyi*	
* Pine Vole	*Microtus pinetorum*	Only one specimen trapped. Probably common in woodlands.
* Raccoon	*Procyon lotor*	
Red Bat	*Lasiurus borealis*	
* Red Fox	*Vulpes fulva*	Seen all over the installation. One specimen found in weakened condition in 1980. It was almost devoid of fur due to infection with mange mite (*Sarcoptes*). In 1980, 22 were reported taken by trappers.
Red Squirrel	*Tamiascurrus hudsonicus*	
Rice Rat	*Oryzomys palustris*	
* River Otter	*Lutra canadensis*	Three captured in traps set for beaver in February-March, 1980.
* Short-tailed Shrew	*Blarina brevicauda*	Abundant. Frequently captured in mousetraps in wooded areas.
Silver-haired Bat	*Lasionycteris noctivagars*	
* Southern Flying Squirrel	*Glauconys volans*	More abundant than realized. This was the most abundant mammal encountered in the artificial nest boxes erected on tree tunks for fox or gray squirrels.
Star-nosed Mole	*Condylura cristata*	

TABLE E-3 (Continued)

ANNOTATED LIST OF MAMMALS[a]

Common Name	Scientific Name	Remarks
* Striped Skunk	*Mephitis mephitis*	Abundant. Seen frequently at night.
* White-footed Mouse	*Peromyscus leucopus*	Abundant. The most frequently captured mammal in mousetraps set in or near woodlands.
* White-tailed Deer	*Odocoileus virginianus*	Introduced in 1932 and fluorished. By the late 1940s, they had reached troublesome proportions. With the help of the Maryland Game and Inland Fish Department, approximately 2,000 were removed and relocated throughout the State. The deer reached peak numbers in the 1950s and then declined. They are smaller sized, have a heavier parasite load and have a lower fawn production per female than do deer elsewhere in Maryland. Deer are so overly abundant that they seriously hinder growth and development of natural vegetation, including forests.
* Woodchuck	*Marmota monax*	Seen along roadsides and grasslands. Sometimes a nuisance, especially in built-up areas, because they burrow into lawns and eat flowers and shrubs.

* Species which have been reliably reported from the Proving Ground. Mammals which are not denoted by an asterisk occur in the general area and possibly on the Proving Ground although they have not been recorded there.

[a] Source: Aberdeen Proving Ground. 1987. Natural Resources Management Plan, Part IV Fish and Wildlife Management. Aberdeen Proving Ground Environmental Management Office. Aberdeen Proving Ground, Maryland.

TABLE E-4

REPTILES AND AMPHIBIANS RECORDED AT ABERDEEN[a]

Common Name	Scientific Name	Status
American Toad	*Bufo a. americanus*	Common
Black Rat Snake	*Elaphe o. obsoleta*	Common
Bog Turtle	*Clemmys muhlenbergi*	Abundant
Bullfrog	*Rana catesbeiana*	Abundant
Eastern Box Turtle	*Terrepene c. carolina*	Abundant
Eastern Garter Snake	*Thamnophis s. sirtalis*	Common
Eastern Gray Treefrog	*Hyla v. versicolor*	Abundant
Eastern Hognose Snake	*Heterodon platyrhinos*	Rare
Eastern Kingsnake	*Lampropeltis g. getulus*	Rare
Eastern Milk Snake	*Lampropeltis doliata*	Rare
Eastern Mud Turtle	*Kinosternon s. subrubrum*	Abundant
Eastern Painted Turtle	*Chrysemys p. picta*	Abundant
Eastern Ribbon Snake	*Thamnophis s. sauritus*	Uncommon
Eastern Worm Snake	*Carphophis a. amoenus*	Uncommon
Five-Lined Skink	*Eumaces fasciatus*	Common
Fowler's Toad	*Bufo woodhousei fowleri*	Abundant
Green Frog	*Rana clamitans melanota*	Abundant
Green Treefrog	*Hyla cinerea*	Uncommon
Marbled Salamander	*Ambystoma opacum*	Uncommon
Northern Black Racer	*Coluber c. constrictor*	Common
Northern Cricket Frog	*Acris crepitans crepitans*	Abundant
Northern Diamondback Terrapin	*Malaclemys t. terrapin*	Abundant
Northern Fence Lizard	*Sceloporus undulatus hyacinthinus*	Rare
Northern Leopard Frog	*Rana p. pipiens*	Rare
Northern Ringneck Snake	*Diadophis punctatus edwardsi*	Rare
Northern Spring Peeper	*Hyla c. crucifer*	Abundant
Northern Water Snake	*Natrix s. sipedon*	Abundant
Pickeral Frog	*Rana palustris palustris*	Uncommon
Queen Snake	*Regina s. septemvittata*	Rare
Red-backed Salamader	*Plethodon c. cinereus*	Common
Red-bellied Turtle	*Chrysemys rubriventris*	Rare
Red-eared Turtle	*Chrysemys scripta clegans*	Uncommon
Snapping Turtle	*Chelydra s. serpentina*	Abundant
Southern Leopard Frog	*Rana p. sphenocephala*	Abundant
Spotted Salamander	*Ambystoma maculatum*	Common
Spotted Turtle	*Clemmys guttata*	Abundant
Upland Chorus Frog	*Pseudacris triseriata feriarum*	Common

[a] Source: Aberdeen Proving Ground. 1987. Natural Resources Management Plan, Part IV Fish and Wildlife Management. Aberdeen Proving Ground Environmental Management Office. Aberdeen Proving Ground, Maryland.

TABLE E-5

FISH SPECIES FOUND AT ABERDEEN PROVING GROUND
AND IN THE SURROUNDING WATERS
OF THE UPPER CHESAPEAKE BAY[a]

FAMILY/COMMON NAME	SCIENTIFIC NAME
Anguillidae (Freshwater eels)	
American eel	*Anguila rostrata*
Atherinidae (Silversides)	
Rough silverside	*Membras martinica*
Tidewater silverside	*Menidia beryllina*
Atlantic silverside	*Menidia menidia*
Blenniidae (Blennies)	
Blennies	*Chasmodes bosquianus, Hypsoblennius hentz,* and/or *Blennius fucorum*
Catostomidae (Suckers)	
Quillback sucker	*Carpoides cyprinus*
White sucker	*Catostomus commersoni*
Creek chubsucker	*Erimyzon oblongus*
Spotted sucker	*Minytrema melanops*
Centrarchidae (Sunfishes)	
Blue spotted sunfish	*Enneacanthus gloriosus*
Red breast sunfish	*Lepomis auritus*
Pumpkinseed	*Lepomis gibbosus*
Bluegill	*Lepomis macrochirus*
Largemouth bass	*Micropterus salmoides*
White crappie	*Pomoxis ammularis*
Black crappie	*Pomoxis nigromaculatus*
Clupeidae (Herrings)	
Blueblack herring	*Alosa aestivalis*
Hickory shad	*Alosa mediocris*
Alewife	*Alosa pseudoharengus*
American shad	*Alosa sapidissima*
Bay anchovy	*Anchoa mitchilli*
Atlantic menhaden	*Brevoortia tyrannus*
Gizzard shad	*Drosoma cepedianum*

TABLE E-5 (Continued)

FISH SPECIES FOUND AT ABERDEEN PROVING GROUND
AND IN THE SURROUNDING WATERS
OF THE UPPER CHESAPEAKE BAY[a]

FAMILY/COMMON NAME	SCIENTIFIC NAME
Cyprinodontidae (Killifishes)	
Banded killifish	*Fundulus diaphanus*
Mummichog	*Fundulus heteroclitus*
Striped killifish	*Fundulus majalis*
Cyprinidae (Minnows)	
Goldfish	*Carassius auratus*
Carp	*Cyprinus carpio*
Silvery minnow	*Hybognathus nuchalis*
Comley shiner	*Notropis amoenus*
Satinfin shiner	*Notropis analostanus*
Bridle shiner	*Notropis bifrenatus*
Spottail shiner	*Notropis hudsonius*
Golden shiner	*Notemigonus crysoleucas*
Esocidae (Pikes)	
Pickerel	*Esox spp.*
Gasterosteidae (Sticklebacks)	
Stickleback	*Gasterosteus spp.* or *Apeltes spp.*
Gobiidae (Gobies)	
Naked goby	*Gobisoma bosci*
Ictaluridae (Bullhead catfishes)	
White catfish	*Ictalurus catus*
Brown bullhead	*Ictalurus nebulosus*
Black bullhead	*Ictalurus melas*
Channel catfish	*Ictalurus punctatus*
Percichthyidae (Temperate basses)	
White perch	*Morone americana*
Striped bass	*Morone saxatilis*

TABLE E-5 (Continued)

FISH SPECIES FOUND AT ABERDEEN PROVING GROUND
AND IN THE SURROUNDING WATERS
OF THE UPPER CHESAPEAKE BAY[a]

FAMILY/COMMON NAME	SCIENTIFIC NAME
Percidae (Perches)	
Tessalated darter	*Etheostoma olmstedi*
Maryland darter	*Etheostoma sellare*
Glassy darter	*Etheostoma vitreum*
Yellow perch	*Perca flavescens*
Pleuronectidae (Flounders)	
Winter flounder	*Pseudopleuronecthes americanus*
Poeciliidae (Livebearers)	
Mosquito fish	*Gambusia affinis*
Sciaenidae (Croakers and Drums)	
Spot	*Leiostomus xanthurus*
Atlantic croaker	*Micropogonias undulatus*
Black drum	*Pogonias cromis*
Red drum	*Sciaenops ocellatus*
Soleidae (Soles)	
Hogchoker	*Trinectes maculatus*

[a] Source: Aberdeen Proving Ground. 1987. Natural Resources Management Plan, Part IV Fish and Wildlife Management. Aberdeen Proving Ground Environmental Management Office. Aberdeen Proving Ground, Maryland.